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A Twin Study Examining Rumination as a Transdiagnostic Correlate of Psychopathology

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Abstract

This study examined the genetic and environmental influences on rumination and its associations with several forms of psychopathology in a sample of adult twins (N = 744). Rumination was significantly associated with major depressive disorder, depressive symptoms, generalized anxiety disorder, eating pathology, and substance dependence symptoms. There were distinct patterns of etiological overlap between rumination and each form of psychopathology; rumination had considerable genetic overlap with depression, modest genetic overlap with eating pathology, and almost no genetic overlap with substance dependence. Findings further suggest considerable overlap between genetic and environmental influences on rumination and those contributing to the covariance between forms of psychopathology. Results were specific to ruminative thought and

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did not extend to self-reflection. These findings support the conceptualization of rumination as a transdiagnostic correlate and risk factor for psychopathology and also suggest that the biological and environmental mechanisms linking rumination to psychopathology may differ depending on the disorder.

Keywords

rumination; depression; transdiagnostic; twin study; comorbidity

Rumination is a pattern of repetitive, self-directed thought, focused on symptoms of distress, potential causes of symptoms, and the implications of symptoms (Nolen-Hoeksema & Morrow, 1991). This thought pattern does not lead to effective action or problem-solving, but rather increases distress, perpetuates symptoms, and enhances functional impairment. The detrimental effects of rumination include increases in negative thinking (e.g., negative interpretations of events, self-criticism), poor problem-solving, inhibition of instrumental behavior, impaired concentration and cognition, increases in stressors, and decreases in social support (for a review, see Lyubomirsky & Tkach, 2004). Furthermore, rumination has been shown to increase risk for onset of depression, increase severity and duration of symptoms, and increase risk for depressive relapse (Nolen-Hoeksema et al, 2008). In addition to the robust association between rumination and depression, burgeoning evidence indicates that rumination also predicts the onset and course of other forms of psychopathology (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Ehring & Watkins, 2008; Nolen-Hoeksema & Watkins, 2011). A recent meta-analysis of studies examining emotion-regulation strategies and psychopathology in adults found significant associations between rumination and depression ($r = .55$; $k = 51$); anxiety disorders ($r = .42$; $k = 23$); eating disorders ($r = .26$; $k = 3$); and substance use disorders ($r = .21$; $k = 7$; Aldao et al., 2010). Additional evidence that ruminative thinking is associated with many psychiatric disorders (e.g., Nolen-Hoeksema & Watkins, 2011; for a review see Ehring & Watkins, 2008) suggests that rumination may play an important role across psychopathologies and contribute to the high rates of comorbidity among psychiatric diagnoses. From this perspective, researchers have suggested that rumination may be an important transdiagnostic factor, defined as an environmental, biological, or intrapersonal process that is linked distally or proximally to multiple forms of psychopathology (Nolen-Hoeksema & Watkins, 2011; Ehring & Watkins, 2008).

Importantly, not all self-focused, repetitive thought is associated with increased risk for depression and other negative mental health outcomes. Research supports the specificity of rumination as a risk factor for negative outcomes and suggests that other forms of thought, such as experiential self-focus and intellective self-consciousness (i.e., self-reflection), can have constructive consequences and can contribute to effective coping, adaptive preparation, and psychological well-being (e.g., Trapnell & Campbell, 1999; Watkins, 2008; Watkins & Teasdale, 2004). Researchers have suggested that distinguishing between rumination and other forms of self-focused, repetitive thought remains an important consideration in studies investigating vulnerabilities for psychopathology (Watkins & Teasdale, 2004) given the

range in functional outcomes associated with different types of self-focused thought (e.g., Sanders & Lam, 2010; Watkins & Moulds, 2005).

To date, limited research has examined the genetic influences on rumination and the extent to which rumination and different forms of psychopathology have overlapping genetic influences. Several studies have investigated the link between rumination and specific genetic polymorphisms, but with conflicting results. Specifically, three studies have evaluated the association between rumination and the Val66Met polymorphism of the BDNF gene (Beevers, Wells & McGeary, 2009; Hilt, Sander, Nolen-Hoeksema & Simen, 2007; Juhasz et al., 2011) based on evidence that BNDF is implicated in neuroplasticity pathways and stress reactivity (for a review, see Castrén & Rantamaki, 2010). Results of these three studies show some convergence regarding a potential link between BDNF and rumination, but yield inconsistent findings that mirror patterns in the candidate gene literature on psychiatric phenotypes in general (Duncan & Keller, 2011). Thus, these results should be interpreted with caution.

Three twin studies have examined the heritability of rumination and the extent to which genetic and environmental influences contribute to its association with depression. Moore et al. (2013) examined 12- to 14-year-old twins and found that rumination and depressive symptoms were both heritable (h^2 = .17 and .54, respectively), and that their association was largely genetic (genetic correlation [rA] = .83). These findings were supported by a study of Chinese twins aged 11-17 years (Chen & Li, 2013), which found modest heritability for rumination (h^2 = .24) and substantial genetic overlap between rumination and depressive symptoms (rA = .99). A recent study from our group (Johnson et al., 2014) yielded similar results using multiple measures of rumination (h^2 = .37 – .41) and similar findings for depressive symptoms (rA = .71 – .77) and major depressive disorder (MDD; rA = .68) in young adults (ages 21 – 26). In concert, these studies suggest that rumination is a heritable construct in adolescence and young adulthood. They also indicate a statistically significant and robust genetic correlation (and a moderate nonshared environmental correlation) between rumination and depression, suggesting considerable etiological overlap between these constructs.

No research to date has examined genetic and environmental influences on rumination with forms of psychopathology other than depression. This question is of critical importance given emerging evidence suggesting that rumination is associated with a range of psychopathologies. Furthermore, there is evidence of strong genetic correlations between depression and other forms of psychopathology, including anxiety disorders (Hettema, 2008), eating disorders (Wade et al., 2000), and substance use disorders (SUDs) (Olvera, Bearden, Velligan, et al., 2011), which may be explained by an underlying genetically influenced vulnerability, such as rumination, that contributes to the high rates of comorbidity among psychiatric disorders.

This study examined rumination and self-reflection as potential transdiagnostic correlates of psychopathology in early adulthood and is the first study to investigate the role of genetic and environmental influences on the associations between rumination and different forms of psychopathology. We hypothesized that rumination, but not self-reflection, would be

robustly associated with symptoms and diagnoses of MDD, generalized anxiety disorder, eating pathology, and substance dependence symptoms. The twin design enabled us to examine the magnitude of the variance in the study variables explained by genetic and environmental influences. We hypothesized that there would be overlap in the genetic and environmental influences on rumination and self-reflection, but that self-reflection would also have genetic and environmental influences not shared with rumination. Finally, we hypothesized that rumination, but not self-reflection, would share common sources of genetic variance and environmental variance with forms of psychopathology (i.e., we hypothesized there would be genetic covariance and environmental covariance among these constructs).

Method

Study participants

Analyses were conducted on data from 744 participants enrolled in the Longitudinal Twin Study (LTS) who also participated in the Executive Function and Self Regulation (EFSR) and Center on Antisocial Drug Dependence (CADD) studies. The LTS consists of same-sex twin pairs recruited through the Colorado Department of Health born between 1984 and 1990 in Colorado. Of the parents initially contacted, more than 50% of the families who lived within a 2-hour drive of Boulder, Colorado enrolled in the study. Most of the sample (92.6%) identified as White, and the remaining individuals identified as either “more than one race” (5.0%), American Indian/Alaskan Native (<1%), Pacific Islander (<1%), or did not report their race (1.2%). Individuals who identified as Hispanic comprised 9.1% of the sample. For additional information on the sample, see Rhea et al. (2006; 2013). Data from 386 families were analyzed in the current study, including 170 male twin pairs (87 monozygotic [MZ]; 83 dizygotic [DZ]), 14 male singletons, 195 female twin pairs (107 MZ; 88 DZ), and 7 female singletons.

Zygosity determination—Zygosity was determined using in-person ratings from raters on 10 physical characteristics across time. Pairs were considered unambiguously MZ or DZ if 85% of the raters agreed on their zygosity. These ratings were later confirmed using 11 polymorphic microsatellite markers.

Procedures

Data collection—Self-report measures of rumination, self-reflection, and depressive symptoms were collected in the EFSR study when twin pairs were between the ages of 21 and 28 (M = 22.84, SD = 1.29). Contemporaneously, as part of assessments for the CADD study, diagnostic information regarding past year and lifetime endorsement of psychiatric disorders were assessed in twins using structured diagnostic interviews. A self-report questionnaire of eating pathology symptoms was also collected at this assessment. On average, the EFSR study measures (rumination, self-reflection and depressive symptoms) were completed within 14 days of the CADD study measures (diagnostic interview and eating pathology questionnaire).

Measures

Rumination and self-reflection—Two measures of rumination were collected. The 10-item version of the 22-item Ruminative Response Scale (RRS; Nolen-Hoeksema & Morrow, 1991) was developed by Treynor and colleagues (2003) by eliminating RRS items overlapping substantially with items on depression inventories and factor analyzing the remaining 10 items to obtain two factors: brooding (RRS-B) and reflection (RRS-R). Brooding represents passive, perseverative, maladaptive, self-focused thought, whereas reflection represents less maladaptive self-reflective strategies. Items on both subscales assess a respondent's general tendency to engage in these types of thinking/behaviors when he or she is feeling depressed. Brooding and reflection are positively associated with each other and with concurrent depression; however, brooding is a stronger predictor of depression and other negative psychosocial outcomes (Nolen-Hoeksema et al., 2008; Treynor et al., 2003). Thus, these two subscales represent variations of the same construct, rather than orthogonal forms of self-focused thought (e.g., Siegle et al., 2004). Both scales have been shown to have adequate psychometric properties in previous studies (RRS-B, α = .74 – .77; RRS-R, α = .66 – .72; Siegle et al., 2004; Treynor et al., 2003). The reliabilities of these measures were comparable in our sample (see Table 1).

Second, the Rumination-Reflection Questionnaire (RRQ; Trapnell & Campbell, 1999) is a 24-item assessment that reliably (α> .90) measures two types of self-focused thought: rumination and reflection. Rumination (RRQ-RU), or "self-attentiveness motivated by perceived threat, losses or injustices to the self," (Trapnell & Campbell, 1999, p. 297) is strongly associated with neuroticism (Trapnell & Campbell, 1999), depressive symptoms, and the RRS subscales (Siegle et al., 2004). Conversely, reflection (RRQ-RE) is conceptualized as "self-attentiveness motivated by curiosity and interest in the self," (Trapnell & Campbell, 1999, p. 297) and has been found to be strongly associated with personality constructs of openness to experience and motivation. Items on the RRQ assess a respondent's general tendency to engage in self-focused thought. The reliabilities of these measures were comparable in our sample (see Table 1).

It is important to note that the RRQ-RE measures self-focused thought that is based on self-awareness and curiosity and is not necessarily a reaction to distress. Furthermore, studies show that the RRQ-RE is distinct from measures of rumination, yielding only very modest associations with rumination measures and depression measures (e.g., Siegle et al., 2004). Given these distinctions, we used the RRQ-RE subscale as a measure of "self-reflection" or adaptive/benign self-focused thought.

Depressive symptoms—The Center for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977) is a frequently used 20-item scale for measuring depressive symptoms that was developed by the National Institute of Mental Health and has strong psychometric properties in this sample (α = .90; see Table 1). Respondents were asked about the frequency with which they experienced depressive symptoms in the past week; their total score was used.

Eating pathology—Eating pathology was assessed with the total score from the Eating Disorder Examination Questionnaire (EDEQ; Fairburn & Beglin, 1994), which assesses restraint (e.g., avoidance of food), eating concern (e.g., preoccupation with food), shape concern (e.g., importance of body shape), and weight concern (e.g., importance of weight) in the past 28 days. This measure has shown good psychometric properties in prior studies (e.g., Berg et al., 2012) and did so our sample (see Table 1).

Psychiatric diagnoses—Two diagnostic interviews were used to assess psychiatric diagnoses. First, the Diagnostic Interview Schedule for the DSM-IV (DIS-IV; Robins et al., 2000) is a structured interview designed to diagnose in a reliable and valid fashion the major psychiatric disorders according to the DSM-IV. The current study analyzed lifetime diagnoses of MDD and Generalized Anxiety Disorder (GAD), where individuals were coded as endorsing no symptoms (0), some symptoms, but not enough to meet criteria for diagnosis (1), or meeting criteria for a diagnosis (2). The psychometric properties of the DIS have been studied extensively and diagnosis of MDD and GAD have yielded good inter-rater reliability in other samples (for a review, see Compton & Cottler, 2004).

Second, the Composite International Diagnostic Interview – Substance Abuse Module (CIDI-SAM; Robins et al., 1990) is a self-report structured interview that assesses symptoms and diagnoses of abuse and dependence for tobacco, alcohol, and eight classes of illicit drugs in a reliable and valid fashion (Compton et al., 1996; Cottler, Robins & Helzer, 1989). We examined dependence vulnerability (DV), as research suggests it represents a clinically valid, familial, and heritable construct (e.g., Button et al., 2006). This index was derived by taking a total count of lifetime dependence criteria endorsed across all classes of substances, then dividing the total count by the number of substances used. Those who had never used any substance more than five times were assigned a DV score of zero. DV scores were corrected for gender and age using standard regression procedures.

Analyses

General analysis procedures—All analyses were conducted on raw data and allowed missing data. Structural equation models, both phenotypic and genetic, were implemented using Mplus 7 (Muthén & Muthén, 2013). Analyses adjusted for non-independence using the TYPE=COMPLEX option, which provides adjusted standard errors and model fit statistics based on the maximum likelihood estimation with robust standard errors (MLR) estimator. When analyses included both continuous and ordinal variables, the weighted least square mean and variance (WLSMV) estimation method was used. Statistical significance of the parameters was determined by *p*-values for *z*-tests based on ratios of parameters/standard errors and verified by χ^2 difference tests (scaled for non-independence when appropriate; Satorra & Bentler, 2001). Given that the χ^2 is sensitive to sample size, additional fit indices were assessed, including the Tucker-Lewis index (TLI; Bentler, 1990) and the root mean square error of approximation (RMSEA; Browne & Cudeck, 1987). A TLI >.95 and RMSEA <.06 indicate good model fit (Hu & Bentler, 1998).

To test the hypothesis that rumination is a transdiagnostic correlate of psychopathology, and that self-reflection is not, structural equation models were conducted to examine

associations between each construct (independent of the other) and psychopathology. In all models, a residual correlation between RRS-R and RRQ-RE was included. This was statistically motivated to improve model fit and also aligns with theoretical considerations of the RRS-R as a measure of reflection – a type of rumination that may be less maladaptive than other types and perhaps related to adaptive self-reflection.

Twin models—The twin method was used to examine the pattern and magnitude of genetic and environmental influences on the study measures. This method is based on the fact that MZ twins share 100% of their genes whereas DZ twins share 50% of their genes identical by descent on average, and both types of twins are reared together (i.e., have shared environmental influences). When correlations of a measure are greater within MZ twin pairs than within DZ twin pairs (rMZ > rDZ), there is evidence of genetic influences on the phenotype. If rMZ is greater than twice rDZ, this suggests the influence of non-additive genetic effects, whereas if rMZ is less than twice rDZ, there is evidence of shared environmental effects on the phenotype. When the rMZ is <1.0, nonshared environmental effects are indicated. These interpretations of patterns of twin correlations can also be applied to the cross-twin correlations between phenotypes.

After examining twin correlations, univariate models were conducted to estimate the magnitude of additive genetic (A), shared environmental (C), non-additive genetic (D), and nonshared environmental (E) influences on a phenotype. Nonshared environmental influences also include measurement error. A limitation of the traditional twin design is that C and D cannot be estimated in the same model. Therefore, in the present study, the pattern of twin correlations was used to decide whether an ACE or ADE model was most appropriate for these data. For example, if rMZ was greater than rDZ (e.g., rMZ = 0.6, rDZ = 0.4), then an ACE model was fit to the data, as this correlation pattern suggests a role for additive genetic influences (A), shared environmental influences (C), and nonshared environmental influences (E).

Next, a multivariate Cholesky decomposition including measures of rumination, self-reflection, and psychopathology (e.g., depression) was used to estimate genetic influences that are shared in common by all variables, those shared in common by self-reflection and depression after controlling for rumination, and those genetic influences that are unique to depression. The covariances between these constructs due to A, C or D, and E were also estimated.

The results from the Cholesky decomposition include the percentage of the genetic and environmental variance of psychopathology shared in common with rumination and those specific to psychopathology. They also provide the percentage of variance of psychopathology and the covariance between rumination and psychopathology that is due to genetic, shared environmental, and nonshared environmental influences. We used a series of multivariate Cholesky decompositions to examine overlapping and unique genetic and environmental influences on rumination, self-reflection, and their associations with multiple forms of psychopathology. We examined gender differences in these models by comparing the fit of models with separate parameters for each gender to models with parameters fixed to be equal across gender.

Results

Descriptive Statistics

Means and standard deviations, reliability, and sample sizes for continuous measures are presented in Table 1. Rumination measures and the self-reflection measure were normally distributed, with acceptable skewness and kurtosis values (between 1.00 and -1.00). Distributions of depressive symptoms (CES-D), eating pathology symptoms (EDEQ), and substance dependence vulnerability (DV) were skewed; thus, scores were log-transformed to better approximate a normal distribution. Lifetime prevalence of MDD symptoms (11.6%) and diagnosis (12.5%), as well as GAD symptoms (5.3%) and diagnosis (3.3%), were slightly lower than prevalence of diagnosis reported in large, population-based young adult samples (e.g., 15.4% for MDD, 4.1% for GAD, by Kessler et al., 2005; 14.1% for MDD by Reichborn-Kjennerud et al., 2010). Compared to women, men endorsed fewer rumination and eating pathology symptoms and were less likely to endorse symptoms and diagnoses of MDD ($\chi^2[2] = 9.48$, $p = .01$) and GAD ($\chi^2[2] = 15.78$, $p < .01$).

Phenotypic Associations among Rumination, Self-Reflection, and Psychopathology

The three measures of rumination were moderately to highly correlated with each other ($r = .44 – .70$) and also significantly associated with all measures of psychopathology ($r = .13 – .51$). The self-reflection measure (RRQ-RE) was significantly correlated with measures of rumination; however, these associations were modest in magnitude for RRS-B and RRQ-RU ($r = .11 – .19$) and moderate for RRS-R ($r = .34 – .42$). The correlations all had p values below .01 except the correlation between RRS-R and EDEQ in men ($p = .03$). Additionally, RRQ-RE was significantly associated with some psychopathology measures (MDD, GAD, DV; $r = .11$ - $.32$; p values from <.001 to .04), but these associations were smaller in magnitude than those between rumination measures and psychopathology.

We used the three rumination measures (RRQ-RU, RRS-B, RRS-R) as indicators of a rumination latent variable (RLV) given the considerable overlap between them. This model was just identified (zero degrees of freedom), so there was no test of overall model fit. Each indicator loaded significantly on the latent factor (factor loadings can be found in Supplemental Material 3). There were significant, albeit small, gender differences in the factor loadings ($\chi^2_{diff}[3] = 12.23$, $p = .01$), so subsequent analyses were conducted allowing the RLV factor loadings to differ between men and women. This decision was supported by the significant gender difference in rumination found in the literature (see Johnson & Whisman, 2013, for a meta-analysis).

Structural equation models predicting psychopathology with correlated RLV and RRQ-RE variables indicated that RLV was significantly associated with all five measures of psychopathology when controlling for RRQ-RE: MDD (standardized $b = .36$ [men], .42 [women]), CES-D ($b = .60$ [men], .58 [women]), GAD ($b = .43$ [men], .38 [women]), EDEQ ($b = .24$ [men], .37 [women]), and DV ($b = .22$ [men], .20 [women]). Conversely, only associations between RRQ-RE and MDD ($b = .21$[men], .15[women]) and GAD (for women; $b = .25$) remained significant when controlling for RLV. These results suggest that associations between rumination and psychopathology are independent of self-reflection,

whereas associations between self-reflection and psychopathology are largely accounted for by rumination. Only one gender difference was significant, which was for the association between EDEQ and RLV.

Genetic and Environmental Influences on Rumination, Self-Reflection, and Psychopathology

Given evidence that rumination is associated phenotypically with several forms of psychopathology, we next examined our hypothesis that these associations would be explained by both genetic and environmental overlap between rumination and psychopathology. Twin correlations suggested significant genetic influences on all constructs, with MZ twin correlations greater than DZ twin correlations (presented separately for men and women in Supplemental Material 1). One exception was RRQ-RE in women, for which the MZ and DZ correlations were similar, suggesting that shared environmental influences may play a substantial role for this construct in women. There were too few individuals with symptoms and diagnoses of GAD to examine separate MZ and DZ groups; therefore, GAD was not included in the genetic analyses.

Supplemental Material 1 presents cross-twin correlations for men and women separately to illustrate that, qualitatively, the patterns of correlations differed between men and women for some variables. However, these gender differences were not statistically significant, so our decisions to apply ACE or ADE models to the data for each variable were made based on the pattern of MZ/DZ correlations in the full sample (i.e., twin correlations fixed to be equal for men and women).[1] In the full the sample, MZ twin correlations for measures of psychopathology (r = .35 – .68) and self-focused thought (r = .28 – .43) were substantially larger than DZ twin correlations for these measures (r = .12 – .26 and r = .08 – .26), suggesting a role for genetic influences.

The majority of within-trait twin correlations suggested shared environmental influences and thus the use of ACE univariate models for most phenotypes. There appeared to be evidence of non-additive genetic influences (D) for CES-D in men and EDEQ in men and women. However, when taking into account cross-trait twin correlations to inform the multivariate modeling, there was a clear pattern suggesting effects of C on the phenotypes and only limited evidence for the effects of D.[2] Further examination indicated the influences of D were nonsignificant in multivariate models. Thus, for the purposes of clarity and succinctness in presenting the results, we focus our discussion on ACE univariate and multivariate models and do not further discuss ADE models.

Univariate ACE models were compared to more parsimonious univariate models that constrained all non-significant parameter estimates of C to zero (models labeled “1” and “2”, respectively, in Supplemental Material 2). The reduced model did not fit significantly worse

[1]The decision to examine twin correlations in the full sample (i.e., both genders) did not preclude us from examining gender differences in the genetic models. By comparing the fit of models with parameters free to differ between men and women to models with parameters fixed to be equal for men and women, we were able to examine gender differences in subsequent analyses.
[2]Univariate twin models derived from within-trait twin correlations are underpowered to detect shared environmental (C) influences and therefore, C may be undetected or underestimated. However, the cross-trait correlations in multivariate models provide additional pieces of information to the model, thus increasing power to detect the influence of C on individual phenotypes and the associations between them.

than the ACE model for any variable (all $\Delta\chi^2(2) < 4.10$, $p > .13$), and all reduced models fit the data well. These results suggest that C paths could be dropped in all models. However, they do not imply that C influences are absent, but instead, may reflect the low power of the twin design to distinguish them from A influences (Martin, Eaves, Kearsey, & Davies, 1978). Research suggests that disregarding nonsignificant estimates of the shared environment (C) can lead to estimates of A that are biased upwards, thus overemphasizing the role of genetic influences on certain phenotypes (e.g. Burt, 2014; 2009; Keller, Medland & Duncan, 2010). Therefore, the focus in our results and discussion is primarily on ACE models rather than the AE models, even when the C influences were not statistically significant.

The RLV was heritable for men ($h^2 = .40$) and women ($h^2 = .34$) and was also influenced substantially by nonshared environmental factors, as presented in Supplemental Material 2. There were no significant gender differences in the etiological influences on the latent variable itself; however, the measure-specific influences on RRS-R did differ between men and women, with higher genetic influences on RRS-R for men than for women. Estimates of genetic and environmental influences on RRQ-RE were similar in magnitude to those for RLV, although there was evidence of shared environmental influences for women. Measures of psychopathology showed modest to moderate genetic influences and modest influences of the shared environment, with nonshared environmental influences explaining the majority of variance in each construct. For example, shared environmental influences were modest but not negligible in magnitude in the ACE models for DV, explaining 28% of the variance in DV in men and 13% of the variance in women. Modest C estimates were also found for MDD in both groups and CES-D and RRQ-RE in women. We found no significant gender differences in the genetic and environmental influences on any form of psychopathology in the univariate ACE models.

With evidence that RLV, RRQ-RE, and psychopathology are heritable, we next examined overlapping genetic and environmental influences among RLV, RRQ-RE, and each measure of psychopathology in separate Cholesky decompositions. Supplemental Material 4 presents figures including the parameter estimates from trivariate analyses examining RLV, RRQ-RE, and psychopathology (i.e., MDD, CES-D, and DV). Table 2 presents the A, C, and E influences on the variance of each form of psychopathology that are shared in common with rumination and self-reflection and those that are unique to psychopathology. Table 2 also presents the covariances between psychopathology and rumination/self-reflection due to A, C, and E, which were calculated using path-tracing based on values found in Supplemental Material 4.[3]

A different model was used to examine the hypotheses for the EDEQ because the genetic models including the EDEQ and the RLV fit poorly. This poor model fit was likely due to the fact that in female MZ twins, the correlations between EDEQ and RLV indicators varied widely— $r_{EDEQ\text{-}RRS\text{-}R}$ ($r = .19$) compared to $r_{EDEQ\text{-}RRS\text{-}B}$ ($r = .40$) and $r_{EDEQ\text{-}RRQ\text{-}RU}$ ($r = .$

[3]For example, the first value in Table 2, the estimate of genetic variance (A) in CES-D shared in common with RLV and RRQ-RE (.29), is calculated by the squaring the path estimate from A1 to CES-D (.54 * .54 = .29) from Supplemental Material 4: Panel A. The estimate of genetic covariance between CES-D and RLV (.31) is calculated by multiplying the path from A_1 to RLV and the path from A_1 to CES-D (.57 * .54 = .31) in Supplemental Material 4: Panel A.

51) — suggesting that the use of the RLV was not appropriate for this analysis. Thus, a multivariate Cholesky decomposition including the three RLV indicators individually and the EDEQ was used (see Supplemental Material 5 for figures and parameter estimates). The RRQ-RE was not included because there was little phenotypic association between EDEQ and RRQ-RE. Table 3 presents the variance of EDEQ shared in common with rumination and self-reflection and that unique to EDEQ. It also presents the covariances between EDEQ and RLV/RRQ-RE due to A, C, and E, which were calculated using path-tracing rules.

The only multivariate model with significant gender differences was the model including rumination measures and EDEQ, $\chi^2_{diff}(30) = 60.06$, $p < .01$; thus, these results are discussed separately for men and women. For the MDD trivariate models examining gender differences, there were too few individuals in each group to have the power to detect significant genetic and shared environmental influences. Therefore, subsequent analyses for MDD combined men and women and included gender as a covariate. For CES-D and DV, results from the models with parameters fixed across gender are discussed (i.e., models that assume there are no significant gender differences).

Rumination, self-reflection, and depression symptoms (CES-D) and diagnosis (MDD)

A bivariate examination of rumination and depression using a subgroup of this sample is reported elsewhere (Johnson et al., 2014). Results of the current analysis using this larger sample were consistent with those from the previous study. Results suggested considerable genetic overlap between rumination and both CES-D and MDD and that the majority of genetic influences on RRQ-RE were separate from those influencing depression and rumination.

Trivariate analyses examining the rumination latent variable (RLV), self-reflection (RRQ-RE), and CES-D suggested that all genetic influences on CES-D were shared in common with RLV and RRQ-RE, but only 9% was shared exclusively with RRQ-RE; 25% of nonshared environmental influences on CES-D was shared in common with both RLV and RRQ-RE, and 75% was unique to CES-D (Table 2). Trivariate analyses examining RLV, RRQ-RE, and MDD yielded similar results. However, there was evidence of modest shared environmental influences unique to MDD, explaining approximately 17% of the variance. For both CES-D and MDD, the phenotypic covariance between depression and RLV was explained by approximately equal parts genetic (~50%) and nonshared environmental (~50%) influences.

Rumination, self-reflection, and vulnerability to substance dependence (DV)

The pattern of results for DV differed from those for depression. All of the genetic influences on DV were shared exclusively with RRQ-RE (i.e., not shared in common with RLV), and most of the nonshared environmental influences (93%) were unique to DV. There were modest shared environmental influences on DV, which were explained entirely by influences shared in common with RLV (78%) and RRQ-RE (22%). Furthermore, results suggested that the covariance between RLV and DV was due only to environmental influences, with 30% and 70% of the covariance due to shared and nonshared environmental influences, respectively.

Rumination, self-reflection, and eating pathology (EDEQ)

For men, the majority of etiological influences on EDEQ were not shared with the rumination measures, with only 12% of genetic influences and 14% of environmental influences on EDEQ overlapping with any of the rumination measures (see Table 3). The phenotypic associations between EDEQ and RRS-B and RRS-R (Ruminative Response Scale, Brooding, and Reflection subscales) were primarily explained by overlapping nonshared environmental influences, whereas the association between RRQ-RU (Rumination-Reflection Questionnaire – Rumination) and EDEQ was due to genetic (62%) and environmental (38%) influences. For men, there was no evidence of shared environmental influences on EDEQ or the covariance between EDEQ and rumination measures.

Results for women indicated that 10% of genetic influences on EDEQ were shared in common with rumination measures. Though shared environmental influences on EDEQ were only modest in magnitude (7%), they overlapped entirely with rumination measures. In contrast, most of the nonshared environmental influences on EDEQ were unique to it. Despite the modest magnitude of genetic and shared environmental influences overlapping between EDEQ and rumination, genetic influences explained up to 69% and shared environmental influences explained up to 23% of the covariance between EDEQ and the rumination measures.

Rumination and covariance between disorders

Our results indicated significant correlations between rumination and all forms of psychopathology, and modest to moderate correlations between forms of psychopathology. Furthermore, there was modest to moderate genetic overlap between rumination and individual disorders. These results, in conjunction with evidence of strong genetic correlations between depression and other forms of psychopathology (e.g., Braun et al., 1994, Hettema, 2008, Olvera et al., 2011), led us to question whether genetic influences on rumination contribute to the genetic correlations between different forms of psychopathology. We conducted post hoc analyses to estimate the extent to which genetic and environmental influences on the covariance between forms of psychopathology overlapped with the genetic and environmental influences on rumination. Trivariate Cholesky decompositions including rumination followed by two forms of psychopathology (e.g., RLV, CES-D, and DV in one model) were used to examine this hypothesis (Table 4). Similar to previous analyses, models including the EDEQ allowed the parameter estimates to differ between men and women to examine gender differences, whereas models that did not include the EDEQ did not allow estimates to differ between men and women (i.e. gender differences were not examined).

Results showed that slightly more than half of the covariance between CES-D and DV (r = . 24) was due to genetic influences. Of these genetic influences, 40% was shared in common with genetic influences on RLV and 60% was unique, or not shared with genetic influences on RLV (i.e., first and second row of “CES-D with DV Covariance” section in Table 4). This finding suggests that a substantial portion of the genetic correlation between CES-D and DV was explained by genetic influences on RLV. Additionally, 73% of the nonshared

environmental influences on this association was shared with environmental influences on RLV. There was no evidence that shared environmental factors contributed to the covariance between disorders.

The models including EDEQ examined the three rumination measures separately and included separate parameters for men and women. (Table 4 shows the covariance shared in common with any measure of rumination.) With respect to the CES-D-EDEQ association in men (r = .23), 0% of the genetic influences on this correlation was shared with rumination measures (i.e., 100% was unique to psychopathology). In contrast, 100% of genetic influences on this phenotypic association in women (r = .28) overlapped with rumination measures. Men and women showed similar patterns with regard to nonshared environmental influences on the CES-D-EDEQ association, with ~50% of the nonshared environmental influences on this correlation overlapping with nonshared environmental influences on rumination. Shared environmental influences did not contribute to the covariance between EDEQ and CES-D for men or women.

EDEQ and DV were modestly correlated in women (r = .16) and the entirety of genetic influences on this association was common with rumination measures, whereas nonshared environmental influences were primarily unique to the EDEQ-DV association in women (82%). For men, EDEQ and DV were not significantly associated.

Discussion

Results from this study have several important implications for understanding rumination as a transdiagnostic correlate and risk factor for psychopathology. First, rumination was associated with multiple psychopathologies in a young adult sample of twins, including MDD, GAD, substance dependence symptoms, and eating pathology, suggesting the maladaptive effects of rumination may transcend the boundaries of disorders or syndromes. Second, the genetic and environmental influences on these associations differed by phenotype, indicating that there may be differential etiological pathways linking rumination to forms of psychopathology. Whereas the link between rumination and depression was largely explained by overlapping genetic influences on these constructs, results suggested a more significant role of environmental influences in the associations between rumination and other disorders (e.g., eating pathology, substance abuse). Third, genetic and environmental influences on rumination contributed to the covariance among the different psychopathologies. This finding provides initial behavior genetic support for theoretical frameworks that emphasize the role of rumination in psychiatric comorbidity and co-occurrence (e.g. Nolen-Hoeksema & Watkins, 2011). Fourth, self-reflection was associated with fewer phenotypes and to a lesser extent than rumination and shared little or no etiological influences with psychopathology, suggesting that the association between self-focused thought and psychopathology is specific to rumination.

Rumination as a Transdiagnostic Risk Factor

Our results strongly support the idea that rumination is associated with several forms of psychopathology and may serve as a transdiagnostic risk factor for psychopathology (Nolen-Hoeksema & Watkins, 2011). Phenotypic results suggested that rumination was positively

associated with self-report symptom measures of depression and eating pathology, and interview-based symptoms and diagnoses of MDD, GAD, and SUDs.

These results are strengthened by several methodological aspects of the current study. First, we conducted our analyses with a latent variable of rumination, including three commonly used measures of rumination (i.e., RRS-B, RRS-R, RRQ-RU) as indicators. Thus, the pattern of results increases confidence that these results extend beyond a specific measure of rumination. Second, psychopathology was measured by self-report and structured clinical interview where available, suggesting these results may hold for both continuous measures of symptoms and clinical diagnoses. Third, the majority of research examining rumination as a transdiagnostic risk factor has investigated specific disorders individually, requiring cross-disorder comparisons to be made across samples, study design, and measures. Our sample and methodological approach enabled us to circumvent this limitation.

Our results also extend the current literature by suggesting there is specificity in the association of self-focused thought and psychopathology. Self-reflection, a form of self-focused, repetitive thought that is considered less maladaptive than rumination, did not show the same pattern of associations with psychopathology that was found for rumination. In general, self-reflection was not associated or only modestly associated with psychopathology, after controlling for the effects of rumination. This is an important finding in that it guides efforts to identify specific forms of self-focused thought that are maladaptive and increase risk for psychopathology. Our results support the conceptualization of rumination as a pattern of repetitive, self-directed thought that is a unique and specific risk factor for several forms of pathology.

Shared Genetic and Environmental Influences on Rumination, Self-Reflection and Psychopathology

Depression—Our results largely replicated those from other recent studies (Chen & Li, 2013; Moore et al., 2013) and were similar to those from our previous study examining genetic and environmental influences on rumination and depression in a subgroup of the current sample (Johnson et al., 2014). Rumination was found to be moderately heritable and the majority of genetic influences on depression overlapped with rumination. This general pattern of results did not depend on the measure of depression (although the proportion of overlapping genetic influences was higher for depressive symptoms than MDD diagnosis), suggesting it is largely consistent across dimensional and categorical conceptualizations of depression. Results for nonshared environmental influences showed a similar pattern, although the overlap of influences was smaller than that for genetic influences. The covariances between depression measures and rumination were explained by approximately equal parts genetic (~50%) and nonshared environmental (~50%) influences. Self-reflection was shown to have moderate genetic and environmental influences, but it shared little or no etiological influences with depression after controlling for those in common with rumination. This differentiation between rumination and self-reflection is supported by studies suggesting different neural mechanisms behind these two forms of self-focused thought (e.g., Hamilton et al., 2011) and further affirms rumination’s unique role as a correlate of and risk factor for psychopathology.

The substantial genetic overlap between rumination and depression suggests that rumination may serve as a cognitive mediator between genetic risk for depression and the onset and course of depression. This interpretation is consistent with a recent theoretical model of psychopathology risk (Nolen-Hoeksema & Watkins, 2011), which posits that genetic susceptibility acts as a distal risk factor for depression, "setting the stage" for rumination (a proximal risk factor), which in turn increases risk for onset of depression through changes in cognition and behavior (e.g., perseverative thinking, avoidance, reduced problem-solving behavior).

These results also align with recent research indicating specific biologically-based mechanisms that may link genetic risk for rumination and depression. A study by Mandell et al. (2014) identified several neural substrates associated with rumination, the most substantial of which was elevated amygdala activity. In a sample of clinically depressed adults, rumination was associated with sustained activity in the amygdala throughout emotionally valenced and emotionally neutral cognitive tasks, suggesting this activation was sustained even when ruminators had ostensibly shifted their attention to a neutral task and a new goal. This inability to disengage from stimuli that are no longer relevant is consistent with evidence of a related mechanism behind rumination and depression, namely executive function deficits. Certain executive functions, which are highly heritable (Friedman et al., 2008), enable individuals to disengage from information and stimuli that are no longer relevant or rewarding, allowing cognitive resources to be used efficiently and effectively. Reviews of the literature suggest that depressed individuals (Snyder, 2013) and individuals who ruminate (Whitmer & Gotlib, 2013) exhibit deficits in these functions, consistent with subjective reports of rumination and cognitive impairments in depression. However, there is some debate about the nature of the associations between rumination, repetitive thought, and executive functions (McVay & Kane, 2010). Nevertheless, the conjunction of evidence of neural mechanisms associated with rumination and recent theoretical models emphasizing the role of executive function deficits in rumination depression (Whitmer & Gotlib, 2013) provides an exciting framework to elucidate the genetic overlap between rumination and depression.

Substance dependence vulnerability—In contrast to depression, we found evidence of very modest genetic overlap between vulnerability to substance dependence (DV) and rumination. The phenotypic association between rumination and DV was due to overlapping shared (30%) and nonshared (70%) environmental influences, and the preponderance of genetic and environmental influences on DV were not shared with rumination. In fact, the genetic variance in DV was related to self-reflection, rather than rumination, a pattern that was unique to this phenotype. The literature on substance use and rumination is far sparser than the literature on depression, although several studies have found associations between rumination and substance problems. For example, rumination prospectively predicted greater alcohol use in adults following alcohol abuse treatment (Caselli et al., 2010), greater substance misuse following life stressors in adolescents (Skitch & Abela, 2008), and greater problematic substance use in adolescents, controlling for concurrent depressive symptoms (Willem et al., 2011).

Much less is known about the mechanisms linking rumination and substance use disorders, and thus, we believe the current study provides an important contribution to this literature. The association between rumination and DV was due primarily, if not entirely, to overlapping environmental influences between the two constructs, indicating minimal genetic overlap. This finding suggests that future research efforts may focus on specific environmental contexts that generate risk for both rumination and substance use, and the potential interplay between environmental contexts and genetic risk for these associated phenotypes.

Eating pathology—The majority of genetic and environmental variance in eating pathology was separate from those influences on measures of rumination for both men and women. However, results differed between men and women when considering the factors influencing the association between each rumination measure — which were considered independently in this analysis — and eating pathology. For men, nonshared environmental factors explained the majority (~85%) of the covariance among RRS-B, RRS-R, and EDEQ, and the association between RRQ-RU and EDEQ was due to genetic and environmental influences (62% and 38% of the covariance, respectively). For women, genetic influences explained a greater percentage of the covariance between eating pathology and RRS-B, RRQ-RU, and RRS-R (27%, 25%, and 69%, respectively), and shared environmental influences explained up to 23% of these associations; nonshared environmental influences explained between one third to half of the covariance between rumination measures and EDEQ. Though relatively few studies have examined the association between rumination and eating disorders symptoms (only three studies were included in a meta-analysis by Aldao et al., 2010), our results suggest that this is an important area for future research.

Rumination and covariance between disorders—Researchers have suggested that transdiagnostic risk factors for psychopathology, such as rumination, may serve as mechanisms by which two disorders co-occur (e.g., Nolen-Hoeksema & Watkins, 2011). To explore this question in our sample, we estimated the extent to which genetic and environmental covariance between different psychopathologies overlapped with influences on rumination. We found that for some co-occurring disorders, there was considerable overlap between the etiological influences on their co-occurrence and the etiological influences on rumination. For example, 40% of the genetic covariance and 73% of the environmental covariance between depression and dependence vulnerability overlapped with influences on rumination. Additionally, covariance between eating pathology and other disorders (depression, dependence vulnerability) shared a substantial amount of genetic overlap with rumination, especially in women. Modest to moderate nonshared environmental overlap with rumination was found in both genders. These results, in concert with the results discussed above, not only strengthen the case for rumination as a transdiagnostic correlate of psychopathology, but also provide support for identifying it as a risk factor for multiple psychopathologies and as one mechanism by which disorders co-occur.

Limitations of the Study

The results of the current study should be considered with some limitations in mind. First, the design of the study was cross-sectional, so we cannot make inferences about the

temporal association between rumination and psychopathology in our sample. There is significant evidence to suggest that rumination precedes onset of depression and relapse (Nolen-Hoeksema et al., 2008), and some evidence that rumination prospectively predicts substance use problems (Skitch & Abela, 2008) and binge eating (Nolen-Hoeksema et al, 2007) in youth; however, there is also evidence of bidirectional associations between rumination and psychopathology over time (e.g., Nolen-Hoeksema et al., 2007; Willem et al., 2014). As we did not measure rumination at earlier time points, we cannot rule out the possibility that current psychopathology preceded rumination in our sample. Additionally, the time frame assessed by these measures varied, including past week (depressive symptoms), "typical" responses over time (rumination and self-reflection), and lifetime (e.g. substance dependence). This variability in assessment also limits our ability to make temporal inferences about rumination and psychopathology in this study. Thus, it is important for future research to examine these associations prospectively in a twin sample and to use measures that assess the experiences of rumination and psychopathology across the same period of time.

Second, our sample was relatively small for twin analyses. As mentioned, limited statistical power may have influenced our results. For example, this limitation can reduce the ability to detect significant shared environmental influences (C) on these constructs and to differentiate additive (A) and non-additive (D) genetic influences. Additionally, limited statistical power can make it more difficult to detect significant gender differences and can impact the magnitude of multivariate parameter estimates. Thus, replication in a larger sample would be useful in terms of generalizability.

Third, our measures of rumination, self-reflection, and some measures of psychopathology (i.e., CES-D, EDEQ) were self-report measures, and thus associations may be affected by method covariance. However, our results were also significant and consistent for MDD, GAD, and DV, all of which were based on structured clinical interviews, which are less prone to this limitation. Additionally, we measured self-reflection using a manifest variable (i.e. the RRQ-RE) and rumination with a latent variable, which takes into account measurement error. Our comparisons between rumination and self-reflection should be considered in the context of this difference in measurement.

Fourth, although the twin design provides a powerful method to examine rumination as a transdiagnostic correlate and risk factor, there are limitations to this method (for a review, see Tenesa & Haley, 2013). Heritability estimates can vary from study to study, depending on measurement, sample characteristics, and study design. Further, the twin design relies on several assumptions, such as the equal environments assumption (EEA), which states that the environments of MZ twins are no more similar than those of DZ twins. If these assumptions are not met, then heritability estimates may be biased. It is important to note that studies of the EEA have generally found little evidence for its violation influencing twin similarities (e.g., Kendler, Kessler, Neale, Heath, & Eaves, 1993). Furthermore, our heritability estimates are largely consistent with prior twin studies examining psychopathology (Sullivan et al., 2000; Prescott, Madden & Stallings, 2006; Thornton, Mazzeo & Bulik, 2011) and rumination and depressive symptoms (Chen & Li, 2013; Moore et al., 2013), reducing concern of biased estimates.

Finally, associations between forms of psychopathology were modest ($r < .30$), and this introduces error in the estimation of genetic and environmental influences on covariance. These results must be replicated in a larger sample before firm conclusions can be drawn regarding the role of rumination in the co-occurrence among disorders.

Conclusions

The results of the present study suggest that rumination is associated with several forms of psychopathology, including depression, generalized anxiety, substance dependence symptoms, and eating pathology. Furthermore, the genetic and environmental influences on the associations between rumination and these psychopathologies differed by phenotypes, suggesting unique etiological pathways of risk between rumination and these psychopathologies. Specifically, rumination was genetically correlated most with depression, somewhat with eating disorders, and least with vulnerability to substance use disorders. Finally, results suggested that genetic and environmental influences on rumination overlapped considerably with those contributing to covariance between forms of psychopathology. As the first behavior genetic study to examine rumination as a transdiagnostic correlate of psychopathology, this study provides a strong foundation for exploring new avenues of research that could guide prevention and treatment efforts in individuals suffering from comorbid psychiatric disorders.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Reliability, Mean (Standard Deviation), and Sample Size for Study Measures

Measure	α	Men	*N*	Women	*N*
RRS-B[a]	.80	1.87 (.57)	346	2.04 (.64)	395
RRS-R[a]	.81	1.95 (.68)	346	2.13 (.70)	396
RRQ-RU[a]	.91	2.69 (.72)	345	2.93 (.75)	395
RRQ-RE	.91	3.13 (.72)	343	3.10 (.77)	393
CES-D	.90	10.34 (8.63)	337	11.36 (9.09)	389
EDEQ[a]	.94	0.54 (.71)	338	1.27 (1.18)	388
DS	--	3.35 (4.19)	345	2.35 (4.49)	396

		Men			Women		
Ordinal Variables		0	1	2	0	1	2
MDD[a]	N	278	39	30	285	47	63
	%	80.1	11.2	8.6	72.9	11.9	15.9
GAD[a]	N	318	25	4	341	26	28
	%	91.6	7.2	1.2	86.3	6.6	7.1

Note.

[a]Significant gender difference (women higher; $p<.05$).

RRS-B=Ruminative Responses Scale-Brooding; RRS-R = Ruminative Responses Scale-Reflection; RRQ-Ru = Rumination-Reflection Questionnaire-Rumination; RRQ-Re = Rumination-Reflection Questionnaire-Reflection; CES-D = Center for Epidemiological Studies-Depression; EDEQ = Eating Disorder Examination Questionnaire; DS = mean number of dependence symptoms endorsed across 10 substance classes (based on Composite International Diagnostic Interview – Substance Abuse Module); MDD = Major Depressive Disorder; GAD = Generalized Anxiety Disorder (MDD and GAD based on Diagnostic Interview Schedule). MDD and GAD coded as endorsing: no symptoms (0), some symptoms, but not enough to meet criteria for diagnosis (1), or meeting criteria for a diagnosis (2). CES-D means are for the untransformed total score; EDEQ means are for the untransformed average item score; CES-D and EDEQ were log-transformed for subsequent analyses.

Table 2

Variance of Psychopathology and Covariance Between Psychopathology and Rumination/Self-Reflection Explained by A, C and E Components

	Variance of Psychopathology				Covariance Between Psychopathology and	
	Common with		Unique	Total	RLV	RRQ-RE
	RLV+ RRQ-RE	RRQ-RE only[d]				
CES-D						
A	.29	.03	.00	.32	.31	.08
[%][a]	[91]	[9]	[0]	[100]	[52]	[89]
C	.02	.01	.00	.03	-.03	.01
[%][b]	[67]	[33]	[0]	[100]	[-4]	[11]
E	.16	.00	.49	.65	.31	.00
[%][c]	[25]	[0]	[75]	[100]	[52]	[0]
Total	.47	.04	.49	1	.59	.10
					[100]	[100]
MDD						
A	.09	.01	.07	.17	.20	.09
[%][a]	[53]	[6]	[41]	[100]	[43]	[39]
C	.00	.00	.17	.17	.00	.00
[%][b]	[0]	[0]	[100]	[100]	[0]	[.00]
E	.13	.02	.49	.64	.27	.14
[%][c]	[20]	[3]	[77]	[100]	[57]	[61]
Total	.22	.03	.73	.98	.47	.23
Sex		.02[e]			[100]	[100]
DV						
A	.00	.21	.00	.21	.00	.23
[%][a]	[0]	[100]	[0]	[100]	[0]	[191]
C	.14	.04	.00	.18	.07	-.07
[%][b]	[78]	[22]	[0]	[100]	[30]	[-58]
E	.04	.00	.57	.61	.16	-.04

	Variance of Psychopathology				Covariance Between Psychopathology and	
	Common with		Unique	Total	RLV	RRQ-RE
	RLV+ RRQ-RE	RRQ-RE only[d]				
[%][c]	[7]	[0]	[93]	[100]	[70]	[-33]
Total	.18	.25	.57	1	.23	.12
					[100]	[100]

Note. Values presented were calculated by squaring the parameter estimates presented in Supplemental Material 4. Percentages above 100% indicate that the genetic or environmental covariances that add up to the total positive covariance have opposite signs (+/-).

RLV = Rumination Latent Variable; RRQ-Re = Rumination-Reflection Questionnaire-Reflection; CES-D = Center for Epidemiological Studies-Depression; MDD = Major Depressive Disorder; DV = Dependence vulnerability; A = genetic influences; C = shared environmental influences; E = noshared environmental influences.

[a] Percentage of genetic variance/covariance.

[b] Percentage of shared environmental variance/covariance.

[c] Percentage of nonshared environmental variance/covariance.

[d] Controlling for influences in common with RLV.

[e] Sex was included as a covariate in the model examining MDD; men and women were combined in the same group, as there were too few individuals in each group to have the power to detect significant genetic and shared environmental influences.

Table 3

Variance of EDEQ and Covariance Between EDEQ and Rumination/Self-Reflection Explained by A, C, and E Components

	Variance of EDEQ					Covariance Between EDEQ and		
	Common with			Unique	Total	RRS-B	RRQ-RU	RRS-R
	RRS-B +RRQ-RU +RRS-B	RRQ-RU +RRS-R[a]	RRS-R only[b]					
Men								
A	.00	.05	.01	.44	.50	.03	.11	.02
[%][d]	[0]	[10]	[2]	[88]	[100]	[14]	[62]	[17]
C	.00	.00	.00	.00	.00	-.01	.00	.00
[%][e]	[0]	[0]	[0]	[0]	[0]	[-4]	[0]	[0]
E	.06	.01	.00	.43	.50	.19	.07	.10
[%][f]	[12]	[2]	[0]	[86]	[100]	[90]	[38]	[83]
Total	.06	.06	.01	.87	1	.21	.18	.12
						[100]	[100]	[100]
Women								
A	.04	.00[c]	.00[c]	.37	.41	.08	.09	.09
[%][d]	[10]	[0]	[0]	[90]	[100]	[27]	[25]	[69]
C	.07	.00	.00	.00	.07	.07	.08	-.01
[%][e]	[100]	[0]	[0]	[0]	[100]	[23]	[22]	[-8]
E	.03	.03	.00	.46	.52	.15	.19	.05
[%][f]	[6]	[4]	[0]	[90]	[100]	[50]	[53]	[38]
Total	.14	.03	.00	.83	1	.30	.36	.13
						[100]	[100]	[100]

Note. Values presented were calculated by squaring the parameter estimates presented in Supplemental Material 5.

RRS-B = Ruminative Responses Scale-Brooding; RRQ-Ru = Rumination-Reflection Questionnaire-Rumination; RRS-R = Ruminative Responses Scale-Reflection; EDEQ = Eating Disorder Examination Questionnaire; A = genetic influences; C = shared environmental influences; E = noshared environmental influences.

[a]Controlling for influences in common with RRS-B.

[b]Controlling for influences in common with RRS-B and RRQ-RU.

[c]Paths representing shared genetic variance between RRQ-RU/RRS-R and EDEQ were fixed to zero due to negligible loadings on the rumination measures.

[d] Percentage of genetic variance/covariance.

[e] Percentage of shared environmental variance/covariance.

[f] Percentage of nonshared environmental variance/covariance.

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Table 4

Phenotypic Covariance Between Psychopathologies Explained by A, C, and E Components Common with Rumination and Unique to Psychopathology

	A. CES-D with DV Covariance		**B. CES-D with EDEQ Covariance**		**C. DV with EDEQ Covariance**	
	Common with Rumination	**Unique**	**Common with Rumination**	**Unique**	**Common with Rumination**	**Unique**
			Men		Men	
A	.05	.08	A	-.06	A	.03	-.10
[%][a]	[40]	[60]	[%][a]	[-51]	[%][a]	[-46]	[146]
C	.00	.00	C	.00	.00	C	.05	.00
[%][b]	[0]	[0]	[%][b]	[0]	[0]	[%][b]	[100]	[0]
E	.08	.03	E	.05	.05	E	.06	.04
[%][c]	[73]	[27]	[%][c]	[50]	[50]	[%][c]	[63]	[37]
Total	.24		Total	.23		Total	.07	
			Women			Women		
			A	.15	.00	A	.06	.00
			[%][a]	[100]	[0]	[%][a]	[340]	[-240]
			C	.00	.00	C	-.01	.00
			[%][b]	[0]	[0]	[%][b]	[100]	[0]
			E	.07	.06	E	.02	.09
			[%][c]	[54]	[46]	[%][c]	[18]	[82]
			Total	.28		Total	.16	

Note. Percentages above 100% indicate that the genetic or environmental covariances that add up to the total positive covariance have opposite signs (+/-).

A = genetic influences; C = shared environmental influences; E = nonshared environmental influences; CES-D = Center for Epidemiological Studies-Depression; DV = Dependence vulnerability.

[a]Percentage of genetic covariance between psychopathologies that is common with rumination or unique (i.e. not common with rumination).

[b]Percentage of shared environmental covariance between psychopathologies that is common with rumination or unique (i.e. not common with rumination).

[c]Percentage of nonshared environmental covariance between psychopathologies that is common with rumination or unique (i.e. not common with rumination).

[d]Rumination Latent Variable was not used in analyses including EDEQ; covariance values reflect those across independent measures of rumination.