Family Risk and Protective Factors for Binge Eating-Related Concerns in a Nationally Representative Sample of Young Adults in the United States

by

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Dedication

This dissertation is dedicated to the memory of my father, who would be glad to see I pursued a path in science.

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Abstract

Binge eating-related concerns (i.e., emotions and cognitions associated with binge eating, such as embarrassment over amount eaten and fear of losing control over eating) have been found to prospectively predict eating disorder onset. Therefore, reducing binge eating-related concerns may be a promising target for eating disorders prevention and early intervention, particularly for eating disorders involving binge eating. Using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative sample of the United States, the overarching objective of this dissertation project was to examine family risk and protective factors for binge eating-related concerns in young adulthood, as well as to investigate potential mediators and moderators. Using a person-centered approach, the first aim identified five childhood maltreatment latent classes: "no/low maltreatment," "physical abuse only," "multi-type maltreatment," "physical neglect only," and "sexual abuse only." Participants assigned to the "multi-type maltreatment" class were more likely to report binge eating-related concerns compared to those assigned to the "no/low maltreatment" class. Self-esteem in adolescence mediated a statistically significant but modest proportion of this association. However, no associations were observed between the single-type childhood maltreatment classes and binge eating-related concerns. In the second aim, higher mother-child connectedness in adolescence, but not father-child connectedness in adolescence, was found to be associated with lower odds of binge eating-related concerns in the whole sample, but differences by sex emerged. Both higher mother-child connectedness and higher father-child connectedness in adolescence were associated with lower odds of binge eating-related concerns

among females, but neither mother-child connectedness nor father-child connectedness in adolescence were associated with binge eating-related concerns among males. The third aim explored the differential susceptibility hypothesis, which posits that genetic variants such as the S allele of 5-HTTLPR confer increased sensitivity not only to environmental risk factors, but also to environmental protective factors. Neither childhood abuse nor parent-child connectedness in adolescence was found to interact with 5-HTTLPR genotype in predicting binge eating-related concerns; thus, the differential susceptibility hypothesis was not supported. These findings suggest that eating disorders interventions should focus on decreasing risk factors such as childhood maltreatment and promoting protective factors such as parent-child connectedness.

Chapter 1

Introduction

Binge Eating and Related Concerns

Binge eating and clinical eating disorders

Binge eating episodes are characterized by overeating (i.e., eating an unusually large quantity of food in a discrete period of time) accompanied by a sense of loss of control over eating (American Psychiatric Association, 2013). Binge eating is a common symptom across several eating disorder diagnoses, including binge eating disorder, bulimia nervosa, and anorexia nervosa, binge-eating/purging type (American Psychiatric Association, 2013). Typical age of onset for eating disorders falls within adolescence and young adulthood (Hudson, Hiripi, Pope, & Kessler, 2007; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011), and based on criteria for eating disorders in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013), prevalence estimates of any eating disorder among young adults range from 1.2% - 2.9% for males and 5.7% - 15.2% for females (Allen, Byrne, Oddy, & Crosby, 2013; Smink, van Hoeken, Oldehinkel, & Hoek, 2014; Stice, Marti, & Rohde, 2013). Eating disorders are highly comorbid with other psychiatric disorders and strongly associated with medical complications, psychosocial impairment, and suicidality (Hudson et al., 2007; Mitchell & Crow, 2006; Swanson et al., 2011). Given their early age of onset, prevalence, and burden of disease, eating disorders are of significant public health concern.

Subthreshold binge eating

Because not all individuals with binge eating meet criteria for a clinical eating disorder, subthreshold binge eating is more prevalent than clinical eating disorders involving binge eating (e.g., binge eating disorder, bulimia nervosa). Among community samples of young adults, prevalence estimates of at least weekly binge eating episodes range from 1.1% - 8.0% for males and 3.1% - 10.0% for females (Sonneville et al., 2013; Striegel-Moore et al., 2009). Therefore, compared with 0.7% - 1.2% of young adult males and 2.3% - 3.0% of young adult females meeting criteria for a clinical diagnosis of binge eating disorder and 0.1% - 1.6% of young adult males and 0.8% - 2.6% of young adult females meeting criteria for a clinical diagnosis of bulimia nervosa (Allen et al., 2013; Smink et al., 2014; Stice et al., 2013), binge eating is relatively common. Additionally, subthreshold binge eating is still associated with substantial psychosocial impairment, as binge eating is cross-sectionally and prospectively associated with higher depressive symptoms (Bentley, Gratwick-Sarll, Harrison, & Mond, 2015; Goldschmidt, Wall, Choo, Larson, & Neumark-Sztainer, 2015; Sehm & Warschburger, 2016; Sinclair-McBride & Cole, 2017; Skinner, Haines, Austin, & Field, 2012; Sonneville et al., 2013; Vannucci et al., 2013). Associations between binge eating and depressive symptoms may be explained, in part, by the ego-dystonic nature of binge eating (Hail & Le Grange, 2018) – the act of binge eating often conflicts with one's ideal self-image (e.g., the thin body ideal), which may contribute to psychological distress.

Binge eating-related concerns

Related to the ego-dystonic nature of binge eating, emotions and cognitions associated with binge eating (hereafter referred to as binge eating-related concerns) include embarrassment over amount eaten and fear of losing control over eating (Gormally, Black, Daston, & Rardin,

1982). Evidence suggests these concerns are distinct from the component constructs of binge eating to which they relate. For example, fear of losing control over eating, but not loss of control eating, has been found to be associated with dietary restraint among eating disorder patients (Ricca et al., 2012). It has recently been proposed that the subjective experience of binge eating, which may include a sense of loss of control over eating as well as binge eating-related concerns, may be a more important indicator of distress and impairment than more objective aspects of binge eating, namely overeating (Goldschmidt, 2017). Supporting this idea, loss of control eating (Tanofsky-Kraff et al., 2011) and binge eating-related concerns (Hazzard, Hahn, Bauer, & Sonneville, 2019), but not overeating (Sonneville et al., 2013), have been found to be longitudinally associated with higher depressive symptoms. Further, binge eating-related concerns have been identified as key characteristics prospectively predicting eating disorder onset (Fairburn, Cooper, Doll, & Davies, 2005), suggesting that binge eating-related concerns may be important precursors to eating disorders. Reducing binge eating-related concerns may therefore be a promising target for eating disorders prevention and early intervention, particularly for eating disorders involving binge eating.

Attachment Theory

Attachment theory provides an important framework for understanding how family factors in early life may influence eating disorder risk. Although the origins of attachment theory pertain primarily to infancy and child development, empirical support has since extended the theory to help explain functioning and mental health in adulthood (Cassidy & Shaver, 2016). John Bowlby (1969) proposed that natural selection favored infant attachment behaviors (e.g., crying, smiling, vocalizing, following) because they increase proximity of the child to the

caregiver, which in turn increases likelihood of protection and provides survival advantage. Availability and responsiveness of the caregiver to the child are essential to the development of secure attachment, an emotional bond that acts as a secure base from which the child can explore (Bowlby, 1969). Repeated attachment-related experiences become organized into scripts that guide emotion regulation and coping behavior in threatening situations (Bowlby, 1969). Whereas secure attachment fosters adaptive emotion regulation strategies, insecure attachment contributes to the development of maladaptive emotion regulation strategies (Bowlby, 1969; Cassidy & Shaver, 2016). Evidence suggests that these maladaptive emotion regulation strategies, in turn, contribute to symptoms of eating disorders – including binge eating – among adults with insecure attachment (Han & Lee, 2017; Keating, Mills, & Rawana, 2018; Tasca et al., 2009).

Childhood maltreatment

While individual differences in attachment security (i.e., secure attachment versus insecure attachment) typically develop in early childhood, severe threats to caregiver availability or responsiveness during middle childhood and adolescence can disrupt attachment relationships (Cassidy & Shaver, 2016). When these disruptions go unresolved, they can contribute to maladaptive emotion regulation strategies and adverse mental health outcomes later in life (Cassidy & Shaver, 2016). There are two key types of attachment disruptions: (1) severe threats to caregiver availability via situations such as abandonment or death and (2) severe threats to caregiver responsiveness via situations such as betrayal or frightening behavior, indicating failure of the caregiver to provide protection (Cassidy & Shaver, 2016). Maltreatment (i.e., abuse and neglect) by caregivers falls under the latter type of attachment disruption.

In addition to the consequences of attachment disruptions, there is evidence to suggest that although individual differences in attachment security tend to be relatively stable across the

lifespan (Cassidy & Shaver, 2016), they are to some extent malleable in response to experiences that threaten relationships with caregivers. For example, abuse by a family member has been found to predict changes from secure attachment to insecure attachment between infancy and young adulthood (Waters, Merrick, Treboux, Crowell, & Albersheim, 2000). This evidence, when considered along with the extensive literature finding strong associations between childhood maltreatment and insecure attachment (Baer & Martinez, 2006; Morton & Browne, 1998), suggests that childhood maltreatment might be associated with maladaptive emotion regulation strategies, and, in turn, higher levels of eating disorders symptoms, including binge eating. Indeed, childhood maltreatment has been found to be associated with eating disorders (Afifi et al., 2017; Caslini et al., 2016; Molendijk, Hoek, Brewerton, & Elzinga, 2017), with particularly strong and consistent evidence for associations with eating disorders involving binge eating (Caslini et al., 2016; Molendijk et al., 2017). Maladaptive emotion regulation strategies have been found to help explain associations between childhood maltreatment and eating disorder symptoms (Burns, Fischer, Jackson, & Harding, 2012; Mills, Newman, Cossar, & Murray, 2015), which aligns with the known link between childhood maltreatment and insecure attachment. Self-esteem may also help explain associations between childhood maltreatment and eating disorder symptoms, as insecure attachment, childhood maltreatment, and eating disorder symptoms are all associated with lower self-esteem (Goldschmidt et al., 2016; Greger, Myhre, Klöckner, & Jozefiak, 2017; Huntsinger & Luecken, 2004; Ju & Lee, 2018).

Parent-child connectedness

The influence of early attachment on later development depends, to some extent, on the quality of parental care throughout childhood and adolescence (Cassidy & Shaver, 2016). Parent-child connectedness, which has been defined as closeness, caring, and satisfaction in parent-child

relationships, is grounded in attachment theory but relates to the ongoing dynamics of how parents and children interact with one another not only in infancy, but throughout childhood and adolescence (Lezin, Rolleri, Bean, & Taylor, 2004). Parent-child connectedness has been found to be protective against binge eating among adolescents (Berge et al., 2014), but the ways in which mother-child connectedness and father-child connectedness may differentially shape eating disorder risk and the ways in which these associations may differ by the sex of the child are not well understood.

Differential Susceptibility Hypothesis

A widely studied candidate gene involved in the serotonergic system is 5-HTTLPR, a polymorphism in the promoter region of the gene that codes for the serotonin transporter (SLC6A4; Heils et al., 1996). The two most frequent alleles resulting from this polymorphism are the short (S) allele and the long (L) allele (Heils et al., 1996). Gene x environment (G x E) interaction findings in the depression field have suggested that 5-HTTLPR genotype moderates associations between major life stressors and depression, such that the S allele is associated with increased risk for depression in the context of major life stressors (Bleys, Luyten, Soenens, & Claes, 2018; Caspi et al., 2003; Haberstick et al., 2016; Karg, Burmeister, Shedden, & Sen, 2011; Sharpley, Palanisamy, Glyde, Dillingham, & Agnew, 2014). A small number of studies in the eating disorders field have since examined G x E interaction between 5-HTTLPR genotype and major life stressors, and the S allele has been found to be associated with increased risk for bulimia nervosa among participants with a history of childhood physical and/or sexual abuse (Rozenblat et al., 2017). Relating back to attachment theory, the S allele has also been found to be associated with insecure attachment among bulimia nervosa patients with a history of

childhood physical and/or sexual abuse (Steiger et al., 2007). However, the generalizability of these findings may be limited, as these studies have examined small, demographically homogenous samples. Additionally, the eating disorders field has yet to take a differential susceptibility approach to exploring G x E interactions, as has been done in the depression field.

The differential susceptibility hypothesis theorizes that genetic variants such as the S allele of 5-HTTLPR confer increased sensitivity not only to environmental risk factors, but also to environmental protective factors (Belsky et al., 2009; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009). The depression literature has found evidence to support this hypothesis, such that the S allele of 5-HTTLPR has been found to be associated with lower depressive symptoms among individuals with high family support (Hankin et al., 2011; Li, Berk, & Lee, 2013; Taylor et al., 2006). To our knowledge, no studies in the eating disorders field have investigated whether the S allele is associated with increased sensitivity to protective factors. If the S allele is associated with lower binge eating-related concerns among individuals with higher levels of factors related to family support, such as parent-child connectedness, future research could investigate whether interventions to promote these protective factors show greater efficacy among individuals with greater genetic susceptibility, such as those with the S allele of 5-HTTLPR.

Dissertation Aims and Hypotheses

Using data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative sample of the United States, the overarching objective of this dissertation project was to examine family risk and protective factors for binge eating-related concerns in young adulthood, as well as to investigate potential mediators and moderators. The

findings of this project have the potential to inform interventions to reduce binge eating-related concerns, which may be important for eating disorders prevention and early intervention. The aims of the project are illustrated in Figure 1.1 and described below.

The first aim identifies distinct childhood maltreatment profiles, examines associations between childhood maltreatment profiles and binge eating-related concerns, and evaluates the extent to which self-esteem during adolescence mediates observed associations. We hypothesized that childhood maltreatment would be associated with greater odds of binge eating-related concerns, with the magnitude of association varying by childhood maltreatment profile but the direction of association consistent across childhood maltreatment profiles. We expected self-esteem to partially mediate observed associations.

The second aim investigates the extent to which mother-child connectedness and father-child connectedness in adolescence are associated with binge eating-related concerns and examines differences in associations by sex. We hypothesized that mother-child connectedness and father-child connectedness in adolescence would be associated with lower odds of binge eating-related concerns. We anticipated that protective associations would hold for both sexes but might be strongest for father-daughter connectedness.

The third aim explores the extent to which associations of childhood abuse and parent-child connectedness in adolescence with binge eating-related concerns differ by 5-HTTLPR genotype. We expected to find that with increasing numbers of the S allele, childhood abuse would be more strongly associated with greater odds of binge eating-related concerns, while parent-child connectedness in adolescence would be more strongly associated with lower odds of binge eating-related concerns.

By examining family risk and protective factors for binge eating-related concerns in young adulthood and investigating potential mediators and moderators, this dissertation project offers important epidemiologic contributions to understanding the etiology of binge eating-related concerns and, ultimately, to informing eating disorders prevention and treatment efforts.

References

Afifi, T. O., Sareen, J., Fortier, J., Taillieu, T., Turner, S., Cheung, K., & Henriksen, C. A. (2017). Child maltreatment and eating disorders among men and women in adulthood: Results from a nationally representative United States sample. *International Journal of Eating Disorders*, *50*(11), 1281–1296. http://doi.org/10.1002/eat.22783

Allen, K. L., Byrne, S. M., Oddy, W. H., & Crosby, R. D. (2013). DSM-IV-TR and DSM-5 eating disorders in adolescents: Prevalence, stability, and psychosocial correlates in a population-based sample of male and female adolescents. *Journal of Abnormal Psychology*, *122*(3), 720–732. http://doi.org/10.1037/a0034004

American Psychiatric Association. (2013). Feeding and Eating Disorders. In *Diagnostic and Statistical Manual of Mental Disorders (DSM-5®)*. American Psychiatric Association. http://doi.org/https://doi.org/10.1176/appi.books.9780890425596.dsm10

Baer, J. C., & Martinez, C. D. (2006). Child maltreatment and insecure attachment: A meta-analysis. *Journal of Reproductive and Infant Psychology*, *24*(3), 187–197. http://doi.org/10.1080/02646830600821231

Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). For better and for worse: Differential susceptibility to environmental influences. *Current Directions in Psychological Science*, *16*(6), 300–304. http://doi.org/10.1111/j.1467-8721.2007.00525.x

Belsky, J., Jonassaint, C., Pluess, M., Stanton, M., Brummett, B., & Williams, R. (2009). Vulnerability genes or plasticity genes? *Molecular Psychiatry*, *14*(8), 746–54. http://doi.org/10.1038/mp.2009.44

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin*, *135*(6), 885–908.

http://doi.org/10.1037/a0017376

Bentley, C., Gratwick-Sarll, K., Harrison, C., & Mond, J. (2015). Sex differences in psychosocial impairment associated with eating disorder features in adolescents: A school-based study. *International Journal of Eating Disorders*, *48*(6), 633–640. http://doi.org/10.1002/eat.22396

Berge, J. M., Wall, M., Larson, N., Eisenberg, M. E., Loth, K. A., & Neumark-Sztainer, D. (2014). The unique and additive associations of family functioning and parenting practices with disordered eating behaviors in diverse adolescents. *Journal of Behavioral Medicine*, *37*(2), 205–17. http://doi.org/10.1007/s10865-012-9478-1

Bleys, D., Luyten, P., Soenens, B., & Claes, S. (2018). Gene-environment interactions between stress and 5-HTTLPR in depression: A meta-analytic update. *Journal of Affective Disorders*, *226*, 339–345. http://doi.org/10.1016/J.JAD.2017.09.050

Bowlby, J. (1969). *Attachment and Loss Vol. 1. Attachment* (Vol. 1). http://doi.org/10.1177/000306518403200125

Burns, E. E., Fischer, S., Jackson, J. L., & Harding, H. G. (2012). Deficits in emotion regulation mediate the relationship between childhood abuse and later eating disorder symptoms. *Child Abuse & Neglect*, *36*(1), 32–9. http://doi.org/10.1016/j.chiabu.2011.08.005

Caslini, M., Bartoli, F., Crocamo, C., Dakanalis, A., Clerici, M., & Carrà, G. (2016). Disentangling the association between child abuse and eating disorders: A systematic review and meta-analysis. *Psychosomatic Medicine*, *78*(1), 79–90. http://doi.org/10.1097/PSY.0000000000000233

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., … Poulton, R. (2003). Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science (New York, N.Y.)*, *301*(5631), 386–9. http://doi.org/10.1126/science.1083968

Cassidy, J., & Shaver, P. R. (Eds.). (2016). *Handbook of Attachment: Theory, Research, and Clinical Applications* (3rd ed). New York, NY: Guilford Publications.

Fairburn, C. G., Cooper, Z., Doll, H. A., & Davies, B. A. (2005). Identifying dieters who will develop an eating disorder: a prospective, population-based study. *American Journal of Psychiatry*, *162*(12), 2249–2255. http://doi.org/10.1176/appi.ajp.162.12.2249

Goldschmidt, A. B. (2017). Are loss of control while eating and overeating valid constructs? A critical review of the literature. *Obesity Reviews*, *18*(4), 412–449. http://doi.org/10.1111/obr.12491

Goldschmidt, A. B., Wall, M. M., Choo, T.-H. J., Larson, N. I., & Neumark-Sztainer, D. (2015). Mediators involved in the relation between depressive symptoms and weight status in female adolescents and young adults. *International Journal of Obesity (2005)*, *39*(6), 1027–1029. http://doi.org/10.1038/ijo.2015.6

Goldschmidt, A. B., Wall, M. M., Zhang, J., Loth, K. A., Neumark-sztainer, D., Goldschmidt, A. B., … Neumark-sztainer, D. (2016). Overeating and binge eating in emerging adulthood: 10-year stability and risk factors. *Developmental Psychology*, *52*(3), 475–483. http://doi.org/10.1037/dev0000086

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors*, *7*(1), 47–55. http://doi.org/10.1016/0306-4603(82)90024-7

Greger, H. K., Myhre, A. K., Klöckner, C. A., & Jozefiak, T. (2017). Childhood maltreatment, psychopathology and well-being: The mediator role of global self-esteem, attachment difficulties and substance use. *Child Abuse & Neglect*, *70*, 122–133. http://doi.org/10.1016/j.chiabu.2017.06.012

Haberstick, B. C., Boardman, J. D., Wagner, B., Smolen, A., Hewitt, J. K., Killeya-Jones, L. A., … Mullan Harris, K. (2016). Depression, stressful life events, and the impact of variation in the serotonin transporter: Findings from the National Longitudinal Study of Adolescent to Adult Health (Add Health). *PloS One*, *11*(3), e0148373. http://doi.org/10.1371/journal.pone.0148373

Hail, L., & Le Grange, D. (2018). Bulimia nervosa in adolescents: Prevalence and treatment challenges. *Adolescent Health, Medicine and Therapeutics*, *9*, 11–16. http://doi.org/10.2147/AHMT.S135326

Han, S., & Lee, S. (2017). College student binge eating: Attachment, psychological needs satisfaction, and emotion regulation. *Journal of College Student Development*, *58*(7), 1074–1086. http://doi.org/10.1353/csd.2017.0084

Hankin, B. L., Nederhof, E., Oppenheimer, C. W., Jenness, J., Young, J. F., Abela, J. R. Z., … Oldehinkel, A. J. (2011). Differential susceptibility in youth: Evidence that 5-HTTLPR x positive parenting is associated with positive affect for better and worse. *Translational Psychiatry*, *1*(9), e44-47. http://doi.org/10.1038/tp.2011.44

Hazzard, V. M., Hahn, S. L., Bauer, K. W., & Sonneville, K. R. (2019). Binge eating-related concerns and depressive symptoms in young adulthood: Seven-year longitudinal associations and differences by race/ethnicity. *Eating Behaviors*, *32*, 90–94. http://doi.org/10.1016/j.eatbeh.2019.01.004

Heils, A., Teufel, A., Petri, S., Stöber, G., Riederer, P., Bengel, D., & Lesch, K. P. (1996). Allelic variation of human serotonin transporter gene expression. *Journal of Neurochemistry*, *66*(6), 2621–2624. http://doi.org/10.1046/j.1471-4159.1996.66062621.x

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of

eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*, *61*(3), 348–358. http://doi.org/10.1016/j.biopsych.2006.03.040

Huntsinger, E. T., & Luecken, L. J. (2004). Attachment relationships and health behavior: The mediational role of self-esteem. *Psychology & Health*, *19*(4), 515–526. http://doi.org/10.1080/0887044042000196728

Ju, S., & Lee, Y. (2018). Developmental trajectories and longitudinal mediation effects of self-esteem, peer attachment, child maltreatment and depression on early adolescents. *Child Abuse & Neglect*, *76*, 353–363. http://doi.org/10.1016/j.chiabu.2017.11.015

Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: Evidence of genetic moderation. *Archives of General Psychiatry*, *68*(5), 444–54. http://doi.org/10.1001/archgenpsychiatry.2010.189

Keating, L., Mills, J. S., & Rawana, J. S. (2018). Momentary predictors of binge eating: An attachment perspective. *Eating Behaviors*, *32*, 44–52. http://doi.org/10.1016/j.eatbeh.2018.12.003

Lezin, N., Rolleri, L. A., Bean, S., & Taylor, J. (2004). *Parent-child connectedness: Implications for research, interventions and positive impacts on adolescent health*. Santa Cruz, CA.

Li, J. J., Berk, M. S., & Lee, S. S. (2013). Differential susceptibility in longitudinal models of gene-environment interaction for adolescent depression. *Development and Psychopathology*, *25*(4pt1), 991–1003. http://doi.org/10.1017/S0954579413000321

Mills, P., Newman, E. F., Cossar, J., & Murray, G. (2015). Emotional maltreatment and disordered eating in adolescents: Testing the mediating role of emotion regulation. *Child Abuse & Neglect*, *39*, 156–166. http://doi.org/10.1016/J.CHIABU.2014.05.011

Mitchell, J. E., & Crow, S. (2006). Medical complications of anorexia nervosa and bulimia nervosa. *Current Opinion in Psychiatry*, *19*(4), 438–443. http://doi.org/10.1097/01.yco.0000228768.79097.3e LK - https://aalborguh.tdnetdiscover.com/resolver/full?&sid=EMBASE&issn=09517367&id=doi:10.1097%2F01.yco.0000228768.79097.3e&atitle=Medical+complications+of+anorexia+nervosa+and+bulimia+nervosa&stitle=Curr.+Opin.+Psychiatry&title=Current+Opinion+in+Psychiatry&volume=19&issue=4&spage=438&epage=443&aulast=Mitchell&aufirst=James+E.&auinit=J.E.&aufull=Mitchell+J.E.&coden=COPPE&isbn=&pages=438-443&date=2006&auinit1=J&auinitm=E.

Molendijk, M. L., Hoek, H. W., Brewerton, T. D., & Elzinga, B. M. (2017). Childhood maltreatment and eating disorder pathology: a systematic review and dose-response meta-analysis. *Psychological Medicine*, *47*(8), 1402–1416. http://doi.org/10.1017/S0033291716003561

Morton, N., & Browne, K. D. (1998). Theory and observation of attachment and its relation to child maltreatment: A review. *Child Abuse & Neglect*, *22*(11), 1093–1104.

Ricca, V., Castellini, G., Fioravanti, G., Lo Sauro, C., Rotella, F., Ravaldi, C., … Faravelli, C. (2012). Emotional eating in anorexia nervosa and bulimia nervosa. *Comprehensive Psychiatry*, *53*(3), 245–251. http://doi.org/10.1016/j.comppsych.2011.04.062

Rozenblat, V., Ong, D., Fuller-Tyszkiewicz, M., Akkermann, K., Collier, D., Engels, R. C. M. E., … Krug, I. (2017). A systematic review and secondary data analysis of the interactions between the serotonin transporter 5-HTTLPR polymorphism and environmental and psychological factors in eating disorders. *Journal of Psychiatric Research*, *84*, 62–72. http://doi.org/10.1016/j.jpsychires.2016.09.023

Sehm, M., & Warschburger, P. (2016). Prospective associations between binge eating and psychological risk factors in adolescence. *Journal of Clinical Child and Adolescent Psychology*, *47*(5), 770–784. http://doi.org/10.1080/15374416.2016.1178124

Sharpley, C. F., Palanisamy, S. K. A., Glyde, N. S., Dillingham, P. W., & Agnew, L. L. (2014). An update on the interaction between the serotonin transporter promoter variant (5-HTTLPR), stress and depression, plus an exploration of non-confirming findings. *Behavioural Brain Research*, *273*, 89–105. http://doi.org/10.1016/j.bbr.2014.07.030

Sinclair-McBride, K., & Cole, D. A. (2017). Prospective relations between overeating, loss of control eating, binge eating, and depressive symptoms in a school-based sample of adolescents. *Journal of Abnormal Child Psychology*, *45*(4), 693–703. http://doi.org/10.1007/s10802-016-0186-0

Skinner, H. H., Haines, J., Austin, S. B., & Field, A. E. (2012). A prospective study of overeating, binge eating, and depressive symptoms among adolescent and young adult women. *The Journal of Adolescent Health*, *50*(5), 478–83. http://doi.org/10.1016/j.jadohealth.2011.10.002

Smink, F. R. E., van Hoeken, D., Oldehinkel, A. J., & Hoek, H. W. (2014). Prevalence and severity of DSM-5 eating disorders in a community cohort of adolescents. *International Journal of Eating Disorders*, *47*(6), 610–619. http://doi.org/10.1002/eat.22316

Sonneville, K. R., Horton, N. J., Micali, N., Crosby, R. D., Swanson, S. A., Solmi, F., & Field, A. E. (2013). Longitudinal associations between binge eating and overeating and adverse outcomes among adolescents and young adults: Does loss of control matter? *JAMA Pediatrics*, *167*(2), 149–155. http://doi.org/10.1001/2013.jamapediatrics.12

Steiger, H., Richardson, J., Joober, R., Gauvin, L., Israel, M., Bruce, K. R., … Young, S. N.

(2007). The 5HTTLPR polymorphism, prior maltreatment and dramatic-erratic personality manifestations in women with bulimic syndromes. *Journal of Psychiatry & Neuroscience*, *32*(5), 354–62.

Stice, E., Marti, C. N., & Rohde, P. (2013). Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. *Journal of Abnormal Psychology*, *122*(2), 445–457. http://doi.org/10.1037/a0030679

Striegel-Moore, R. H., Rosselli, F., Perrin, N., DeBar, L., Wilson, G. T., May, A., & Kraemer, H. C. (2009). Gender difference in the prevalence of eating disorder symptoms. *International Journal of Eating Disorders*, *42*(5), 471–474. http://doi.org/10.1002/eat.20625

Swanson, S. A., Crow, S. J., Le Grange, D., Swendsen, J., & Merikangas, K. R. (2011). Prevalence and correlates of eating disorders in adolescents. Results from the national comorbidity survey replication adolescent supplement. *Archives of General Psychiatry*, *68*(7), 714–723. http://doi.org/10.1001/archgenpsychiatry.2011.22

Tanofsky-Kraff, M., Shomaker, L. B., Olsen, C., Roza, C. A., Wolkoff, L. E., Columbo, K. M., … Yanovski, J. A. (2011). A prospective study of pediatric loss of control eating and psychological outcomes. *Journal of Abnormal Psychology*, *120*(1), 108–18. http://doi.org/10.1037/a0021406

Tasca, G. A., Szadkowski, L., Illing, V., Trinneer, A., Grenon, R., Demidenko, N., … Bissada, H. (2009). Adult attachment, depression, and eating disorder symptoms: The mediating role of affect regulation strategies. *Personality and Individual Differences*, *47*(6), 662–667. http://doi.org/10.1016/j.paid.2009.06.006

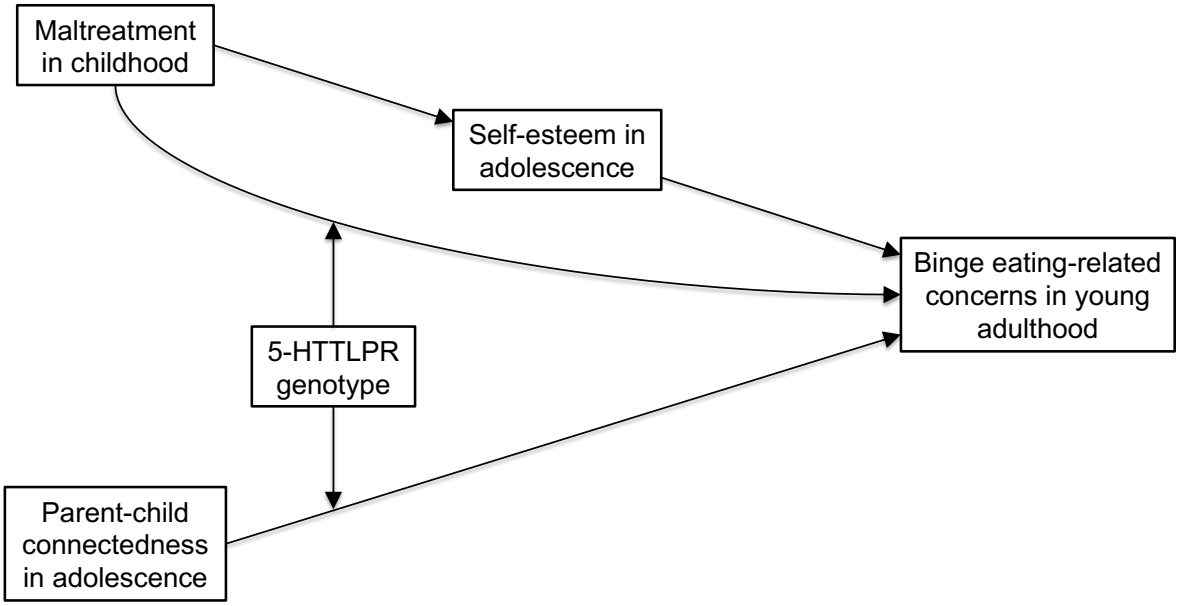
Taylor, S. E., Way, B. M., Welch, W. T., Hilmert, C. J., Lehman, B. J., & Eisenberger, N. I.

(2006). Early family environment, current adversity, the serotonin transporter promoter polymorphism, and depressive symptomatology. *Biological Psychiatry*, *60*(7), 671–676. http://doi.org/10.1016/j.biopsych.2006.04.019

Vannucci, A., Tanofsky-Kraff, M., Crosby, R. D., Ranzenhofer, L. M., Shomaker, L. B., Field, S. E., … Yanovski, J. A. (2013). Latent profile analysis to determine the typology of disinhibited eating behaviors in children and adolescents. *Journal of Consulting and Clinical Psychology*, *81*(3), 494–507. http://doi.org/10.1037/a0031209

Waters, E., Merrick, S., Treboux, D., Crowell, J., & Albersheim, L. (2000). Attachment security in infancy and early adulthood: A twenty-year longitudinal study. *Child Development*, *71*(3), 684–689. http://doi.org/10.1111/1467-8624.00176

Figure 1.1. Overall conceptual model.



Chapter 2

Associations Between Childhood Maltreatment and Binge Eating-Related Concerns Among Young Adults

Introduction

A binge eating episode is characterized by overeating (i.e., eating an unusually large quantity of food in a discrete period of time) accompanied by a sense of loss of control over eating (American Psychiatric Association, 2013). Binge eating-related concerns, which include embarrassment over amount eaten and fear of losing control over eating (Gormally, Black, Daston, & Rardin, 1982), have been identified as key characteristics prospectively predicting eating disorder onset (Fairburn, Cooper, Doll, & Davies, 2005). Given their predictive utility, reducing binge eating-related concerns may be a promising target for eating disorders intervention.

Childhood maltreatment, which encompasses various forms of childhood abuse and neglect, is common, with global prevalence estimates ranging from 4-23% for physical abuse, 10-16% for physical neglect, and 5-13% for sexual abuse (Gilbert et al., 2009; Hussey, Chang, & Kotch, 2006; Stoltenborgh, Bakermans-Kranenburg, Alink, & van IJzendoorn, 2015). Childhood maltreatment has been found to be associated with eating disorders (Afifi, Sareen, et al., 2017; Caslini et al., 2016; Molendijk, Hoek, Brewerton, & Elzinga, 2017), with particularly strong and consistent evidence for associations with eating disorders involving binge eating (Caslini et al., 2016; Molendijk et al., 2017). A history of childhood maltreatment is also associated with

greater psychiatric comorbidity, greater treatment attrition, greater diagnostic crossover, and lower rates of full recovery among eating disorder patients (Castellini et al., 2018). Although there is convincing evidence that childhood maltreatment plays an important role in the development and clinical course of eating disorders, it is not known whether childhood maltreatment is associated with binge eating-related concerns.

Types of childhood maltreatment often co-occur (Higgins & McCabe, 2001). Among individuals with a history of any childhood maltreatment, prevalence estimates of multi-type childhood maltreatment (i.e., experiencing more than one type of childhood maltreatment) range from 35% in community samples (Edwards, Holden, Felitti, & Anda, 2003) to 65% in samples identified through Child Protective Services case records (Kim, Mennen, & Trickett, 2017). Because types of childhood maltreatment often co-occur, examining types of childhood maltreatment in isolation may overestimate the unique associations between each individual type of childhood maltreatment and health outcomes (Armour, Elklit, & Christoffersen, 2014; Pears, Kim, & Fisher, 2008). Illustrating this idea, Afifi et al. (2017) found physical abuse, physical neglect, emotional abuse, emotional neglect, and sexual abuse to be associated with binge eating disorder among women when examining each type of childhood maltreatment in isolation, but only associations for sexual abuse and emotional abuse held when controlling for all types of childhood maltreatment. Additionally, growing evidence suggests that distinct childhood maltreatment profiles (e.g., exposure to physical abuse and sexual abuse versus exposure to physical abuse and physical neglect) have qualitatively different associations with health outcomes (Berzenski & Yates, 2011; Curran, Adamson, Stringer, Rosato, & Leavey, 2016). Thus, taking a person-centered approach (Von Eye & Bergman, 2003) would advance

understanding of how specific patterns of childhood maltreatment shape eating disorder risk and could help inform interventions to treat and prevent eating disorders.

While preventing childhood maltreatment from occurring in the first place is of utmost importance, current childhood maltreatment prevention efforts have limited reach. Thus, we must also develop ways to mitigate the consequences of childhood maltreatment once it has occurred. The identity disruption model is a theoretical model that seeks to explain the link between early life adversity and disordered eating behaviors (Vartanian, Hayward, Smyth, Paxton, & Touyz, 2018). The identity disruption model posits that early life adversity leads to less clearly defined views of the self, which leads to greater internalization of appearance ideals, and, in turn, greater levels of disordered eating behaviors, including binge eating (Vartanian et al., 2018). Individuals with less clearly defined views of the self tend to have lower self-esteem (Campbell, 1990), and childhood maltreatment is associated with lower self-esteem (Greger, Myhre, Klöckner, & Jozefiak, 2017; Ju & Lee, 2018). Thus, low self-esteem may be a mediator in the pathway from childhood maltreatment to binge eating-related consequences. Improving self-esteem may be a way to mitigate the consequences of childhood maltreatment once it has occurred, as low self-esteem is modifiable (Haney & Durlak, 1998). Adolescence is a key developmental period for the formation of self-esteem (Robins & Trzesniewski, 2005), and, as such, could be an important developmental period to target for intervention. If self-esteem in adolescence mediates associations between childhood maltreatment and binge eating-related concerns, eating disorders treatment and prevention efforts for individuals with a history of childhood maltreatment could focus on improving self-esteem in adolescence.

Using a person-centered approach and data from a large, nationally representative sample of young adults in the United States, the objectives of this study were to (a) identify distinct

childhood maltreatment profiles, (b) examine associations between childhood maltreatment profiles and binge eating-related concerns in young adulthood, and, if such associations exist, (c) assess the extent to which self-esteem in adolescence mediates such associations.

Methods

Participants

This study used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health; Harris, 2009). 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 I data were collected in 1994-1995 when participants were in grades 7-12, Wave II data were collected in 1996 when participants were in grades 8-12, and Wave III data were collected in 2001-2002 when participants were 18-26 years old (Harris et al., 2009). Of the 15,197 participants interviewed at Wave III, 875 participants were excluded due to missing sampling weights, leaving 14,322 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 (Harris et al., 2009).

Measures

Childhood maltreatment. Childhood maltreatment was assessed retrospectively at Wave III using Computer-Assisted Self-Interview (Harris et al., 2009), a method that has been shown to elicit more accurate reporting than interviewer-administered assessment for questions of a sensitive nature (Metzger et al., 2000). Participants were asked about the following occurrences prior to sixth grade: “How often had your parents or other adult care-givers slapped,

hit, or kicked you?" (physical abuse), "How often had your parents or other adult care-givers not taken care of your basic needs, such as keeping you clean or providing food or clothing?" (physical neglect), and "How often had one of your parents or other adult care-givers touched you in a sexual way, forced you to touch him or her in a sexual way, or forced you to have sexual relations?" (sexual abuse). Possible response options for each question were *one time*, *two times*, *three to five times*, *six to ten times*, *more than ten times*, and *this has never happened*. We collapsed these response options in two ways: we created dichotomous variables representing each type of maltreatment having never occurred versus having ever occurred (one time or more), and we created ordinal variables representing each type of maltreatment having never occurred, having occurred one to two times, or having occurred three or more times. We also created a dichotomous variable (hereafter referred to as "any childhood maltreatment") representing none of the three types of maltreatment having ever occurred versus any of the three types of maltreatment having ever occurred. In sensitivity analyses, we substituted physical abuse assessed at Wave IV (which does not include slapping) with the question "Before your 18th birthday, how often did a parent or adult caregiver hit you with a fist, kick you, or throw you down on the floor, into a wall, or down stairs?" for physical abuse assessed at Wave III (which does include slapping), restricting physical abuse assessed at Wave IV to occurrences that began before age 12.

Binge eating-related concerns in young adulthood. Binge eating-related concerns were assessed at Wave III via self-report with the following items derived from the Eating Concern subscale of the Eating Disorder Examination-Questionnaire (Fairburn & Beglin, 1994): "In the past seven days, have you eaten so much in a short period that you would have been embarrassed if others had seen you do it?" and "In the past seven days, have you been afraid to start eating

because you thought you wouldn't be able to stop or control your eating?" A positive response to the dichotomous variable for binge eating-related concerns was assigned to participants with an affirmative response to at least one of these items.

Self-esteem in adolescence. Self-esteem was assessed at Waves I and II with six items modified from the Rosenberg Self-Esteem Scale, a measure of global self-esteem (Rosenberg, 1965). A five-point agreement scale was used for the following items: "You have a lot of good qualities," "You have a lot to be proud of," "You like yourself just the way you are," "You feel like you are doing everything just about right," "You feel socially accepted," and "You feel loved and wanted." Wave I data were used for participants with complete data at Wave I; Wave II data were used for participants with missing data at Wave I but complete data at Wave II. We averaged responses to yield a continuous variable with possible scores ranging from 1-5, with higher scores indicating lower levels of self-esteem. The scale demonstrated good internal consistency in this sample ($\alpha = .84$).

Demographic covariates. The following variables were included as demographic covariates: age at Wave III (continuous), sex (dichotomous), race/ethnicity (categorical: non-Hispanic white, non-Hispanic black, or 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 during adolescence, and 1994 federal poverty guidelines).

Statistical analysis

All analyses accounted for the complex sampling design used in Add Health, and all analyses were conducted with SAS 9.4 unless otherwise noted.

Descriptive statistics. We computed univariate statistics for childhood maltreatment, binge eating-related concerns, self-esteem, and demographic covariates. We also computed bivariate statistics by sex, by binge eating-related concerns, by any childhood maltreatment, and by childhood maltreatment latent classes.

Latent class analysis. We utilized latent class analysis (LCA) to identify distinct childhood maltreatment profiles. LCA is a person-centered approach (Von Eye & Bergman, 2003) that has facilitated important insights regarding eating disorders classification (Peterson et al., 2011; Swanson et al., 2014) and childhood maltreatment patterns (Brumley, Brumley, & Jaffee, 2018; Debowska, Willmott, Boduszek, & Jones, 2017). We conducted two latent class analyses using physical abuse, physical neglect, and sexual abuse as indicators: one with dichotomous childhood maltreatment indicators, and one with ordinal childhood maltreatment indicators. We conducted both analyses because based on a previous study which used LCA to examine maltreatment profiles (Berzenski & Yates, 2011), we expected to identify only two latent classes – no/low probability of maltreatment and high probability of maltreatment – in the analysis with dichotomous indicators. Rather than conduct a subsequent analysis among participants who reported maltreatment to identify classes defined by specific maltreatment types as in the study by Berzenski & Yates, we anticipated that adding information on frequency of each type of maltreatment might enable LCA to reveal the most informative and relevant patterns of maltreatment. We conducted an additional LCA with dichotomous childhood maltreatment indicators, substituting the Wave IV physical abuse variable for the Wave III physical abuse variable. For each analysis, minimum Bayesian Information Criterion (BIC; Schwarz, 1978) was used to determine which number of classes provided the optimal balance between model fit and parsimony, as BIC has been shown to outperform other information criteria in LCA model

selection (Nylund, Asparouhov, & Muthén, 2007). For comparison, we also reported Akaike's Information Criterion (AIC; Akaike, 1987). After identifying the models providing the optimal balance between model fit and parsimony, each participant was assigned to latent classes based on maximum posterior probability. We conducted LCA with Latent GOLD 5.1, using the Advanced/Syntax add-on to account for the complex sampling design (Vermunt & Magidson, 2016).

Multiple imputation. Data were missing at the following rates: 25% for percent federal poverty level, 7% for childhood maltreatment, 5% for highest parental education, and less than 1% for binge eating-related concerns, self-esteem, 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 (SAS Institute Inc., 2015a). Auxiliary variables in the imputation model included childhood physical neglect, physical abuse, and sexual abuse assessed at Wave III, childhood physical abuse, sexual abuse, and emotional abuse assessed at Wave IV, depressive symptoms in adolescence, parent-reported household income, household size during adolescence, parent-reported receipt of public assistance, body mass index at Wave III, embarrassment over amount eaten reported at Wave III, fear of losing control over eating reported at Wave III, lifetime eating disorder diagnosis reported at Wave III, and disordered weight control behaviors (fasting, using diet pills, and purging) reported at Wave III. In sensitivity analyses, we conducted analyses with only demographic covariates imputed and using complete case data only.

Logistic regression. We ran unadjusted and adjusted logistic regression models examining associations between childhood maltreatment latent classes and binge eating-related

concerns. The demographic covariates described above were included in adjusted models. We ran separate models for latent class membership from the model identified in the LCA with dichotomous indicators and latent class membership from the model identified in the LCA with ordinal indicators. We also ran a model for any childhood maltreatment (the dichotomous variable representing none of the three types of maltreatment having ever occurred versus any of the three types of maltreatment having ever occurred) to compare with models for the latent classes. Because sex differences in associations between childhood maltreatment and eating disorders have been observed (Afifi, Sareen, et al., 2017), we assessed for effect modification by sex by adding cross-product terms (childhood maltreatment x sex) to the adjusted models.

Combining inference from multiply imputed datasets. Results from logistic regression analyses were combined and summarized with the MIANALYZE procedure in SAS 9.4 (SAS Institute Inc., 2015b), using both within-imputation and between-imputation variance to reflect uncertainty due to the missing data (Little & Rubin, 2002).

Mediation analysis. To assess for mediation by self-esteem in adolescence, we ran the logistic regression models described above additionally adjusting for self-esteem in adolescence, and we ran linear regression models examining associations between childhood maltreatment variables and self-esteem in adolescence, adjusted for demographic covariates. For observed associations between childhood maltreatment variables and binge eating-related concerns, we used the results from these models to calculate the natural direct effect, natural indirect effect, and proportion mediated by self-esteem in adolescence using the approach described by Vanderweele and Vansteelandt (2010) for mediation analysis with dichotomous outcomes in a counterfactual framework. We obtained 95% confidence intervals via bootstrapping with 1,000 resamples.

Results

Descriptives

Univariate descriptive statistics and bivariate statistics by sex are presented in Table 2.1. In this sample of young adults (mean age = 21.82 years), binge eating-related concerns were reported by 7.3% of participants. Prevalence of binge eating-related concerns differed by sex, with females reporting higher prevalence than males ($p < .001$). Childhood physical abuse was reported by 28.4% of participants, childhood physical neglect was reported by 11.6% of participants, and childhood sexual abuse was reported by 4.5% of participants, with an overall prevalence of 34.2% for any childhood maltreatment. Prevalence of childhood physical abuse ($p = .01$) and childhood physical neglect ($p < .001$) differed by sex, with males reporting higher prevalence than females for each. Prevalence of childhood sexual abuse did not differ by sex ($p = .79$).

Bivariate descriptive statistics by binge eating-related concerns are presented in Table 2.2. Race/ethnicity ($p = .008$) and self-esteem in adolescence ($p < .001$) differed between participants reporting binge eating-related concerns and those not reporting binge eating-related concerns. Bivariate descriptive statistics by any childhood maltreatment are presented in Table 2.3. With the exception of age, all variables examined differed between participants reporting childhood maltreatment and those not reporting childhood maltreatment (all p's $< .001$).

Latent class analysis

LCA model fit indices are presented in Table 2.4. Both BIC and AIC were lowest for a two-class model in the LCA with dichotomous indicators and for a five-class model in the LCA with ordinal indicators, indicating that these models provided the optimal balance between model fit and parsimony. Probability estimates of each type of childhood maltreatment for each latent

class from the models identified in each LCA are displayed in Figure 2.1. In the model identified from the LCA with dichotomous indicators, Class 1 was characterized by low probability of maltreatment ("no/low maltreatment") and comprised 92.2% of the sample, and Class 2 was characterized by high probability of each type of maltreatment ("maltreatment") and comprised 7.8% of the sample. In the model identified from the LCA with ordinal indicators, Class 1 was characterized by low probability of childhood maltreatment ("no/low maltreatment") and comprised 78.5% of the sample, Class 2 was characterized by high probability of childhood physical abuse only ("physical abuse only") and comprised 11.0% of the sample, Class 3 was characterized by high probability of each childhood maltreatment type ("multi-type maltreatment") and comprised 7.8% of the sample, Class 4 was characterized by high probability of childhood physical neglect only ("physical neglect only") and comprised 2.1% of the sample, and Class 5 was characterized by high probability of childhood sexual abuse only ("sexual abuse only") and comprised 0.6% of the sample. Participants assigned to Class 2 in the LCA with dichotomous indicators were the same participants assigned to Class 3 in the LCA with ordinal indicators.

Bivariate descriptive statistics by childhood maltreatment latent classes are presented in Tables 2.5 and 2.6. With the exception of age, all variables examined differed between the latent classes from the LCA with dichotomous indicators (all p's < .001). All variables examined differed between the latent classes from the LCA with ordinal indicators (age: $p = .01$; all other variables: $p < .001$).

LCA model fit indices from the additional LCA with dichotomous childhood maltreatment indicators substituting physical abuse assessed at Wave IV for physical abuse assessed at Wave III are presented in Table 2.7. Both BIC and AIC were lowest for a two-class

model. Probability estimates of each type of childhood maltreatment for each latent class from the model identified are displayed in Figure 2.2. Class 1 was characterized by low probability of maltreatment ("no/low maltreatment") and comprised 85.6% of the sample, and Class 2 was characterized by high probability of each type of maltreatment ("maltreatment") and comprised 14.4% of the sample.

Associations between childhood maltreatment and binge eating-related concerns

Unadjusted and adjusted associations between childhood maltreatment and binge eating-related concerns are presented in Table 2.8. After adjusting for age, sex, race/ethnicity, highest parental education, and percent federal poverty level in adolescence, participants who reported any childhood maltreatment were 1.67 times more likely to report binge eating-related concerns (95% confidence interval [CI]: 1.42, 1.97) than those reporting no childhood maltreatment. Similarly, participants assigned to the "maltreatment" class in the LCA with dichotomous indicators were 1.93 times more likely to report binge eating-related concerns (95% CI: 1.50, 2.48) than those assigned to the "no/low maltreatment" class, and participants assigned to the "multi-type maltreatment" class in the LCA with ordinal indicators were 1.97 times more likely to report binge eating-related concerns (95% CI: 1.52, 2.56) than those assigned to the "no/low maltreatment" class. None of the single-type maltreatment classes ("physical abuse only," "physical neglect only," or "sexual abuse only") were significantly associated with binge eating-related concerns. There was no evidence for effect modification of associations between childhood maltreatment and binge eating-related concerns by sex for any childhood maltreatment variable (all cross-product p's > .40).

Sensitivity analysis results comparing estimates using Wave IV physical abuse (which does not include slapping; 8.0% prevalence) to estimates using Wave III physical abuse (which

does include slapping; 28.4% prevalence) are presented in Table 2.9. While results using latent classes were similar, the association for any maltreatment (21.6% prevalence using Wave IV physical abuse versus 34.2% prevalence using Wave III physical abuse) was stronger when using Wave IV physical abuse (odds ratio [OR] = 1.86; 95% CI: 1.41, 2.46) than when using Wave III physical abuse (OR = 1.67; 95% CI: 1.42, 1.97).

Sensitivity analysis results using complete case data, imputed demographic covariates, and all variables imputed are presented in Table 2.10. Results were not substantially different in analyses using complete case data only or imputing only demographic covariates, with the exception that when imputing only demographic covariates, a stronger and statistically significant association emerged for the "sexual abuse only" class from the LCA with ordinal indicators. Participants assigned to the "sexual abuse only" class were 2.37 times more likely to report binge eating-related concerns (95% CI: 1.04, 5.39) than those assigned to the "no/low maltreatment" class.

Mediation by self-esteem in adolescence

Results of the mediation analyses are presented in Table 2.11. The odds ratio for the natural direct effect of any maltreatment was 1.63 (95% CI: 1.57, 1.70), and the odds ratio for the natural indirect effect of any maltreatment through self-esteem was 1.03 (95% CI: 1.02, 1.03). The proportion of the association between any maltreatment and binge eating-related concerns mediated by self-esteem in adolescence was 6.6% (95% CI: 5.6%, 7.8%).

The odds ratio for the natural direct effect of the "maltreatment" class from the LCA with dichotomous indicators was 1.87 (95% CI: 1.77, 1.99), and the odds ratio for the natural indirect effect of the "maltreatment" class through self-esteem was 1.03 (95% CI: 1.03, 1.04). The

proportion of the association between the "maltreatment" class and binge eating-related concerns mediated by self-esteem in adolescence was 6.2% (95% CI: 5.3%, 7.7%).

The odds ratio for the natural direct effect of the "multi-type maltreatment" class from the LCA with ordinal indicators was 1.90 (95% CI: 1.79, 2.03), and the odds ratio for the natural indirect effect of the "multi-type maltreatment" class through self-esteem was 1.04 (95% CI: 1.03, 1.04). The proportion of the association between the "multi-type maltreatment" class and binge eating-related concerns mediated by self-esteem in adolescence was 6.9% (95% CI: 6.0%, 8.7%).

Discussion

The aims of this study were to identify distinct childhood maltreatment classes via latent class analysis based on the occurrence of physical abuse, physical neglect, and sexual abuse in childhood, to examine associations between childhood maltreatment classes and binge eating-related concerns in young adulthood, and to assess the extent to which self-esteem in adolescence mediates such associations. When only considering whether or not each type of childhood maltreatment had ever occurred, we identified two childhood maltreatment classes: "no/low maltreatment" and "maltreatment." When preserving more information in the observed variables by also considering the frequency with which each type of childhood maltreatment had occurred, we identified five childhood maltreatment classes: "no/low maltreatment," "physical abuse only," "multi-type maltreatment," "physical neglect only," and "sexual abuse only." Participants who reported any childhood maltreatment were more likely to report binge eating-related concerns compared to those not reporting any childhood maltreatment, and participants assigned to the "maltreatment" and "multi-type maltreatment" classes were more likely to report

binge eating-related concerns compared to those assigned to the "no/low maltreatment" classes. Self-esteem in adolescence mediated a statistically significant but modest proportion of each of these associations. However, we did not observe associations between the single-type childhood maltreatment classes and binge eating-related concerns. These findings highlight the importance of considering the overall childhood maltreatment profile rather than focusing on individual types of childhood maltreatment.

Over 1,000 participants (7.8% of the sample) in this study were assigned to the "multi-type maltreatment" class, supporting previous findings that different types of childhood maltreatment often co-occur (Edwards et al., 2003; Higgins & McCabe, 2001; Kim et al., 2017). Our results suggest that exposure to multiple types of childhood maltreatment may increase risk for binge eating-related concerns, whereas exposure to isolated types of childhood maltreatment may not. These results cohere with previous findings that individuals with a history of multi-type childhood maltreatment, but not single-type childhood maltreatment, have greater depressive symptoms and suicidality than individuals with no history of childhood maltreatment (Arata, Langhinrichsen-Rohling, Bowers, & O'Farrill-Swails, 2005). Multi-type childhood maltreatment may confer greater risk for adverse mental health outcomes than single-type childhood maltreatment not only because of the cumulative effects of the multiple types of maltreatment, but also because multi-type childhood maltreatment be a marker of a more adverse home environment overall. For instance, exposure to more types of childhood maltreatment has been found to be associated with lower caretaker functioning and more exposure to domestic violence in the home (Kim et al., 2017). However, other studies have found dose-response relationships between the number of types of childhood maltreatment reported and adverse mental health outcomes, such that individuals reporting single-type childhood maltreatment were more likely

to have adverse mental health outcomes than their non-maltreated counterparts (Edwards et al., 2003). Thus, single-type childhood maltreatment also has detrimental consequences, but the detrimental consequences may not be as severe or may manifest in different ways compared to multi-type childhood maltreatment. Taking a person-centered approach enables us to gain insight as to how detrimental consequences may manifest differently across unique childhood maltreatment profiles.

When using a childhood physical abuse measure that includes slapping, results across childhood maltreatment variables demonstrated that while any childhood maltreatment was associated with binge eating-related concerns, associations were stronger for the "maltreatment" class in the LCA with dichotomous indicators and the "multi-type maltreatment" class in the LCA with ordinal indicators. However, in sensitivity analyses using a childhood physical abuse measure that does not include slapping, associations for any childhood maltreatment and the "maltreatment" class identified via LCA were more similar in strength. Taken together, these results suggest that slapping (which includes spanking) may be less strongly associated with binge eating-related concerns than other forms of physical abuse. However, this does not mean that slapping is not harmful in other domains, as previous work has found spanking to be associated with a variety of other adverse mental health outcomes (Afifi, Ford, et al., 2017).

Self-esteem in adolescence did not emerge as a salient factor driving the observed associations between childhood maltreatment variables and binge eating-related concerns. Although it mediated statistically significant proportions of these associations, the proportions were modest, which may be related to the fact that low self-esteem is a somewhat non-specific risk factor for a broad range of adverse mental health outcomes (Mann, Hosman, Schaalma, & de Vries, 2004). The non-specificity of low self-esteem as a risk factor may make it a less potent

mediator on the pathway from childhood maltreatment to binge eating-related concerns. Nevertheless, the non-specific nature of self-esteem may also translate to a valuable role for self-esteem in transdiagnostic treatment and prevention efforts. For example, interventions targeting self-esteem have been found to not only improve self-esteem, but also reduce symptoms of depression, anxiety, and eating disorders (Musiat et al., 2014; Tirlea, Truby, & Haines, 2016). Future studies should examine other potential mediators that could be more specific to binge eating-related outcomes, such as dissociation and impulsivity (Lee-Winn, Townsend, Reinblatt, & Mendelson, 2016; Mason et al., 2017).

This study had several strengths. We used data from a large, nationally representative sample of young adults in the United States. Using a community sample rather than a clinical sample avoids bias introduced by studying treatment-seeking individuals, and young adulthood is a critical period, as levels of cognitive features of eating disorders have been found to increase during this period (Slane, Klump, McGue, & Iacono, 2014). Additionally, our sample included males, a group that has been severely underrepresented in research examining associations between childhood maltreatment and eating disorders (Caslini et al., 2016). We did not find evidence for differences by sex, highlighting the importance of including males in this area of research. Further, using LCA allowed us to efficiently address the interrelatedness yet distinct qualities of multiple types of childhood maltreatment, harnessing a person-centered approach to foster better understanding of pathways from childhood maltreatment to binge eating-related concerns.

Despite these strengths, this study also had limitations, which included the retrospective self-report of childhood maltreatment and the use of single-item measures with a seven-day assessment time frame to assess binge eating-related concerns. Another limitation was the

narrow range of childhood maltreatment types that were assessed (e.g., emotional abuse and neglect were not assessed). Additionally, residual confounding is a possibility, and latent class assignment does not convey the probabilistic nature of the latent class model. Not accounting for the uncertainty in class assignment can lead to underestimation of standard errors in logistic regression. Despite these limitations, findings from this study offer important contributions to understanding the relationship between childhood maltreatment and eating disorders.

The findings from this study provide evidence for the existence of distinct childhood maltreatment profiles that are differentially associated with binge eating-related concerns. Exposure to multiple types of childhood maltreatment may confer risk for binge eating-related concerns, while exposure to isolated types of childhood maltreatment may not. Thus, individuals exposed to multiple types of childhood maltreatment may be at particularly high risk for eating disorders. Accurately classifying childhood maltreatment profiles is not only valuable for identifying high-risk subgroups, but also a necessary step towards better informing eating disorders treatment and prevention for those high-risk subgroups. From a treatment perspective, using trauma-informed approaches (Brewerton, 2018; Trottier, Monson, Wonderlich, MacDonald, & Olmsted, 2017) may be necessary to effectively treat eating disorders among patients with a history of childhood maltreatment, and the appropriate course of treatment may differ depending on the childhood maltreatment profile. From a prevention perspective, in addition to strengthening strategies to prevent childhood maltreatment from occurring in the first place, targeted prevention programs for individuals with a history of childhood maltreatment – particularly during the developmental period of adolescence – may maximize effectiveness by taking a personalized prevention approach, providing tailored feedback based on risk profiling. Ongoing research that uncovers modifiable mediators of the association between childhood

maltreatment and eating disorders and tests the efficacy of trauma-informed treatment approaches and targeted prevention programs are needed to help minimize the long-lasting consequences of childhood maltreatment and reduce the public health burden of eating disorders.

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References

Afifi, T. O., Ford, D., Gershoff, E. T., Merrick, M., Grogan-Kaylor, A., Ports, K. A., … Peters Bennett, R. (2017). Spanking and adult mental health impairment: The case for the designation of spanking as an adverse childhood experience. *Child Abuse & Neglect*, *71*, 24–31. http://doi.org/10.1016/j.chiabu.2017.01.014

Afifi, T. O., Sareen, J., Fortier, J., Taillieu, T., Turner, S., Cheung, K., & Henriksen, C. A. (2017). Child maltreatment and eating disorders among men and women in adulthood: Results from a nationally representative United States sample. *International Journal of Eating Disorders*, *50*(11), 1281–1296. http://doi.org/10.1002/eat.22783

Akaike, H. (1987). Factor analysis and AIC. *Psychometrika*, *52*(3), 317–332.

American Psychiatric Association. (2013). Feeding and Eating Disorders. In *Diagnostic and Statistical Manual of Mental Disorders (DSM-5®)*. American Psychiatric Association. http://doi.org/https://doi.org/10.1176/appi.books.9780890425596.dsm10

Arata, C. M., Langhinrichsen-Rohling, J., Bowers, D., & O'Farrill-Swails, L. (2005). Single versus multi-type maltreatment. *Journal of Aggression, Maltreatment & Trauma*, *11*(4), 29–52. http://doi.org/10.1300/J146v11n04_02

Armour, C., Elklit, A., & Christoffersen, M. N. (2014). A latent class analysis of childhood maltreatment: Identifying abuse typologies. *Journal of Loss and Trauma*, *19*(1), 23–39. http://doi.org/10.1080/15325024.2012.734205

Berzenski, S. R., & Yates, T. M. (2011). Classes and consequences of multiple maltreatment: A person-centered analysis. *Child Maltreatment*, *16*(4), 250–261. http://doi.org/10.1177/1077559511428353

Brewerton, T. D. (2018). An overview of trauma-informed care and practice for eating disorders.

Journal of Aggression, Maltreatment & Trauma, 1–18. http://doi.org/10.1080/10926771.2018.1532940

Brumley, L. D., Brumley, B. P., & Jaffee, S. R. (2018). Comparing cumulative index and factor analytic approaches to measuring maltreatment in the National Longitudinal Study of Adolescent to Adult Health. *Child Abuse & Neglect*. http://doi.org/10.1016/J.CHIABU.2018.08.014

Campbell, J. D. (1990). Self-esteem and clarity of the self-concept. *Journal of Personality and Social Psychology*, *59*(3), 538–549. http://doi.org/10.1037/0022-3514.59.3.538

Caslini, M., Bartoli, F., Crocamo, C., Dakanalis, A., Clerici, M., & Carrà, G. (2016). Disentangling the association between child abuse and eating disorders: A systematic review and meta-analysis. *Psychosomatic Medicine*, *78*(1), 79–90. http://doi.org/10.1097/PSY.0000000000000233

Castellini, G., Lelli, L., Cassioli, E., Ciampi, E., Zamponi, F., Campone, B., … Ricca, V. (2018). Different outcomes, psychopathological features, and comorbidities in patients with eating disorders reporting childhood abuse: A 3-year follow-up study. *European Eating Disorders Review*. http://doi.org/10.1002/erv.2586

Curran, E., Adamson, G., Stringer, M., Rosato, M., & Leavey, G. (2016). Severity of mental illness as a result of multiple childhood adversities: US National Epidemiologic Survey. *Social Psychiatry and Psychiatric Epidemiology*, *51*(5), 647–657. http://doi.org/10.1007/s00127-016-1198-3

Debowska, A., Willmott, D., Boduszek, D., & Jones, A. D. (2017). What do we know about child abuse and neglect patterns of co-occurrence? A systematic review of profiling studies and recommendations for future research. *Child Abuse & Neglect*, *70*, 100–111.

http://doi.org/10.1016/J.CHIABU.2017.06.014

Edwards, V. J., Holden, G. W., Felitti, V. J., & Anda, R. F. (2003). Relationship between multiple forms of childhood maltreatment and adult mental health in community respondents: Results from the Adverse Childhood Experiences Study. *The American Journal of Psychiatry*, *160*(8), 1453–1460. http://doi.org/10.1176/appi.ajp.160.8.1453

Fairburn, C., & Beglin, S. (1994). Assessment of eating disorders: interview or self-report questionnaire? *International Journal of Eating Disorders*, *16*(4), 363–370.

Fairburn, C. G., Cooper, Z., Doll, H. A., & Davies, B. A. (2005). Identifying dieters who will develop an eating disorder: a prospective, population-based study. *American Journal of Psychiatry*, *162*(12), 2249–2255. http://doi.org/10.1176/appi.ajp.162.12.2249

Gilbert, R., Widom, C. S., Browne, K., Fergusson, D., Webb, E., & Janson, S. (2009). Burden and consequences of child maltreatment in high-income countries. *Lancet*, *373*(9657), 68–81. http://doi.org/10.1016/S0140-6736(08)61706-7

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors*, *7*(1), 47–55. http://doi.org/10.1016/0306-4603(82)90024-7

Greger, H. K., Myhre, A. K., Klöckner, C. A., & Jozefiak, T. (2017). Childhood maltreatment, psychopathology and well-being: The mediator role of global self-esteem, attachment difficulties and substance use. *Child Abuse & Neglect*, *70*, 122–133. http://doi.org/10.1016/j.chiabu.2017.06.012

Haney, P., & Durlak, J. A. (1998). Changing self-esteem in children and adolescents: A meta-analytical review. *Journal of Clinical Child Psychology*, *27*(4), 423–433. http://doi.org/10.1207/s15374424jccp2704_6

Harris, K. M. (2009). The National Longitudinal Study of Adolescent to Adult Health (Add Health), Waves I & II, 1994–1996; Wave III, 2001–2002; Wave IV, 2007-2009 [machine-readable data file and documentation]. Chapel Hill, NC: Carolina Population Center, University of North Carolina at Chapel Hill. http://doi.org/10.3886/ICPSR27021.v9

Harris, K. M., Halpern, C. T., Whitsel, E., Hussey, J., Tabor, J., Entzel, P., & Udry, J. R. (2009). The National Longitudinal Study of Adolescent to Adult Health: Research Design [WWW document]. Retrieved April 1, 2016, from http://www.cpc.unc.edu/projects/addhealth/design

Higgins, D. J., & McCabe, M. P. (2001). Multiple forms of child abuse and neglect: Adult retrospective reports. *Aggression and Violent Behavior*, *6*(6), 547–578. http://doi.org/10.1016/S1359-1789(00)00030-6

Hussey, J. M., Chang, J. J., & Kotch, J. B. (2006). Child maltreatment in the United States: prevalence, risk factors, and adolescent health consequences. *Pediatrics*, *118*(3), 933–942. http://doi.org/10.1542/peds.2005-2452

Ju, S., & Lee, Y. (2018). Developmental trajectories and longitudinal mediation effects of self-esteem, peer attachment, child maltreatment and depression on early adolescents. *Child Abuse & Neglect*, *76*, 353–363. http://doi.org/10.1016/j.chiabu.2017.11.015

Kim, K., Mennen, F. E., & Trickett, P. K. (2017). Patterns and correlates of co-occurrence among multiple types of child maltreatment. *Child & Family Social Work*, *22*(1), 492–502. http://doi.org/10.1111/cfs.12268

Lee-Winn, A. E., Townsend, L., Reinblatt, S. P., & Mendelson, T. (2016). Associations of neuroticism and impulsivity with binge eating in a nationally representative sample of adolescents in the United States. *Personality and Individual Differences*, *90*, 66–72.

http://doi.org/10.1016/j.paid.2015.10.042

Little, R. J. A., & Rubin, D. B. (2002). *Statistical Analysis with Missing Data* (2nd ed). Hoboken, NJ: John Wiley & Sons, Inc.

Mann, M., Hosman, C. M. H., Schaalma, H. P., & de Vries, N. K. (2004). Self-esteem in a broad-spectrum approach for mental health promotion. *Health Education Research*, *19*(4), 357–372. http://doi.org/10.1093/her/cyg041

Mason, T. B., Lavender, J. M., Wonderlich, S. A., Steiger, H., Cao, L., Engel, S. G., … Crosby, R. D. (2017). Comfortably numb: The role of momentary dissociation in the experience of negative affect around binge eating. *The Journal of Nervous and Mental Disease*, *205*(5), 335–339. http://doi.org/10.1097/NMD.0000000000000658

Metzger, D. S., Koblin, B., Turner, C., Navaline, H., Valenti, F., Holte, S., … Seage, G. R. (2000). Randomized controlled trial of audio computer-assisted self-interviewing: utility and acceptability in longitudinal studies. *American Journal of Epidemiology*, *152*(2), 99–106. http://doi.org/10.1093/aje/152.2.99

Molendijk, M. L., Hoek, H. W., Brewerton, T. D., & Elzinga, B. M. (2017). Childhood maltreatment and eating disorder pathology: a systematic review and dose-response meta-analysis. *Psychological Medicine*, *47*(8), 1402–1416. http://doi.org/10.1017/S0033291716003561

Musiat, P., Conrod, P., Treasure, J., Tylee, A., Williams, C., & Schmidt, U. (2014). Targeted prevention of common mental health disorders in university students: Randomised controlled trial of a transdiagnostic trait-focused web-based intervention. *PLoS ONE*, *9*(4), 1–10. http://doi.org/10.1371/journal.pone.0093621

Nylund, K. L., Asparouhov, T., & Muthén, B. O. (2007). Deciding on the number of classes in

latent class analysis and growth mixture modeling: A Monte Carlo simulation study. *Structural Equation Modeling*, *14*(4), 535–569. http://doi.org/10.1080/10705510701575396

Pears, K. C., Kim, H. K., & Fisher, P. A. (2008). Psychosocial and cognitive functioning of children with specific profiles of maltreatment. *Child Abuse & Neglect*, *32*(10), 958–971. http://doi.org/10.1016/j.chiabu.2007.12.009

Peterson, C. B., Crow, S. J., Swanson, S. A., Crosby, R. D., Wonderlich, S. A., Mitchell, J. E., … Halmi, K. A. (2011). Examining the stability of DSM-IV and empirically derived eating disorder classification: implications for DSM-5. *Journal of Consulting and Clinical Psychology*, *79*(6), 777–83. http://doi.org/10.1037/a0025941

Robins, R. W., & Trzesniewski, K. H. (2005). Self-esteem development across the lifespan. *Current Directions in Psychological Science*, *14*(3), 158–162.

Rosenberg, M. (1965). *Society and the adolescent self-image*. Princeton (NJ): Princeton University Press.

SAS Institute Inc. (2015a). The MI Procedure. In *SAS/STAT® 14.1 User's Guide*. Cary, NC: SAS Institute Inc.

SAS Institute Inc. (2015b). The MIANALYZE Procedure. In *SAS/STAT® 14.1 User's Guide*. Cary, NC: SAS Institute Inc.

Schwarz, G. (1978). Estimating the dimension of a model. *The Annals of Statistics*, *6*(2), 461–464. http://doi.org/10.1214/aos/1176344136

Slane, J. D., Klump, K. L., McGue, M., & Iacono, W. G. (2014). Developmental trajectories of disordered eating from early adolescence to young adulthood: a longitudinal study. *The International Journal of Eating Disorders*, *47*(7), 793–801. http://doi.org/10.1002/eat.22329

Stoltenborgh, M., Bakermans-Kranenburg, M. J., Alink, L. R. A., & van IJzendoorn, M. H.

(2015). The prevalence of child maltreatment across the globe: Review of a series of meta-analyses. *Child Abuse Review*, *24*(1), 37–50. http://doi.org/10.1002/car.2353

Swanson, S. A., Horton, N. J., Crosby, R. D., Micali, N., Sonneville, K. R., Eddy, K., & Field, A. E. (2014). A latent class analysis to empirically describe eating disorders through developmental stages. *The International Journal of Eating Disorders*, *47*(7), 762–72. http://doi.org/10.1002/eat.22308

Tirlea, L., Truby, H., & Haines, T. P. (2016). Pragmatic, randomized controlled trials of the Girls on the Go! Program to improve self-esteem in girls. *American Journal of Health Promotion*, *30*(4), 231–41. http://doi.org/10.1177/0890117116639572

Trottier, K., Monson, C. M., Wonderlich, S. A., MacDonald, D. E., & Olmsted, M. P. (2017). Frontline clinicians' perspectives on and utilization of trauma-focused therapy with individuals with eating disorders. *Eating Disorders*, *25*(1), 22–36. http://doi.org/10.1080/10640266.2016.1207456

Vanderweele, T. J., & Vansteelandt, S. (2010). Odds ratios for mediation analysis for a dichotomous outcome. *American Journal of Epidemiology*, *172*(12), 1339–48. http://doi.org/10.1093/aje/kwq332

Vartanian, L. R., Hayward, L. E., Smyth, J. M., Paxton, S. J., & Touyz, S. W. (2018). Risk and resiliency factors related to body dissatisfaction and disordered eating: The identity disruption model. *International Journal of Eating Disorders*, *51*(4), 322–330. http://doi.org/10.1002/eat.22835

Vermunt, J. K., & Magidson, J. (2016). *Technical Guide for Latent GOLD 5 .1: Basic, Advanced, and Syntax*. *Statistical Innovations Inc.* Belmont, MA.

Von Eye, A., & Bergman, L. R. (2003). Research strategies in developmental psychopathology:

Dimensional identity and the person-oriented approach. *Development and Psychopathology*, *15*(3), 553–580. http://doi.org/10.1017/S0954579403000294

Table 2.1. Sample characteristics, overall and by sex.

	Overall (N = 14,322)	Males (N = 6,759)	Females (N = 7,563)	
	Sampled Frequency (Weighted Percent)			***p***
Race/ethnicity				
Non-Hispanic white	7,728 (67.6)	3,649 (67.3)	4,079 (67.8)	
Non-Hispanic black	3,038 (16.0)	1,323 (15.7)	1,715 (16.3)	.38
Other	3,514 (16.4)	1,761 (17.0)	1,753 (15.9)	
Percent federal poverty level				
< 100%	1,783 (16.4)	818 (16.0)	965 (16.8)	
100-199%	2,340 (20.9)	1,126 (21.1)	1,214 (20.6)	.63
200-399%	4,061 (38.1)	1,958 (38.7)	2,103 (37.5)	
≥ 400%	2,624 (24.7)	1,257 (24.2)	1,367 (25.1)	
Highest parental education				
Less than high school	1,694 (12.0)	751 (11.8)	943 (12.2)	
High school graduate or equivalent	3,974 (31.6)	1,880 (31.5)	2,094 (31.6)	.23
Some college/trade school	2,875 (21.4)	1,327 (20.6)	1,548 (22.1)	
Graduated college or above	5,083 (35.1)	2,473 (36.1)	2,610 (34.0)	
Any childhood maltreatment	4,717 (34.2)	2,355 (36.5)	2,362 (32.0)	<.001
Physical abuse	3,996 (28.4)	1,998 (29.9)	1,998 (27.0)	.01
Physical neglect	1,558 (11.6)	895 (14.5)	663 (8.8)	<.001
Sexual abuse	645 (4.5)	279 (4.5)	366 (4.6)	.79
Binge eating-related concerns	1,095 (7.3)	427 (5.9)	668 (8.8)	<.001
		Mean (Standard Error)		***p***
Age (years)	21.82 (0.12)	21.91 (0.12)	21.73 (0.12)	<.001
Self-esteem in adolescence	1.89 (0.01)	1.79 (0.01)	1.98 (0.01)	<.001

Higher self-esteem scores indicate lower levels of self-esteem.

Table 2.2. Bivariate descriptives by binge eating-related concerns.

	No Binge Eating-Related Concerns (N = 13,200)	Binge Eating-Related Concerns (N = 1,095)	
	Sampled Frequency (Weighted Percent)		***p***
Race/ethnicity			
Non-Hispanic white	7,186 (68.1)	533 (62.4)	
Non-Hispanic black	2,788 (15.8)	240 (17.3)	.008
Other	3,186 (16.1)	320 (20.4)	
Percent federal poverty level			
< 100%	1,614 (16.2)	163 (18.0)	
100-199%	2,152 (20.8)	184 (21.9)	.53
200-399%	3,765 (38.2)	290 (37.9)	
≥ 400%	2,444 (24.9)	176 (22.3)	
Highest parental education			
Less than high school	1,543 (11.8)	145 (14.4)	
High school graduate or equivalent	3,661 (31.5)	305 (31.6)	.25
Some college/trade school	2,654 (21.3)	214 (21.4)	
Graduated college or above	4,707 (35.3)	373 (32.6)	
	Mean (Standard Error)		***p***
Age (years)	21.82 (0.12)	21.79 (0.14)	.62
Self-esteem in adolescence	1.88 (0.01)	1.99 (0.03)	<.001

Higher self-esteem scores indicate lower levels of self-esteem.

Table 2.3. Bivariate descriptives by childhood maltreatment.

	No Childhood Maltreatment (N = 8,773)	Any Childhood Maltreatment (N = 4,717)	
	Sampled Frequency (Weighted Percent)		***p***
Sex			
Male	3,907 (48.3)	2,355 (53.3)	<.001
Female	4,866 (51.7)	2,362 (46.7)	
Race/ethnicity			
Non-Hispanic white	5,025 (70.6)	2,355 (63.4)	
Non-Hispanic black	1,870 (15.6)	959 (16.2)	<.001
Other	1,857 (13.8)	1,386 (20.5)	
Percent federal poverty level			
< 100%	990 (14.7)	669 (18.9)	
100-199%	1,342 (19.3)	828 (23.0)	<.001
200-399%	2,585 (39.9)	1,301 (36.3)	
≥ 400%	1,729 (26.1)	763 (21.8)	
Highest parental education			
Less than high school	943 (11.0)	624 (13.5)	
High school graduate or equivalent	2,392 (30.6)	1,314 (32.4)	<.001
Some college/trade school	1,726 (21.0)	1,011 (22.9)	
Graduated college or above	3,324 (37.4)	1,521 (31.3)	
Binge eating-related concerns	553 (6.1)	480 (9.7)	<.001
	Mean (Standard Error)		***p***
Age (years)	21.81 (0.12)	21.85 (0.13)	.48
Self-esteem in adolescence	1.84 (0.01)	1.96 (1.93)	<.001

Higher self-esteem scores indicate lower levels of self-esteem.

Table 2.4. Model fit indices for childhood maltreatment latent class analyses.

	BIC	AIC
LCA with dichotomous indicators (never, ≥ 1 time)		
1-class model	46,258,703.58	46,258,659.06
2-class model	**42,778,733.78**	**42,778,629.91**
LCA with ordinal indicators (never, 1-2 times, ≥ 3 times)		
1-class model	58,586,069.60	58,585,980.57
2-class model	56,000,066.97	55,999,918.59
3-class model	55,522,768.51	55,522,560.78
4-class model	55,143,193.93	55,142,926.84
5-class model	**55,012,346.82**	**55,012,020.38**
6-class model	55,013,689.81	55,013,304.02

BIC = Bayesian Information Criterion; AIC = Akaike's Information Criterion; LCA = latent class analysis. Lower BIC and AIC values indicate better model fit. Models with ≥ 3 classes and ≥ 7 classes were not identified for analyses with dichotomous and ordinal indicators, respectively. Bold indicates optimal balance between model fit and parsimony.

Figure 2.1. Probability estimates of each type of childhood maltreatment for each latent class from latent class analyses with dichotomous indicators (top) and ordinal indicators (bottom).

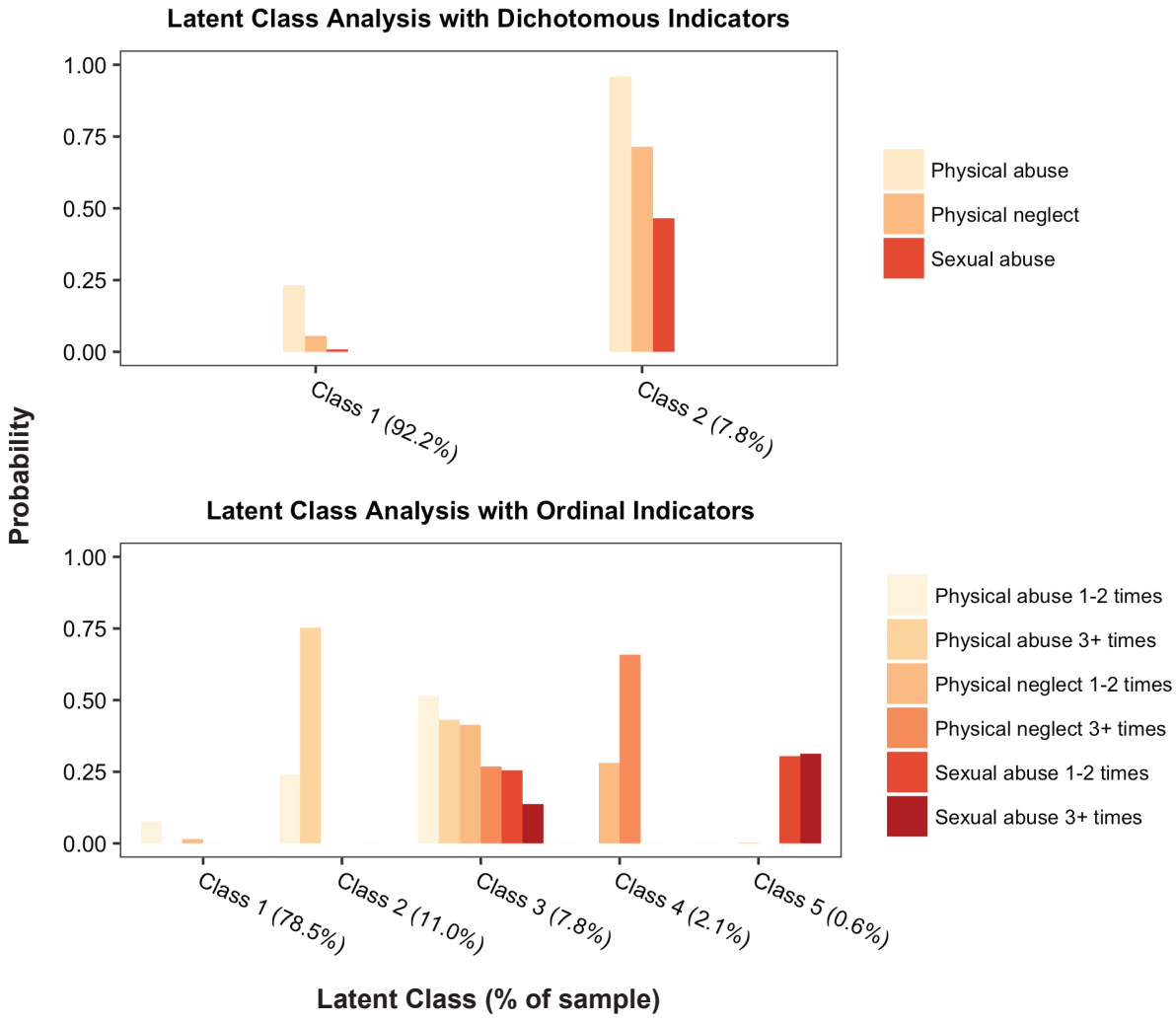

Table 2.5. Bivariate descriptives by childhood maltreatment latent classes from latent class analysis with dichotomous indicators.

	"No/Low Maltreatment" Class (N = 12,181)	"Maltreatment" Class (N = 1,085)	
	Sampled Frequency (Weighted Percent)		***p***
Sex			
Male	5,562 (49.3)	578 (57.3)	<.001
Female	6,619 (50.7)	507 (42.7)	
Race/ethnicity			
Non-Hispanic white	6,792 (69.3)	489 (56.7)	
Non-Hispanic black	2,517 (15.3)	263 (21.8)	<.001
Other	2,836 (15.5)	331 (21.5)	
Percent federal poverty level			
< 100%	1,404 (14.9)	204 (26.7)	
100-199%	1,931 (20.1)	209 (26.5)	<.001
200-399%	3,581 (39.5)	248 (30.5)	
≥ 400%	2,339 (25.5)	128 (16.4)	
Highest parental education			
Less than high school	1,344 (11.2)	184 (19.1)	
High school graduate or equivalent	3,308 (30.8)	336 (35.4)	<.001
Some college/trade school	2,479 (21.7)	214 (21.2)	
Graduated college or above	4,518 (36.3)	267 (24.2)	
Binge eating-related concerns	878 (6.9)	138 (12.9)	<.001
	Mean (Standard Error)		***p***
Age (years)	21.83 (0.12)	21.84 (0.15)	.84
Self-esteem in adolescence	1.87 (0.01)	1.99 (0.02)	<.001

Higher self-esteem scores indicate lower levels of self-esteem.

Table 2.6. Bivariate descriptives by childhood maltreatment latent classes from latent class analysis with ordinal indicators.

	"No/Low Maltreatment" Class (N = 10,322)	"Physical Abuse Only" Class (N = 1,499)	"Multi-Type Maltreatment" Class (N = 1,085)	"Physical Neglect Only" Class (N = 271)	"Sexual Abuse Only" Class (N = 89)	
	Sampled Frequency (Weighted Percent)					***p***
Sex						
Male	4,647 (48.7)	762 (53.5)	578 (57.3)	142 (56.8)	11 (15.5)	<.001
Female	5,675 (51.3)	737 (46.5)	507 (42.7)	129 (43.2)	78 (84.5)	
Race/ethnicity						
Non-Hispanic white	5,856 (70.2)	764 (64.8)	489 (56.7)	124 (59.1)	48 (66.9)	
Non-Hispanic black	2,142 (15.3)	285 (14.1)	263 (21.8)	70 (19.7)	20 (18.7)	<.001
Other	2,293 (14.5)	446 (21.2)	331 (21.5)	77 (21.1)	20 (14.4)	
Percent federal poverty level						
< 100%	1,174 (14.7)	166 (15.3)	204 (26.7)	48 (20.6)	16 (20.1)	
100-199%	1,606 (19.6)	243 (21.1)	209 (26.5)	58 (29.5)	24 (29.2)	<.001
200-399%	3,018 (39.6)	488 (41.2)	248 (30.5)	57 (29.4)	18 (32.1)	
≥ 400%	2,040 (26.1)	251 (22.4)	128 (16.4)	38 (20.4)	10 (18.6)	
Highest parental education						
Less than high school	1,127 (11.0)	159 (11.0)	184 (19.1)	46 (17.7)	12 (7.4)	
High school graduate or equivalent	2,791 (30.4)	382 (30.7)	336 (35.4)	101 (44.7)	34 (49.7)	<.001
Some college/trade school	2,060 (21.4)	357 (25.3)	214 (21.2)	45 (15.3)	17 (18.3)	
Graduated college or above	3,893 (37.2)	541 (32.9)	267 (24.2)	64 (22.3)	20 (24.6)	
Binge eating-related concerns	711 (6.7)	128 (7.3)	138 (12.9)	28 (9.5)	11 (16.3)	<.001
			Mean (Standard Error)			***p***
Age (years)	21.80 (0.12)	22.08 (0.13)	21.84 (0.15)	21.56 (0.21)	21.84 (0.28)	.01
Self-esteem in adolescence	1.86 (0.01)	1.97 (0.02)	1.99 (0.02)	1.77 (0.05)	2.05 (0.09)	<.001

Higher self-esteem scores indicate lower levels of self-esteem.

Table 2.7. Model fit indices for childhood maltreatment latent class analysis, substituting Wave IV physical abuse (which does not include slapping) for Wave III physical abuse (which does include slapping).

	BIC	AIC
1-class model	28,637,210.93	28,637,166.87
2-class model	**27,134,390.18**	**27,134,287.38**

BIC = Bayesian Information Criterion; AIC = Akaike's Information Criterion. Lower BIC and AIC values indicate better model fit. Models with ≥ 3 classes were not identified. Bold indicates optimal balance between model fit and parsimony.

Figure 2.2. Probability estimates of each type of childhood maltreatment for each latent class from latent class analysis with dichotomous indicators substituting Wave IV physical abuse (which does not include slapping) for Wave III physical abuse (which does include slapping).

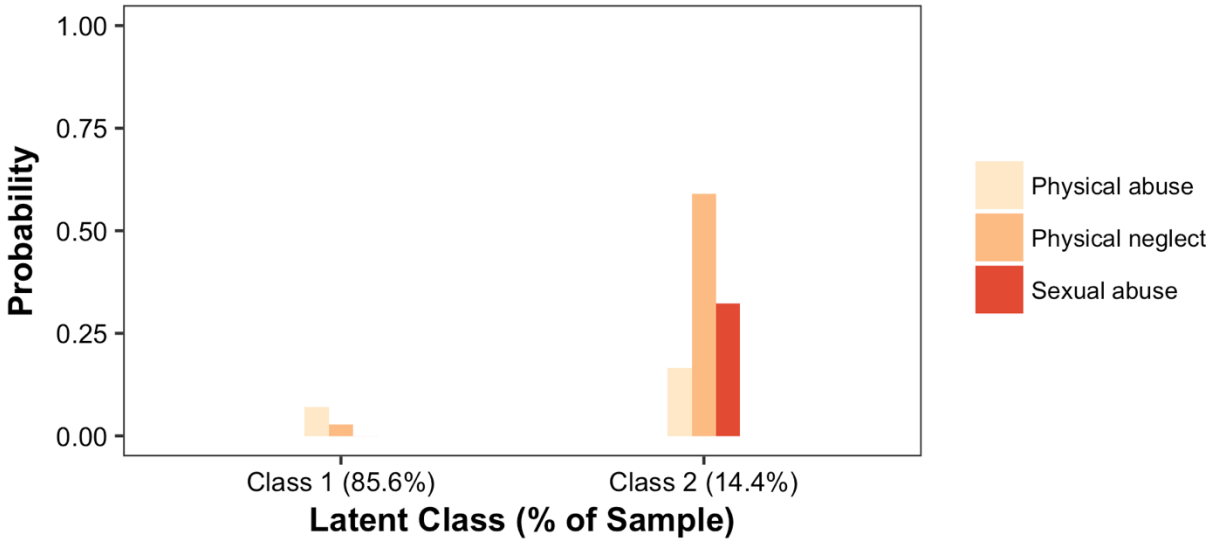

Table 2.8. Associations between childhood maltreatment and binge eating-related concerns.

	Unadjusted Model	Adjusted Model
	OR (95% CI)	
Any childhood maltreatment		
No maltreatment	1.00 (Ref)	1.00 (Ref)
Any maltreatment	1.66 (1.42, 1.96)***	1.67 (1.42, 1.97)***
Latent classes from LCA with dichotomous indicators		
Class 1 (“no/low maltreatment”)	1.00 (Ref)	1.00 (Ref)
Class 2 (“maltreatment”)	1.91 (1.49, 2.46)***	1.93 (1.50, 2.48)***
Latent classes from LCA with ordinal indicators		
Class 1 (“no/low maltreatment”)	1.00 (Ref)	1.00 (Ref)
Class 2 (“physical abuse only”)	1.10 (0.84, 1.43)	1.11 (0.84, 1.45)
Class 3 (“multi-type maltreatment”)	1.96 (1.52, 2.54)***	1.97 (1.52, 2.56)***
Class 4 (“physical neglect only”)	1.44 (0.85, 2.45)	1.47 (0.85, 2.52)
Class 5 (“sexual abuse only”)	2.37 (1.05, 5.37)*	2.08 (0.92, 4.70)

OR = odds ratio; CI = confidence interval; LCA = latent class analysis. Adjusted models controlled for age, sex, race/ethnicity, highest parental education, and percent federal poverty level in adolescence. All p’s > .05 for interactions by sex.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 2.9. Associations between childhood maltreatment and binge eating-related concerns, comparing estimates using Wave III physical abuse (which does include slapping) and Wave IV physical abuse (which does not include slapping).

	Using Wave III Physical Abuse	Using Wave IV Physical Abuse
	OR (95% CI)	
Any childhood maltreatment		
No maltreatment	1.00 (Ref)	1.00 (Ref)
Any maltreatment	1.67 (1.42, 1.97)***	1.86 (1.41, 2.46)***
Latent classes from LCA with dichotomous indicators		
Class 1 ("no/low maltreatment")	1.00 (Ref)	1.00 (Ref)
Class 2 ("maltreatment")	1.93 (1.50, 2.48)***	1.97 (1.50, 2.59)***

OR = odds ratio; CI = confidence interval; LCA = latent class analysis. Models adjusted for age, sex, race/ethnicity, highest parental education, and percent federal poverty level in adolescence.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 2.10. Associations between childhood maltreatment and binge eating-related concerns, comparing complete case, demographic covariates imputed, and all variables imputed.

	Complete Case Analysis	Only Demographic Covariates Imputed	Fully Imputed
		OR (95% CI)	
Any childhood maltreatment			
No maltreatment	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Any maltreatment	1.68 (1.40, 2.02)***	1.66 (1.41, 1.95)***	1.67 (1.42, 1.97)***
Latent classes from LCA with dichotomous indicators			
Class 1 ("no/low maltreatment")	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Class 2 ("maltreatment")	2.06 (1.50, 2.83)***	2.03 (1.57, 2.63)***	1.93 (1.50, 2.48)***
Latent classes from LCA with ordinal indicators			
Class 1 ("no/low maltreatment")	1.00 (Ref)	1.00 (Ref)	1.00 (Ref)
Class 2 ("physical abuse only")	1.07 (0.77, 1.47)	1.12 (0.85, 1.47)	1.11 (0.84, 1.45)
Class 3 ("multi-type maltreatment")	2.12 (1.55, 2.91)***	2.10 (1.61, 2.73)***	1.97 (1.52, 2.56)***
Class 4 ("physical neglect only")	1.64 (0.89, 3.02)	1.47 (0.84, 2.58)	1.47 (0.85, 2.52)
Class 5 ("sexual abuse only")	2.07 (0.69, 6.26)	2.37 (1.04, 5.39)*	2.08 (0.92, 4.70)

OR = odds ratio; CI = confidence interval; LCA = latent class analysis. All models adjusted for age, sex, race/ethnicity, highest parental education, and percent federal poverty level in adolescence.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 2.11. Mediation of observed associations between childhood maltreatment and binge eating-related concerns by self-esteem.

	Natural Direct Effect OR	Natural Indirect Effect OR	Proportion Mediated
		Estimate (95% CI)	
Any maltreatment	1.63 (1.57, 1.70)	1.03 (1.02, 1.03)	6.6% (5.6%, 7.8%)
"Maltreatment" latent class	1.87 (1.77, 1.99)	1.03 (1.03, 1.04)	6.2% (5.3%, 7.7%)
"Multi-type maltreatment" latent class	1.90 (1.79, 2.03)	1.04 (1.03, 1.04)	6.9% (6.0%, 8.7%)

OR = odds ratio; CI = confidence interval. Models adjusted for age, sex, race/ethnicity, highest parental education, and percent federal poverty level in adolescence.

Chapter 3

Associations Between Parent-Child Connectedness in Adolescence and Binge Eating-Related Concerns in Young Adulthood

Introduction

Parent-child connectedness, defined as closeness, caring, and satisfaction in parent-child relationships (Lezin, Rolleri, Bean, & Taylor, 2004), has emerged as a protective factor across a wide range of domains. It has been found to be protective against adverse outcomes including emotional distress, suicidality, violence, substance abuse, and poor physical health (Durlak, 1998; Resnick et al., 1997). Parent-child connectedness is grounded in attachment theory, which posits that healthy child development depends upon an infant's ability to form a lasting emotional bond with at least one primary caregiver (Ainsworth, Blehar, Waters, & Wall, 1978; Bowlby, 1969). Parent-child connectedness extends the origins of attachment theory, such that it is conceptualized as a more bidirectional process resulting from the ongoing dynamics of how parents and children interact with one another not only in infancy, but through adolescence (Lezin et al., 2004). Parent-child connectedness is closely related to dimensions of parenting that have been established as important for healthy child development (Lezin et al., 2004), namely warmth (Baumrind, 1971; Maccoby & Martin, 1983) and connection (Barber & Olsen, 1997). Given its close ties with constructs well established as important for healthy child development, it is not surprising that parent-child connectedness has emerged as a protective factor across numerous domains.

Parent-child connectedness and family connectedness (i.e., connectedness at the family level rather than the parent-child dyadic level) have been explored as protective factors in the domain of eating disorders as well. Family connectedness has been found to be protective against binge eating and extreme weight control behaviors among adolescents (Croll, Neumark-Sztainer, Story, & Ireland, 2002; Fonseca, Ireland, & Resnick, 2002; Linde, Wall, Haines, & Neumark-Sztainer, 2009; Neumark-Sztainer et al., 2007), as have both mother-child connectedness and father-child connectedness (Ackard, Neumark-Sztainer, Story, & Perry, 2006; Berge et al., 2014). However, to our knowledge, no studies have examined whether such associations extend beyond adolescence into young adulthood. Additionally, the ways in which mother-child connectedness and father-child connectedness may differentially shape eating disorder risk and the ways in which these associations may differ by the sex of the child are not well understood.

Evidence suggests that mothers and fathers interact differently with their children and that parents interact differently with sons and daughters (Ranson & Urichuk, 2008). Accordingly, distinct attachment relationships across mother-child and father-child dyads have been found to contribute to divergent outcomes, such that secure mother-child attachment is associated with higher levels of self-esteem, while secure father-child attachment is associated with lower levels of internalizing and externalizing behaviors (Ranson & Urichuk, 2008). Similar results have been found for mother-child connectedness and father-child connectedness; mother-child connectedness is associated with higher levels of prosocial behaviors, while father-child connectedness is associated with lower levels of internalizing and externalizing behaviors (Day & Padilla-Walker, 2009). In addition, attachment relationships with fathers have been found to be particularly protective for daughters (Ranson & Urichuk, 2008), indicating that the sex of the child is important to consider when examining how parent-child relationships may influence

development. Elucidating how mother-child connectedness and father-child connectedness may differentially shape eating disorder risk for sons and daughters could help inform eating disorders treatment and prevention.

Improving parent-child connectedness may be a useful target for eating disorders treatment and prevention, as evidence suggests that parent-child connectedness is modifiable (Feinberg & Kan, 2008; Kaminski, Valle, Filene, & Boyle, 2008; Letourneau et al., 2001). Examining the role of parent-child connectedness in the development of eating disorders could therefore have important clinical and public health implications. Binge eating-related concerns (i.e., emotions and cognitions associated with binge eating, such as embarrassment over amount eaten and fear of losing control over eating; Gormally, Black, Daston, & Rardin, 1982) appear to play a role in the development of eating disorders, as they have been found to prospectively predict eating disorder onset (Fairburn, Cooper, Doll, & Davies, 2005). Thus, they may be a valuable target for eating disorders intervention.

Using data from a large, nationally representative sample in the United States, the objectives of this study were to (a) investigate the extent to which mother-child connectedness and father-child connectedness in adolescence are associated with binge eating-related concerns in young adulthood and (b) examine differences in associations by sex.

Methods

Participants

This study used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health; Harris, 2009). 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 I data were collected in 1994-1995 when participants were in grades 7-12, Wave II data were collected in 1996 when participants were in grades 8-12, and Wave III data were collected in 2001-2002 when participants were 18-26 years old (Harris et al., 2009). Of the 15,197 participants interviewed at Wave III, 875 participants were excluded due to missing sampling weights and 790 participants that 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 (Harris et al., 2009).

Measures

Parent-child connectedness in adolescence. Mother-child connectedness and father-child connectedness were assessed at Wave I with the Relationship with Mother and Relationship with Father subscales of the Youth Asset Survey (Oman et al., 2018; Oman, Vesely, Tolma, Aspy, & Marshall, 2010). Five-point Likert-type scales was 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 (Ackard et al., 2006; Foster et al., 2017; Resnick et al., 1997; Sieving et al., 2001). We averaged responses to yield a continuous variable with possible scores ranging from 1-5, with higher scores indicating higher levels of mother-child connectedness (Cronbach's α = .86 in this sample) and father-child connectedness (Cronbach's α = .90 in this sample).

Binge eating-related concerns in young adulthood. Binge eating-related concerns were assessed at Wave III via self-report with the following items derived from the Eating Concern subscale of the Eating Disorder Examination-Questionnaire (Fairburn & Beglin, 1994): "In the past seven days, have you eaten so much in a short period that you would have been embarrassed if others had seen you do it?" and "In the past seven days, have you been afraid to start eating because you thought you wouldn't be able to stop or control your eating?" A positive response to the dichotomous variable for binge eating-related concerns was assigned to participants with an affirmative response to at least one of these items.

Demographic covariates. The following variables were included as demographic covariates: age at Wave I (continuous), sex (dichotomous), race/ethnicity (categorical: non-Hispanic white, non-Hispanic black, or other), family structure (categorical: two-parent, 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 during adolescence, and 1994 federal poverty guidelines).

Depressive symptoms in adolescence. In sensitivity analyses, we adjusted for depressive symptoms in adolescence, as depressive symptoms depressive symptoms have been found to predict decreased parent-child connectedness over time and thus could potentially confound associations between parent-child connectedness and binge eating-related concerns (Boutelle, Eisenberg, Gregory, & Neumark-Sztainer, 2009). Depressive symptoms were assessed at Wave I with 19 items modified from the Center for Epidemiologic Studies Depression Scale (CES-D;

Radloff, 1977). These items assessed depressive symptoms over the past seven days with response options ranging from 0 (*never or rarely*) to 3 (*most of the time or all of the time*). Appropriate items were reverse-scored, and responses were summed to yield continuous variables for each wave. Possible scores ranged from 0 to 57, with higher scores indicating higher levels of depressive symptoms. The scale demonstrated good internal consistency in this sample ($\alpha = .86$).

Statistical analysis

All analyses were conducted with SAS 9.4 and accounted for the complex sampling design used in Add Health.

Descriptive statistics. We computed univariate statistics for mother-child connectedness, father-child connectedness, binge eating-related concerns, demographic covariates, and depressive symptoms. We also computed bivariate statistics by sex, as well as sex-stratified bivariate statistics by binge eating-related concerns, by levels of mother-child connectedness in adolescence, and by levels of father-child connectedness in adolescence. For the purpose of computing bivariate statistics by levels of parent-child connectedness, we dichotomized parent-child connectedness scores as low (scores < 4) versus high (scores ≥ 4), using cut-offs corresponding to those used in the Youth Asset Survey (Oman et al., 2010).

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), binge eating-related concerns, age, sex, race/ethnicity, and depressive symptoms. 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 (SAS Institute Inc., 2015a). Auxiliary variables in the imputation model included mother-child connectedness and father-child connectedness at Wave II, self-esteem at Wave I, parent-reported household income, household size during adolescence, parent-reported receipt of public assistance, body mass index at Wave III, embarrassment over amount eaten reported at Wave III, fear of losing control over eating reported at Wave III, lifetime eating disorder diagnosis reported at Wave III, and disordered weight control behaviors (fasting, using diet pills, and purging) reported at Wave III. In sensitivity analyses, we conducted analyses with only demographic covariates imputed and using complete case data only.

Logistic regression. We ran unadjusted and demographics-adjusted logistic regression models examining associations of mother-child connectedness and father-child connectedness in adolescence with binge eating-related concerns in young adulthood. We ran separate models for mother-child connectedness and father-child connectedness, including the demographic covariates described above in adjusted models. We assessed for effect modification by sex by adding cross-product terms (mother-child connectedness x sex, father-child connectedness x sex) to the models. Irrespective of evidence for effect modification by sex, we also ran sex-stratified models, as both psychoanalytic theory and gender theory provide theoretical support for relationship distinctness between mother-son, mother-daughter, father-son, and father-daughter dyads (Richters & Waters, 1991; Russell & Saebel, 1997; Thompson & Walker, 1989).

Combining inference from multiply imputed datasets. Results from logistic regression analyses were combined and summarized with the MIANALYZE procedure in SAS 9.4 (SAS Institute Inc., 2015b), using both within-imputation and between-imputation variance to reflect uncertainty due to the missing data (Little & Rubin, 2002).

Results

Descriptives

Descriptive statistics are presented in Table 3.1. In young adulthood (mean age = 21.75 years), binge eating-related concerns were reported by 7.2% of participants. Prevalence of binge eating-related concerns differed by sex, with females reporting higher prevalence than males ($p < .001$). Mean (standard error [SE]) mother-child connectedness and father-child connectedness levels in adolescence were 4.33 (0.01) and 4.13 (0.02) respectively, with higher levels of both among males than females (both p's < .001). Mother-child connectedness and father-child connectedness were positively correlated ($r = .47$, $p < .001$).

Sex-stratified bivariate descriptive statistics by binge eating-related concerns are presented in Table 3.2. Among males, race/ethnicity ($p = .02$), father type ($p = .04$), and depressive symptoms in adolescence ($p < .001$) differed between participants reporting binge eating-related concerns and those not reporting binge eating-related concerns. Among females, depressive symptoms ($p < .001$), mother-child connectedness ($p = .007$), and father-child connectedness ($p = .003$) in adolescence differed between participants reporting binge eating-related concerns and those not reporting binge eating-related concerns.

Sex-stratified bivariate descriptive statistics by mother-child connectedness in adolescence are presented in Table 3.3. Among males, mother type ($p < .001$), age at baseline and follow-up (both p's < .001), and depressive symptoms in adolescence ($p < .001$) differed between participants reporting low versus high mother-child connectedness in adolescence. Among females, percent federal poverty level in adolescence ($p = .02$), family structure ($p = .03$), binge eating-related concerns ($p = .001$), age at baseline and follow-up (both p's < .001),

and depressive symptoms in adolescence ($p < .001$) differed between participants reporting low versus high mother-child connectedness in adolescence.

Sex-stratified bivariate descriptive statistics by father-child connectedness in adolescence are presented in Table 3.4. Among males, father type ($p < .001$), age at baseline and follow-up (both p's $< .001$), and depressive symptoms in adolescence ($p < .001$) differed between participants reporting low versus high father-child connectedness in adolescence. Among females, race/ethnicity ($p < .001$), percent federal poverty level in adolescence ($p = .009$), father type ($p < .001$), binge eating-related concerns ($p = .02$), age at baseline and follow-up (both p's $< .001$), and depressive symptoms in adolescence ($p < .001$) differed between participants reporting low versus high father-child connectedness in adolescence.

Overall associations between parent-child connectedness and binge eating-related concerns

Unadjusted and demographics-adjusted associations of mother-child connectedness and father-child connectedness with binge eating-related concerns are presented in Tables 3.5 and 3.6. After adjusting for age, sex, race/ethnicity, highest parental education, percent federal poverty level in adolescence, family structure, and mother/father type, higher mother-child connectedness in adolescence was associated with lower odds of binge eating-related concerns in young adulthood (odds ratio [OR] = 0.85; 95% confidence interval [CI]: 0.76, 0.96), but father-child connectedness in adolescence was not associated with binge eating-related concerns in young adulthood (OR = 0.91; 95% CI: 0.80, 1.03).

In sensitivity analyses further adjusting for depressive symptoms, neither mother-child connectedness (OR = 1.00; 95% CI: 0.88, 1.13) nor father-child connectedness (OR = 1.02; 95% CI: 0.89, 1.18) were associated with binge eating-related concerns.

Sensitivity analysis results from demographics-adjusted models using complete case data, imputed demographic covariates, and all variables imputed are presented in Table 3.7. Results were not substantially different in sensitivity analyses using complete cases only and imputing only demographic covariates.

Differences by sex

The association between father-child connectedness and binge eating-related concerns significantly differed by sex (p = .007), but the association between mother-child connectedness and binge-eating related concerns did not (p = .62). However, differences emerged in sex-stratified models for both mother-child connectedness and father-child connectedness. Sex-stratified unadjusted and demographics-adjusted associations of mother-child connectedness and father-child connectedness with binge eating-related concerns are presented in Tables 3.5 and 3.6. Among females, both higher mother-child connectedness (OR = 0.83; 95% CI: 0.74, 0.94) and higher father-child connectedness (OR = 0.79; 95% CI: 0.69, 0.91) were associated with lower odds of binge eating-related concerns after adjusting for demographic variables. Among males, neither mother-child connectedness (OR = 0.89; 95% CI: 0.70, 1.15) nor father-child connectedness (OR = 1.19; 95% CI: 0.93, 1.53) were associated with binge eating-related concerns after adjusting for demographic variables.

In sensitivity analyses further adjusting for depressive symptoms, neither mother-child connectedness (OR = 0.99; 95% CI: 0.86, 1.13) nor father-child connectedness (OR = 0.89; 95% CI: 0.78, 1.02) were associated with binge eating-related concerns among females, and mother-child connectedness was not associated with binge eating-related concerns among males (OR = 1.02; 95% CI: 0.78, 1.33), but higher father-child connectedness was associated with greater odds of binge eating-related concerns among males (OR = 1.35; 95% CI: 1.01, 1.80).

Discussion

The objectives of this study were to investigate the extent to which mother-child connectedness and father-child connectedness in adolescence are associated with binge eating-related concerns in young adulthood and to examine differences in associations by sex. In overall analyses (i.e., before examining differences by sex), we found higher mother-child connectedness in adolescence, but not father-child connectedness in adolescence, to be associated with lower odds of binge eating-related concerns in young adulthood. While only the association between father-child connectedness and binge eating-related concerns differed significantly by sex, sex-stratified analyses revealed differences for mother-child connectedness as well. Both higher mother-child connectedness and higher father-child connectedness in adolescence were associated with lower odds of binge eating-related concerns in young adulthood among females, but neither mother-child connectedness nor father-child connectedness in adolescence were associated with binge eating-related concerns in young adulthood among males. These results suggest that improving mother-daughter connectedness and father-daughter connectedness in adolescence may be important targets for intervention.

Our results build upon previous findings that mother-child connectedness and father-child connectedness are protective against binge eating behaviors among adolescents (Berge et al., 2014) by providing evidence that among females, mother-child connectedness and father-child connectedness in adolescence are protective against binge eating-related concerns into young adulthood. While Berge et al. (2014) found mother-child connectedness and father-child connectedness to be protective against binge eating behaviors for both girls and boys in adolescence, our findings suggest that parent-child connectedness is not protective against binge eating-related concerns for men in young adulthood. The differences by sex observed in the

present study may be specific to binge eating-related concerns, as Berge et al. (2014) examined binge eating behaviors. Prior research suggests that binge eating is not as distressing for males as for females, as young adult men have reported comparable rates of binge eating behaviors but lower levels of concerns about their eating behavior compared to young adult women (Lavender, De Young, & Anderson, 2010; Luce, Crowther, & Pole, 2008). In line with this idea, we observed a lower prevalence of binge eating-related concerns among males than females in the present study. This lower prevalence may help explain why the protective association between higher mother-child connectedness and binge eating-related concerns was not statistically significant among males.

Though the association between father-child connectedness and binge eating-related concerns among males was not statistically significant, the direction of this association indicated that higher father-son connectedness may be associated with greater odds of binge eating-related concerns. The counterintuitive direction of this association may be related to traditional gender roles. Binge eating may generally be less distressing for males as compared to females because males consider consuming large amounts of food to be "masculine" (Carey, Saules, & Carr, 2017), while items used to assess parent-child connectedness draw upon traditionally feminine traits, such as warm and loving (Bem, 1974). Thus, higher father-child connectedness may be reported for fathers that endorse more traditionally feminine traits, and those traits may be passed on from father to son (Bem, 1981). If males reporting higher father-child connectedness endorse more traditionally feminine traits, they may be more likely to express binge eating-related concerns.

We conducted sensitivity analyses additionally adjusting for depressive symptoms in adolescence, as depressive symptoms have been found to predict decreased parent-child

connectedness over time and thus could potentially confound associations between parent-child connectedness and binge eating-related concerns (Boutelle et al., 2009). After further adjusting for depressive symptoms, neither mother-child connectedness nor father-child connectedness were associated with binge eating-related concerns in overall analyses or among females. Among males, mother-child connectedness was not associated with binge eating-related concerns, but higher father-child connectedness was significantly associated with greater odds of binge eating-related concerns among males. However, given that the relationship between parent-child connectedness and depressive symptoms is bidirectional (Boutelle et al., 2009; Branje, Hale, Frijns, & Meeus, 2010), depressive symptoms likely act as both a confounder and a mediator of these associations. Therefore, the associations which were attenuated after adjusting for depressive symptoms may have been attenuated as a result of adjusting not only for confounding, but for mediation.

A key strength of this study is the availability of data from a large, nationally representative sample of participants in the United States followed from adolescence into young adulthood. Young adulthood is a critical period, as levels of cognitive features of eating disorders have been found to increase during this period (Slane, Klump, McGue, & Iacono, 2014). Another strength of this study was the use of reliable and valid measures for parent-child connectedness. Further, 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 seven-day assessment time frame to assess binge eating-related concerns. Due to the manner in which Add Health data were collected, we were unable to examine associations for same-sex parents and household members reported as the husband/wife or partner of the mother/father. We

did not account for country of origin or acculturation stress, factors which may influence adolescent report of parent-child connectedness (Hardway & Fuligni, 2006; Smokowski, Rose, & Bacallao, 2008). Additionally, there may be residual confounding. Regardless, findings from this study offer important contributions to understanding how parent-child connectedness may influence the development of eating disorders.

The findings from this study suggest that while more research is needed to better understand how mother-son connectedness and father-son connectedness shape eating disorder risk, improving mother-daughter connectedness and father-daughter connectedness in adolescence may be important intervention targets to reduce the burden of eating disorders. As binge eating-related concerns have been found to be precursors to eating disorders, reducing binge eating-related concerns through improving mother-daughter connectedness and father-daughter connectedness in adolescence may be valuable for eating disorders prevention and early intervention, particularly those involving binge eating. Further, 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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References

Ackard, D. M., Neumark-Sztainer, D., Story, M., & Perry, C. (2006). Parent-child connectedness and behavioral and emotional health among adolescents. *American Journal of Preventive Medicine*, *30*(1), 59–66. http://doi.org/10.1016/j.amepre.2005.09.013

Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of Attachment: A Psychological Study of the Strange Situation*. Lawrence Erlbaum Associates.

Barber, B. K., & Olsen, J. A. (1997). Socialization in context: Connection, regulation and autonomy in the family, school and neighborhood, and with peers. *Journal of Adolescent Research*, *12*(2), 287–315.

Baumrind, D. (1971). Harmonious parents and their preschool children. *Developmental Psychology*, *4*(1), 99–102.

Bem, S. L. (1974). The measurement of psychological androgyny. *Journal of Consulting and Clinical Psychology*, *42*(2), 155–162.

Bem, S. L. (1981). Gender schema theory: A cognitive account of sex typing. *Psychological Review*, *88*(4), 354–364. http://doi.org/10.1037/0033-295X.88.4.354

Berge, J. M., Wall, M., Larson, N., Eisenberg, M. E., Loth, K. A., & Neumark-Sztainer, D. (2014). The unique and additive associations of family functioning and parenting practices with disordered eating behaviors in diverse adolescents. *Journal of Behavioral Medicine*, *37*(2), 205–17. http://doi.org/10.1007/s10865-012-9478-1

Boutelle, K., Eisenberg, M. E., Gregory, M. L., & Neumark-Sztainer, D. (2009). The reciprocal relationship between parent – child connectedness and adolescent emotional functioning over 5 years. *Journal of Psychosomatic Research*, *66*(4), 309–316. http://doi.org/10.1016/j.jpsychores.2008.10.019

Bowlby, J. (1969). *Attachment and Loss Vol. 1. Attachment* (Vol. 1). http://doi.org/10.1177/000306518403200125

Branje, S. J. T., Hale, W. W., Frijns, T., & Meeus, W. H. J. (2010). Longitudinal associations between perceived parent-child relationship quality and depressive symptoms in adolescence. *Journal of Abnormal Child Psychology*, *38*(6), 751–763. http://doi.org/10.1007/s10802-010-9401-6

Carey, J. B., Saules, K. K., & Carr, M. M. (2017). A qualitative analysis of men's experiences of binge eating. *Appetite*, *116*, 184–195. http://doi.org/10.1016/j.appet.2017.04.030

Croll, J., Neumark-Sztainer, D., Story, M., & Ireland, M. (2002). Prevalence and risk and protective factors related to disordered eating behaviors among adolescents: relationship to gender and ethnicity. *Journal of Adolescent Health*, *31*(2), 166–175. http://doi.org/10.1016/S1054-139X(02)00368-3

Day, R. D., & Padilla-Walker, L. M. (2009). Mother and father connectedness and involvement during early adolescence. *Journal of Family Psychology*, *23*(6), 900–904. http://doi.org/10.1037/a0016438

Durlak, J. A. (1998). Common risk and protective factors in successful prevention programs. *American Journal of Orthopsychiatry*, *68*(4), 512–520. http://doi.org/10.1037/h0080360

Fairburn, C., & Beglin, S. (1994). Assessment of eating disorders: interview or self-report questionnaire? *International Journal of Eating Disorders*, *16*(4), 363–370.

Fairburn, C. G., Cooper, Z., Doll, H. A., & Davies, B. A. (2005). Identifying dieters who will develop an eating disorder: a prospective, population-based study. *American Journal of Psychiatry*, *162*(12), 2249–2255. http://doi.org/10.1176/appi.ajp.162.12.2249

Feinberg, M. E., & Kan, M. L. (2008). Establishing family foundations: Intervention effects on

coparenting, parent/infant well-being, and parent-child relations. *Journal of Family Psychology*, *22*(2), 253–263. http://doi.org/10.1037/0893-3200.22.2.253

Fonseca, H., Ireland, M., & Resnick, M. D. (2002). Familial correlates of extreme weight control behaviors among adolescents. *International Journal of Eating Disorders*, *32*(4), 441–448. http://doi.org/10.1002/eat.10078

Foster, C. E., Horwitz, A., Thomas, A., Opperman, K., Gipson, P., Burnside, A., … King, C. A. (2017). Connectedness to family, school, peers, and community in socially vulnerable adolescents. *Children and Youth Services Review*, *81*, 321–331. http://doi.org/10.1016/j.childyouth.2017.08.011

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors*, *7*(1), 47–55. http://doi.org/10.1016/0306-4603(82)90024-7

Hardway, C., & Fuligni, A. J. (2006). Dimensions of family connectedness among adolescents with Mexican, Chinese, and European backgrounds. *Developmental Psychology*, *42*(6), 1246–1258. http://doi.org/10.1037/0012-1649.42.6.1246

Harris, K. M. (2009). The National Longitudinal Study of Adolescent to Adult Health (Add Health), Waves I & II, 1994–1996; Wave III, 2001–2002; Wave IV, 2007-2009 [machine-readable data file and documentation]. Chapel Hill, NC: Carolina Population Center, University of North Carolina at Chapel Hill. http://doi.org/10.3886/ICPSR27021.v9

Harris, K. M., Halpern, C. T., Whitsel, E., Hussey, J., Tabor, J., Entzel, P., & Udry, J. R. (2009). The National Longitudinal Study of Adolescent to Adult Health: Research Design [WWW document]. Retrieved April 1, 2016, from http://www.cpc.unc.edu/projects/addhealth/design

Kaminski, J. W., Valle, L. A., Filene, J. H., & Boyle, C. L. (2008). A meta-analytic review of components associated with parent training program effectiveness. *Journal of Abnormal Child Psychology*, *36*(4), 567–589. http://doi.org/10.1007/s10802-007-9201-9

Lavender, J. M., De Young, K. P., & Anderson, D. A. (2010). Eating Disorder Examination Questionnaire (EDE-Q): Norms for undergraduate men. *Eating Behaviors*, *11*(2), 119–121. http://doi.org/10.1016/j.eatbeh.2009.09.005

Letourneau, N., Drummond, J., Fleming, D., Kysela, G., McDonald, L., & Stewart, M. (2001). Supporting parents: Can intervention improve parent-child relationships? *Journal of Family Nursing*, *7*(2), 159–187. http://doi.org/10.1177/107484070100700203

Lezin, N., Rolleri, L. A., Bean, S., & Taylor, J. (2004). *Parent-child connectedness: Implications for research, interventions and positive impacts on adolescent health*. Santa Cruz, CA.

Linde, J. A., Wall, M. M., Haines, J., & Neumark-Sztainer, D. (2009). Predictors of initiation and persistence of unhealthy weight control behaviours in adolescents. *International Journal of Behavioral Nutrition and Physical Activity*, *6*, 1–10. http://doi.org/10.1186/1479-5868-6-72

Little, R. J. A., & Rubin, D. B. (2002). *Statistical Analysis with Missing Data* (2nd ed). Hoboken, NJ: John Wiley & Sons, Inc.

Luce, K. H., Crowther, J. H., & Pole, M. (2008). Eating Disorder Examination Questionnaire (EDE-Q): Norms for undergraduate women. *International Journal of Eating Disorders*, *41*, 273–276.

Maccoby, E. E., & Martin, J. A. (1983). Socialization in the context of the family: Parent-child interaction. In P. H. Mussen (Series Ed.) & E. M. Hetherington (Vol. Ed.) (Eds.), *Handbook of Child Psychology: Vol. 4: Socialization, Personality and Social Development* (4th ed, pp.

1–101). New York: Wiley.

Neumark-Sztainer, D. R., Wall, M. M., Haines, J. I., Story, M. T., Sherwood, N. E., & van den Berg, P. A. (2007). Shared risk and protective factors for overweight and disordered eating in adolescents. *American Journal of Preventive Medicine*, *33*(5), 359–369. http://doi.org/10.1016/j.amepre.2007.07.031

Oman, R. F., Lensch, T., Amroussia, N., Clements-Nolle, K., Lu, M., & Yang, Y. (2018). The Revised Youth Asset Survey (YAS-R). *American Journal of Health Promotion*. http://doi.org/10.1177/0890117118814390

Oman, R. F., Vesely, S. K., Tolma, E. L., Aspy, C. B., & Marshall, L. (2010). Reliability and validity of the Youth Asset Survey: An update. *American Journal of Health Promotion*, *25*(1), e13–e24. http://doi.org/10.4278/ajhp.081009-QUAN-242

Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, *1*(3), 385–401. http://doi.org/10.1177/014662167700100306

Ranson, K. E., & Urichuk, L. J. (2008). The effect of parent–child attachment relationships on child biopsychosocial outcomes: A review. *Early Child Development and Care*, *178*(2), 129–152. http://doi.org/10.1080/03004430600685282

Resnick, M. D., Bearman, P. S., Blum, R. W., Bauman, K. E., Harris, K. M., Jones, J., … Udry, J. R. (1997). Protecting adolescents from harm: Findings from the National Longitudinal Study on Adolescent Health. *Journal of the American Medical Association*, *278*(10), 823–832.

Richters, J. E., & Waters, E. (1991). Attachment and socialization: The positive side of social influence. In M. Lewis & S. Feinman (Eds.), *Social influences and socialization in infancy*

(pp. 185–213). New York: Plenum.

Russell, A., & Saebel, J. (1997). Mother-son, mother-daughter, father-son, and father-daughter: Are they distinct relationships? *Developmental Review*, *17*(2), 111–147. http://doi.org/10.1006/drev.1996.0431

SAS Institute Inc. (2015a). The MI Procedure. In *SAS/STAT® 14.1 User's Guide*. Cary, NC: SAS Institute Inc.

SAS Institute Inc. (2015b). The MIANALYZE Procedure. In *SAS/STAT® 14.1 User's Guide*. Cary, NC: SAS Institute Inc.

Sieving, R. E., Beuhring, T., Resnick, M. D., Bearinger, L. H., Shew, M., Ireland, M., & Blum, R. W. (2001). Development of adolescent self-report measures from the National Longitudinal Study of Adolescent Health. *The Journal of Adolescent Health*, *28*(1), 73–81. http://doi.org/S1054-139X(00)00155-5 [pii]

Slane, J. D., Klump, K. L., McGue, M., & Iacono, W. G. (2014). Developmental trajectories of disordered eating from early adolescence to young adulthood: a longitudinal study. *The International Journal of Eating Disorders*, *47*(7), 793–801. http://doi.org/10.1002/eat.22329

Smokowski, P. R., Rose, R., & Bacallao, M. L. (2008). Acculturation and Latino family processes: How cultural involvement, biculturalism, and acculturation gaps influence family dynamics. *Family Relations*, *57*(3), 295–308.

Thompson, L., & Walker, A. J. (1989). Gender in families: Women and men in marriage, work, and parenthood. *Journal of Marriage and Family*, *51*(4), 845–871.

Table 3.1. Sample characteristics, overall and by sex.

	Overall (N = 13,532)	Males (N = 6,432)	Females (N = 7,100)	
	Sampled Frequency (Weighted Percent)			***p***
Race/ethnicity				
Non-Hispanic white	7,376 (68.1)	3,488 (67.6)	3,888 (68.5)	
Non-Hispanic black	2,734 (14.8)	1,205 (14.4)	1,529 (15.3)	.09
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)	
100-199%	2,223 (20.5)	1,082 (20.8)	1,141 (20.2)	.69
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)	
High school graduate or equivalent	3,768 (31.1)	1,788 (31.1)	1,980 (31.1)	.12
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				
Two-parent	9,507 (71.1)	4,590 (71.9)	4,917 (70.3)	
Mother-only	3,523 (25.1)	1,567 (23.8)	1,956 (26.6)	<.001
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)	
Binge eating-related concerns	1,017 (7.2)	399 (5.7)	618 (8.8)	<.001
	Mean (Standard Error)			***p***
Age at baseline (years)	15.37 (0.12)	15.46 (0.12)	15.29 (0.12)	<.001
Age at follow-up (years)	21.75 (0.12)	21.84 (0.12)	21.66 (0.12)	<.001
Depressive symptoms in adolescence	10.79 (0.14)	9.93 (0.15)	11.68 (0.19)	<.001
Mother-child connectedness in adolescence	4.33 (0.01)	4.41 (0.02)	4.24 (0.02)	<.001
Father-child connectedness in adolescence	4.13 (0.02)	4.20 (0.02)	4.05 (0.02)	<.001

Table 3.2. Sex-stratified bivariate descriptives by binge eating-related concerns.

	Males			*Females*		
	No Binge Eating-Related Concerns (N = 6,023)	Binge Eating-Related Concerns (N = 399)		No Binge Eating-Related Concerns (N = 6,468)	Binge Eating-Related Concerns (N = 618)	
	Sampled Frequency (Weighted Percent)		***p***	**Sampled Frequency (Weighted Percent)**		***p***
Race/ethnicity						
Non-Hispanic white	3,298 (68.2)	186 (59.0)		3,569 (68.8)	316 (66.3)	
Non-Hispanic black	1,114 (14.1)	88 (17.4)	.02	1,399 (15.4)	124 (14.0)	.14
Other	1,599 (17.7)	124 (23.6)		1,474 (15.8)	176 (19.7)	
Percent federal poverty level						
< 100%	705 (15.3)	57 (15.0)		795 (15.9)	93 (18.8)	
100-199%	1,010 (20.5)	71 (25.3)	.47	1,036 (20.3)	102 (19.2)	.37
200-399%	1,793 (39.4)	103 (36.8)		1,875 (37.9)	181 (40.2)	
≥ 400%	1,167 (24.8)	68 (22.8)		1,228 (25.9)	101 (21.8)	
Highest parental education						
Less than high school	676 (11.5)	46 (14.9)		800 (12.0)	88 (13.4)	
High school graduate or equivalent	1,674 (31.0)	110 (31.8)	.36	1,798 (31.2)	179 (31.1)	.85
Some college/trade school	1,180 (20.6)	91 (21.8)		1,392 (22.6)	115 (21.5)	
Graduated college or above	2,254 (37.0)	141 (31.5)		2,293 (34.2)	215 (34.0)	
Family structure						
Two-parent	4,312 (72.0)	274 (71.3)		4,489 (70.6)	422 (68.6)	
Mother-only	1,459 (23.6)	103 (24.2)	.97	1,773 (26.2)	175 (28.7)	.44
Father-only	252 (4.3)	22 (4.5)		206 (3.2)	21 (2.6)	
Mother type						
Biological/adoptive	5,619 (97.3)	363 (97.0)	.79	6,132 (98.1)	584 (98.2)	.95
Step/other	152 (2.7)	14 (3.0)		130 (1.9)	13 (1.8)	

Father type						
Biological/adoptive	4,046 (88.7)	256 (83.1)	.04	4,136 (88.2)	380 (84.9)	.14
Step/other	518 (11.3)	40 (16.9)		559 (11.8)	63 (15.1)	
	Mean (Standard Error)		***p***	**Mean (Standard Error)**		***p***
Age at baseline (years)	15.46 (0.12)	15.44 (0.18)	.91	15.29 (0.12)	15.22 (0.14)	.40
Age at follow-up (years)	21.84 (0.12)	21.87 (0.18)	.83	21.66 (0.12)	21.55 (0.14)	.16
Depressive symptoms in adolescence	9.78 (0.14)	12.41 (0.58)	<.001	11.38 (0.19)	14.86 (0.59)	<.001
Mother-child connectedness in adolescence	4.41 (0.02)	4.37 (0.05)	.40	4.25 (0.02)	4.13 (0.04)	.007
Father-child connectedness in adolescence	4.20 (0.02)	4.28 (0.07)	.24	4.06 (0.02)	3.86 (0.07)	.003

Table 3.3. Sex-stratified bivariate descriptives by mother-child connectedness in adolescence.

	Males			*Females*		
	Low Connectedness (N = 1,005)	High Connectedness (N = 5,138)		Low Connectedness (N = 1,729)	High Connectedness (N = 5,125)	
	Sampled Frequency (Weighted Percent)		***p***	**Sampled Frequency (Weighted Percent)**		***p***
Race/ethnicity						
Non-Hispanic white	566 (69.5)	2,751 (67.3)		907 (67.6)	2,840 (68.7)	
Non-Hispanic black	163 (12.7)	994 (14.8)	.49	356 (15.1)	1,126 (15.4)	.66
Other	274 (17.8)	1,383 (17.9)		459 (17.3)	1,139 (16.0)	
Percent federal poverty level						
< 100%	113 (13.9)	619 (15.5)		244 (18.1)	627 (15.8)	
100-199%	152 (20.0)	886 (21.0)	.79	302 (23.5)	804 (19.3)	.02
200-399%	285 (40.7)	1,532 (39.0)		446 (34.6)	1,551 (38.9)	
≥ 400%	194 (25.4)	992 (24.5)		300 (23.7)	987 (26.0)	
Highest parental education						
Less than high school	119 (13.4)	570 (11.1)		241 (13.8)	604 (11.1)	
High school graduate or equivalent	244 (28.9)	1,457 (31.6)	.26	472 (30.3)	1,423 (31.2)	.05
Some college/trade school	200 (19.1)	1,021 (21.1)		381 (24.2)	1,085 (22.2)	
Graduated college or above	396 (38.6)	1,914 (36.3)		580 (31.7)	1,875 (35.5)	
Family structure						
Two-parent	735 (73.2)	3,845 (75.6)	.32	1,212 (69.9)	3,691 (73.4)	.03
Mother-only	270 (26.8)	1,293 (24.4)		517 (30.1)	1,434 (26.6)	
Mother type						
Biological/adoptive	925 (92.5)	5,051 (98.1)	<.001	1,676 (97.5)	5,037 (98.3)	.09
Step/other	80 (7.5)	87 (1.9)		53 (2.5)	88 (1.7)	
Binge eating-related concerns	73 (6.9)	303 (5.4)	.22	184 (11.7)	413 (8.0)	.001

	Mean (Standard Error)		*p*	Mean (Standard Error)		*p*
Age at baseline (years)	16.10 (0.11)	15.33 (0.13)	<.001	15.65 (0.10)	15.16 (0.13)	<.001
Age at follow-up (years)	22.48 (0.12)	21.72 (0.13)	<.001	22.03 (0.11)	21.53 (0.13)	<.001
Depressive symptoms in adolescence	12.94 (0.35)	9.29 (0.15)	<.001	15.75 (0.31)	10.31 (0.19)	<.001

Low mother-child connectedness defined by scores < 4 and high mother-child connectedness defined by scores ≥ 4 for this table.

Table 3.4. Sex-stratified bivariate descriptives by father-child connectedness in adolescence.

	Males			*Females*		
	Low Connectedness (N = 1,253)	High Connectedness (N = 3,603)		Low Connectedness (N = 1,818)	High Connectedness (N = 3,312)	
	Sampled Frequency (Weighted Percent)		***p***	**Sampled Frequency (Weighted Percent)**		***p***
Race/ethnicity						
Non-Hispanic white	692 (71.5)	2,157 (72.9)		981 (69.4)	2,097 (75.6)	
Non-Hispanic black	170 (8.6)	473 (9.0)	.63	277 (10.1)	474 (9.4)	<.001
Other	389 (19.9)	964 (18.1)		554 (20.5)	725 (15.0)	
Percent federal poverty level						
< 100%	105 (9.8)	292 (10.1)		155 (11.1)	235 (9.1)	
100-199%	185 (19.7)	538 (18.2)	.79	279 (19.4)	426 (15.2)	.009
200-399%	393 (43.1)	1,143 (42.4)		539 (38.4)	1,111 (44.9)	
≥ 400%	251 (27.4)	849 (29.4)		393 (31.1)	776 (30.8)	
Highest parental education						
Less than high school	133 (10.3)	333 (9.1)		207 (9.9)	303 (8.7)	
High school graduate or equivalent	309 (26.8)	958 (29.7)	.49	483 (29.8)	850 (27.9)	.12
Some college/trade school	239 (20.7)	715 (20.9)		404 (24.1)	691 (22.7)	
Graduated college or above	519 (42.2)	1,487 (40.4)		689 (36.2)	1,378 (40.8)	
Family structure						
Two-parent	1,173 (93.1)	3,408 (94.7)	.12	1,738 (96.0)	3,166 (95.6)	.65
Father-only	80 (6.9)	195 (5.3)		80 (4.0)	146 (4.4)	
Father type						
Biological/adoptive	1,019 (81.0)	3,278 (90.7)	<.001	1,505 (82.6)	3,006 (90.6)	<.001
Step/other	234 (19.0)	325 (9.3)		313 (17.4)	306 (9.4)	
Binge eating-related concerns	90 (5.6)	206 (5.7)	.98	190 (10.2)	252 (7.7)	.02

	Mean (Standard Error)		*p*	Mean (Standard Error)		*p*
Age at baseline (years)	15.93 (0.13)	15.29 (0.13)	<.001	15.64 (0.11)	15.07 (14.8)	<.001
Age at follow-up (years)	22.33 (0.13)	21.66 (0.13)	<.001	22.02 (0.11)	21.43 (0.13)	<.001
Depressive symptoms in adolescence	12.55 (0.25)	8.64 (0.15)	<.001	14.17 (0.34)	9.80 (0.21)	<.001

Low father-child connectedness defined by scores < 4 and high mother-child connectedness defined by scores ≥ 4 for this table.

Table 3.5. Associations between mother-child connectedness and binge eating-related concerns, overall and by sex.

	Unadjusted Model	Model Adjusted for Demographics
	OR (95% CI)	
Overall (N = 13,030)	0.83 (0.74, 0.93)**	0.85 (0.76, 0.96)**
Among females (N = 6,873)	0.84 (0.74, 0.95)**	0.83 (0.74, 0.94)**
Among males (N = 6,157)	0.91 (0.72, 1.15)	0.89 (0.70, 1.15)

OR = odds ratio; CI = confidence interval. Adjusted models controlled for sex (in overall model), age, race/ethnicity, highest parental education, percent federal poverty level in adolescence, family structure, and mother type. In overall adjusted model, interaction by sex $p = .62$.
* $p < .05$, ** $p < .01$, *** $p < .001$

Table 3.6. Associations between father-child connectedness and binge eating-related concerns, overall and by sex.

	Unadjusted Model	Model Adjusted for Demographics
	OR (95% CI)	
Overall (N = 10,009)	0.88 (0.77, 0.99)*	0.91 (0.80, 1.03)
Among females (N = 5,144)	0.79 (0.69, 0.91)**	0.79 (0.69, 0.91)***
Among males (N = 4,865)	1.15 (0.90, 1.47)	1.19 (0.93, 1.53)

OR = odds ratio; CI = confidence interval. Adjusted models controlled for sex (in overall model), age, race/ethnicity, highest parental education, percent federal poverty level in adolescence, family structure, and father type. In overall adjusted model, interaction by sex $p = .007$.
* $p < .05$, ** $p < .01$, *** $p < .001$

Table 3.7. Associations between parent-child connectedness and binge eating-related concerns, comparing complete case, demographic covariates imputed, and all variables imputed.

	Complete Case Analysis	Only Demographic Covariates Imputed	Fully Imputed
		OR (95% CI)	
Mother-child connectedness	0.84 (0.73, 0.97)*	0.85 (0.76, 0.95)**	0.85 (0.76, 0.96)**
Father-child connectedness	0.94 (0.82, 1.08)	0.91 (0.80, 1.03)	0.91 (0.80, 1.03)

OR = odds ratio; CI = confidence interval. All models adjusted for age, sex, race/ethnicity, highest parental education, percent federal poverty level in adolescence, family structure, and mother/father type.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Chapter 4

Associations of Childhood Abuse and Parent-Child Connectedness in Adolescence with Binge Eating-Related Concerns Among Young Adults Do Not Differ by 5-HTTLPR Genotype

Introduction

Eating disorders have a substantial heritable component, with estimates from twin studies ranging from 40% to 60% (Trace, Baker, Peñas-Lledó, & Bulik, 2013). However, specific genes contributing to such heritability remain largely unidentified (Bulik, Kleiman, & Yilmaz, 2016). Findings from candidate gene studies have been inconsistent, and genome-wide association studies in the eating disorders field are still in their infancy (Bulik et al., 2016; Duncan et al., 2017). A number of candidate gene studies have investigated genes involved in the serotonergic system as potential risk factors for eating disorders, as serotonin plays an important role in appetite regulation as well as mood (Voigt & Fink, 2015).

A widely studied candidate gene involved in the serotonergic system is 5-HTTLPR, an insertion/deletion polymorphism in the promoter region of the gene that codes for the serotonin transporter (SLC6A4; Heils et al., 1996). The two most frequent alleles resulting from this polymorphism are named for their length: the short (S) allele contains 14 copies of a repetitive sequence and the long (L) allele contains 16 copies (Heils et al., 1996). In vitro studies have found that gene promoter activity is about three times greater for the L allele than the S allele, resulting in increased reuptake of serotonin into the presynaptic vesicle from the synaptic cleft (Heils et al., 1996). Although this suggests the short allele may act similarly to a selective

serotonin reuptake inhibitor (SSRI; Sangkuhl, Klein, & Altman, 2009), serotonin has also been found to exert neurotrophic action during brain development (Jonassen & Landrø, 2014; Persico, 2010). Decreased serotonin reuptake results in increased extracellular levels of serotonin (Fuller, 1994), and animal models have demonstrated that extracellular levels above or below certain thresholds disrupt brain pathway development (Nordquist & Oreland, 2010; Persico, 2010), even with only slight levels of alteration (Daws & Gould, 2011). Demonstrating that a phenotype associated with 5-HTTLPR genotype may exist, the S allele has been found to be associated with certain traits, such as low self-esteem, mood variability, proclivity for fear, and sensitivity (Gonda et al., 2009).

The S allele of 5-HTTLPR has been explored as a possible risk factor for binge eating and eating disorders involving binge eating. Findings from these studies have been mixed, however, possibly related to small sample sizes and differences in study populations. The S allele has been found to be associated with increased binge eating severity (Akkermann, Nordquist, Oreland, & Harro, 2010) but also decreased risk for binge eating disorder (Monteleone, Tortorella, Castaldo, & Maj, 2006), and other findings have been null (Chen, Qian, Pu, Ge, & Wu, 2015; Lauzurica et al., 2003; Lee & Lin, 2010; Munn-Chernoff et al., 2012).

After encountering similar inconsistencies when examining main effects of 5-HTTLPR genotype in the depression field, investigation into gene x environment (G x E) interaction yielded findings suggesting that 5-HTTLPR genotype moderates associations between major life stressors and depression. Specifically, the S allele has been linked to increased risk for depression when triggered by major life stressors (Bleys, Luyten, Soenens, & Claes, 2018; Caspi et al., 2003; Haberstick et al., 2016; Karg, Burmeister, Shedden, & Sen, 2011; Sharpley, Palanisamy, Glyde, Dillingham, & Agnew, 2014). Interpersonal stressors – experiences that

predominantly impact the quality or quantity of an individual's relationships (Vrshek-Schallhorn et al., 2013), such as childhood maltreatment – have been found to be particularly salient for such a G x E interaction (Vrshek-Schallhorn et al., 2013). However, controversy exists regarding the interaction between 5-HTTLPR genotype and major life stressors in predicting depression risk, as several meta-analyses have found no evidence for this G x E interaction (Culverhouse et al., 2018; Munafò, Durrant, Lewis, & Flint, 2009; Risch et al., 2009).

A small number of studies in the eating disorders field have since emulated the approach used in the depression field examining G x E interaction between 5-HTTLPR genotype and major life stressors. Among females, 5-HTTLPR genotype has been found to moderate the association between major life stressors and eating disorder symptoms (Akkermann et al., 2012; Karwautz et al., 2011; Stoltenberg, Anderson, Nag, & Anagnopoulos, 2012), including binge eating (Akkermann et al., 2012). However, these studies examined small, demographically homogenous samples; thus, the generalizability of these findings may be limited. Drawing upon these and similar studies, a recent meta-analysis examined a G x E interaction between 5-HTTLPR genotype and childhood abuse in predicting bulimia nervosa, with a total sample size of 1,097 participants (Rozenblat et al., 2017). This meta-analysis found a significant G x E interaction in a sample of male and female participants, such that the S allele was associated with increased risk for bulimia nervosa among participants who had experienced childhood physical and/or sexual abuse (Rozenblat et al., 2017). However, the sample size available for this meta-analysis was small for detecting G x E interactions, and the significant meta-analytic finding may be related to publication bias. Despite these limitations, the results of this meta-analysis suggest this G x E interaction warrants further investigation. In addition, Rozenblat et al. (2017)

suggested that future studies in the eating disorders field may benefit from adopting a differential susceptibility approach to exploring G x E interactions, as has been done in the depression field.

The differential susceptibility hypothesis theorizes that genetic variants such as the S allele of 5-HTTLPR confer increased sensitivity not only to environmental risk factors, but also to environmental protective factors (Belsky et al., 2009; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Belsky & Pluess, 2009). The depression literature has found evidence to support this hypothesis; the S allele of 5-HTTLPR has been found to be associated with lower depressive symptoms among individuals with high family support (Hankin et al., 2011; Li, Berk, & Lee, 2013; Taylor et al., 2006). To our knowledge, no studies in the eating disorders field have investigated whether the S allele is associated with increased sensitivity to protective factors.

Binge eating-related concerns, such as embarrassment over amount eaten and fear of losing control over eating (Gormally, Black, Daston, & Rardin, 1982), have been found to prospectively predict eating disorder onset (Fairburn, Cooper, Doll, & Davies, 2005) and therefore may be important precursors to eating disorders. Thus, reducing binge eating-related concerns may be valuable for eating disorders prevention and early intervention.

Considering 5-HTTLPR genotype in tandem with potential risk and protective may inform future research avenues, which could in turn lead to personalized intervention approaches in the eating disorders field. If the S allele and childhood abuse act synergistically to predict outcomes such as binge eating-related concerns, future research could focus on elucidating factors that promote resilience within this particularly high-risk subgroup. Additionally, if the S allele is associated with lower binge eating-related concerns among individuals with higher levels of factors related to family support, such as parent-child connectedness (Lezin, Rolleri, Bean, & Taylor, 2004), future research could investigate whether interventions to promote

protective factors such as these show greater efficacy among individuals with greater genetic susceptibility, such as those with the S allele of 5-HTTLPR. Initial findings of emerging gene by intervention (G x I) research have supported the differential susceptibility hypothesis, reporting that the S allele is associated with greater intervention effects for cognitive behavioral therapy (Eley et al., 2012), attention bias modification (Fox, Zougkou, Ridgewell, & Garner, 2011), and a home-visiting intervention designed to improve mother-infant attachment (Morgan et al., 2017). Personalized treatment and prevention approaches could draw upon findings such as these to better predict responsiveness to interventions (Pashayan et al., 2013; Thibodeau, August, Cicchetti, & Symons, 2016; van den Brekel-Dijkstra, Rengers, Niessen, de Wit, & Kraaijenhagen, 2016). If the highest risk subgroup is also particularly responsive to intervention, targeting such a subgroup could increase intervention efficacy as well as cost-effectiveness (Pashayan et al., 2013).

Using data from a large, nationally representative sample in the United States, the objective of this study was to investigate the extent to which associations of childhood abuse and parent-child connectedness in adolescence with binge eating-related concerns in young adulthood differ by 5-HTTLPR genotype.

Methods

Participants

This study used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health; Harris, 2009). 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 I data were collected in 1994-1995 when participants were in grades 7-12, Wave II data were collected in 1996 when participants were in grades 8-12, Wave III data were collected in 2001-2002 when participants were 18-26 years old, and Wave IV data were collected in 2008 when participants were 24-32 years old (Harris et al., 2009). Of the 15,197 participants interviewed at Wave III, 875 participants were excluded due to missing sampling weights and 2,624 participants without genotype data from Wave IV were excluded, leaving 11,698 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 (Harris et al., 2009).

Measures

Childhood abuse. Childhood abuse was assessed retrospectively at Wave III using Computer-Assisted Self-Interview (Harris et al., 2009), a method that has been shown to elicit more accurate reporting than interviewer-administered assessment for questions of a sensitive nature (Metzger et al., 2000). Participants were asked about the following occurrences prior to sixth grade: "How often had your parents or other adult care-givers slapped, hit, or kicked you?" (physical abuse) and "How often had one of your parents or other adult care-givers touched you in a sexual way, forced you to touch him or her in a sexual way, or forced you to have sexual relations?" (sexual abuse). Possible response options for each question were *one time*, *two times*, *three to five times*, *six to ten times*, *more than ten times*, and *this has never happened*. We created dichotomous variables representing each type of abuse having never occurred versus having ever occurred (one time or more), and we created a dichotomous childhood abuse variable representing neither type of abuse having ever occurred versus either type of abuse having ever occurred.

Parent-child connectedness in adolescence. Mother-child connectedness and father-child connectedness were assessed at Wave I with the Relationship with Mother and Relationship with Father subscales of the Youth Asset Survey (Oman et al., 2018; Oman, Vesely, Tolma, Aspy, & Marshall, 2010). Five-point Likert-type scales was 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 (Ackard, Neumark-Sztainer, Story, & Perry, 2006; Foster et al., 2017; Resnick et al., 1997; Sieving et al., 2001). We averaged responses to yield a continuous variable with possible scores ranging from 1-5, 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). We then created dichotomous variables representing low (scores < 4) versus high (scores ≥ 4) mother-child connectedness and father-child connectedness, using cut-offs corresponding to those used in the Youth Asset Survey (Oman et al., 2010).

5-HTTLPR genotype. Saliva was collected by trained and certified field interviewers at Wave IV. The 43 base-pair insertion/deletion polymorphism in the 5' regulatory region of the serotonin transporter gene (SLC6A4; 5-HTTLPR) and the A/G single nucleotide polymorphism (SNP) rs25331 in the long repeat unit of 5-HTTLPR were characterized using polymerase chain reaction assays and genomic DNA isolated from buccal cells using Zymo Research Silicon-A™ Plates and Oragene™ solution (Smolen et al., 2013). Alleles for biallelic classification of 5-HTTLPR used in the present study were characterized as either S (14 repeat units) or L (16 repeat units). Alleles for triallelic classification of 5-HTTLPR, which take into account the SNP

rs25531, were characterized as either S, L_G, or L_A. Under the triallelic classification, S and L_G alleles were combined for analyses, as has been recommended because the functional activity of L_G is believed to be comparable to that of S (Hu et al., 2006; Hu et al., 2005). However, because the functionality of rs25531 has been disputed, we reported results using both biallelic and triallelic classifications (Jonassen & Landrø, 2014). We created categorical variables for biallelic and triallelic classifications of 5-HTTLPR genotype with higher numbers representing a greater number of short alleles. The biallelic classification was coded as 0 = L/L, 1 = S/L, 2 = S/S, and the triallelic classification was coded as 0 = L_A/L_A (referred to as L/L), 1 = S/L_A or L_G/L_A (referred to as S/L), 2 = S/S, S/L_G, or L_G/L_G (referred to as S/S). We treated these as nominal variables in analyses, as prior research has been inconclusive regarding whether 5-HTTLPR functions according to an additive, dominant, or recessive genetic model (Jonassen & Landrø, 2014).

Binge eating-related concerns in young adulthood. Binge eating-related concerns were assessed at Wave III via self-report with the following items derived from the Eating Concern subscale of the Eating Disorder Examination-Questionnaire (Fairburn & Beglin, 1994): "In the past seven days, have you eaten so much in a short period that you would have been embarrassed if others had seen you do it?" and "In the past seven days, have you been afraid to start eating because you thought you wouldn't be able to stop or control your eating?" A positive response to the dichotomous variable for binge eating-related concerns was assigned to participants with an affirmative response to at least one of these items.

Demographic covariates. The following variables were included as demographic covariates in all analyses: age at Wave I (continuous), sex (dichotomous), race/ethnicity (categorical: non-Hispanic white, non-Hispanic black, or 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 during adolescence, and 1994 federal poverty guidelines). Mother type (dichotomous: biological/adoptive or step/other) and father type (dichotomous: biological/adoptive or step/other) were also included as demographic covariates in mother-child connectedness and father-child connectedness models, respectively.

Statistical analysis

All analyses were conducted with SAS 9.4 and accounted for the complex sampling design used in Add Health.

Descriptive statistics. We computed univariate statistics for childhood abuse, mother-child connectedness, father-child connectedness, 5-HTTLPR genotype, binge eating-related concerns, and demographic covariates. We also computed bivariate statistics by sex and by binge eating-related concerns, as well as abuse-stratified bivariate statistics by mother-child connectedness and father-child connectedness in adolescence. Additionally, we assessed for Hardy-Weinberg equilibrium for biallelic and triallelic 5-HTTLPR genotype classifications.

Multiple imputation. Data were missing at the following rates: 21% for percent federal poverty level, 6% for highest parental education, 5% for childhood abuse, 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), binge eating-related concerns, age, sex, and race/ethnicity. To preserve sample size, we conducted multiple imputation for all variables except 5-HTTLPR genotype 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 (SAS Institute Inc., 2015a). Auxiliary variables in the imputation model included childhood physical abuse, sexual abuse, and emotional abuse assessed at Wave IV, mother-child connectedness and father-child connectedness at Wave II, self-esteem in adolescence, depressive symptoms in adolescence, parent-reported household income, household size during adolescence, parent-reported receipt of public assistance, body mass index at Wave III, embarrassment over amount eaten reported at Wave III, fear of losing control over eating reported at Wave III, lifetime eating disorder diagnosis reported at Wave III, and disordered weight control behaviors (fasting, using diet pills, and purging) reported at Wave III. In sensitivity analyses, we conducted analyses with only demographic covariates imputed and using complete case data only.

Logistic regression. We ran logistic regression models to examine whether associations of childhood abuse, mother-child connectedness in adolescence, and father-child connectedness in adolescence with binge eating-related concerns in young adulthood differed by 5-HTTLPR genotype. We adjusted for the demographic covariates described above and ran separate models for each environmental exposure (childhood abuse, mother-child connectedness, and father-child connectedness), as well as for biallelic and triallelic classifications of 5-HTTLPR genotype. We used cross-product terms (childhood abuse x 5-HTTLPR genotype, mother-child connectedness x 5-HTTLPR genotype, father-child connectedness x 5-HTTLPR genotype) in the models to assess for gene x environment interaction. While it has been recommended that covariate x gene and covariate x environment terms should be included in models when assessing for gene x environment interaction in order to properly control for confounding (Keller, 2014), gene-environment independence was found for all gene x environment combinations in the present study, indicating that confounding bias is not a concern for interaction tests (Vanderweele, Ko, &

Mukherjee, 2013). Because a three-way interaction between 5-HTTLPR genotype, childhood trauma, and sex in predicting eating disorder symptoms has previously been observed (Stoltenberg et al., 2012), we assessed for differences by sex by adding three-way cross-product terms (childhood abuse x 5-HTTLPR genotype x sex, mother-child connectedness x 5-HTTLPR genotype x sex, father-child connectedness x 5-HTTLPR genotype x sex) and appropriate lower order interaction terms to the models. Additionally, we ran models to identify associations for the main effects of each environmental exposure and both classifications of 5-HTTLPR genotype, and we ran models to identify associations for the main effects of each environmental exposure stratified by each genotype and of each genotype stratified by each environmental exposure.

Combining inference from multiply imputed datasets. Results from logistic regression analyses were combined and summarized with the MIANALYZE procedure in SAS 9.4 (SAS Institute Inc., 2015b), using both within-imputation and between-imputation variance to reflect uncertainty due to the missing data (Little & Rubin, 2002).

Assessing for additive interaction. Interaction on the additive scale is important to assess because it is of more relevance to public health than interaction on the multiplicative scale (Rothman, Greenland, & Walker, 1980), and it has been recommended that interaction measures should be reported on both additive and multiplicative scales (Knol & VanderWeele, 2012). Therefore, in addition to calculating measures of interaction on the multiplicative scale (namely, ratios of odds ratios), we calculated relative excess risk due to interaction (RERI), a measure of interaction on the additive scale (VanderWeele & Knol, 2014). Using output from the logistic regression models described above and SAS code developed by VanderWeele & Knol (2014), we estimated RERI for odds ratios and corresponding standard errors using the delta method.

Results

Descriptives

Descriptive statistics are presented in Table 4.1. In young adulthood (mean age = 21.78 years), binge eating-related concerns were reported by 7.2% of participants. Prevalence of binge eating-related concerns differed by sex, with females reporting higher prevalence than males ($p <$ .001). Childhood abuse was reported by 29.3% of participants, with 28.7% reporting childhood physical abuse and 4.8% reporting childhood sexual abuse. Prevalence of childhood abuse did not differ by sex ($p = .36$). In adolescence, 79.8% of participants reported high mother-child connectedness and 70.7% reported high father-child connectedness, with males reporting higher prevalence than females for both (both p's < .001).

Bivariate descriptive statistics by binge eating-related concerns are presented in Table 4.2. Race/ethnicity ($p = .009$) differed between participants reporting binge eating-related concerns and those not reporting binge eating-related concerns.

Mother-child connectedness differed by abuse history ($p < .001$), such that high mother-child connectedness was reported by 82.9% of participants with no abuse history and 72.7% of those with a history of abuse. Abuse-stratified bivariate descriptive statistics by mother-child connectedness in adolescence are presented in Table 4.3. Among participants reporting no abuse, sex ($p < .001$), mother type ($p < .001$), binge eating-related concerns (($p = .004$), and age at baseline and follow-up (both p's < .001) differed between participants reporting low versus high mother-child connectedness in adolescence. Among participants reporting abuse, sex ($p < .001$), highest parental education ($p = .006$), and age at baseline and follow-up (both p's < .001) differed between participants reporting low versus high mother-child connectedness in adolescence.

Father-child connectedness differed by abuse history ($p < .001$), such that high father-child connectedness was reported by 74.5% of participants with no abuse history and 61.1% of those with a history of abuse. Abuse-stratified bivariate descriptive statistics by father-child connectedness in adolescence are presented in Table 4.4. Among participants reporting no abuse, sex ($p < .001$), race/ethnicity ($p = .006$), father type ($p < .001$), and age at baseline and follow-up (both p's $< .001$) differed between participants reporting low versus high father-child connectedness in adolescence. Among participants reporting abuse, sex, father type, and age at baseline and follow-up differed between participants reporting low versus high father-child connectedness in adolescence (all p's $< .001$).

5-HTTLPR genotype distribution met Hardy-Weinberg Equilibrium for biallelic ($\chi^2 = 2.64$, $df = 1$, $p > .05$) and triallelic ($\chi^2 = 4.77$, $df = 2$, $p > .05$) genotype classifications. Under the biallelic classification of 5-HTTLPR, 32.9% of participants had the L/L genotype, 47.9% had the S/L genotype, and 19.2% had the S/S genotype. Under the triallelic classification of 5-HTTLPR, 22.9% of participants had the L/L genotype, 49.5% had the S/L genotype, and 27.7% had the S/S genotype.

Main effects

Main effect analyses revealed that childhood abuse was associated with greater odds of binge eating-related concerns in young adulthood (odds ratio [OR] = 1.57; 95% confidence interval [CI]: 1.32, 1.86; see tables 4.5 and 4.6) and high mother-child connectedness in adolescence was associated with lower odds of binge eating-related concerns in young adulthood (OR = 0.69; 95% CI: 0.55, 0.87; see tables 4.7 and 4.8), but neither father-child connectedness in adolescence nor 5-HTTLPR genotype were associated with binge eating-related concerns in young adulthood (see Tables 4.9 and 4.10).

5-HTTLPR genotype x childhood abuse

As shown in Tables 4.5 and 4.6, there was no evidence of interaction between 5-HTTLPR genotype and childhood abuse for the biallelic or triallelic classification of 5-HTTLPR on additive or multiplicative scales. There was also no evidence of a three-way interaction by sex under the biallelic (p = .57) or triallelic (p = .43) classification of 5-HTTLPR.

5-HTTLPR genotype x mother-child connectedness in adolescence

As shown in Tables 4.7 and 4.8, there was no evidence of interaction between 5-HTTLPR genotype and mother-child connectedness for the biallelic classification of 5-HTTLPR on additive or multiplicative scales and no evidence of interaction between 5-HTTLPR genotype and childhood abuse for the triallelic classification of 5-HTTLPR on the additive scale. There was evidence of interaction between 5-HTTLPR genotype and mother-child connectedness for the triallelic classification of 5-HTTLPR on the multiplicative scale when conducting a joint test (p = .03) but not when testing individual comparisons between S/L and L/L genotypes (p = .49) or S/S and L/L genotypes (p = 13). Although none of the associations from the interaction model were statistically significant when compared to a single reference category, differences were observed with regard to direction of association. Compared to participants with low mother-child connectedness and L/L genotype, participants with low mother-child connectedness and S/L genotype were more likely to report binge eating-related concerns (OR = 1.26; 95% CI: 0.76, 2.09) while participants with low mother-child connectedness and S/S genotype were less likely to report binge eating-related concerns (OR = 0.60; 95% CI: 00.34, 1.05). Participants of any genotype with high mother-child connectedness were less likely to report binge-eating concerns compared to participants with low mother-child connectedness and L/L genotype. There was no

evidence of a three-way interaction by sex under the biallelic (p = .39) or triallelic (p = .69) classification of 5-HTTLPR.

5-HTTLPR genotype x father-child connectedness in adolescence

As shown in Tables 4.9 and 4.10, there was no evidence of interaction between 5-HTTLPR genotype and father-child connectedness for the biallelic or triallelic classification of 5-HTTLPR on additive or multiplicative scales. There was also no evidence of a three-way interaction by sex under the biallelic (p = .41) or triallelic (p = .06) classification of 5-HTTLPR.

Discussion

The objective of this study was to investigate the extent to which associations of childhood abuse and parent-child connectedness in adolescence with binge eating-related concerns in young adulthood differ by 5-HTTLPR genotype. In main effect analyses, we found that a history of physical and/or sexual abuse in childhood was associated with greater odds of binge eating-related concerns in young adulthood, while high mother-child connectedness in adolescence was associated with lower odds of binge eating-related concerns in young adulthood. No associations were found for father-child connectedness or 5-HTTLPR genotype. In G x E interaction analyses, we generally did not find associations of childhood abuse, mother-child connectedness in adolescence, or father-child connectedness in adolescence with binge eating-related concerns in young adulthood to differ by 5-HTTLPR genotype. Overall, the findings from this study suggest that while childhood abuse and parent-child connectedness in adolescence are important predictors of binge eating-related concerns in young adulthood, susceptibility to these environmental factors does not differ by 5-HTTLPR genotype.

Our null findings for a main effect of 5-HTTLPR genotype coheres with previous null findings for 5-HTTLPR genotype and binge eating-related outcomes (Chen et al., 2015; Lauzurica et al., 2003; Lee & Lin, 2010; Munn-Chernoff et al., 2012). However, the lack of evidence we found for an interaction between 5-HTTLPR genotype and childhood abuse is not consistent with previous studies finding the S allele to be associated with greater binge eating-related outcomes among participants who had experienced childhood abuse (Akkermann et al., 2012; Rozenblat et al., 2017; Stoltenberg et al., 2012). Also in contrast with previous work (Stoltenberg et al., 2012), we did not find evidence for differences by sex. To our knowledge, this study is the first in the eating disorders field to investigate whether susceptibility to protective factors differs by 5-HTTLPR genotype, but we did not find evidence for such an interaction.

While our null findings for an interaction between 5-HTTLPR genotype and childhood abuse are not consistent with the small existing body of G x E work in the eating disorders field, they cohere with null findings from several meta-analyses published in the depression field investigating an interaction between 5-HTTLPR genotype and life stressors (Culverhouse et al., 2018; Munafò et al., 2009; Risch et al., 2009). There are several possible reasons that could help explain why our results conflict with prior G x E findings in the eating disorders field, including differences related to sample characteristics and sample size, as well as differences related to measurement. Our sample size was over ten times that of the sample in the meta-analysis by Rozenblat et al. (2017) and was nationally representative of the United States, whereas samples in previous studies were small and demographically homogenous (Akkermann et al., 2012; Stoltenberg et al., 2012). In the depression G x E literature, it has been found that studies with null findings tend to have larger sample sizes than those with significant findings (Sharpley et

al., 2014), suggesting that significant findings in smaller studies may represent false positives. With regard to measurement, however, our study examined binge eating-related concerns as the outcome rather than binge eating as examined in previous studies, and our study relied on single-item measures for childhood abuse and binge eating-related concerns, whereas previous studies have used multiple-item scales for exposure and outcome variables (Akkermann et al., 2012; Rozenblat et al., 2017; Stoltenberg et al., 2012).

A key strength of this study is the availability of data from a large, nationally representative sample of participants in the United States followed from adolescence into young adulthood. Another strength of this study was that we assessed for G x E interaction on both additive and multiplicative scales, as most previous studies using logistic regression have only assessed for interaction on the multiplicative scale (VanderWeele & Knol, 2014).

This study also had limitations, which, as mentioned above, included the use of single-item measures to assess childhood abuse and binge eating-related concerns. Further, childhood abuse was assessed retrospectively, and binge eating-related concerns were assessed with a seven-day assessment time frame, which may underestimate our outcome of interest. In addition, although the actions of 5-HTTLPR genotype via brain development are believed to be more predictive of adverse outcomes than current serotonin levels (Kobiella et al., 2011), another limitation of this study is that data for antidepressant use were not collected in Add Health at or before the time binge eating-related concerns were assessed, prohibiting the possibility of conducting sensitivity analyses excluding respondents taking antidepressants at or before the time binge eating-related concerns were assessed. Nonetheless, findings from this study are an important contribution to the G x E literature in the eating disorders field.

The results of this study indicate that childhood abuse is a risk factor for binge eating-related concerns in young adulthood and high mother-child connectedness in adolescence is a protective factor for binge eating-related concerns in young adulthood, but that susceptibility to these environmental factors does not differ by 5-HTTLPR genotype. Therefore, although previous research finding greater intervention effects among individuals with greater genetic susceptibility has shown promise for personalized intervention approaches, our findings do not support the differential susceptibility hypothesis. Instead, our findings suggest that eating disorders intervention approaches should focus on decreasing risk factors, such as childhood abuse, and promoting protective factors, such as parent-child connectedness.

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References

Ackard, D. M., Neumark-Sztainer, D., Story, M., & Perry, C. (2006). Parent-child connectedness and behavioral and emotional health among adolescents. *American Journal of Preventive Medicine*, *30*(1), 59–66. http://doi.org/10.1016/j.amepre.2005.09.013

Akkermann, K., Kaasik, K., Kiive, E., Nordquist, N., Oreland, L., & Harro, J. (2012). The impact of adverse life events and the serotonin transporter gene promoter polymorphism on the development of eating disorder symptoms. *Journal of Psychiatric Research*, *46*(1), 38–43. http://doi.org/10.1016/j.jpsychires.2011.09.013

Akkermann, K., Nordquist, N., Oreland, L., & Harro, J. (2010). Serotonin transporter gene promoter polymorphism affects the severity of binge eating in general population. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *34*(1), 111–4. http://doi.org/10.1016/j.pnpbp.2009.10.008

Belsky, J., Bakermans-Kranenburg, M. J., & van IJzendoorn, M. H. (2007). For better and for worse: Differential susceptibility to environmental influences. *Current Directions in Psychological Science*, *16*(6), 300–304. http://doi.org/10.1111/j.1467-8721.2007.00525.x

Belsky, J., Jonassaint, C., Pluess, M., Stanton, M., Brummett, B., & Williams, R. (2009). Vulnerability genes or plasticity genes? *Molecular Psychiatry*, *14*(8), 746–54. http://doi.org/10.1038/mp.2009.44

Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin*, *135*(6), 885–908. http://doi.org/10.1037/a0017376

Bleys, D., Luyten, P., Soenens, B., & Claes, S. (2018). Gene-environment interactions between stress and 5-HTTLPR in depression: A meta-analytic update. *Journal of Affective*

Disorders, *226*, 339–345. http://doi.org/10.1016/J.JAD.2017.09.050

Bulik, C. M., Kleiman, S. C., & Yilmaz, Z. (2016). Genetic epidemiology of eating disorders. *Current Opinion in Psychiatry*, *29*(6), 383–388. http://doi.org/10.1097/YCO.0000000000000275

Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., … Poulton, R. (2003). Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene. *Science (New York, N.Y.)*, *301*(5631), 386–9. http://doi.org/10.1126/science.1083968

Chen, W., Qian, J., Pu, D., Ge, H., & Wu, J. (2015). The association of 5-HTTLPR gene polymorphisms and eating disorder: A meta-analysis. *Journal of Psychology & Psychotherapy*, *05*(06). http://doi.org/10.4172/2161-0487.1000214

Culverhouse, R. C., Saccone, N. L., Horton, A. C., Ma, Y., Anstey, K. J., Banaschewski, T., … Bierut, L. J. (2018). Collaborative meta-analysis finds no evidence of a strong interaction between stress and 5-HTTLPR genotype contributing to the development of depression. *Molecular Psychiatry*, *23*(1), 133. http://doi.org/10.1038/mp.2017.44

Daws, L. C., & Gould, G. G. (2011). Ontogeny and regulation of the serotonin transporter: providing insights into human disorders. *Pharmacology & Therapeutics*, *131*(1), 61–79. http://doi.org/10.1016/j.pharmthera.2011.03.013

Duncan, L., Yilmaz, Z., Gaspar, H., Walters, R., Goldstein, J., Anttila, V., … Bulik, C. M. (2017). Significant locus and metabolic genetic correlations revealed in genome-wide association study of anorexia nervosa. *American Journal of Psychiatry*, *174*(9), 850–858. http://doi.org/10.1176/appi.ajp.2017.16121402

Eley, T., Hudson, J., Creswell, C., Tropeano, M., Lester, K., Cooper, P., … Collier, D. (2012). Therapygenetics: the 5HTTLPR and response to psychological therapy. *Molecular*

Psychiatry, *17*, 236–237. http://doi.org/10.1038/mp.2011.132

Fairburn, C., & Beglin, S. (1994). Assessment of eating disorders: interview or self-report questionnaire? *International Journal of Eating Disorders*, *16*(4), 363–370.

Fairburn, C. G., Cooper, Z., Doll, H. A., & Davies, B. A. (2005). Identifying dieters who will develop an eating disorder: a prospective, population-based study. *American Journal of Psychiatry*, *162*(12), 2249–2255. http://doi.org/10.1176/appi.ajp.162.12.2249

Foster, C. E., Horwitz, A., Thomas, A., Opperman, K., Gipson, P., Burnside, A., … King, C. A. (2017). Connectedness to family, school, peers, and community in socially vulnerable adolescents. *Children and Youth Services Review*, *81*, 321–331. http://doi.org/10.1016/j.childyouth.2017.08.011

Fox, E., Zougkou, K., Ridgewell, A., & Garner, K. (2011). The serotonin transporter gene alters sensitivity to attention bias modification: Evidence for a plasticity gene. *Biological Psychiatry*, *70*(11), 1049–1054. http://doi.org/10.1016/j.biopsych.2011.07.004

Fuller, R. W. (1994). Uptake inhibitors increase extracellular serotonin concentration measured by brain microdialysis. *Life Sciences*, *55*(3), 163–7.

Gonda, X., Fountoulakis, K. N., Rihmer, Z., Lazary, J., Laszik, A., Akiskal, K. K., … Bagdy, G. (2009). Towards a genetically validated new affective temperament scale: a delineation of the temperament phenotype of 5-HTTLPR using the TEMPS-A. *Journal of Affective Disorders*, *112*(1–3), 19–29. http://doi.org/10.1016/j.jad.2008.03.012

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive Behaviors*, *7*(1), 47–55. http://doi.org/10.1016/0306-4603(82)90024-7

Haberstick, B. C., Boardman, J. D., Wagner, B., Smolen, A., Hewitt, J. K., Killeya-Jones, L. A.,

… Mullan Harris, K. (2016). Depression, stressful life events, and the impact of variation in the serotonin transporter: Findings from the National Longitudinal Study of Adolescent to Adult Health (Add Health). *PloS One*, *11*(3), e0148373. http://doi.org/10.1371/journal.pone.0148373

Hankin, B. L., Nederhof, E., Oppenheimer, C. W., Jenness, J., Young, J. F., Abela, J. R. Z., … Oldehinkel, A. J. (2011). Differential susceptibility in youth: Evidence that 5-HTTLPR x positive parenting is associated with positive affect for better and worse. *Translational Psychiatry*, *1*(9), e44-47. http://doi.org/10.1038/tp.2011.44

Harris, K. M. (2009). The National Longitudinal Study of Adolescent to Adult Health (Add Health), Waves I & II, 1994–1996; Wave III, 2001–2002; Wave IV, 2007-2009 [machine-readable data file and documentation]. Chapel Hill, NC: Carolina Population Center, University of North Carolina at Chapel Hill. http://doi.org/10.3886/ICPSR27021.v9

Harris, K. M., Halpern, C. T., Whitsel, E., Hussey, J., Tabor, J., Entzel, P., & Udry, J. R. (2009). The National Longitudinal Study of Adolescent to Adult Health: Research Design [WWW document]. Retrieved April 1, 2016, from http://www.cpc.unc.edu/projects/addhealth/design

Heils, A., Teufel, A., Petri, S., Stöber, G., Riederer, P., Bengel, D., & Lesch, K. P. (1996). Allelic variation of human serotonin transporter gene expression. *Journal of Neurochemistry*, *66*(6), 2621–2624. http://doi.org/10.1046/j.1471-4159.1996.66062621.x

Hu, X.-Z., Lipsky, R. H., Zhu, G., Akhtar, L. A., Taubman, J., Greenberg, B. D., … Goldman, D. (2006). Serotonin transporter promoter gain-of-function genotypes are linked to obsessive-compulsive disorder. *The American Journal of Human Genetics*, *78*(5), 815–826. http://doi.org/10.1086/503850

Hu, X., Oroszi, G., Chun, J., Smith, T. L., Goldman, D., & Schuckit, M. A. (2005). An expanded evaluation of the relationship of four alleles to the level of response to alcohol and the alcoholism risk. *Alcoholism: Clinical and Experimental Research*, *29*(1), 8–16. http://doi.org/10.1097/01.ALC.0000150008.68473.62

Jonassen, R., & Landrø, N. I. (2014). Serotonin transporter polymorphisms (5-HTTLPR) in emotion processing: implications from current neurobiology. *Progress in Neurobiology*, *117*, 41–53. http://doi.org/10.1016/j.pneurobio.2014.02.003

Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: Evidence of genetic moderation. *Archives of General Psychiatry*, *68*(5), 444–54. http://doi.org/10.1001/archgenpsychiatry.2010.189

Karwautz, A., Wagner, G., Waldherr, K., Nader, I., Fernandez-Aranda, F., & Estivill, X. (2011). Gene-environment interaction in anorexia nervosa: relevance of non-shared environment and the serotonin transporter gene. *Molecular Psychiatry*, *16*, 590–592.

Keller, M. C. (2014). Gene × environment interaction studies have not properly controlled for potential confounders: The problem and the (simple) solution. *Biological Psychiatry*, *75*(1), 18–24. http://doi.org/10.1016/j.biopsych.2013.09.006

Knol, M. J., & VanderWeele, T. J. (2012). Recommendations for presenting analyses of effect modification and interaction. *International Journal of Epidemiology*, *41*(2), 514–20. http://doi.org/10.1093/ije/dyr218

Kobiella, A., Reimold, M., Ulshöfer, D. E., Ikonomidou, V. N., Vollmert, C., Vollstädt-Klein, S., … Smolka, M. N. (2011). How the serotonin transporter 5-HTTLPR polymorphism influences amygdala function: the roles of in vivo serotonin transporter expression and

amygdala structure. *Translational Psychiatry*, *1*, e37. http://doi.org/10.1038/tp.2011.29

Lauzurica, N., Hurtado, A., Escartí, A., Delgado, M., Barrios, V., Morandé, G., … Fuentes, J. . (2003). Polymorphisms within the promoter and the intron 2 of the serotonin transporter gene in a population of bulimic patients. *Neuroscience Letters*, *352*(3), 226–230. http://doi.org/10.1016/j.neulet.2003.08.058

Lee, Y., & Lin, P.-Y. (2010). Association between serotonin transporter gene polymorphism and eating disorders: a meta-analytic study. *The International Journal of Eating Disorders*, *43*(6), 498–504. http://doi.org/10.1002/eat.20732

Lezin, N., Rolleri, L. A., Bean, S., & Taylor, J. (2004). *Parent-child connectedness: Implications for research, interventions and positive impacts on adolescent health*. Santa Cruz, CA.

Li, J. J., Berk, M. S., & Lee, S. S. (2013). Differential susceptibility in longitudinal models of gene-environment interaction for adolescent depression. *Development and Psychopathology*, *25*(4pt1), 991–1003. http://doi.org/10.1017/S0954579413000321

Little, R. J. A., & Rubin, D. B. (2002). *Statistical Analysis with Missing Data* (2nd ed). Hoboken, NJ: John Wiley & Sons, Inc.

Metzger, D. S., Koblin, B., Turner, C., Navaline, H., Valenti, F., Holte, S., … Seage, G. R. (2000). Randomized controlled trial of audio computer-assisted self-interviewing: utility and acceptability in longitudinal studies. *American Journal of Epidemiology*, *152*(2), 99–106. http://doi.org/10.1093/aje/152.2.99

Monteleone, P., Tortorella, A., Castaldo, E., & Maj, M. (2006). Association of a functional serotonin transporter gene polymorphism with binge eating disorder. *American Journal of Medical Genetics Part B, Neuropsychiatric Genetics*, *141B*(1), 7–9. http://doi.org/10.1002/ajmg.b.30232

Morgan, B., Kumsta, R., Fearon, P., Moser, D., Skeen, S., Cooper, P., … Tomlinson, M. (2017). Serotonin transporter gene (SLC6A4) polymorphism and susceptibility to a home-visiting maternal-infant attachment intervention delivered by community health workers in South Africa: Reanalysis of a randomized controlled trial. *PLOS Medicine*, *14*(2), e1002237. http://doi.org/10.1371/journal.pmed.1002237

Munafò, M. R., Durrant, C., Lewis, G., & Flint, J. (2009). Gene x environment interactions at the serotonin transporter locus. *Biological Psychiatry*, *65*(3), 211–9. http://doi.org/10.1016/j.biopsych.2008.06.009

Munn-Chernoff, M. A., McQueen, M. B., Stetler, G. L., Haberstick, B. C., Rhee, S. H., Sobik, L. E., … Stallings, M. C. (2012). Examining associations between disordered eating and serotonin transporter gene polymorphisms. *International Journal of Eating Disorders*, *45*(4), 556–561. http://doi.org/10.1002/eat.22001

Nordquist, N., & Oreland, L. (2010). Serotonin, genetic variability, behaviour, and psychiatric disorders--a review. *Upsala Journal of Medical Sciences*, *115*(1), 2–10. http://doi.org/10.3109/03009730903573246

Oman, R. F., Lensch, T., Amroussia, N., Clements-Nolle, K., Lu, M., & Yang, Y. (2018). The Revised Youth Asset Survey (YAS-R). *American Journal of Health Promotion*. http://doi.org/10.1177/0890117118814390

Oman, R. F., Vesely, S. K., Tolma, E. L., Aspy, C. B., & Marshall, L. (2010). Reliability and validity of the Youth Asset Survey: An update. *American Journal of Health Promotion*, *25*(1), e13–e24. http://doi.org/10.4278/ajhp.081009-QUAN-242

Pashayan, N., Hall, A., Chowdhury, S., Dent, T., Pharoah, P. D. P., & Burton, H. (2013). Public health genomics and personalized prevention: Lessons from the COGS project. *Journal of*

Internal Medicine, *274*(5), 451–456. http://doi.org/10.1111/joim.12094

Persico, A. M. (2010). Developmental roles for the serotonin transporter. In A. V Kalueff & J. L. Laporte (Eds.), *Experimental Models in Serotonin Transporter Research* (pp. 78–104). Cambridge University Press. http://doi.org/10.1017/CBO9780511729935.004

Resnick, M. D., Bearman, P. S., Blum, R. W., Bauman, K. E., Harris, K. M., Jones, J., … Udry, J. R. (1997). Protecting adolescents from harm: Findings from the National Longitudinal Study on Adolescent Health. *Journal of the American Medical Association*, *278*(10), 823–832.

Risch, N., Herrell, R., Lehner, T., Liang, K.-Y., Eaves, L., Hoh, J., … Merikangas, K. R. (2009). Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: A meta-analysis. *JAMA*, *301*(23), 2462–2471. http://doi.org/10.1001/jama.2009.878

Rothman, K. J., Greenland, S., & Walker, A. M. (1980). Concepts of interaction. *American Journal of Epidemiology*, *112*(4), 467–470.

Rozenblat, V., Ong, D., Fuller-Tyszkiewicz, M., Akkermann, K., Collier, D., Engels, R. C. M. E., … Krug, I. (2017). A systematic review and secondary data analysis of the interactions between the serotonin transporter 5-HTTLPR polymorphism and environmental and psychological factors in eating disorders. *Journal of Psychiatric Research*, *84*, 62–72. http://doi.org/10.1016/j.jpsychires.2016.09.023

Sangkuhl, K., Klein, T. E., & Altman, R. B. (2009). Selective serotonin reuptake inhibitors pathway. *Pharmacogenetics and Genomics*, *19*(11), 907–9. http://doi.org/10.1097/FPC.0b013e32833132cb

SAS Institute Inc. (2015a). The MI Procedure. In *SAS/STAT® 14.1 User's Guide*. Cary, NC:

SAS Institute Inc.

SAS Institute Inc. (2015b). The MIANALYZE Procedure. In *SAS/STAT® 14.1 User's Guide*. Cary, NC: SAS Institute Inc.

Sharpley, C. F., Palanisamy, S. K. A., Glyde, N. S., Dillingham, P. W., & Agnew, L. L. (2014). An update on the interaction between the serotonin transporter promoter variant (5-HTTLPR), stress and depression, plus an exploration of non-confirming findings. *Behavioural Brain Research*, *273*, 89–105. http://doi.org/10.1016/j.bbr.2014.07.030

Sieving, R. E., Beuhring, T., Resnick, M. D., Bearinger, L. H., Shew, M., Ireland, M., & Blum, R. W. (2001). Development of adolescent self-report measures from the National Longitudinal Study of Adolescent Health. *The Journal of Adolescent Health*, *28*(1), 73–81. http://doi.org/S1054-139X(00)00155-5 [pii]

Smolen, A., Whitsel, E. A., Tabor, J., Killeya-Jones, L. A., Cuthbertson, C. C., Hussey, J. M., … Harris, K. M. (2013). *Add Health Wave IV Documentation: Candidate Genes*. Chapel Hill, NC.

Stoltenberg, S. F., Anderson, C., Nag, P., & Anagnopoulos, C. (2012). Association between the serotonin transporter triallelic genotype and eating problems is moderated by the experience of childhood trauma in women. *The International Journal of Eating Disorders*, *45*(4), 492–500. http://doi.org/10.1002/eat.20976

Taylor, S. E., Way, B. M., Welch, W. T., Hilmert, C. J., Lehman, B. J., & Eisenberger, N. I. (2006). Early family environment, current adversity, the serotonin transporter promoter polymorphism, and depressive symptomatology. *Biological Psychiatry*, *60*(7), 671–676. http://doi.org/10.1016/j.biopsych.2006.04.019

Thibodeau, E. L., August, G. J., Cicchetti, D., & Symons, F. J. (2016). Application of

environmental sensitivity theories in personalized prevention for youth substance abuse: a transdisciplinary translational perspective. *Translational Behavioral Medicine*, *6*(1), 81–89. http://doi.org/10.1007/s13142-015-0374-4

Trace, S. E., Baker, J. H., Peñas-Lledó, E., & Bulik, C. M. (2013). The genetics of eating disorders. *Annual Review of Clinical Psychology*, *9*(1), 589–620. http://doi.org/10.1146/annurev-clinpsy-050212-185546

van den Brekel-Dijkstra, K., Rengers, A. H., Niessen, M. A., de Wit, N. J., & Kraaijenhagen, R. A. (2016). Personalized prevention approach with use of a web-based cardiovascular risk assessment with tailored lifestyle follow-up in primary care practice - A pilot study. *European Journal of Preventive Cardiology*, *23*(5), 544–551. http://doi.org/10.1177/2047487315591441

VanderWeele, T. J., & Knol, M. J. (2014). A tutorial on interaction. *Epidemiologic Methods*, *3*(1), 33–72. http://doi.org/10.1515/em-2013-0005

Vanderweele, T. J., Ko, Y.-A., & Mukherjee, B. (2013). Environmental confounding in gene-environment interaction studies. *American Journal of Epidemiology*, *178*(1), 144–52. http://doi.org/10.1093/aje/kws439

Voigt, J.-P., & Fink, H. (2015). Serotonin controlling feeding and satiety. *Behavioural Brain Research*, *277*, 14–31. http://doi.org/10.1016/j.bbr.2014.08.065

Vrshek-Schallhorn, S., Mineka, S., Zinbarg, R. E., Craske, M. G., Griffith, J. W., Sutton, J., … Adam, E. K. (2013). Refining the candidate environment: Interpersonal stress, the serotonin transporter polymorphism, and gene-environment interactions in major depression. *Clinical Psychological Science*, *2*(3), 235–248. http://doi.org/10.1177/2167702613499329

Table 4.1. Sample characteristics, overall and by sex.

	Overall (N = 11,698)	Males (N = 5,324)	Females (N = 6,374)	
	Sampled Frequency (Weighted Percent)			***p***
Race/ethnicity				
Non-Hispanic white	6,531 (69.3)	3,007 (69.3)	3,524 (69.2)	
Non-Hispanic black	2,397 (14.9)	984 (14.1)	1,413 (15.7)	.10
Other	2,730 (15.8)	1,322 (16.6)	1,408 (15.1)	
Percent federal poverty level				
< 100%	1,440 (16.0)	623 (15.6)	817 (16.5)	
100-199%	1,937 (21.1)	910 (21.4)	1,027 (20.8)	.76
200-399%	3,378 (38.0)	1,576 (38.5)	1,802 (37.5)	
≥ 400%	2,198 (24.9)	1,032 (24.6)	1,166 (25.3)	
Highest parental education				
Less than high school	1,376 (12.1)	569 (11.6)	807 (12.5)	
High school graduate or equivalent	3,249 (31.6)	1,477 (31.5)	1,772 (31.6)	.08
Some college/trade school	2,373 (21.7)	1,052 (20.7)	1,321 (22.6)	
Graduated college or above	4,080 (34.7)	1,941 (36.1)	2,139 (33.3)	
Mother type				
Biological/adoptive	10,425 (97.7)	4,736 (97.2)	5,689 (98.2)	.001
Step/other	254 (2.3)	135 (2.8)	119 (1.8)	
Father type				
Biological/adoptive	7,261 (88.2)	3,429 (88.5)	3,832 (88.0)	.36
Step/other	970 (11.8)	440 (11.5)	530 (12.0)	
Biallelic 5-HTTLPR genotype				
L/L	3,986 (32.9)	1,773 (31.8)	2,213 (34.0)	
S/L	5,394 (47.9)	2,491 (49.3)	2,903 (46.4)	.07
S/S	2,318 (19.2)	1,060 (18.9)	1,258 (19.5)	
Triallelic 5-HTTLPR genotype				
L/L	2,660 (22.9)	1,185 (21.8)	1,475 (23.9)	
S/L	5,574 (49.5)	2,582 (50.8)	2,992 (48.2)	.06
S/S	3,356 (27.7)	1,505 (27.3)	1,851 (28.0)	
Any childhood abuse	3,353 (29.3)	1,581 (29.9)	1,772 (28.7)	.36
Physical abuse	3,320 (28.7)	1,596 (29.9)	1,724 (27.7)	.10
Sexual abuse	558 (4.8)	237 (4.8)	321 (4.8)	.98

Mother-child connectedness in adolescence				
Low	2,279 (20.2)	804 (15.9)	1,475 (24.4)	<.001
High	8,378 (79.8)	4,058 (84.1)	4,320 (75.6)	
Father-child connectedness in adolescence				
Low	2,546 (29.3)	1,006 (25.3)	1,540 (33.4)	<.001
High	5,667 (70.7)	2,856 (74.7)	2,811 (66.6)	
Binge eating-related concerns	885 (7.2)	328 (5.3)	557 (8.9)	<.001
	Mean (Standard Error)			***p***
Age at baseline (years)	15.40 (0.12)	15.49 (0.12)	15.31 (0.12)	<.001
Age at follow-up (years)	21.78 (0.12)	21.88 (0.12)	21.68 (0.12)	<.001

Table 4.2. Bivariate descriptives by binge eating-related concerns.

	No Binge Eating-Related Concerns (N = 10,794)	Binge Eating-Related Concerns (N = 885)	
	Sampled Frequency (Weighted Percent)		***p***
Race/ethnicity			
Non-Hispanic white	6,074 (69.7)	451 (64.2)	.009
Non-Hispanic black	2,198 (14.8)	192 (15.6)	
Other	2,484 (15.5)	240 (20.2)	
Percent federal poverty level			
< 100%	1,295 (15.7)	142 (19.3)	.27
100-199%	1,785 (21.1)	148 (20.7)	
200-399%	3,142 (38.0)	232 (37.9)	
≥ 400%	2,049 (25.2)	147 (22.1)	
Highest parental education			
Less than high school	1,258 (11.8)	113 (14.4)	.45
High school graduate or equivalent	2,997 (31.6)	247 (31.2)	
Some college/trade school	2,196 (21.8)	171 (20.3)	
Graduated college or above	3,780 (34.8)	299 (34.1)	
Mother type			
Biological/adoptive	9,641 (97.6)	768 (98.7)	.07
Step/other	235 (2.4)	19 (1.3)	
Father type			
Biological/adoptive	6,724 (88.4)	529 (85.5)	.13
Step/other	894 (11.6)	76 (14.5)	
	Mean (Standard Error)		***p***
Age at baseline (years)	15.40 (0.12)	15.37 (0.14)	.67
Age at follow-up (years)	21.78 (0.12)	21.72 (0.14)	.47

Table 4.3. Abuse-stratified bivariate descriptives by mother-child connectedness in adolescence.

	No Abuse			*Abuse*		
	Low Mother-Child Connectedness (N = 1,290)	High Mother-Child Connectedness (N = 5,777)		Low Mother-Child Connectedness (N = 848)	High Mother-Child Connectedness (N = 2,164)	
	Sampled Frequency (Weighted Percent)		***p***	**Sampled Frequency (Weighted Percent)**		***p***
Sex						
Male	445 (37.8)	2,655 (49.6)	<.001	301 (38.0)	1,129 (54.1)	<.001
Female	845 (62.2)	3,122 (50.4)		547 (62.0)	1,035 (45.9)	
Race/ethnicity						
Non-Hispanic white	756 (72.1)	3,456 (72.7)	.93	455 (68.8)	1,131 (65.8)	.42
Non-Hispanic black	251 (13.9)	1,154 (13.3)		138 (12.5)	412 (14.6)	
Other	279 (14.0)	1,151 (14.0)		252 (18.7)	612 (19.5)	
Percent federal poverty level						
< 100%	166 (16.6)	634 (13.8)	.38	124 (18.4)	292 (17.7)	.80
100-199%	198 (19.1)	916 (19.2)		160 (24.7)	395 (22.7)	
200-399%	349 (39.0)	1,839 (40.1)		230 (34.8)	644 (37.5)	
≥ 400%	257 (25.3)	1,193 (26.8)		136 (22.1)	396 (22.1)	
Highest parental education						
Less than high school	149 (11.8)	610 (10.5)	.71	127 (16.5)	250 (10.5)	.006
High school graduate or equivalent	339 (29.6)	1,627 (30.9)		230 (29.7)	597 (31.8)	
Some college/trade school	282 (21.2)	1,160 (21.3)		185 (25.0)	511 (24.5)	
Graduated college or above	480 (37.3)	2,228 (37.3)		273 (28.9)	740 (33.3)	
Mother type						
Biological/adoptive	1,229 (95.9)	5,679 (98.3)	<.001	814 (96.5)	2,120 (97.9)	.13
Step/other	61 (4.1)	98 (1.7)		34 (3.5)	44 (2.1)	
Binge eating-related concerns	103 (8.6)	339 (5.6)	.004	92 (10.8)	209 (9.1)	.29

				Mean (Standard Error)		
Age at baseline (years)	15.78 (0.11)	15.20 (0.12)	<.001	15.77 (0.13)	15.26 (0.14)	<.001
Age at follow-up (years)	22.16 (0.11)	21.58 (0.13)	<.001	22.15 (0.14)	21.62 (0.14)	<.001

Table 4.4. Abuse-stratified bivariate descriptives by father-child connectedness in adolescence.

	No Abuse			*Abuse*		
	Low Father-Child Connectedness (N = 1,510)	High Father-Child Connectedness (N = 4,040)		Low Father-Child Connectedness (N = 893)	High Father-Child Connectedness (N = 1,353)	
	Sampled Frequency (Weighted Percent)		***p***	**Sampled Frequency (Weighted Percent)**		***p***
Sex						
Male	590 (43.0)	1,892 (49.8)	<.001	351 (42.5)	774 (58.8)	<.001
Female	920 (57.0)	2,148 (50.2)		542 (57.5)	579 (41.2)	
Race/ethnicity						
Non-Hispanic white	872 (72.8)	2,705 (78.3)	.006	487 (72.7)	800 (72.0)	.88
Non-Hispanic black	243 (9.7)	536 (8.2)		103 (7.6)	166 (8.4)	
Other	391 (17.4)	787 (13.5)		300 (19.7)	380 (19.6)	
Percent federal poverty level						
< 100%	120 (9.8)	289 (8.9)	.13	78 (11.5)	106 (10.2)	.76
100-199%	210 (18.2)	547 (16.1)		147 (19.3)	208 (19.7)	
200-399%	456 (39.5)	1,399 (44.5)		278 (43.1)	434 (41.2)	
≥ 400%	361 (32.5)	983 (30.5)		165 (26.0)	321 (28.9)	
Highest parental education						
Less than high school	148 (8.6)	334 (8.2)	.89	108 (11.9)	134 (8.5)	.13
High school graduate or equivalent	384 (28.7)	1,066 (29.0)		235 (28.8)	327 (27.7)	
Some college/trade school	302 (20.5)	823 (21.6)		206 (25.9)	317 (25.1)	
Graduated college or above	640 (42.2)	1,712 (41.2)		321 (33.4)	539 (38.7)	
Father type						
Biological/adoptive	1,242 (82.4)	3,720 (91.7)	<.001	726 (80.6)	1,198 (89.0)	<.001
Step/other	268 (17.6)	320 (8.3)		167 (19.4)	155 (11.0)	
Binge eating-related concerns	115 (7.4)	237 (5.7)	.09	107 (10.2)	112 (8.7)	.31

				Mean (Standard Error)		
Age at baseline (years)	15.78 (0.11)	15.13 (0.13)	<.001	15.75 (0.14)	15.24 (0.14)	<.001
Age at follow-up (years)	22.19 (0.11)	21.50 (0.13)	<.001	22.11 (0.15)	21.59 (0.14)	<.001

Table 4.5. Interaction between biallelic 5-HTTLPR genotype and childhood abuse on the odds of binge eating-related concerns.

	No Abuse		Abuse		**Abuse vs. No Abuse OR (95% CI), by Genotype**	**Abuse vs. No Abuse OR (95% CI), Irrespective of Genotype**
	N With / Without Outcome	**OR (95% CI)**	**N With / Without Outcome**	**OR (95% CI)**		
L/L	166 / 2,486	1.00 (Ref)	106 / 1,002	1.73 (1.25, 2.38)***	1.63 (1.19, 2.24)**	
S/L	218 / 3,338	1.10 (0.85, 1.43)	167 / 1,353	1.82 (1.38, 2.40)***	1.67 (1.27, 2.20)***	1.57 (1.32, 1.86)***
S/S	110 / 1,364	1.05 (0.76, 1.45)	71 / 650	1.39 (0.93, 2.06)	1.35 (0.88, 2.07)	
Genotype OR (95% CI), by Abuse History						
L/L	1.00 (Ref)		1.00 (Ref)			
S/L	1.10 (0.85, 1.43)		1.07 (0.76, 1.50)			
S/S	1.05 (0.76, 1.46)		0.80 (0.52, 1.24)			
Genotype OR (95% CI), Irrespective of Abuse History						
L/L	1.00 (Ref)					
S/L	1.09 (0.89, 1.33)					
S/S	0.95 (0.73, 1.24)					

OR = odds ratio; CI = confidence interval; RERI = relative excess risk due to interaction.
Interaction on additive scale:
RERI (95% CI) for S/L vs. L/L = -0.005 (-0.67, 0.66), $p = .49$
RERI (95% CI) for S/S vs. L/L = -0.39 (-1.09, 0.32), $p = .14$
Interaction on multiplicative scale:
Ratio of ORs (95% CI) for S/L vs. L/L = 0.96 (0.62, 1.48), $p = .85$
Ratio of ORs (95% CI) for S/S vs. L/L = 0.77 (0.46, 1.28), $p = .31$
Joint test $p = .58$
Models adjusted for age, sex, race/ethnicity, percent federal poverty level in adolescence, and highest level of parental education.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 4.6. Interaction between triallelic 5-HTTLPR genotype and childhood abuse on the odds of binge eating-related concerns.

	No Abuse		Abuse		**Abuse vs. No Abuse OR (95% CI), by Genotype**	**Abuse vs. No Abuse OR (95% CI), Irrespective of Genotype**
	N With / Without Outcome	**OR (95% CI)**	**N With / Without Outcome**	**OR (95% CI)**		
L/L	112 / 1,676	1.00 (Ref)	71 / 655	1.88 (1.25, 2.82)**	1.80 (1.20, 2.69)**	
S/L	238 / 3,432	1.11 (0.81, 1.51)	169 / 1,420	1.67 (1.22, 2.28)**	1.53 (1.17, 2.00)**	1.57 (1.32, 1.86)***
S/S	139 / 2,005	0.90 (0.67, 1.22)	101 / 910	1.45 (1.03, 2.03)*	1.59 (1.14, 2.23)**	
	Genotype OR (95% CI), by Abuse History					
L/L	1.00 (Ref)		1.00 (Ref)			
S/L	1.11 (0.81, 1.52)		0.89 (0.62, 1.28)			
S/S	0.91 (0.67, 1.23)		0.76 (0.51, 1.14)			
	Genotype OR (95% CI), Irrespective of Abuse History					
L/L	1.00 (Ref)					
S/L	1.03 (0.82, 1.29)					
S/S	0.86 (0.68, 1.07)					

OR = odds ratio; CI = confidence interval; RERI = relative excess risk due to interaction.
Interaction on additive scale:
RERI (95% CI) for S/L vs. L/L = -0.32 (-1.11, 0.47), $p = .21$
RERI (95% CI) for S/S vs. L/L = -0.34 (-1.13, 0.46), $p = .20$
Interaction on multiplicative scale:
Ratio of ORs (95% CI) for S/L vs. L/L = 0.80 (0.49, 1.32), $p = .38$
Ratio of ORs (95% CI) for S/S vs. L/L = 0.85 (0.51, 1.44), $p = .55$
Joint test $p = .68$
Models adjusted for age, sex, race/ethnicity, percent federal poverty level in adolescence, and highest level of parental education.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 4.7. Interaction between biallelic 5-HTTLPR genotype and mother-child connectedness on the odds of binge eating-related concerns.

	Low Mother-Child Connectedness		High Mother-Child Connectedness		**High vs. Low Connectedness OR (95% CI), by Genotype**	**High vs. Low Connectedness OR (95% CI), Irrespective of Genotype**
	N With / Without Outcome	**OR (95% CI)**	**N With / Without Outcome**	**OR (95% CI)**		
L/L	60 / 656	1.00 (Ref)	190 / 2,713	0.75 (0.50, 1.12)	0.73 (0.49, 1.09)	
S/L	105 / 970	1.31 (0.83, 2.09)	267 / 3,567	0.80 (0.55, 1.15)	0.58 (0.41, 0.81)**	0.69 (0.55, 0.87)**
S/S	42 / 441	0.84 (0.49, 1.42)	123 / 1,507	0.76 (0.50, 1.15)	0.96 (0.55, 1.67)	
Genotype OR (95% CI), by Connectedness						
L/L	1.00 (Ref)		1.00 (Ref)			
S/L	1.34 (0.84, 2.12)		1.05 (0.81, 1.36)			
S/S	0.87 (0.51, 1.48)		1.00 (0.71, 1.39)			
Genotype OR (95% CI), Irrespective of Connectedness						
L/L	1.00 (Ref)					
S/L	1.09 (0.89, 1.33)					
S/S	0.95 (0.73, 1.24)					

OR = odds ratio; CI = confidence interval; RERI = relative excess risk due to interaction.
Interaction on additive scale:
RERI (95% CI) for S/L vs. L/L = -0.27 (-0.91, 0.38), $p = .21$
RERI (95% CI) for S/S vs. L/L = 0.17 (-0.34, 0.67), $p = .74$
Interaction on multiplicative scale:
Ratio of ORs (95% CI) for S/L vs. L/L = 0.81 (0.47, 1.39), $p = .44$
Ratio of ORs (95% CI) for S/S vs. L/L = 1.20 (0.65, 2.22), $p = .56$
Joint test $p = .44$
Models adjusted for age, sex, race/ethnicity, percent federal poverty level in adolescence, highest level of parental education, and mother type.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 4.8. Interaction between triallelic 5-HTTLPR genotype and mother-child connectedness on the odds of binge eating-related concerns.

	Low Mother-Child Connectedness		High Mother-Child Connectedness		**High vs. Low Connectedness OR (95% CI), by Genotype**	**High vs. Low Connectedness OR (95% CI), Irrespective of Genotype**
	N With / Without Outcome	**OR (95% CI)**	**N With / Without Outcome**	**OR (95% CI)**		
L/L	46 / 447	1.00 (Ref)	117 / 1,816	0.71 (0.44, 1.13)	0.69 (0.43, 1.11)	
S/L	106 / 966	1.26 (0.76, 2.09)	290 / 3,702	0.72 (0.47, 1.10)	0.55 (0.40, 0.77)***	0.69 (0.55, 0.87)**
S/S	53 / 634	0.60 (0.34, 1.05)	168 / 2,200	0.70 (0.45, 1.07)	1.26 (0.76, 2.08)	
Genotype OR (95% CI), by Connectedness						
L/L	1.00 (Ref)		1.00 (Ref)			
S/L	1.28 (0.78, 2.09)		1.02 (0.75, 1.38)			
S/S	0.62 (0.36, 1.06)		0.97 (0.72, 1.32)			
Genotype OR (95% CI), Irrespective of Connectedness						
L/L	1.00 (Ref)					
S/L	1.03 (0.82, 1.29)					
S/S	0.86 (0.68, 1.07)					

OR = odds ratio; CI = confidence interval; RERI = relative excess risk due to interaction.
Interaction on additive scale:
RERI (95% CI) for S/L vs. L/L = -0.24 (-0.92, 0.43), $p = .24$
RERI (95% CI) for S/S vs. L/L = 0.39 (-0.02, 0.80), $p = .97$
Interaction on multiplicative scale:
Ratio of ORs (95% CI) for S/L vs. L/L = 0.81 (0.45, 1.47), $p = .49$
Ratio of ORs (95% CI) for S/S vs. L/L = 1.64 (0.86, 3.14), $p = .13$
Joint test $p = .03$
Models adjusted for age, sex, race/ethnicity, percent federal poverty level in adolescence, highest level of parental education, and mother type.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 4.9. Interaction between biallelic 5-HTTLPR genotype and father-child connectedness on the odds of binge eating-related concerns.

	Low Father-Child Connectedness		High Father-Child Connectedness		**High vs. Low Connectedness OR (95% CI), by Genotype**	**High vs. Low Connectedness OR (95% CI), Irrespective of Genotype**
	N With / Without Outcome	**OR (95% CI)**	**N With / Without Outcome**	**OR (95% CI)**		
L/L	66 / 740	1.00 (Ref)	112 / 1,712	0.97 (0.64, 1.44)	1.02 (0.68, 1.53)	
S/L	120 / 1,092	1.26 (0.83, 1.92)	168 / 2,466	0.93 (0.62, 1.38)	0.72 (0.51, 1.02)	0.85 (0.68, 1.07)
S/S	51 / 475	1.09 (0.58, 2.05)	87 / 1,116	0.83 (0.55, 1.27)	0.77 (0.43, 1.38)	
Genotype OR (95% CI), by Connectedness						
L/L	1.00 (Ref)		1.00 (Ref)			
S/L	1.29 (0.85, 1.96)		0.94 (0.66, 1.34)			
S/S	1.13 (0.60, 2.15)		0.83 (0.56, 1.25)			
Genotype OR (95% CI), Irrespective of Connectedness						
L/L	1.00 (Ref)					
S/L	1.09 (0.89, 1.33)					
S/S	0.95 (0.73, 1.24)					

OR = odds ratio; CI = confidence interval; RERI = relative excess risk due to interaction.
Interaction on additive scale:
RERI (95% CI) for S/L vs. L/L = -0.30 (-0.94, 0.34), $p = .18$
RERI (95% CI) for S/S vs. L/L = -0.23 (-1.04, 0.58), $p = .29$
Interaction on multiplicative scale:
Ratio of ORs (95% CI) for S/L vs. L/L = 0.76 (0.44, 1.31), $p = .32$
Ratio of ORs (95% CI) for S/S vs. L/L = 0.79 (0.37, 1.66), $p = .53$
Joint test $p = .61$
Models adjusted for age, sex, race/ethnicity, percent federal poverty level in adolescence, highest level of parental education, and father type.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Table 4.10. Interaction between triallelic 5-HTTLPR genotype and father-child connectedness on the odds of binge eating-related concerns.

	Low Father-Child Connectedness		High Father-Child Connectedness		**High vs. Low Connectedness OR (95% CI), by Genotype**	**High vs. Low Connectedness OR (95% CI), Irrespective of Genotype**
	N With / Without Outcome	**OR (95% CI)**	**N With / Without Outcome**	**OR (95% CI)**		
L/L	49 / 507	1.00 (Ref)	85 / 1,210	1.07 (0.67, 1.69)	1.09 (0.69, 1.72)	
S/L	120 / 1,106	1.27 (0.78, 2.04)	164 / 2,458	0.88 (0.56, 1.39)	0.67 (0.48, 0.94)*	0.85 (0.68, 1.07)
S/S	67 / 673	0.97 (0.54, 1.75)	114 / 1,575	0.80 (0.51, 1.25)	0.84 (0.52, 1.35)	
Genotype OR (95% CI), by Connectedness						
L/L	1.00 (Ref)		1.00 (Ref)			
S/L	1.27 (0.79, 2.06)		0.82 (0.56, 1.19)			
S/S	0.98 (0.54, 1.78)		0.74 (0.52, 1.05)			
Genotype OR (95% CI), Irrespective of Connectedness						
L/L	1.00 (Ref)					
S/L	1.03 (0.82, 1.29)					
S/S	0.86 (0.68, 1.07)					

OR = odds ratio; CI = confidence interval; RERI = relative excess risk due to interaction.
Interaction on additive scale:
RERI (95% CI) for S/L vs. L/L = -0.45 (-1.19, 0.29), $p = .11$
RERI (95% CI) for S/S vs. L/L = -0.24 (-0.97, 0.48), $p = .26$
Interaction on multiplicative scale:
Ratio of ORs (95% CI) for S/L vs. L/L = 0.65 (0.36, 1.16), $p = .15$
Ratio of ORs (95% CI) for S/S vs. L/L = 0.77 (0.39, 1.51), $p = .44$
Joint test $p = .34$
Models adjusted for age, sex, race/ethnicity, percent federal poverty level in adolescence, highest level of parental education, and father type.
* $p < .05$, ** $p < .01$, *** $p < .001$.

Chapter 5

Conclusion

Summary of Main Findings

The overall purpose of this dissertation project was to examine family risk and protective factors for binge eating-related concerns in young adulthood, as well as to investigate potential mediators and moderators. More specifically, we examined associations of childhood maltreatment and mother-child connectedness and father-child connectedness in adolescence with binge eating-related concerns in young adulthood, and we assessed for moderation of these associations by 5-HTTLPR genotype. We also investigated the extent to which associations between childhood maltreatment and binge eating-related concerns are mediated by self-esteem in adolescence, and we examined whether associations of mother-child connectedness and father-child connectedness in adolescence with binge eating-related concerns in young adulthood differ by sex. To address these questions, we used data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a nationally representative sample of the United States.

Childhood maltreatment as a risk factor for binge eating-related concerns

In Chapter 2, we identified distinct childhood maltreatment profiles, examined associations between childhood maltreatment profiles and binge eating-related concerns, and evaluated the extent to which self-esteem during adolescence mediates observed associations. When considering the frequency with which each type of childhood maltreatment had occurred,

we identified five childhood maltreatment latent classes: “no/low maltreatment,” “physical abuse only,” “multi-type maltreatment,” “physical neglect only,” and “sexual abuse only.” A substantial proportion of participants were assigned to the “multi-type maltreatment” class, supporting previous findings that different types of childhood maltreatment often co-occur (Edwards, Holden, Felitti, & Anda, 2003; Higgins & McCabe, 2001; Kim, Mennen, & Trickett, 2017). Participants assigned to the “multi-type maltreatment” class were more likely to report binge eating-related concerns compared to those assigned to the “no/low maltreatment” class. Self-esteem in adolescence mediated a statistically significant but modest proportion of this association. However, we did not observe associations between the single-type childhood maltreatment classes and binge eating-related concerns. These results cohere with previous findings that individuals with a history of multi-type childhood maltreatment, but not single-type childhood maltreatment, have greater depressive symptoms and suicidality than individuals with no history of childhood maltreatment (Arata, Langhinrichsen-Rohling, Bowers, & O’Farrill-Swails, 2005), highlighting the importance of considering the overall childhood maltreatment profile rather than focusing on individual types of childhood maltreatment.

Parent-child connectedness as a protective factor against binge eating-related concerns

In Chapter 3, we investigated the extent to which mother-child connectedness and father-child connectedness in adolescence are associated with binge eating-related concerns and examined differences in associations by sex. In the whole sample, higher mother-child connectedness in adolescence, but not father-child connectedness in adolescence, was associated with lower odds of binge eating-related concerns in young adulthood. However, differences by sex emerged. Both higher mother-child connectedness and higher father-child connectedness in adolescence were associated with lower odds of binge eating-related concerns in young

adulthood among females, but neither mother-child connectedness nor father-child connectedness in adolescence were associated with binge eating-related concerns in young adulthood among males. These results suggest that improving mother-daughter connectedness and father-daughter connectedness in adolescence may be important targets for intervention. Our results build upon previous findings that mother-child connectedness and father-child connectedness are protective against binge eating behaviors among adolescents (Berge et al., 2014) by providing evidence that among females, mother-child connectedness and father-child connectedness in adolescence are protective against binge eating-related concerns into young adulthood.

No evidence of moderation by 5-HTTLPR genotype

In Chapter 4, we explored the extent to which associations of childhood abuse and parent-child connectedness in adolescence with binge eating-related concerns differ by 5-HTTLPR genotype. There was no evidence of interaction on multiplicative or additive scales, suggesting that susceptibility to binge eating-related concerns based on these environmental risk and protective factors does not differ by 5-HTTLPR. While our null findings for an interaction between 5-HTTLPR genotype and childhood abuse is not consistent with previous studies finding the S allele to be associated with greater binge eating-related outcomes among participants who had experienced childhood abuse (Akkermann et al., 2012; Rozenblat et al., 2017; Stoltenberg, Anderson, Nag, & Anagnopoulos, 2012), they do parallel null findings from several meta-analyses published in the depression field investigating an interaction between 5-HTTLPR genotype and life stressors (Culverhouse et al., 2018; Munafò, Durrant, Lewis, & Flint, 2009; Risch et al., 2009).

Strengths and Limitations

This dissertation project had several strengths. We used data from a large, nationally representative sample of participants in the United States followed from adolescence into young adulthood. Using a community sample rather than a clinical sample avoids bias introduced by studying treatment-seeking individuals, and binge eating-related concerns were assessed in young adulthood, a critical period during which levels of cognitive features of eating disorders have been found to increase (Slane, Klump, McGue, & Iacono, 2014). Additionally, our sample included males, a group that has been severely underrepresented in eating disorders research (Murray et al., 2017). Using latent class analysis in Chapter 2 allowed us to efficiently address the interrelatedness yet distinct qualities of multiple types of childhood maltreatment, harnessing a person-centered approach to foster better understanding of pathways from childhood maltreatment to binge eating-related concerns. The use of reliable and valid measures for parent-child connectedness in Chapters 3 and 4 was also a strength (Oman, Vesely, Tolma, Aspy, & Marshall, 2010), as was assessing father-child relationships, which have been less studied than mother-child relationships. As most previous studies using logistic regression have only assessed for interaction on the multiplicative scale (VanderWeele & Knol, 2014), another strength of this project was that we assessed for gene x environment interaction on both additive and multiplicative scales in Chapter 4.

This project also had notable limitations. A key limitation was that binge eating-related concerns were assessed via single-item measures, which threatens the validity of the outcome data. Additionally, these measures used a seven-day assessment time frame, which may underestimate the proportion of participants that experience binge eating-related concerns. However, this would likely result in bias toward the null rather than away from the null. Another

limitation of this project was the retrospective, self-report assessment of childhood maltreatment, which likely underestimates the proportion of participants that had been maltreated during childhood (Hardt & Rutter, 2004). This would again likely result in bias toward the null; thus, the magnitude of association we reported between childhood maltreatment and binge eating-related concerns is likely conservative. Another limitation of this project was the narrow range of childhood maltreatment types that were assessed. Emotional maltreatment has been found to be an important risk factor for eating disorder symptoms (Burns, Fischer, Jackson, & Harding, 2012; Mills, Newman, Cossar, & Murray, 2015), but emotional abuse and emotional neglect were not assessed with physical abuse, sexual abuse, and physical neglect. Another limitation of this project is that in Chapter 2, latent class assignment does not convey the probabilistic nature of the latent class model, and not accounting for the uncertainty in class assignment can lead to underestimation of standard errors in logistic regression. In Chapters 3 and 4, there were limitations related to the data collection approaches used in Add Health. There may be misclassification of some mothers and fathers in households with same-sex parents, as data on same-sex parents were not adequately measured. In addition, no parent-child connectedness data were collected for household members reported by the participant as the husband/wife or partner of the mother/father; therefore, although these household members may serve as mother or father figures, these observations were excluded from analyses. A limitation in Chapter 4 is that although the actions of 5-HTTLPR genotype via brain development are believed to be more predictive of adverse outcomes than current serotonin levels (Kobiella et al., 2011), data for antidepressant use were not collected in Add Health at or before the time binge eating-related concerns were assessed. Thus, we could not conduct sensitivity analyses excluding respondents taking antidepressants at or before the time binge eating-related concerns were assessed. Other

methodologic limitations of this project include the inability to determine causality due to the observational study design, the possibility of residual confounding, and the age of the Add Health data. Despite these limitations, findings from this project offer important contributions to understanding how family risk and protective factors may influence the development of binge eating-related concerns in young adulthood.

Future Research

Our counterintuitive finding suggesting father-son connectedness in adolescence may be associated with increased odds of binge eating-related concerns in young adulthood warrants further investigation. The direction of this association may be related to traditional gender roles, as binge eating may generally be less distressing for males as compared to females because males consider consuming large amounts of food to be "masculine" (Carey, Saules, & Carr, 2017), while items used to assess parent-child connectedness draw upon traditionally feminine traits, such as warm and loving (Bem, 1974). The direction of this association may also be thought of in the context of sex differences in developmental changes around the age at which parent-child connectedness was assessed in adolescence. Participants' mean age was 15 years when parent-child connectedness was assessed, which coincides with the age at which girls' – but not boys' – perceptions of support from fathers have been found to begin increasing after a period of decline in perceived support from fathers between early to middle adolescence (De Goede, Branje, & Meeus, 2009). Thus, as we would expect boys' perceptions of support from fathers to continue to decrease around this age, boys reporting higher levels of father-son connectedness around this age may simply represent a different subset of boys. Future research

should elucidate why patterns may differ for father-son dyads, but it seems unlikely that interventions promoting father-son connectedness would have detrimental effects.

Given that we did not find evidence for gene x environment interaction, the findings of this project suggest that for the purpose of informing eating disorders interventions, future research may benefit from focusing on environmental risk and protective factors irrespective of genetic susceptibility. Future research should also examine the role of protective factors in buffering the negative sequelae of risk factors such as childhood maltreatment. Researching effective ways to prevent childhood maltreatment from occurring in the first place is critical, but more research on moderation by protective factors could help mitigate the consequences of childhood maltreatment once it has occurred. In this project, while high parent-child connectedness was less prevalent among participants with a history of abuse than among participants with no history of abuse, over half of participants with a history of abuse still reported high parent-child connectedness. We were unable to meaningfully assess whether parent-child connectedness moderates associations between childhood abuse and binge eating-related concerns in the current project, as we did not have data on which parent or adult caregiver participants were abused by, but a recent study suggests that body compassion buffers associations between life stressors and binge eating (Barata-Santos, Marta-Simões, & Ferreira, 2018). More such research is needed to help inform targeted interventions for high-risk populations.

In addition to more work advancing our understanding of how protective factors can help mitigate consequences among high-risk populations, future research should also focus on modifiable protective factors to inform universal prevention programs. While many risk factors, including childhood maltreatment, are non-specific (i.e., they are associated with adverse

outcomes across multiple domains; Anda et al., 2006; Durlak, 1998), many protective factors, including parent-child connectedness, are also non-specific (Durlak, 1998; Levine & Smolak, 2016). Considering that eating disorders are highly comorbid with other psychiatric disorders and etiological processes contributing to eating disorders and other psychiatric disorders often overlap (Caspi & Moffitt, 2018; Hudson, Hiripi, Pope, & Kessler, 2007), adopting a transdiagnostic approach to prevention of psychiatric disorders may translate to improved efficiency and dissemination reach of preventive interventions.

In order to inform effective transdiagnostic interventions for a range of psychiatric disorders, it is necessary to understand which factors are most central to the development and maintenance of psychiatric disorders and comorbidities. Network analysis is one tool that shows promise to help advance understanding in this area, as it can help identify these core etiological and maintenance factors that, if disrupted, may help prevent further symptom occurrence (Smith et al., 2018). For example, among individuals with a history of trauma, re-experiencing trauma has been found to be the most central symptom in a network structure of eating disorder and post-traumatic stress disorder symptoms, suggesting utility of re-experiencing as a key intervention target (Liebman et al., 2019). Similarly, network analysis has the potential to help identify protective factors that are most central to good mental health, which could inform effective mental health promotion efforts. Future research employing network analysis using prospective data could therefore help pave the way for interventions to target particularly salient risk and protective factors for eating disorders and other psychiatric disorders.

Public Health Implications

Eating disorders represent an important public health problem. Not only do they have an early age of onset and relatively high prevalence (Allen, Byrne, Oddy, & Crosby, 2013; Hudson et al., 2007; Smink, van Hoeken, Oldehinkel, & Hoek, 2014; Stice, Marti, & Rohde, 2013; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011), they are highly comorbid with other psychiatric disorders and strongly associated with medical complications, psychosocial impairment, and suicidality (Hudson et al., 2007; Mitchell & Crow, 2006; Swanson et al., 2011). Prevention and early intervention are crucial to reducing the burden of disease associated with eating disorders. As binge eating-related concerns have been found to be precursors to eating disorders (Fairburn, Cooper, Doll, & Davies, 2005), reducing binge eating-related concerns may be an important target for eating disorders prevention and early intervention.

The findings from this dissertation project indicate that individuals exposed to multiple types of childhood maltreatment may be at particularly high risk for eating disorders. Accurately classifying childhood maltreatment profiles is not only valuable for identifying high-risk subgroups but also necessary for providing trauma-informed eating disorders treatment and prevention for those high-risk subgroups. As unaddressed trauma can perpetuate eating disorder symptoms (Brewerton, Alexander, & Schaefer, 2018), it is essential that clinicians screen for trauma and utilize trauma-focused treatment modalities such as eye-movement desensitization and reprocessing, cognitive processing therapy, and trauma-focused cognitive-behavioral therapy to address the range of traumas patients report (Brewerton, 2018). As importantly, clinicians should also be careful to select appropriate treatment modalities given their patients' trauma histories, as family-based treatment could be counterproductive if the parents involved in treatment were responsible for those trauma histories. From a prevention standpoint, targeted

prevention programs should address trauma – the full range of traumas experienced – before eating disorders develop. Moreover, given the substantial proportion of individuals who experience multiple types of maltreatment and the unequivocal adverse consequences of multi-type maltreatment, more resources must be devoted to preventing childhood maltreatment from occurring in the first place.

The results from this project also suggest that improving parent-child connectedness in adolescence, particularly mother-daughter connectedness and father-daughter connectedness, may be important targets for eating disorders interventions. Given its non-specific nature (Durlak, 1998), interventions to improve parent-child connectedness have the potential to reduce risk for a wide range of adverse outcomes. Preventive interventions could come in the form of universal interventions for all families or in the form of targeted interventions for high-risk families (e.g., families experiencing high levels of conflict or families in the process of restructuring). In addition, clinicians working with eating disorder patients and their families may consider working to improve parent-child connectedness throughout treatment. However, in both treatment and prevention approaches, some situations of extreme parent-child connectedness deficits may warrant alternative strategies, such as promoting other sources of adult connection (e.g., teachers, mentors).

We did not find susceptibility to environmental risk or protective factors to differ by 5-HTTLPR. Therefore, although previous research finding greater intervention effects among individuals with greater genetic susceptibility has shown promise for personalized intervention approaches (Morgan et al., 2017), our findings do not support the differential susceptibility hypothesis. Instead, our findings suggest that eating disorders intervention approaches should focus on decreasing risk factors, such as childhood maltreatment, and promoting protective

factors, such as parent-child connectedness. Further, given that childhood maltreatment has been established as a risk factor across a wide range of domains (Anda et al., 2006) and parent-child connectedness has been established as a protective factor across a wide range of domains (Durlak, 1998), effective interventions to prevent childhood maltreatment or mitigate its effects and increase parent-child connectedness could have widespread positive impact beyond reducing the burden of eating disorders.

References

Akkermann, K., Kaasik, K., Kiive, E., Nordquist, N., Oreland, L., & Harro, J. (2012). The impact of adverse life events and the serotonin transporter gene promoter polymorphism on the development of eating disorder symptoms. *Journal of Psychiatric Research*, *46*(1), 38–43. http://doi.org/10.1016/j.jpsychires.2011.09.013

Allen, K. L., Byrne, S. M., Oddy, W. H., & Crosby, R. D. (2013). DSM-IV-TR and DSM-5 eating disorders in adolescents: Prevalence, stability, and psychosocial correlates in a population-based sample of male and female adolescents. *Journal of Abnormal Psychology*, *122*(3), 720–732. http://doi.org/10.1037/a0034004

Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C., Perry, B. D., … Giles, W. H. (2006). The enduring effects of abuse and related adverse experiences in childhood. A convergence of evidence from neurobiology and epidemiology. *European Archives of Psychiatry and Clinical Neuroscience*, *256*(3), 174–86. http://doi.org/10.1007/s00406-005-0624-4

Arata, C. M., Langhinrichsen-Rohling, J., Bowers, D., & O'Farrill-Swails, L. (2005). Single versus multi-type maltreatment. *Journal of Aggression, Maltreatment & Trauma*, *11*(4), 29–52. http://doi.org/10.1300/J146v11n04_02

Barata-Santos, M., Marta-Simões, J., & Ferreira, C. (2018). Body compassion safeguards against the impact of major life events on binge eating. *Appetite*. http://doi.org/10.1016/j.appet.2018.12.016

Bem, S. L. (1974). The measurement of psychological androgyny. *Journal of Consulting and Clinical Psychology*, *42*(2), 155–162.

Berge, J. M., Wall, M., Larson, N., Eisenberg, M. E., Loth, K. A., & Neumark-Sztainer, D.

(2014). The unique and additive associations of family functioning and parenting practices with disordered eating behaviors in diverse adolescents. *Journal of Behavioral Medicine*, *37*(2), 205–17. http://doi.org/10.1007/s10865-012-9478-1

Brewerton, T. D. (2018). An overview of trauma-informed care and practice for eating disorders. *Journal of Aggression, Maltreatment & Trauma*, 1–18. http://doi.org/10.1080/10926771.2018.1532940

Brewerton, T. D., Alexander, J., & Schaefer, J. (2018). Trauma-informed care and practice for eating disorders: Personal and professional perspectives of lived experiences. *Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity*. http://doi.org/10.1007/s40519-018-0628-5

Burns, E. E., Fischer, S., Jackson, J. L., & Harding, H. G. (2012). Deficits in emotion regulation mediate the relationship between childhood abuse and later eating disorder symptoms. *Child Abuse & Neglect*, *36*(1), 32–9. http://doi.org/10.1016/j.chiabu.2011.08.005

Carey, J. B., Saules, K. K., & Carr, M. M. (2017). A qualitative analysis of men's experiences of binge eating. *Appetite*, *116*, 184–195. http://doi.org/10.1016/j.appet.2017.04.030

Caspi, A., & Moffitt, T. E. (2018). All for one and one for all: Mental disorders in one dimension. *American Journal of Psychiatry*. http://doi.org/10.1176/appi.ajp.2018.17121383

Culverhouse, R. C., Saccone, N. L., Horton, A. C., Ma, Y., Anstey, K. J., Banaschewski, T., … Bierut, L. J. (2018). Collaborative meta-analysis finds no evidence of a strong interaction between stress and 5-HTTLPR genotype contributing to the development of depression. *Molecular Psychiatry*, *23*(1), 133. http://doi.org/10.1038/mp.2017.44

De Goede, I. H. A., Branje, S. J. T., & Meeus, W. H. J. (2009). Developmental changes in adolescents' perceptions of relationships with their parents. *Journal of Youth and*

Adolescence, *38*(1), 75–88. http://doi.org/10.1007/s10964-008-9286-7

Durlak, J. A. (1998). Common risk and protective factors in successful prevention programs. *American Journal of Orthopsychiatry*, *68*(4), 512–520. http://doi.org/10.1037/h0080360

Edwards, V. J., Holden, G. W., Felitti, V. J., & Anda, R. F. (2003). Relationship between multiple forms of childhood maltreatment and adult mental health in community respondents: Results from the Adverse Childhood Experiences Study. *The American Journal of Psychiatry*, *160*(8), 1453–1460. http://doi.org/10.1176/appi.ajp.160.8.1453

Fairburn, C. G., Cooper, Z., Doll, H. A., & Davies, B. A. (2005). Identifying dieters who will develop an eating disorder: a prospective, population-based study. *American Journal of Psychiatry*, *162*(12), 2249–2255. http://doi.org/10.1176/appi.ajp.162.12.2249

Hardt, J., & Rutter, M. (2004). Validity of adult retrospective reports of adverse childhood experiences: Review of the evidence. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *45*(2), 260–273. http://doi.org/10.1111/j.1469-7610.2004.00218.x

Higgins, D. J., & McCabe, M. P. (2001). Multiple forms of child abuse and neglect: Adult retrospective reports. *Aggression and Violent Behavior*, *6*(6), 547–578. http://doi.org/10.1016/S1359-1789(00)00030-6

Hudson, J. I., Hiripi, E., Pope, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*, *61*(3), 348–358. http://doi.org/10.1016/j.biopsych.2006.03.040

Kim, K., Mennen, F. E., & Trickett, P. K. (2017). Patterns and correlates of co-occurrence among multiple types of child maltreatment. *Child & Family Social Work*, *22*(1), 492–502. http://doi.org/10.1111/cfs.12268

Kobiella, A., Reimold, M., Ulshöfer, D. E., Ikonomidou, V. N., Vollmert, C., Vollstädt-Klein, S.,

… Smolka, M. N. (2011). How the serotonin transporter 5-HTTLPR polymorphism influences amygdala function: the roles of in vivo serotonin transporter expression and amygdala structure. *Translational Psychiatry*, *1*, e37. http://doi.org/10.1038/tp.2011.29

Levine, M. P., & Smolak, L. (2016). The role of protective factors in the prevention of negative body image and disordered eating. *Eating Disorders*, *24*(1), 39–46. http://doi.org/10.1080/10640266.2015.1113826

Liebman, R., Becker, K., Eddy, K., Thomas, J., Cao, L., Smith, K., & Crosby, R. (2019). Network Analysis of Eating Disorders and PTSD in a Community Sample: The Role of Reexperiencing as a Mechanism of Comorbidity. In *International Conference on Eating Disorders*.

Mills, P., Newman, E. F., Cossar, J., & Murray, G. (2015). Emotional maltreatment and disordered eating in adolescents: Testing the mediating role of emotion regulation. *Child Abuse & Neglect*, *39*, 156–166. http://doi.org/10.1016/J.CHIABU.2014.05.011

Mitchell, J. E., & Crow, S. (2006). Medical complications of anorexia nervosa and bulimia nervosa. *Current Opinion in Psychiatry*, *19*(4), 438–443. http://doi.org/10.1097/01.yco.0000228768.79097.3e LK - https://aalborguh.tdnetdiscover.com/resolver/full?&sid=EMBASE&issn=09517367&id=doi:10.1097%2F01.yco.0000228768.79097.3e&atitle=Medical+complications+of+anorexia+nervosa+and+bulimia+nervosa&stitle=Curr.+Opin.+Psychiatry&title=Current+Opinion+in+Psychiatry&volume=19&issue=4&spage=438&epage=443&aulast=Mitchell&aufirst=James+E.&auinit=J.E.&aufull=Mitchell+J.E.&coden=COPPE&isbn=&pages=438-443&date=2006&auinit1=J&auinitm=E.

Morgan, B., Kumsta, R., Fearon, P., Moser, D., Skeen, S., Cooper, P., … Tomlinson, M. (2017).

Serotonin transporter gene (SLC6A4) polymorphism and susceptibility to a home-visiting maternal-infant attachment intervention delivered by community health workers in South Africa: Reanalysis of a randomized controlled trial. *PLOS Medicine*, *14*(2), e1002237. http://doi.org/10.1371/journal.pmed.1002237

Munafò, M. R., Durrant, C., Lewis, G., & Flint, J. (2009). Gene x environment interactions at the serotonin transporter locus. *Biological Psychiatry*, *65*(3), 211–9. http://doi.org/10.1016/j.biopsych.2008.06.009

Murray, S. B., Nagata, J. M., Griffiths, S., Calzo, J. P., Brown, T. A., Mitchison, D., … Mond, J. M. (2017). The enigma of male eating disorders: A critical review and synthesis. *Clinical Psychology Review*, *57*, 1–11. http://doi.org/10.1016/j.cpr.2017.08.001

Oman, R. F., Vesely, S. K., Tolma, E. L., Aspy, C. B., & Marshall, L. (2010). Reliability and validity of the Youth Asset Survey: An update. *American Journal of Health Promotion*, *25*(1), e13–e24. http://doi.org/10.4278/ajhp.081009-QUAN-242

Risch, N., Herrell, R., Lehner, T., Liang, K.-Y., Eaves, L., Hoh, J., … Merikangas, K. R. (2009). Interaction between the serotonin transporter gene (5-HTTLPR), stressful life events, and risk of depression: A meta-analysis. *JAMA*, *301*(23), 2462–2471. http://doi.org/10.1001/jama.2009.878

Rozenblat, V., Ong, D., Fuller-Tyszkiewicz, M., Akkermann, K., Collier, D., Engels, R. C. M. E., … Krug, I. (2017). A systematic review and secondary data analysis of the interactions between the serotonin transporter 5-HTTLPR polymorphism and environmental and psychological factors in eating disorders. *Journal of Psychiatric Research*, *84*, 62–72. http://doi.org/10.1016/j.jpsychires.2016.09.023

Slane, J. D., Klump, K. L., McGue, M., & Iacono, W. G. (2014). Developmental trajectories of

disordered eating from early adolescence to young adulthood: a longitudinal study. *The International Journal of Eating Disorders*, *47*(7), 793–801. http://doi.org/10.1002/eat.22329

Smink, F. R. E., van Hoeken, D., Oldehinkel, A. J., & Hoek, H. W. (2014). Prevalence and severity of DSM-5 eating disorders in a community cohort of adolescents. *International Journal of Eating Disorders*, *47*(6), 610–619. http://doi.org/10.1002/eat.22316

Smith, K. E., Crosby, R. D., Wonderlich, S. A., Forbush, K. T., Mason, T. B., & Moessner, M. (2018). Network analysis: An innovative framework for understanding eating disorder psychopathology. *International Journal of Eating Disorders*, *51*(3), 214–222. http://doi.org/10.1002/eat.22836

Stice, E., Marti, C. N., & Rohde, P. (2013). Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. *Journal of Abnormal Psychology*, *122*(2), 445–457. http://doi.org/10.1037/a0030679

Stoltenberg, S. F., Anderson, C., Nag, P., & Anagnopoulos, C. (2012). Association between the serotonin transporter triallelic genotype and eating problems is moderated by the experience of childhood trauma in women. *The International Journal of Eating Disorders*, *45*(4), 492–500. http://doi.org/10.1002/eat.20976

Swanson, S. A., Crow, S. J., Le Grange, D., Swendsen, J., & Merikangas, K. R. (2011). Prevalence and correlates of eating disorders in adolescents. Results from the national comorbidity survey replication adolescent supplement. *Archives of General Psychiatry*, *68*(7), 714–723. http://doi.org/10.1001/archgenpsychiatry.2011.22

VanderWeele, T. J., & Knol, M. J. (2014). A tutorial on interaction. *Epidemiologic Methods*, *3*(1), 33–72. http://doi.org/10.1515/em-2013-0005