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Genetic and Environmental Influences on Posttraumatic Stress Disorder Symptoms and Disinhibited Eating Behaviors

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Abstract

Posttraumatic stress disorder (PTSD) and eating disorders (ED) frequently co-occur, but the mechanisms underlying this association remain unclear. EDs are characterized by features of maladaptive eating behaviors including, disinhibited eating and cognitive dietary restraint. Identifying the genetic overlap between PTSD symptoms and maladaptive eating behaviors may elucidate biological mechanisms and potential treatment targets. A community sample of 400 same-sex twins (102 monozygotic and 98 dizygotic pairs) completed the PTSD Checklist-Civilian (PCL-C) for PTSD symptoms and the Three-Factor Eating Questionnaire-Reduced (TFEQ-R18) for eating behaviors (uncontrolled eating, emotional eating, and cognitive dietary restraint). We used biometric modeling to examine the genetic and environmental relationships between PCL-C and TFEQ-R18 total and subscales scores. Heritability was estimated at 48% for PTSD symptoms and 45% for eating behavior overall. Bivariate models revealed a significant genetic correlation between PTSD symptoms and eating behavior overall (r_g = .34; CI: .07, .58) and Uncontrolled Eating (r_g = .53; CI: .24, .84), and a significant environmental correlation between PTSD symptoms and Emotional Eating (r_e = .30; CI: .12, .45). These findings suggest the influence of common etiology. Future research and clinical efforts should focus on developing integrated treatments.

Keywords

PTSD; disinhibited eating; cognitive restraint; genetics; twins; eating behavior

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Posttraumatic stress disorder (PTSD) is a psychological condition that may develop following exposure to trauma and is characterized by unwanted thoughts and feelings related to the event, distress in response to trauma-related cues, emotional numbness and avoidance, and increased arousal. PTSD is highly comorbid with eating disorders (ED) including Bulimia Nervosa (BN), Binge Eating Disorder (BED), and Anorexia Nervosa (AN) binge-eating/purging type, and trauma exposure has been suggested as a risk factor for the development of ED (Brewerton, 2007; Mitchell, Mazzeo, Schlesinger, Brewerton, & Smith, 2012). Over 40% of individuals with PTSD surveyed in a large population-based study reported maladaptive eating symptoms (Sommer, Mota, & El-Gabalawy, 2018), where respondents with subthreshold or threshold PTSD reported significantly greater binge eating symptoms than did respondents with a trauma history but no PTSD or low PTSD symptoms (Braun et al., 2019). Patients with co-occurring PTSD and ED show a higher prevalence of psychiatric and substance use comorbidities and more severe ED symptoms than their counterparts with either condition alone (Grilo, White, Barnes, & Masheb, 2012; Scharff, Ortiz, Forrest, & Smith, 2019).

Despite substantial research on the prevalence and negative sequelae of co-occurring PTSD and ED, little is known about shared mechanisms and causal relationships. Shared mechanisms may be genetic, neurobiological, and psychological in nature. PTSD and ED symptoms may be functionally related, such that ED symptoms (e.g., binge eating and emotional eating behaviors, etc.) serve as an effort to downregulate PTSD-consistent hyperarousal or serve trauma-related escape and avoidance functions (Breland, Donalson, Dinh, & Maguen, 2018; Trottier, Wonderlich, Monson, Crosby, & Olmsted, 2016). Elucidating the mechanisms underlying the comorbidity of PTSD and ED symptoms may provide support for developing integrated treatments (Trottier & MacDonald, 2017).

Most research exploring the comorbidity of PTSD and ED has examined individual eating disorders; however, latent class analyses, genetic research, and epidemiological findings support examining maladaptive eating behaviors that are common to several EDs and may also be common phenotypic features of PTSD (Mitchell et al., 2010; Sommer, Mota, & El-Gabalawy, 2018; Von Lojewski, Boyd, Abraham, & Russell, 2012). These maladaptive eating behaviors include, but are not limited to (1) disinhibited eating behavior, and (2) cognitive dietary restraint, characterized by a set of self-imposed rules to control food intake for weight loss or to control body weight. The construct of disinhibited eating subsumes related but distinct eating behaviors, some of which are characterized by loss of control and objectively large intake (i.e., sub-diagnostic threshold binge eating) and others which are characterized by a perceived lack of control and may or may not include objectively large intake but are distressing to the individual (e.g., uncontrolled eating, emotional eating) (Johnson et al., 2012; Vainik et al., 2015). Though past research has demonstrated that these eating behaviors are associated *with* and are potential risk factors *for* disordered eating (Anderson, Reilly, Schaumberg, Dmochowski, & Anderson, 2016; Schaumberg & Anderson, 2016), only a handful of studies have examined the association between PTSD symptoms and disinhibited eating (Mason et al., 2017; Talbot, Maguen, Epel, Metzler, & Neylan, 2013). For example, previous research on the relationship between PTSD and AN has demonstrated the restricting subtype of AN is less strongly associated with PTSD than the AN binge eating/purging type (e.g., Brewerton, 2007; Carter et al, 2006; Reyes-

Rodríguez, 2011), suggesting that binge eating and other disinhibited eating features of EDs may be important for understanding the link between EDs and PTSD. Additionally, the only study evaluating the association of PTSD symptoms with cognitive dietary restraint failed to find a significant relationship (Mason, LeBouthillier, & Asmundson, 2019). Improving our understanding of the relationship between PTSD symptoms and transdiagnostic maladaptive eating behaviors may help elucidate the mechanisms underlying the spectrum of ED and their association with PTSD.

Twin studies, which compare monozygotic (MZ) and dizygotic (DZ) twins using biometric modeling, provide a unique opportunity to test the importance of neurobiological and psychosocial mechanisms by estimating both genetic and environmental influences on complex phenotypes. Twin studies can explain the degree to which phenotypic associations between two or more traits are due to shared genetic or environmental factors. Understanding the extent to which various etiological factors (e.g., genetic or environmental) contribute to the co-occurrence of phenotypes has important implications. For instance, if the same genes are found to contribute to both phenotypes, this lends support for a common biological predisposition or pathway and would have substantial implications for identifying specific common genes. Conversely, if the association among phenotypes is due primarily to environmental reasons, prevention and intervention efforts should focus on appropriate strategies to address environmental factors such as psychosocial adversity or trauma. These scenarios represent two theoretical extremes with a more likely scenario involving both genetic and environmental influences.

Previous twin research has demonstrated moderate levels of heritability for PTSD (Kremen, Koenen, Afari, & Lyons, 2012), ED, and maladaptive eating behaviors (Thornton, Mazzeo, & Bulik, 2011). Heritability estimates for PTSD and PTSD symptoms range from 30% to 72%, depending on the population (Kremen et al., 2012; Sartor et al., 2011). Estimates for the heritability of disinhibited eating alone range from 18% to 45%; findings for cognitive dietary restraint have been mixed due to varying definitions and measures, but estimated genetic influences of up to 59% have been reported (Thornton et al., 2011). Despite the known heritability of these phenotypes individually, no studies have evaluated the contribution of genetic and environmental influences to the co-occurrence of PTSD symptoms and disinhibited and other maladaptive eating behaviors. Thus, the aim of our study was to evaluate potential genetic and environmental sources of co-occurrence between PTSD symptoms and disinhibited eating (i.e., uncontrolled eating and emotional eating) and cognitive dietary restraint in a genetically informatic sample of non-clinical, community-dwelling twins. Based on previous research, we expected that all traits would be genetically influenced, and hypothesized that the covariation between PTSD symptoms and disinhibited eating behaviors would be due, in part, to common genetic factors. Given the very limited prior research, we had no hypotheses regarding PTSD and cognitive dietary restraint.

Methods

Participants

Participants were same-sex twins from the Washington State Twin Registry (WSTR; previously known as the University of Washington Twin Registry), a community-based

sample drawn from information gathered by the Washington State Department of Licensing (Strachan et al., 2013). This randomly selected subset was originally recruited for a study examining the psychosocial, demographic, and clinical factors associated with experimental pain sensitivity. A detailed description of Registry participant recruitment procedures are described elsewhere (Afari et al., 2006; Strachan et al., 2013). Same-sex twin pairs between 18 and 65 years of age were eligible. Of the 752 individuals who were screened for participation, 75 individuals (one or both of a twin pair) did not meet the inclusion criteria. These participants were excluded for use of opioid or other prescription pain medications, immune-modulating medications, a BMI of < 18.5 kg/m^2, current or anticipated pregnancy, a neuropathy, and ongoing cancer treatment, as these could interfere with the parent experimental pain study measurements. In addition, 77 eligible pairs were unable to participate due to scheduling issues. The final sample consisted of 400 non-clinical members of the WSTR consisting of 102 monozygotic (MZ) and 98 dizygotic (DZ) same-sex twin pairs. The twins (63% female) were mostly young to middle-aged adults (M = 29 years, SD = 12, range = 19 – 65), and most were White (80%).

Zygosity was determined using either the AmpFlSTR Identifiler Plus PCR Amplification Kit or the PowerPlex 16 HS System. The two protocols are nearly identical, and all assays were conducted per manufacturer's instructions at the University of Washington Center for Clinical Genomics. Study procedures and protocols were approved by the Institutional Review Board at the University of Washington, and informed consent was obtained from participants.

Measures

PTSD symptoms.—The PTSD Checklist-Civilian Version (PCL-C) (Weathers, Litz, Herman, Huska, & Keane, 1993) is a self-report questionnaire consisting of 17 items that correspond to Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV) (American Psychiatric Association, 2000) diagnostic criteria for PTSD. Participants report the extent to which 17 symptoms (e.g., "repeated, disturbing memories, thoughts, or images of a stressful experience from the past") have caused distress in the past four weeks using a 5-point scale from 1 (*not at all*) to 5 (*extremely*). Because identifying a PTSD Criterion A trauma exposure, is not required on the PCL-C, this measure assesses PTSD symptom burden in the context of stressful life events rather than a specific previous trauma. Item responses are summed to calculate a total score, which ranges from 17 to 85. Higher scores indicate a greater burden of PTSD symptoms. Different cutoff scores are used to examine the estimated prevalence of PTSD in different populations or settings, with a cutoff of 30–35 suggested for general population samples (National Center for PTSD, n.d.). The PCL-C has high internal consistency and strong agreement with the Clinician Administered PTSD Scale (Yeager, Magruder, Knapp, Nicholas, & Frueh, 2007) as well as the Composite International Diagnostic Interview PTSD module, r = 0.90 (Magruder et al., 2015). The mean PCL-C score was 29.7 (SD = 12.5) in a civilian primary care sample (Stein, Mcquaid, Pedrelli, Lenox, & Mcahill, 2000).

Eating Behavior.—The Three-Factor Eating Questionnaire Reduced (TFEQ-R18) (Karlsson, Persson, Sjöström, & Sullivan, 2000) is an 18-item self-report questionnaire used

to assess cognitive dietary restraint (conscious restriction of food intake with the intent to control body weight and/or to promote weight loss), uncontrolled eating (the tendency to eat more than usual due to a loss of control over intake accompanied by a subjective feeling of hunger), and emotional eating (inability to resist emotional cues, eating as a response to different negative emotions). The TFEQ-R18 is a revised and reduced version of the original 51-item TFEQ (Stunkard & Messick, 1985), also called the Eating Inventory, a commonly used measure of eating behaviors. Sample items include: "I consciously hold back at meals in order not to gain weight" (Cognitive Restraint); "Sometimes when I start eating, I just can't seem to stop" (Uncontrolled Eating); and "When I feel lonely, I console myself by eating" (Emotional Eating). Participants rate the extent to which each statement is true on a 4-point scale from 4 (*definitely true*) to 1 (*definitely false*) for the first 17 questions. The final question asks participants to rate the extent of their restrained eating from 1 (*no restraint in eating*) to 8 (*total restraint*). All items are summed into a total score, and subsets are summed to create three subscale scores. Higher scores indicate greater symptoms (De Lauzon et al., 2004). Previous studies have reported strong internal consistency and reliability of the TFEQ-R18 total and subscale scores (Karlsson et al., 2000). To our knowledge, there are no norms for the TFEQ-R18 and no suggested cutoff scores. Mean subscale scores in a study of the psychometric properties of the Spanish version of the measure in a non-clinical sample were: Uncontrolled Eating (17.50±6.20); Cognitive Restraint (11.69±4.11); and Emotional Eating (5.14±2.65) (Jáuregui-Lobera, García-Cruz, Carbonero-Carreño, Magallares, & Ruiz-Prieto, 2014).

Statistical Analyses

Descriptive statistics were calculated using means and standard deviations, and study variables were compared by sex. For the remainder of the analyses, individual variables were age and sex-regressed following standard analytic procedures (McGue & Bouchard, 1984) and log-transformed to better approximate normality. Standard biometric models were used to evaluate the relative contribution of genetic and environmental factors to each phenotype. The classical twin model estimates variance components based on similarities among MZ twins who share 100% and DZ twins who share, on average, 50% of their segregating genes. Using univariate biometric models, phenotypic variance is decomposed into genetic and environmental sources of variance, including additive genetic influence (A), shared environmental influences (C), and non-shared environmental influences (E). Shared environmental influences are influences that make members of a twin pair more similar to one another, while non-shared environmental influences are specific to the individual and also capture measurement error. Three subsequent submodels were computed that dropped shared environmental influences (AE model), genetic influences (CE model), and genetic and shared environmental influences (E model). The univariate models were extended to a bivariate Cholesky decomposition (Neale & Maes, 2004) to determine the degree of genetic and environmental overlap between two variables, such as PTSD symptoms and Emotional Eating (Figure 1a). Latent factors A_1, C_1, and E_1 account for the additive genetic and shared environmental variance in the first phenotype, including the variance that overlaps with risk for the second. Residual additive genetic and non-shared environmental factors (independent of the first phenotype) are captured by Factors A_2, C_2, and E_2. Diagonal paths (a_{21}, c_{21}, e_{21}) represent the genetic or environmental covariance between traits. The

parameter estimates generated by the Cholesky decomposition were used to construct other quantities of interest represented by the correlated factors model (Figure 1b)(Loehlin, 1996). Standardizing the genetic covariance on the genetic variance of the two phenotypes from the bivariate Cholesky decomposition yielded the genetic correlation, $r_g = \frac{a_{11}a_{21}}{\sqrt{a_{11}^2(a_{21}^2 + a_{22}^2)}}$, which indexed the magnitude of the genetic overlap between the two phenotypes, or the extent to which the same genes or environmental factors contribute to the observed phenotypic correlation between two variables (Loehlin, 1996). By estimating the overlap in genetic signal rather than all sources of variance and covariance, genetic correlations are more informative than phenotypic correlations by isolating the source of the association (Neale & Maes, 2004). A genetic correlation of 1.0 suggests that the two variables share of all their genetic influences, while a genetic correlation of 0 suggests genetic independence. High genetic correlations suggest that genes identified for one trait would also likely influence the other trait. Similar analytic procedures can be used to estimate shared (*rc*) and non-shared environmental correlations (*re*). Squaring the genetic correlation provides an estimate of the degree to which the genetic factors associated with one variable account for the genetic risk associated with other. Using the path estimates from the Cholesky decomposition, we also estimated the degree of genetic contribution to the phenotypic correlation between two variables, $prop_A = \frac{r_A\sqrt{a_{11}^2(a_{21}^2 + a_{22}^2)}}{r}$. This approach is particularly valuable when the phenotypic correlations are low, but the observed association may be strongly genetically influenced. Contribution of environmental factors to the phenotypic correlation were calculated in an analogous manner.

Models were fit to the raw data using full information maximum-likelihood in OpenMx software version 2.12.1 (Neale et al., 2016) in the software R (R Core Team, 2016). This approach allows the use of all available information from all cases regardless of missing data and yields less-biased estimates when compared to listwise or pairwise deletion methods (Allison, 2003). Adequacy of model fit was evaluated using −2 times the natural log likelihood (−2lnL) and Akaike's Information Criterion (AIC) between nested models. Lower AIC values signify a better balance between goodness of fit and parsimony. The goal is to identify the most parsimonious model that sufficiently describes the data, and models with fewer parameters are preferred if they do not result in a significant deterioration of fit. Significance of parameters was tested using the ($\chi 2$) difference test, comparing the goodness-of-fit of the reduced model to a fuller model.

Results

The means and standard deviations for all study measures for the entire sample and by sex are presented in Table 1. Only 20 individual item responses were missing (<1%) from both questionnaires. Little's missing completely at random (MCAR) test showed that any missing data were at random (Little's MCAR test: $\chi 2 = 446.377$, df = 440, p = .407). No participants were missing more than one item per scale, and scale scores were prorated for each respondent with missing data. The mean PCL-C and TFEQ-R18 subscale scores were similar to those previously reported in non-clinical samples (Jáuregui-Lobera et al., 2014;

Stein et al., 2000). In our sample, 55 individuals (13.8%), with 25 men (13.3%) and 30 women (14.2%) met the National Center for PTSD (n.d.) suggested PCL-C cut-off of 35 for general population samples. This is similar to rates reported in the general population (National Center for PTSD, n.d.). The total TFEQ-R18 score and the Cognitive Restraint and Emotional Eating subscale scores for female twins were significantly higher than for male twins, supporting the decision to control for sex in the biometric and other analyses. The phenotypic correlations between PCL-C and TFEQ-R18 Total ($r = .32$, $p < .001$), Uncontrolled Eating ($r = .31$, $p < .001$), and Emotional Eating ($r = .29$, $p < .001$) were significant; the phenotypic correlation between PCL-C and Cognitive Restraint subscale score ($r = .08$, $p > .05$) was not significant. Because no phenotypic correlation between PCL-C and Cognitive Restraint was detected, genetic and non-shared environmental correlations were not calculated.

Biometric Model Fitting

Univariate biometric modeling evaluated the contribution of genetic and environmental influences on the variance in PCL-C and TFEQ-R18 total and subscales separately. The model fit results are presented in Supplemental Table 1 and revealed that the AE models were the most parsimonious for all phenotypes, indicating no shared environmental influence on any of the variables. Bivariate Cholesky decompositions were used to estimate the degree of genetic and environmental overlap between PTSD and TFEQ-R18 scores. Model fit results are presented in Supplemental Table 2 and suggest that AE models, containing no shared environmental effects (path estimates from best fitting AE models in Supplemental Table 3), provided the best fit for all comparisons.

Heritability estimates from the bivariate models are presented in Table 2. Heritability was estimated at .48 (95% CI: .33; .61) for PCL-C total score and .45 (95% CI: .29; .58) for TFEQ-R18 total score with the remaining variance due to non-shared environmental influences. Similarly, subscales of the TFEQ-R18 showed moderate heritability ($a^2 = .48$ to .34) with the remaining variance due to non-shared environmental influences.

Genetic and Environmental Correlations

Phenotypic, genetic, and environmental correlations between PCL-C total score and the TFEQ-R18 total and subscale scores are presented in Table 3. The genetic correlation between PCL-C and TFEQ-R18 Total was $rg = .34$ (95% CI: .07; .58) indicating that the genetic factors associated with PTSD symptoms accounted for 12% of the genetic risk associated with eating behavior in general (this statistic was obtained by squaring the genetic correlation: .34 × .34 = .12 × 100% = 12%). Of the subscales, only the association between PCL-C and Uncontrolled Eating showed significant genetic correlation, $rg = .53$ (95% CI: .24; .84), suggesting that the genetic risk for PTSD symptoms accounted for 28% of the genetic risk in uncontrolled eating behaviors. Non-shared environmental correlations (*re*) were significant for PCL-C and TFEQ-R18 Total and Emotional Eating, suggesting moderate environmental overlap among these phenotypes. The non-significant genetic correlation between PCL-C and Emotional Eating, and the non-significant non-shared environmental correlation between PCL-C and Uncontrolled Eating trended towards significance, suggesting that being underpowered may have resulted in these effects being

trend-level and that these should be investigated in future studies. Overall, 51% of the phenotypic association between PCL-C and TFEQ-R18 Total and 69% of the association between PCL-C and Uncontrolled Eating were due to genetic effects, while 61% of the association between PCL-C and Emotional Eating was due to non-shared environmental influences.

Discussion

This is the first study to examine the genetic and environmental relationships of PTSD symptoms with maladaptive eating behaviors, including different forms of disinhibited eating and cognitive dietary restraint. Consistent with previous research (Bulik, Kleiman, & Yilmaz, 2016; Sheerin, Lind, Bountress, Nugent, & Amstadter, 2017), we found that PTSD symptoms and each of the eating behavior phenotypes were moderately heritable, even in this non-clinical sample of men and women. We found a robust phenotypic relationship between PTSD symptoms and eating behaviors in general, and specifically with uncontrolled eating and emotional eating behaviors but not with cognitive dietary restraint. Our findings also revealed genetic overlap between PTSD symptoms and maladaptive eating behavior in general and uncontrolled eating specifically.

Genetic correlations suggest that specific genetic variants that confer an increased risk for PTSD may also be involved in maladaptive eating behavior. The contribution of genetic influences to the phenotypic relationship between PTSD symptoms and uncontrolled eating as an instance of disinhibited eating is consistent with our hypothesis and extends the literature beyond phenotypic analyses to suggest common biological mechanisms may underlie the association between these two domains. Possible mechanisms include dysfunction in the brain reward circuit involved in emotion regulation, impulse control, and motivation, which has been implicated in PTSD (Fenster, Lebois, Ressler, & Suh, 2018). Changes in reward circuitry and associated dopaminergic systems have also been associated with uncontrolled or binge eating, and a handful of small genetic studies have identified potential genetic variants (e.g., *DAT1* dopamine transporter gene, *DRD2* dopamine receptor gene) linked with reward sensitivity in binge eating (Kessler, Hutson, Herman, & Potenza, 2016). Relatedly, research is emerging that trait disinhibition, the propensity to behave impulsively in several domains including eating behaviors, also explains recent engagement in risky/self-destructive behavior in people with high PTSD symptoms (Sadeh, Spielberg, & Hayes, 2018; Sadeh et al., 2015). As with other personality traits, genetic factors may contribute substantially to a disinhibited personality, and some studies have identified genes involved in dopaminergic and serotonergic neurotransmission in relation to trait disinhibition (Gray et al., 2018). Most recently, multivariate genomic structural equation modeling analyses have revealed several hundred unique variants associated with externalizing behavior (Linnér et al., 2020), laying the groundwork for further investigation into disinhibited behavior. Together with this prior research, our findings support the importance of continuing molecular genetics research to identify specific common genes related to these and other biological pathways that may facilitate the development of novel pharmacological therapeutics.

We found that our measure of cognitive dietary restraint was moderately heritable, but not phenotypically associated with PTSD symptoms. Previous heritability estimates for dietary restraint have varied, possibly due to variability in samples, and the use of various measures that capture slightly different constructs, with estimates ranging from 0–59% (Neale, Mazzeo, & Bulik, 2003; Thornton et al., 2011). Some have argued that the Restraint Scale (Polivy, Herman, & Howard, 1988) captures restrictive behaviors more often associated with pathological eating, whereas the restraint scale of the Dutch Eating Behavior Questionnaire (Van Strien, Frijters, Bergers, & Defares, 1986) and cognitive restraint subscale of the original TFEQ (Stunkard & Messick, 1985) focus on restraint behaviors like dieting which are adaptive for weight loss-seeking populations (Schur, Noonan, Polivy, Goldberg, & Buchwald, 2009). However, the TFEQ has undergone several iterations, and the cognitive dietary restraint subscale of the 18-item version no longer contains many of the original items that assessed dieting behavior, and previous efforts to establish heritability of the TFEQ (Neale et al., 2003) do not overlap with the TFEQ-R18. Our estimate of heritability for cognitive restraint, however, was similar to that reported in the only twin study that used the TFEQ-R18 (Keskitalo et al., 2008) which reported 26–63% heritability for cognitive restraint, 45–69% for uncontrolled eating, and 9–45% for emotional eating. Likewise, the only study examining TFEQ-R18 scores and PTSD symptoms (PCL-5) failed to detect an association with cognitive restraint but reported significant correlations with uncontrolled and emotional eating, suggesting that restraint and disinhibited eating may be differentially associated with PTSD (Mason, LeBouthillier, & Asmundson, 2019). Whether this lack of association is due to the quality of measurement by the scale or a true lack of relationship remains unclear. Additional research is needed to refine the construct validity of cognitive restraint and to clarify its relationship to PTSD symptoms.

The findings of a non-shared environmental contribution to the relationship between PTSD symptoms and maladaptive eating behavior have implications for the assessment and treatment of these symptoms. These non-shared, or person-specific environmental influences contribute to differences among siblings (Plomin & Daniels, 2011). Identifying types of psychosocial adversity potentially contributing to both PTSD and maladaptive eating behavior may be important to improving our understanding of this relationship. Influences may include exposure to trauma, illnesses, and accidents. Likewise, protective environmental factors, such as access to and quality of mental healthcare, socioeconomic factors, and social support, may lower the risk for both. Future research efforts may evaluate specific environmental influences associated with both domains.

Evidence for both genetic and environmental overlap between PTSD symptoms and maladaptive eating behavior supports the development of integrated treatments that use evidence-based approaches for co-occurring PTSD and ED conditions with prominent disinhibited eating behavior (Brewerton, 2019). Limited research suggests that concurrent cognitive behavior therapy for PTSD and ED may be effective (Trottier, Monson, Wonderlich, & Olmsted, 2017), and cognitive processing therapy improved both trauma and non-specific ED symptoms in a PTSD sample (i.e., impulse regulation, interoceptive awareness, interpersonal distrust, ineffectiveness, and maturity fears; Mitchell et al., 2012). Future research should evaluate the various treatment approaches with evidence for both PTSD and ED (e.g., cognitive behavioral therapy, dialectical behavioral therapy, exposure

therapies), and determine whether concurrent or sequential treatment is most effective. Frontline ED clinicians report that it is important to address PTSD symptoms in ED treatment and that concurrently treating these conditions may be most beneficial despite concerns about negative side effects (e.g., worsening of PTSD symptoms when ED behavior is reduced). PTSD treatment programs may also be enhanced by incorporating routine screening for disinhibited eating, and if indicated, an evaluation of ED. While pharmacological therapeutics may be developed to impact the shared genetic and biological mechanisms, novel integrated behavioral interventions can also be designed to address the environmental mechanisms that may link these conditions.

This study has several limitations. First, our findings are based on data from a relatively small sample of twins. Although our methods were optimized by a nearly equal ratio of MZ to DZ twin pairs and continuous rather than binary measures of the phenotypes (binary measures decrease statistical power) (Verhulst, 2017), research with larger samples is necessary to replicate our findings. Second, we used standardized, validated self-report measures of PTSD symptoms and eating behaviors that are primarily meant to be used as screening measures. Use of structured diagnostic interview assessments including, clinician-administered PTSD measure that requires endorsement of trauma exposure, would strengthen our conclusions and might extend these findings to diagnostic phenotypes of PTSD and ED. Our measure of eating behaviors (TFEQ-R18) does not have standard cut-offs, making it difficult to compare the prevalence of these eating behaviors in our sample to other populations. However, we were able to examine relationships between this measure and PCL-C in our sample, where the absolute scores and how they compare to other samples is not critical. Third, the use of a non-clinical set of twins with BMI over 18.5 potentially limits generalizability to clinical samples and highlights the need to extend this work to samples with more severe clinical pathology. Nonetheless, our community twin sample provides valuable information about the variability of these phenotypes in non-clinical populations. Fourth, we found and controlled for sex differences on some measures of maladaptive eating; however, we were unable to compare results across sex due to our sample size constraints. Examining the potential moderating effects of sex on heritability and the genetic and environmental correlations among PTSD, ED, and eating behaviors will illuminate the role of sex in the association between these phenotypes. Finally, because our data were cross-sectional, firm conclusions cannot be drawn about causation. Nonetheless, this study contributes to the current understanding of the potential mechanisms underlying the association between PTSD and selected maladaptive eating behaviors characteristic of ED.

In conclusion, we found genetic overlap between PTSD symptoms and uncontrolled eating. In contrast, we found environmental contributions to the association between PTSD symptoms and emotional eating, and no relationship between PTSD symptoms and cognitive dietary restraint. The genetic link between PTSD and uncontrolled eating highlights the need for additional biometric and molecular genetics research to further examine the shared risk factors and biological mechanisms responsible for the co-occurrence of PTSD, maladaptive eating, and ED symptoms. Additional studies should explore the interplay of genetic and environmental factors that protect against the development of PTSD symptoms, maladaptive eating behaviors, and ED. Research is also needed to further elucidate relationships between

PTSD and cognitive dietary restraint. Our findings support development of integrated PTSD and ED treatments that target shared biological mechanisms and common phenotypic presentations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Clinical Implications:

- There is genetic overlap between PTSD symptoms and uncontrolled eating behavior
- There is non-shared environmental overlap between PTSD symptoms and emotional eating
- Further investigation into underlying biological mechanisms shared by PTSD and eating disorder symptoms is warranted
- Identifying common environmental risk and protective factors shared by PTSD and eating disorder symptoms may facilitate treatment

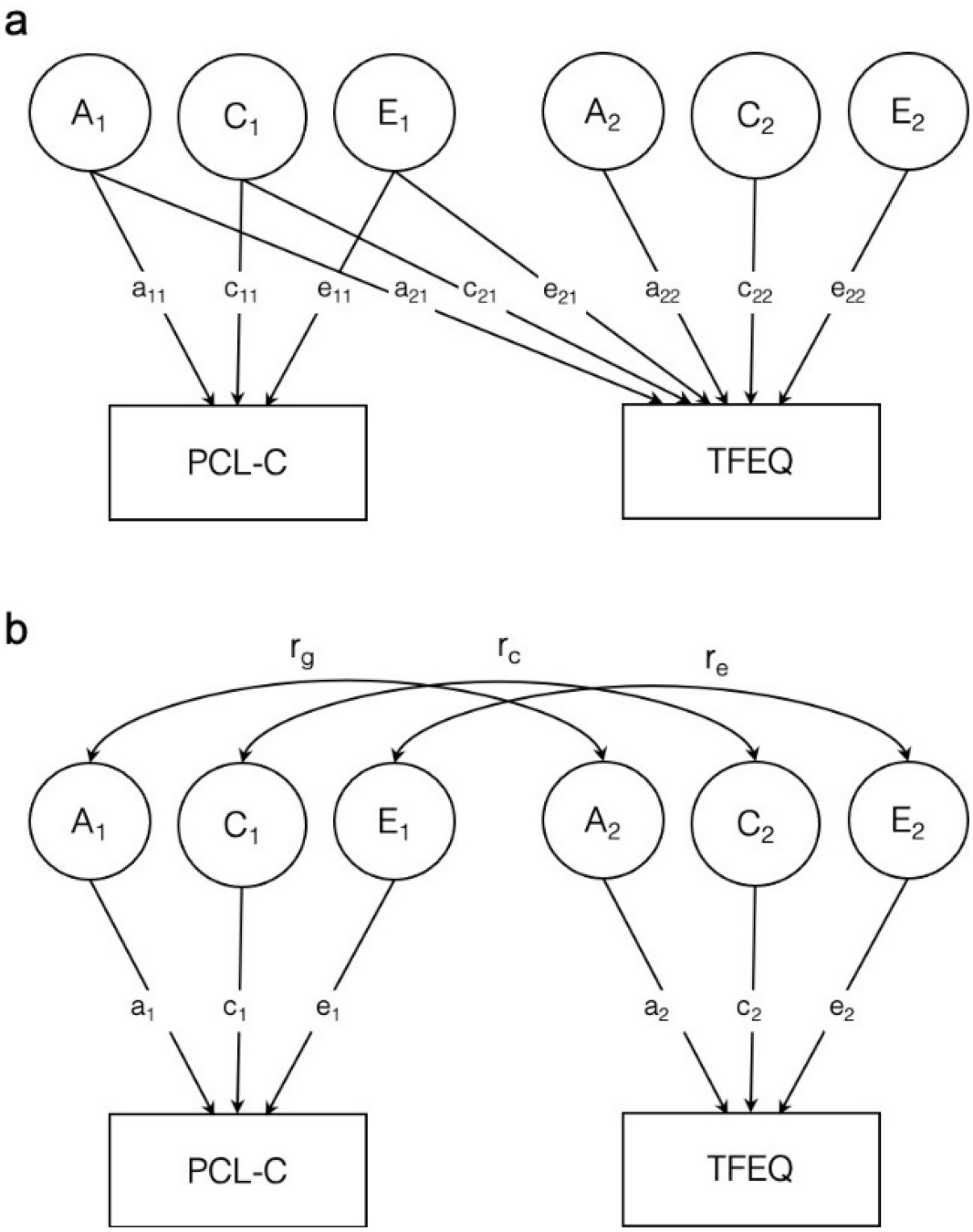

Figure 1.

Path diagram of the bivariate Cholesky decomposition (a) of PCL-C and TFEQ-R18 eating variables and the correlated factors model (b). For simplicity, only one twin of a pair is shown. Variance of each phenotype (in squares) is decomposed into A = additive genetic influences, C = shared environmental influences, E = non-shared environmental influences (in circles). Within the Cholesky decomposition (a), paths a_{11}, c_{11}, and e_{11} are influences on PCL-C, diagonal paths a_{21}, c_{21}, e_{21} are influences on the covariance between PCL-C and TFEQ-R18 eating variables, and paths a_{22}, c_{22}, e_{22} are influences unique to eating variables.

Parameter estimates from the Cholesky decomposition are used to construct other quantities of interest depicted in the correlated factors model (b), including the genetic correlation (r_g) and environmental correlations (r_c, r_e).

Table 1

Means and standard deviations for all study variables for the entire sample and by sex

Measure*	Men (*n* = 188) Mean (*SD*)	Women (*n* = 212) Mean (*SD*)	Total (*N* = 400) Mean (*SD*)	*p*
PCL-C (17–67)	26.26 (9.39)	25.71 (8.85)	25.97 (9.10)	.545
TFEQ Total (18–63)	33.24 (7.67)	37.10 (8.24)	35.28 (8.20)	< .001
Cognitive Restraint (6–23)	11.38 (4.09)	13.62 (3.83)	12.57 (4.10)	< .001
Uncontrolled Eating (9–34)	17.34 (5.28)	17.80 (5.00)	17.58 (5.13)	.375
Emotional Eating (3–12)	4.52 (1.92)	5.68 (2.48)	5.13 (2.31)	< .001

Note. PCL-C = PTSD Checklist – Civilian Version; TFEQ = Three Factor Eating Questionnaire-Reduced 18; Cognitive Restraint = TFEQ-R18 Cognitive Restraint subscale; Uncontrolled Eating = TFEQ-R18 Uncontrolled Eating subscale; Emotional Eating = TFEQ-R18 Emotional Eating subscale.

*Range of obtained scores in parentheses.

Table 2

Standardized variance components from the AE bivariate models

	Standardized Estimates (95% CI)	
Measure	a^2	e^2
PCL-C	.48 (.33; .61)	.52 (.39; .67)
TFEQ Total	.45 (.29; .58)	.55 (.42; .71)
Cognitive Restraint	.48 (.33; .60)	.52 (.40; .67)
Uncontrolled Eating	.34 (.17; .48)	.66 (.52; .83)
Emotional Eating	.34 (.16; .49)	.66 (.51; .84)

Note. PCL-C = PTSD Checklist – Civilian Version; TFEQ = Three Factor Eating Questionnaire-Reduced 18; Cognitive Restraint = TFEQ-R18 Cognitive Restraint subscale; Uncontrolled Eating = TFEQ-R18 Uncontrolled Eating subscale; Emotional Eating = TFEQ-R18 Emotional Eating subscale; CI = Confidence Interval; a^2 = additive genetic; e^2 = non-shared environment.

Table 3.

Phenotypic, additive genetic correlations (r_g), and non-shared environmental correlations (r_e) between PCL-C and TFEQ, along with 95% confidence intervals from AE models.

Measure	Phenotypic Correlation with PCL-C total score (95% CI)	Proportion of Phenotypic Correlation		r_g (95% CI)	r_e (95% CI)
		Genetic Effects	Environmental Effects		
TFEQ Total	**.32 (.25; .40)**	51%	49%	**.34 (.07; .58)**	**.29 (.11; .45)**
Cognitive Restraint	.08 (.00; .21)	_*	_*	_*	_*
Uncontrolled Eating	**.31 (.23; .39)**	69%	31%	**.53 (.24; .84)**	.16 (−.01; .33)
Emotional Eating	**.29 (.22; .34)**	39%	61%	.27 (−.05; .57)	**.30 (.12; .45)**

Note: PCL-C = PTSD Checklist – Civilian Version; TFEQ = Three Factor Eating Questionnaire-Reduced 18; Cognitive Restraint = TFEQ-R18 Cognitive Restraint subscale; Uncontrolled Eating = TFEQ-R18 Uncontrolled Eating subscale; Emotional Eating = TFEQ-R18 Emotional Eating subscale; CI = Confidence Interval; rg = genetic correlation; re = non-shared environmental correlation*

Not computed due to lack of phenotypic association. Significant correlations in bold.