A Twin Study Examining the Development of Rumination and its Role as a Transdiagnostic Risk Factor for Psychopathology

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A large body of research suggests that rumination, or the tendency to engage in passive and repetitive thinking about one's own distress, is a robust risk factor for depression, and is also associated with risk for other psychological disorders. However, much less is known about the influences that lead to ruminative thinking and its associations with psychopathology. The present studies were designed to examine these questions and contribute to the limited body of research investigating the etiology of rumination.

Study 1 examined the genetic and environmental influences on rumination and its associations with several forms of psychopathology (depression, anxiety, eating pathology and substance dependence) in a sample of adult twins. Results suggested that rumination was associated with each form of psychopathology. Furthermore, there was evidence of distinct patterns of etiological overlap between rumination and each disorder; results suggested that rumination had considerable genetic overlap with depression, modest genetic overlap with eating disorder symptoms, and almost no genetic overlap with substance dependence. In general, results were specific to ruminative thought and did not extend to self-reflection. These findings support the conceptualization of rumination as a transdiagnostic risk factor for psychopathology and also suggest that the biological and environmental mechanisms linking rumination to psychopathology may differ depending on the disorder.

Study 2 examined several potential developmental risk factors for rumination. Results suggested that stressful environmental contexts, including exposure to parents in a dissatisfying relationship (for males and females) and negative dependent life events in late childhood and adolescence (for females) were associated with greater rumination in adulthood. Additionally,

mother, father and child neuroticism were associated with rumination in adulthood for both genders. Importantly, these prospective associations were significant even with 10 to 20 years between the assessments of risk factors and rumination.

In concert, results of these two studies lay a foundation to examine further the environmental and biological factors that increase risk for rumination and subsequent risk for psychopathology. Elucidation of the etiological influences on rumination may guide the development and refinement of interventions aimed at reducing rumination, and mitigate rumination's pervasive effects on health and well-being.

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General Introduction

According to cognitive theories of depression, the way in which an individual perceives, attends to, and interprets life experiences can predispose him or her to develop depression (e.g., Beck et al., 1979). An extensive body of research has examined specific cognitive styles and thought content that are most robustly associated with depression, and has yielded a group of risk factors that have been labeled *cognitive vulnerabilities* (CVs) to depression (e.g., Alloy et al., 2006). Examples of CVs include stable, global, negative inferences about the world and life events (e.g., Clark et al., 1999), biased attention to negative stimuli (e.g., MacLeod et al., 2002), and negative self-focused thought (for a review see Nolen-Hoeksema et al, 2008). Cognitive vulnerabilities have been shown to increase risk for onset of depression, maintain depressive symptoms, and increase risk of relapse (for a review see Mathews & MacLeod, 2005).

One CV that has gained considerable attention and empirical support is rumination. 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). The crucial role of rumination in the onset and course of depression is further emphasized by the attention it has garnered in the development of

psychosocial treatments for depression. Reducing rumination has been identified as a core treatment target in several empirically-based interventions for depression, including cognitive therapy, behavioral activation, and mindfulness-based cognitive therapy for depression (e.g., Dimidjian et al. 2006; Segal et al., 2001; Watkins et al., 2011), and reductions in rumination are associated with treatment gains and positive outcomes (e.g., Querstret & Cropley, 2013; van Aalderen et al., 2012; Watkins et al., 2011)

Despite the large body of literature examining rumination, little is known about what factors lead to the development of ruminative thinking. By examining the developmental factors that contribute to rumination, and the relative contributions of genetic and environmental influences to rumination and its association with depression and other forms of psychopathology, we can gain valuable insight into the role rumination plays in the onset, course, and treatment of psychopathology. No prospective, longitudinal examination of the genetic and environmental etiologies of rumination exists in the literature, and thus, the developmental antecedents of rumination remain largely unknown. The present studies address these gaps in the literature by testing a set of hypotheses examining risk for rumination and its association with psychopathology. By elucidating the developmental processes leading to rumination and examining how rumination contributes to risk for psychological disorders, results of these studies may lead to greater knowledge of risk factors for psychopathology and inform empirically-based interventions that target rumination.

Study 1

Rumination as a transdiagnostic risk factor for psychopathology

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 more than one hundred studies (k = 114) 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 pathology (r = .26; k = 3); and substances use disorders (r = .21; k = 7; Aldao et al., 2010). Additional evidence that ruminative thinking is associated with many Axis I diagnoses (e.g., Nolen-Hoeksema & Watkins, 2011; for a review see Ehring & Watkins, 2008) suggests that rumination may play an important role across forms of psychopathology, and may contribute to the high rates of comorbidity among psychiatric diagnoses.

It is important to note that not all self-focused, repetitive thought is associated with increased risk for depression and other negative mental health outcomes. Indeed, non-analytical, experiential self-focus (Watkins & Teasdale, 2004) and "intellective self-consciousness" (or *reflection*; Trapnell & Campbell, 1999) have been identified as potentially adaptive characteristics that can promote well-being and psychological adjustment. Studies have examined the outcomes associated with various forms of thought, and results support the specificity of rumination as a risk factor for negative outcomes, whereas other forms of thought, such as reflection, can have constructive consequences, such as effective coping and adaptive preparation and planning (e.g., Trapnell & Campbell, 1999; Watkins, 2008; Watkins & Teasdale, 2004). Thus, 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).

Behavior genetic studies examining rumination. To date, only a limited body of research has examined the etiology of rumination and the extent to which rumination and different forms of psychopathology share etiological 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 with regards to a potential link between BDNF and rumination, but yield largely 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 12–14-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 ages 11-17 (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) found 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. In concert, these studies suggest rumination is a heritable construct in adolescence and adulthood. They also indicate that there is

a robust genetic correlation between rumination and depression (and, to a lesser extent, a nonshared environmental correlation), suggesting considerable etiological overlap between these constructs.

No research to date has extended the study of genetic and environmental influences on rumination to include other forms of psychopathology. This question is of critical importance, given the growing evidence that rumination is associated with a range of psychopathologies. Furthermore, there is evidence of strong genetic correlations between depression and other forms of psychopathology (e.g., anxiety disorders [Hettema, 2008], eating disorders [Wade et al., 2000]), which may be explained by an underlying genetically-influenced vulnerability, such as rumination, that contributes to the high rates of co-occurrence and comorbidity among psychiatric disorders.

This study will examine rumination and self-reflection as potential transdiagnostic risk factors for psychopathology in early adulthood and will be the first study to investigate the role of genetic and environmental influences on the associations between rumination and different forms of psychopathology. We hypothesize that rumination is a stronger transdiagnostic risk factor for symptoms and diagnoses of major depression, generalized anxiety disorder, eating pathology, and substance use disorders than is self-reflection. We hypothesize that rumination and self-reflection have distinct genetic and environmental influences and that rumination shares common etiological influences with psychopathology, whereas self-reflection does not.

Method

Study participants

Data analyses for Study 1 were conducted on data from participants enrolled in the

Longitudinal Twin Study (LTS) who also participated in the Executive Function and Self Regulation (EFSR) follow-up study. The LTS is a sample of same-sex twin pairs recruited through the Colorado Department of Health born between 1986 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. Data from 386 families were analyzed in Study 1, including 170 male twin pairs (87 monozygotic [MZ]; 83 dizygotic [DZ]) and 14 male singletons and 195 female twin pairs (107 MZ; 88 DZ) and 7 female singletons

Zygosity determination. Zygosity of the twin pairs was determined using ratings from the testers across time. Twin similarity on 10 physical characteristics was rated by the testers each time the twins were seen in person. Twin pairs were considered unambiguously monozygotic (MZ) or dizygotic (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 wave 2 of the EFSR study (N = 751), when twin pairs were between the ages of 21 and 28 (M = 22.84, SD = 1.29). Contemporaneously, as part of assessments for the Center for the Genetics of Antisocial Drug Dependence (CADD) longitudinal study, diagnostic information regarding past year and lifetime endorsement of psychiatric disorders and substance use disorders were assessed in twin pairs using structured diagnostic interviews. A self-report questionnaire of eating pathology symptoms was also collected at this assessment.

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, Gonzalez, and Nolen-Hoeksema (2003). Treynor et al. (2003) eliminated RRS items overlapping substantially with items on depression inventories and factor analyzed 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. Both factors are associated positively 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).

Second, the Rumination-Reflection Questionnaire (RRQ; Trapnell & Campbell, 1999) is a 24-item assessment that is used to measure two types of self-focused thought: rumination and reflection. Reflection on this measure 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. Conversely, rumination, 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).

It is important to note that the RRQ-Re measures self-focused thought that is based in self-awareness and curiosity, not necessarily in reaction to or in the presence of 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 in epidemiological research that was developed by the National Institute of Mental Health. Respondents are asked about the frequency with which they have experienced symptoms of depression in the past 30 days.

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., restraint over eating, avoidance of food), eating concern (e.g., preoccupation with food, eating in secret), shape concern (e.g., desire for a flat stomach, importance of body shape), and weight concern (e.g., importance of weight, desire to lose weight) in the past 28 days. The EDEQ is widely regarded as the instrument of choice for the assessment and diagnosis of eating disorders according to the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, APA, 2000).

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 data collected from the MDD and Generalized Anxiety Disorder (GAD) modules assessment for lifetime diagnosis. For MDD and GAD, individuals were coded as endorsing no symptoms (0), some symptoms (1), or meeting criteria for a diagnosis (2).

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. Based

on prior investigations from our group that included the current sample, we examined dependence vulnerability (DV), as it represents a clinically valid, familial, and heritable construct (Stallings et al., 2003; Button et al., 2006). This index was derived by taking a total count of dependence criteria endorsed across all classes of substances (determined by lifetime symptom counts of each substance) and 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 sex and age using standard regression procedures.

Analyses

All analyses were conducted on raw data and allowed missing data. All structural equation modeling, both phenotypic and genetic, were implemented using Mplus 7 (Muthén & Muthén, 2013). Mplus allows the analyses of a combination of ordinal and continuous data. Given that there were missing observations across time periods, the TYPE=MISSING command was used; this means that the model were estimated using the missing at random missing data method (Little & Rubin, 2002), which allows missingness to be a function of observed covariates and observed outcomes. When conducting phenotypic analyses, non-independence of the data should be considered, given that the data from the two twins in each pair are correlated. Therefore, the TYPE=COMPLEX in the ANALYSIS command was used in order to take into account non-independence of observations when computing standard errors and model fit. Also, given the fact that some of the data (e.g., DV, EDEQ) violated the assumption of normality the MLR estimator was used when possible. The MLR estimator provides maximum likelihood parameter estimates with standard errors and chi-square test statistics that are robust to non-normality. When analyses included both continuous and ordinal variables, the weighted least square mean and variance (WLSMV) estimation method was used. When WLSMV is used,

Mplus uses pairwise deletion. Statistical significance of the parameters was determined by p-values 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 greater than .95 and RMSEA less than .06 indicate good model fit (Hu & Bentler, 1998).

We examined evidence for shared and distinct genetic and environmental factors influencing rumination, self-reflection, and forms of psychopathology in several steps. First, cross-trait phenotypic correlations (e.g., twin 1 rumination with twin 1 self-reflection), within-trait cross-twin correlations (e.g., twin 1 rumination with twin 2 rumination), and cross-trait cross-twin correlations (e.g., twin 1 rumination with twin 2 self-reflection) were calculated to provide initial estimates of genetic and environmental influences on these constructs. Based on the assumption that MZ twins share 100% of their genes and DZ twins share 50% of their genes identical by descent, comparing the magnitude of MZ and DZ within-trait and cross-trait correlations can provide information about genetic and environmental influences on rumination, self-reflection, and their associations with psychopathology. When correlations are greater in MZ twin pairs than DZ twin pairs (rMZ > rDZ), there is evidence of genetic influences on the phenotypes or the covariance between phenotypes. If rMZ is greater than twice rDZ, this suggests the influence of dominant genetic effects, whereas if rMZ is less than twice rDZ, there is evidence of shared environmental effects on the phenotypes. When the rMZ is less than 1.0 nonshared environmental effects are indicated.

To assess the genetic and environmental influences on rumination, self-reflection, and all forms of psychopathology, a multivariate Cholesky decomposition was fit to the data. The

Cholesky decomposes the covariance of these constructs into additive genetic (A), shared environmental (C), non-additive genetic (D) and nonshared environmental (E) influences. A limitation of the traditional twin design is that D and C cannot be estimated in the same model. Therefore, in the present study, the pattern of twin correlations was used to decide whether an ADE or ACE model was most appropriate for these data. Figure 1 provides an example of a multivariate Cholesky decomposition of the genetic influences on rumination, self-reflection, and psychopathology (MDD, GAD, SUDs, eating pathology): A1, genetic influences that all six constructs share in common, A2, genetic influences that are independent from those influencing rumination but that self-reflection and psychopathology share in common, A3, genetic influences common to all four forms of psychopathology but not rumination or self-reflection, and so on for A4 through A7 (only genetic influences are shown in Figure 1 and described here for the sake of simplicity). The full model represented by Figure 1 can be compared to a set of reduced models that drop the paths representing common genetic influences to test specific hypotheses. Comparisons of the fit of these models can be used to test for evidence of shared and distinct genetic effects on rumination and psychopathology and on self-reflection and psychopathology. To examine gender differences, models with separate parameter estimates for males and females were compared to models in which parameters were fixed to be the same for males and females.

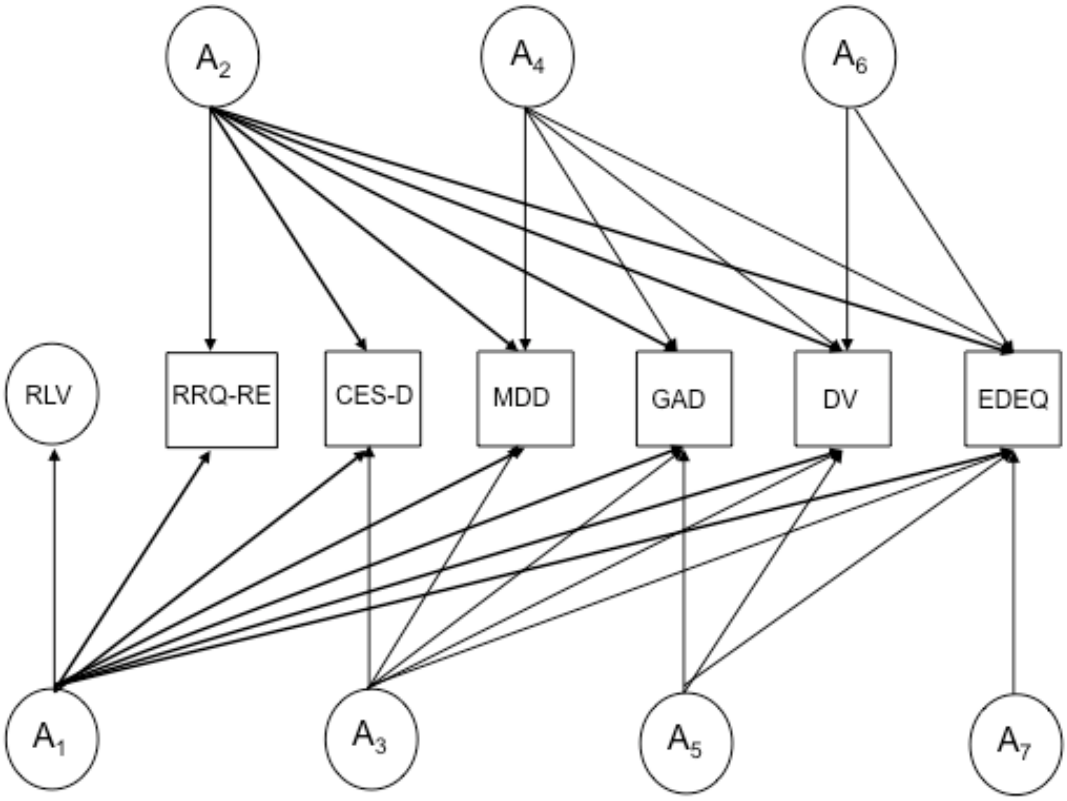

Figure 1. Multivariate Cholesky decomposition of genetic influences (A) for rumination, self-reflection and psychopathology.

Note. RLV = Rumination Latent Variable; RRQ-RE = Rumination-Reflection Questionnaire-Reflection; MDD = Major Depressive Disorder; GAD = Generalized Anxiety Disorder; CES-D=Center for Epidemiological Studies-Depression; EDEQ = Eating Disorder Examination Questionnaire; DV = Dependence vulnerability.

Results

Descriptive Statistics

Means and standard deviations 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). The distributions of CES-D, EDEQ, and DV scores were skewed, so scores were log transformed to achieve a normal distribution for subsequent analyses. Rates of MDD symptoms (11.6%) and diagnosis (12.5%) and GAD symptoms (5.3%) and diagnosis (3.3%) were slightly lower than rates 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 less rumination and eating disorder symptoms and were less likely to endorse symptoms and diagnoses of MDD ($\chi^2[2] = 9.48$, $p = .01$) and GAD ($\chi^2[2] = 15.78$, $p < .01$).

Table 1

Means (Standard Deviations) for Study Measures

Measure		Men			Women		
RRS-B[a]		1.87 (.57)			2.04 (.64)		
RRS-R[a]		1.95 (.68)			2.13 (.70)		
RRQ-RU[a]		2.69 (.72)			2.93 (.75)		
RRQ-RE		3.13 (.72)			3.10 (.77)		
CES-D		10.34 (8.63)			11.36 (9.09)		
EDEQ[a]		0.54 (.71)			1.27 (1.18)		
Dependence Symptoms		3.25 (4.10)			2.32 (4.43)		
Dependence Vulnerability		-.03 (.05)			0.07 (.05)		
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-RE = Rumination-Reflection Questionnaire-Rumination; RRQ-RE = Rumination-Reflection Questionnaire-Reflection; MDD = Major Depressive Disorder; GAD = Generalized Anxiety Disorder; CES-D = Center for Epidemiological Studies-Depression; EDEQ = Eating Disorder Examination Questionnaire; DV = Dependence vulnerability. CES-D means are for the untransformed total score; EDEQ means are for the untransformed average item score; Dependence symptoms is the mean number of dependence symptoms endorsed across 10 substance classes; CES-D and EDEQ were log transformed for subsequent analyses. Dependence symptoms were used to calculate Dependence Vulnerability scores, which were then log transformed.

Phenotypic Associations Among Rumination, Self-Reflection, and Psychopathology

Phenotypic correlations are presented separately by gender in Table 2, although gender

differences in these correlations were not significant. The three measures of rumination were moderately to highly correlated with each other, and also significantly associated with all measures of psychopathology. The self-reflection measure (RRQ-RE) was significantly correlated with measures of rumination; however, these associations were modest in magnitude. Additionally, RRQ-RE was significantly associated with some psychopathology measures, but these associations were also smaller in magnitude than those between than the rumination measures and psychopathology

Table 2
Phenotypic Correlations for Men/Women

Measure	RRS-B	RRS-R	RRQ-RU	RRQ-RE	GAD	MDD	CES-D	EDEQ
RRS-B	--							
RRS-R	**.58/.56**	--						
RRQ-RU	**.68/.70**	**.54/.44**	--					
RRQ-RE	**.13/.11**	**.34/.42**	**.19/.14**	--				
GAD[a]	**.37/.34**	**.42/.28**	**.36/.39**	**.27/.32**	--			
MDD[a]	**.26/.35**	**.31/.36**	**.39/.37**	**.28/.20**	**.27/.55**	--		
CES-D	**.50/.51**	**.37/.32**	**.48/.51**	.06/**.10**	**.37/.40**	**.27/.42**	--	
EDEQ	**.23/.30**	**.12/.14**	**.18/.37**	.05/-.03	.12/.09	.09/.09	**.23/.30**	--
DV	**.23/.20**	**.15/.13**	**.17/.17**	**.13/.12**	**.22/.22**	**.33/.23**	**.21/.28**	.08/**.16**

Note. Bold indicates significance at $p < .05$. All correlations account for missing data and nonindependence using Mplus. [a]Polyserial correlations with continuous variables, polychoric correlations with ordinal variables. RRS-B=Ruminative Responses Scale-Brooding; RRS-R = Ruminative Responses Scale-Reflection; RRQ-RU = Rumination-Reflection Questionnaire-Rumination; RRQ-RE = Rumination-Reflection Questionnaire-Reflection; MDD = Major Depressive Disorder; GAD = Generalized Anxiety Disorder; CES-D = Center for Epidemiological Studies-Depression; EDEQ = Eating Disorder Examination Questionnaire; DV = Dependence vulnerability.

We used the three rumination measures 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. There were significant gender differences ($\chi^2_{diff}[3] = 12.23$, $p = .01$) between the factor loadings for men and women. However, factor loadings were similar, and gender differences (shown later for the genetic analyses) were small, suggesting that the RLV was qualitatively similar for men and women on a phenotypic level. Nonetheless, subsequent analyses were conducted allowing the RLV factor loadings to differ between males and females.

To test the hypothesis that rumination is a transdiagnostic risk factor for psychopathology, and that self-reflection is not, multiple regressions were conducted to examine the associations between rumination and psychopathology, independent of self-reflection and between self-reflection and psychopathology, independent of rumination. In the multiple regression models and all subsequent 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 (Treynor et al., 2003), and possibly more related to measures of adaptive self-reflection.

Results of the multiple regressions (Table 3) indicated that the associations between rumination and psychopathology all remained significant and were modest to moderate in magnitude *(b* = .20 - .58) when controlling for self-reflection. Conversely, only the associations between self-reflection and MDD and GAD remained significant when controlling for rumination. These results suggest that the associations between rumination and psychopathology are independent of self-reflection, whereas the associations between self-reflection and psychopathology are largely, though not entirely, accounted for by rumination. Results were consistent across males and females, except for EDEQ, for which the association with rumination was significantly higher for women than for men.

Table 3
Standardized Regression Coefficients from Multiple Regression Analyses of Psychopathology on Rumination and Self-reflection

Psychopathology	RLV		RRQ-RE	
	Men	Women	Men	Women
GAD	**.43 (.08)**	**.38 (.07)**	*.20 (.11)*	**.25 (.07)**
MDD	**.36 (.07)**	**.42 (.06)**	**.21 (.08)**	**.15 (.06)**
CES-D	**.60 (.05)**	**.58 (.05)**	-.06 (.05)	.01 (.05)
EDEQ	**.24 (.06)**[a]	**.37 (.06)**[a]	.00 (.06)	-.09 (.05)
DV	**.22 (.06)**	**.20 (.06)**	.09 (.05)	.08 (.05)

Note. Bold indicates significance at $p < .05$; italics indicate significance at $p < .10$. [a]Significant gender difference (women higher; $p<.05$). RRQ-RE = Rumination-Reflection Questionnaire-Reflection; RLV = Rumination Latent Variable; MDD = Major Depressive Disorder; GAD = Generalized Anxiety Disorder; CES-D = Center for Epidemiological Studies-Depression; EDEQ = Eating Disorder Examination Questionnaire; DV = Dependence vulnerability.

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

Given the evidence that rumination is associated phenotypically with several forms of psychopathology, we next examined our hypotheses that these associations would be explained by significant etiological overlap between rumination and psychopathology. We examined twin correlations and multivariate twin models to test this hypothesis. Twin correlations suggested significant genetic influences on all constructs, with MZ twin correlations greater than DZ twin correlations. One exception was RRQ-RE in females, for which the MZ and DZ correlations were approximately equal, suggesting 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, and thus GAD was not included in genetic analyses.

Table 4 presents the cross twin correlations for males and females separately. Though there were not significant gender differences in the twin correlations, the patterns of correlations were qualitatively different between men and women. For men, some MZ correlations were larger than twice the DZ correlations (e.g., CES-D, EDEQ, rumination measures), indicating ADE models might best fit these data. However, for women, ACE models were indicated for all constructs (with the exception of EDEQ), as MZ correlations were greater than, but close in magnitude to, DZ correlations.

Table 4
Cross-Twin Correlations for Men (Panel A) and Women (Panel B)

Panel A

MZ	RRSB 1	RRSR 1	RRQRU 1	RRQRE 1	MDD 1	EDEQ 1	CESD 1	DV 1
RRSB2	**0.57**							
RRSR2	**0.36**	**0.50**						
RRQRU 2	**0.47**	**0.35**	**0.57**					
RRQRE2	0.09	**0.23**	0.14	**0.37**				
MDD2	**0.34**	**0.30**	**0.31**	0.12	*0.34*			
EDEQ2	0.09	0.05	0.11	0.04	-0.02	**0.61**		
CESD2	**0.40**	**0.35**	**0.43**	0.10	**0.39**	0.15	**0.55**	
DV2	0.03	0.05	0.03	0.11	**0.23**	0.01	0.05	**0.33**
DZ								
RRSB2	0.1							
RRSR2	0.01	0.14						
RRQRU 2	0	0.02	-0.08					
RRQRE2	0.08	0.07	0.04	0.09				
MDD2	0.05	-0.11	-0.05	0.01	0.2			
EDEQ2	-0.09	-0.05	0.00	0.07	0.03	0.12		
CESD2	-0.03	-0.05	**-0.22**	0.07	-0.06	-0.04	-0.05	
DV2	0.04	-0.13	0.06	0.06	0.09	0.04	0.06	**0.3**

Panel B

MZ	RRSB 1	RRSR 1	RRQRU 1	RRQRE 1	MDD 1	EDEQ 1	CESD 1	DV 1
RRSB2	**0.23**							
RRSR2	*0.14*	*0.17*						
RRQRU 2	**0.29**	**0.22**	**0.30**					
RRQRE2	**0.16**	**0.26**	**0.15**	**0.35**				
MDD2	0.05	0.00	*0.13*	*0.13*	**0.36**			
EDEQ2	**0.15**	*0.12*	**0.16**	0.05	0.11	**0.50**		
CESD2	**0.24**	0.11	**0.28**	0.11	**0.27**	*0.14*	**0.35**	
DV2	0.12	0.07	0.03	**0.26**	**0.22**	0.06	**0.27**	**0.51**
DZ								
RRSB2	0.02							
RRSR2	0.07	0.12						
RRQRU 2	*0.17*	0.09	**0.25**					
RRQRE2	-0.1	0.01	*-0.13*	**0.41**				
MDD2	0.06	0.03	*0.21*	*-0.09*	0.3			
EDEQ2	0.12	-0.04	0.09	**-0.21**	-0.01	0.19		
CESD2	0.07	-0.02	*0.13*	-0.12	0.11	**0.19**	**0.24**	
DV2	0.01	0.04	0.13	-0.05	0.06	0	0.09	**0.25**

Note. Bold indicates significance at $p < .05$, italicized indicates $p < .10$. "1" after variable name indicates measure for first twin in twin pair, "2" after variable name indicates second twin in twin pair. RRS-B = Ruminative Responses Scale-Brooding; RRS-R = Ruminative Responses Scale-Reflection; RRQ-RU = Rumination-Reflection Questionnaire-Rumination; RRQ-RE = Rumination-Reflection Questionnaire-Reflection; MDD = Major Depressive Disorder; GAD = Generalized Anxiety Disorder; CES-D = Center for Epidemiological Studies-Depression; EDEQ = Eating Disorder Examination Questionnaire; DV = Dependence vulnerability.

Univariate ADE and ACE models were compared to more parsimonious univariate models that constrained all non-significant parameter estimates of D/C to zero. The reduced model did not fit significantly worse than the ADE/ACE model for any variable, all $\Delta\chi^2(2) < 4.10$, $p > .13$, and all of the reduced models fit the data well. Results from the ADE/ACE and reduced models are presented in Table 5. Discussion of results will largely focus on the reduced models, though the results of full models also will be discussed when overall conclusions differed between the two.

Table 5

Univariate ACE/ADE Models and Reduced Models for Rumination, Self-reflection and Psychopathology

ACE Models	χ^2	df	TLI	RMSEA	Δχ2(df)	*P*	A	C	E
1. RLV	91.13	74	0.98	0.049	--		**.63(.07)**	0	**.78(.06)**
							.58(.08)	*0*	***.81(.06)***
2. RLV	91.23	82	0.99	0.034	.10(8)	1	**.63(.09)**	--	**.78(.06)**
							.58(.08)	--	***.81(.06)***
1. RRQ-RE	15.34	12	0.98	0.054	--		**.64(.07)**	0	**.77(.06)**
							.42(.28)	***.48(.20)***	***.77(.06)***
2. RRQ-RE	16.7	14	0.98	0.045	1.35(2)	0.51	**.64(.07)**	--	**.77(.06)**
							.66(.06)	--	***.75(.05)***
1. MDD	14.44	14	0.99	0.018	--		.52(.51)	.26(.84)	**.81(.11)**
							.34(.68)	*.49(.40)*	***.80(.09)***
2. MDD	14.97	16	1	0	.41(2)	0.81	**.59(.13)**	--	**.81(.10)**
							.62(.10)	--	**.78(.08)**
1. DV	9.81	12	1	0	--		.19(.72)	**.53(.20)**	**.83(.06)**
							.56(.22)	*.37(.29)*	***.75(.05)***
2. DV	11.73	14	1	0	1.92(2)	0.38	**.60(.07)**	--	**.80(.05)**
							.68(.05)	--	***.74(.05)***
ADE Models							A	D	E
1. CES-D[a]	16.11	12	0.96	0.06	--		0	**.69(.06)**	**.73(.06)**
							.34(.39)	*.42(.27)*	***.84(.05)***
2. CES-D	20.21	14	0.95	0.069	4.10(2)	0.13	**.65(.07)**	--	**.76(.06)**
							.56(.07)	--	***.83(.05)***
1. EDEQ	15.01	12	0.98	0.052	--		0	**.72(.05)**	**.69(.05)**
							.37(.56)	*.62(.35)*	***.69(.05)***
2. EDEQ	17.25	14	0.98	0.05	2.23(2)	0.33	**.70(.06)**	--	**.71(.06)**
							.71(.05)	--	***.71(.05)***

Note. Parameter estimates are shown; these values can be squared to calculated proportions of variance explained. Bold values indicate significance at $p < .05$. Results for women in italics. Standard errors in parentheses. Model 1 is full ACE or ADE model, Model 2 is reduced model (with all non-significant D/C paths fixed at zero). [a]Significant gender difference. RLV = Rumination Latent Variable; RRQ-RE = Rumination-Reflection Questionnaire-Reflection; MDD = Major Depressive Disorder; CES-D = Center for Epidemiological Studies-Depression; EDEQ = Eating Disorder Examination Questionnaire; DV = Dependence vulnerability.

Rumination was heritable for men (h^2 = .40) and women (h^2 = .34) and also influenced substantially by nonshared environmental factors (see Figure 2). There were no significant gender differences in the etiological influences on the latent variable; 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 self-reflection were similar in magnitude to those influencing rumination (h^2 = .41 – .43). Measures of psychopathology were moderately heritable for men and women, and the remaining variance could be explained by nonshared environmental factors. Fixing parameters to be equal across gender significantly worsened model fit in the EDEQ model $\chi^2_{diff}(2) < 17.99$, $p < .01$, suggesting that there may be gender differences in the etiological influences on eating disorder symptoms in men and women. There were no significant gender differences for the other forms of psychopathology.

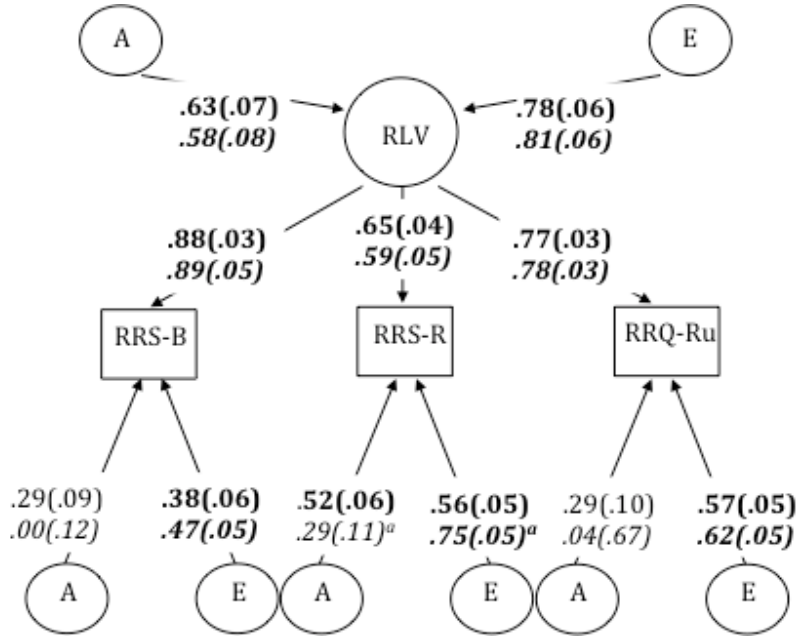

Figure 2. AE model for the rumination latent variable (RLV) with standardized parameter estimates (standard errors in parentheses).
Note. A and E values are parameter estimates; these values can be squared to calculate proportions of variance explained. Bold values indicate significance at $p < .05$. Parameters for

women in italics. [a]Statistically significant gender difference (p<.05) in the unstandardized parameter estimates. A = genetic influences; E = nonshared environmental influences; RRS-B = Ruminative Responses Scale-Brooding; RRS-R = Ruminative Responses Scale-Reflection; RRQ-RU = Rumination-Reflection Questionnaire-Rumination. *p < .05.

The pattern of estimates from the full models (see Table 5) suggested that dominant genetic and shared environmental effects may influence these phenotypes. For example, shared environmental influences were modest, but not negligible in magnitude in the ACE models for DV, explaining 27.8% 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. Finally, evidence of dominant genetic effects was found for EDEQ in both groups and CES-D in men. As discussed above, in all of these models, the reduced models did not fit significantly worse, suggesting that almost all of the D and C parameters could be dropped from the models. These results do not imply that there are no shared environmental or non-additive genetic influences on these constructs, but instead, may reflect the low power of the twin design to distinguish them from additive (A) genetic influences (Martin, Eaves, Kearsey, & Davies, 1978). With this in mind, we consider A to reflect broad sense heritability rather than narrow sense heritability.

With evidence that rumination, self-reflection, and psychopathology are heritable, we next turned to our second set of hypotheses examining the genetic and environmental influences on the associations between rumination and psychopathology. First, a multivariate Cholesky decomposition including rumination, self-reflection, and all measures of psychopathology was tested, as described in the Method section. However, this decomposition fit the data relatively poorly, $\chi^2[472] = 531.53$, $p = .03$, TLI = .92; RMSEA = .04. The poor fit was likely due to the fact that the association between the RLV indicators and EDEQ differed from the associations

between the RLV indicators and the other forms of psychopathology in MZ twin pairs. For example in MZ females, MDD correlated similarly across the three indicators of the RLV (RRS-B [r = .37], RRS-R [r = .48], RRQ-RU [r = .44]) whereas EDEQ correlated less with RRS-R (r = .19), compared to RRS-B (r = .40) and RRQ-RU (r = .51). This led to relatively poor fit in any genetic models including EDEQ and the RLV. Considering the problems with model fit, we decided to examine the associations among rumination, self-reflection, and each measure of psychopathology in separate Cholesky decompositions. These trivariate models are identical to the larger multivariate model, but include only RLV, RRQ-RE and one form of psychopathology.

A different model was used to examine the hypotheses for the EDEQ because the genetic models including the EDEQ and the RLV fit poorly due to differential correlations between EDEQ and the RLV indicators. A multivariate Cholesky decomposition including the three indicators of the RLV (RRS-B, RRQ-RU, RRS-R) individually and the EDEQ was fitted to the data (see Appendix 1). The RRQ-RE was not included because there was almost no phenotypic association between EDEQ and RRQ-RE.

Consistent with results from the regression analyses and the univariate analyses, the only trivariate model with significant gender differences was the model including rumination measures and EDEQ ($\chi^2_{diff}(30) = 60.06$, $p < .01$). Results for the EDEQ models will be 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. Subsequent analyses for MDD include gender as a covariate. For CES-D and DV, results from the models with parameters fixed across gender will be discussed.

Rumination, self-reflection, and depression. 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 largely replicated those from the previous study, suggesting considerable genetic overlap between rumination and both CES-D and MDD, and also unique genetic variance for measures of depression (Table 6; for parameter estimates from the trivariate models, see Appendix 2). Furthermore, results suggested the majority of genetic influences on self-reflection were separate from those influencing depression and rumination. Whereas a large proportion of genetic influences on CES-D were shared in common with rumination and self-reflection (54%), CES-D shared no genetic variance exclusively with self-reflection. A similar pattern was found with environmental influences, although a greater proportion of these influences were unique to CES-D (75%). Furthermore, 90% of the genetic variance in self-reflection was unique to that construct, in other words, not shared with either rumination or CES-D. (These proportions are also presented in Table 6; row titled "(%)[b]" in CES-D section.). A similar pattern was found for MDD, though with lower proportion of genetic variance in MDD shared with rumination (24%). Only 1% of the genetic variance and 4% of nonshared environmental variance in MDD was shared exclusively with self-reflection, suggesting the modest phenotypic correlation between MDD and self-reflection was due mostly to etiological influences that also overlap with those on rumination. For CES-D, the results from the reduced model were very similar to those from full ACE model (results from ACE models presented in Appendix 3). For MDD there were some differences, namely, there was evidence of modest shared environmental influences on RRQ-RE and MDD, explaining approximately 17% of the variance in each construct. These influences were unique to each construct and did not contribute to the covariance between MDD and rumination or MDD and RRQ-RE. With these shared environmental influences accounted for in the model, the proportion of genetic variance in MDD

shared with rumination (52%) aligned with results from the CES-D analyses (54%).

Table 6

Variance in Rumination, Self-reflection, and Psychopathology Explained by Common and Unique A and E Components and Phenotypic Covariance Explained by Common A and E Components

	RLV	RRQ-RE		Psychopathology			Psychopathology Covariance			
	Total Variance	Common with RLV	Unique	Common with RLV	RRQ-RE[d]	Unique	with RLV	with RRQ-RE		
								Common w RLV	Unique	
CES-D										
A	.39	.04	.35	.19	.00	.16	.27	.09	.00	
(%)[b]		(10)	(90)	(54)	(0)	(46)	(47)	(100)	(0)	
E	.61	.00	.60	.16	.00	.49	.31	.02	-.01	
(%)[c]		(0)	(100)	(25)	(0)	(75)	(53)	(100)	(0)	
Total							.58	.10		
MDD										
A	.44	.04	.34	.09	.00	.28	.20	.06	.03	
(%)[b]		(10)	(90)	(24)	(0)	(76)	(43)	(67)	(33)	
E	.56	.00	.62	.13	.02	.46	.27	.02	.12	
(%)[c]		(1)	(99)	(21)	(4)	(75)	(57)	(14)	(86)	
Total							.47	.23		
Sex						.02				
DV										
A	.37	.04	.35	.02	.04	.35	.08	.02	.12	
(%)[b]		(10)	(90)	(5)	(10)	(85)	(33)	(14)	(86)	
E	.63	.00	.61	.04	.00	.55	.15	.01	-.03	
(%)[c]		(0)	(100)	(7)	(0)	(93)	(67)	(-50)	(150)	
Total							.23	.12		

Note. The total variance due to A and E for each variable is similar but not identical across different models because parameters are estimated to minimize the difference between the observed and expected covariance matrices in each model. [a]Controlling for influences in common with RLV. [b]Percentage of genetic variance/covariance. [c]Percentage of nonshared environmental variance/covariance. Estimates presented in the tables may differ slightly from percentages of variance presented in the text due to rounding error.A = genetic influences; E = nonshared environmental influences; RLV = Rumination Latent Variable; RRQ-RE = Rumination-Reflection Questionnaire-Reflection; MDD = Major Depressive Disorder; CES-D = Center for Epidemiological Studies-Depression; DV = Dependence vulnerability.

Rumination, self-reflection and vulnerability to substance dependence. The pattern of results for DV differed from those for depression. The vast majority of genetic (86%) and nonshared environmental (93%) influences on DV were unique to the construct, suggesting only modest etiological overlap with rumination and self-reflection. There was evidence of genetic factors shared between self-reflection and DV after accounting for the genetic influences shared with rumination, and these factors explained 10% of the overall genetic variance on DV. The modest phenotypic correlation between DV and rumination was largely due to nonshared environmental factors, with 67% of the correlation due to environmental and 33% due to genetic influences.

The full ACE model (see Appendix 3) yielded considerably different results than the reduced model for DV. There was modest shared environmental variance on DV (18%) and it was explained entirely by influences shared in common with rumination and self-reflection. Furthermore, results of the ACE model suggested that the covariance between rumination and DV was due only to environmental influences, with 31% and 69% percent of the covariance due to shared and nonshared environmental influences, respectively. Additionally, all of the genetic influences on DV in this model were shared exclusively with self-reflection.

Rumination, self-reflection and eating disorder symptoms. For men, the majority of etiological influences on EDEQ were not shared with the rumination measures, with only 13% of genetic influences and 14% percent of environmental influences on EDEQ overlapping with any of the rumination measures (see Table 7). The phenotypic associations between EDEQ and RRS-R and RRS-B were primarily explained by overlapping nonshared environmental influences, whereas the association between RRQ-RU and EDEQ was due to genetic (61%) and environmental (39%) influences.

Results for women indicated that 28% of genetic influences on EDEQ were shared in common with rumination measures and genetic influences explained 52-55% of the phenotypic associations between EDEQ and each of the rumination measures. In contrast, the vast majority of the nonshared environmental influences on EDEQ were unique to it. The full ACE model yielded very similar results for males, but slightly different results for females (Appendix 4). Though shared environmental influences (C) explained only a modest percentage of variance in EDEQ overall (8%), it did contribute to the covariance between EDEQ and RRS-B and RRQ-RU, explaining approximately 23% of these phenotypic covariances.

Table 7

Variance in RRS-B, RRQ-RU, RRS-R and EDEQ Explained by Common and Unique A and E Components and Phenotypic Covariance Explained by Common A and E Components

	RRS-B	RRQ-RU		RRS-R			EDEQ				EDEQ Covariance with		
	Total Variance	Common with RRS-B	Unique	Common with RRS-B	Common with RRQ-RU[a]	Unique	Common with RRS-B	Common with RRQ-RU[a]	Common with RRS-R[b]	Unique	RRS-B	RRQ-RU	RRS-R
Men													
A	.38	.21	.14	.11	.04	.27	.00	.06	.01	.44	.03	.11	.02
(%)[d]		(60)	(40)	(26)	(10)	(64)	(0)	(12)	(2)	(86)	(13)	(62)	(16)
E	.62	.40	.25	.22	.02	.35	.06	.01	.00	.43	.19	.07	.10
(%)[e]		(62)	(38)	(37)	(3)	(59)	(12)	(2)	(0)	(86)	(86)	(38)	(84)
Total											.22	.18	.12
Women													
A	.22	.33	.00	.14	.00	.09	.11	.00[c]	.03	.36	.16	.19	.07
(%)[d]		(100)	(0)	(61)	(0)	(39)	(22)	(0)	(6)	(72)	(53)	(52)	(55)
E	.78	.24	.43	.19	.00	.58	.02	.03	.00	.46	.14	.17	.06
(%)[e]		(36)	(64)	(25)	(0)	(75)	(4)	(6)	(0)	(90)	(47)	(48)	(45)
Total											.30	.36	.13

Note. The total variance due to A and E for each variable is similar but not identical across different models because parameters are estimated to minimize the difference between the observed and expected covariance matrices in each model. A = genetic influences; E = noshared environmental influences; RRS-B = Ruminative Responses Scale-Brooding; RRS-R = Ruminative Responses Scale-Reflection; RRQ-Ru = Rumination-Reflection Questionnaire-Rumination; EDEQ = Eating Disorder Examination Questionnaire. [a]Controlling for influences in common with RRS-B. [b]Controlling for influences in common with RRS-B and RRQ-RU. [c]This path represents shared genetic variance between rumination measures and EDEQ; it was fixed to zero due to negligible loadings on the rumination measures. [d]Percentage of genetic variance/covariance. [e]Percentage of nonshared environmental variance/covariance. Estimates presented in the tables may differ slightly from percentages of variance presented in the text due to rounding error.

Discussion

Results from the current study have several important implications for understanding rumination as a transdiagnostic risk factor for psychopathology. First, rumination was associated with several forms of psychopathology in an adult sample of twins, including MDD, GAD, SUDs, and eating pathology. Second, the genetic and environmental influences on these associations differed by disorder, suggesting there are differential etiological pathways linking rumination and forms of psychopathology. Third, self-reflection, a more adaptive form of self-focused thought, was associated with fewer phenotypes and to a lesser extent than rumination, and there was little or no common etiological influences with psychopathology. This finding suggests that the risk that is associated with self-focused thought is specific to rumination and does not extend to other forms of thought.

Rumination as a Transdiagnostic Risk Factor

Results of the current study 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 is positively associated with self-report symptom measures of depression and eating pathology, and interview-based symptoms and diagnoses of MDD, GAD, and substance use disorders. 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 of the more commonly used measures of rumination (RRS-B, RRS-R, RRQ-RU) as indicators. Thus, we are confident that these results extend beyond a specific measure of rumination. Second, psychopathology was measured by self-report and structured clinical interview, suggesting these results may hold for both subthreshold symptoms and clinician-rated 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 sample, 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 more adaptive 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 the specific forms of self-focused thought that are maladaptive and increase risk for psychopathology. Our results suggest rumination 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 our previous study examining rumination and depression in a subgroup of the current sample (Johnson et al., 2014) and other recent studies (Chen & Li, 2013; Moore et al., 2013). Rumination was found to be moderately heritable and a majority of the genetic influences on rumination were shared in common with depression. These results did not depend on the measure of depression (CES-D, MDD diagnosis), suggesting they are consistent across dimensional and categorical conceptualizations of depression. Results for nonshared environmental influences showed a similar pattern, although the overlap of influences was less in magnitude than genetic influences. Furthermore, 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 the etiology of 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 risk factor.

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 recent study by Mandell and colleagues (2014) identified several neural substrates associated with rumination, the most substantial of which was elevated amygdala activity. In a sample of clinically depressed adults, the authors found that 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 is 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 is no longer relevant or rewarding, allowing cognitive resources to be used efficiently and effectively. Several lines of research suggest that depressed individuals (for a review and meta-analysis see Snyder, 2013) and individuals who ruminate (for a review see 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, in conjunction, evidence of neural mechanisms associated with rumination, and recent theoretical models emphasizing the role of executive function deficits in rumination depression (Whitmer & Gotlib, 2013), provide an exciting framework to further examine the genetic overlap between rumination and depression.

Dependence vulnerability. In contrast to depression, we found evidence of very modest genetic overlap between vulnerability to substance dependence (DV) and rumination. The majority of the association between rumination and DV was due to overlapping nonshared environmental influences, and the preponderance of genetic and environmental influences on DV were not shared with rumination or self-reflection. 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, suggesting there is a link between the two. 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. In both models (the ACE model including shared environmental influences [C], and the reduced AE model), the association between DV and rumination was due primarily, if not entirely, to overlapping environmental influences between the two constructs, indicating minimal genetic overlap. Although this result must be considered in the context of limited statistical power to distinguish A from C, it 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. Results for symptoms of eating pathology differed for men and women. For men, the majority of genetic and environmental variance in eating disorder symptoms was unshared with those influences on measures of rumination. The etiology of the association between rumination measures — which were considered independently in this analysis — and eating disorder symptoms differed depending on the measure of rumination. Nonshared environmental factors explained the majority (~85%) of the covariance between RRS-B and RRS-R and EDEQ, whereas the association between RRQ-RU and EDEQ was due to genetic and environmental influences (61% and 39% of the covariance, respectively).

For women, 28% of the genetic variance in EDEQ was shared with rumination measures. Additionally, results were similar across rumination measures, with approximately equal contributions of genetic and nonshared environmental influences on the association between rumination and eating disorder symptoms, a pattern similar to those found for measures of depression. Though relatively few studies to date have examined the association between

rumination and symptoms of eating disorders (three studies compared to the 55 on depression and rumination in a recent meta-analysis [Aldao, Nolen-Hoeksema, & Schweizer, 2010]), our results suggest this is an important area for future research.

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. Thus, it will be important for future research to examine these associations prospectively in a twin sample.

Second, our sample was relatively small for twin analyses and replication in a larger sample would be useful in terms of generalizability. As mentioned, limited statistical power may have reduced our ability to detect significant shared environmental influences (C) on these constructs, and the ability to differentiate additive (A) and non-additive (D) genetic influences. Our sample size may also have led to low power to examine gender differences in these associations

Third, our measures of rumination, self-reflection, and some measures of psychopathology (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 conducted by interviewers, which are less prone to this limitation.

Fourth, although the twin design provides a powerful method to examine rumination as a transdiagnostic 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. However, 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.

Conclusions

The results of the present study suggest that rumination is associated with several forms of psychopathology, including depression, anxiety, substance use, 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 traits. Specifically, rumination was genetically correlated most with depression, somewhat with eating disorders, and least with substance use disorders. As the first behavior genetic study to examine rumination as a transdiagnostic risk factor for psychopathology, this study provides a strong foundation for exploring new avenues of research that could guide prevention and treatment efforts for individuals suffering from psychiatric disorders.

Study 2

Developmental Risk Factors for Rumination

Although rumination is included in many theories of depression and psychopathology (e.g. Hankin & Abramson, 2001; Nolen-Hoeksema & Watkins, 2011; Nolen-Hoeksema et al., 2008), only a relatively small literature has examined the effects of an individual's characteristics and environmental contexts on the development of rumination. Theories of depression risk highlight the fact that cognitive vulnerabilities to depression are influenced by biological and developmental contextual factors. Evidence supporting this view has been found for other cognitive vulnerabilities to depression, indicating several developmental events, environmental contexts (for reviews see Alloy et al., 2004; 2006) and biological mechanisms (for a review see Disner et al., 2011) that are associated with cognitive vulnerability to depression. However, despite the considerable interest in rumination as a pervasive risk factor for depression and other forms of psychopathology, the literature examining developmental risk factors for rumination is surprisingly sparse.

In children, a parenting style of "affectionless control," which refers to a pattern of parental rejection and overcontrol, has been found in several studies to be associated with other cognitive vulnerabilities (e.g. negative attributional style; self-criticism; low self-esteem; Alloy et al, 2004 for a review), and may predict rumination later in life (Hilt et al., 2011; Spasojevic & Alloy, 2002). Researchers have theorized that affectionless control involves attempts to suppress children's expression of their thoughts and emotions, and may deprive children of opportunities to develop effective emotion-regulation strategies (e.g. Hilt et al., 2011). Using rumination in an effort to self-regulate may be a consequence of these environmental restrictions on emotional expression (Spasojevic & Alloy, 2002).

In addition to parenting variables, researchers have examined individual characteristics and experiences that may be associated with rumination. Studies have found positive associations between temperament (e.g., negative emotionality) and personality (e.g., neuroticism) and rumination and other cognitive vulnerabilities (Hankin et al., 2007; Mezulis et al., 2006). Furthermore, two studies have found evidence that rumination may mediate the association between neuroticism and depressive symptoms (Roberts et al., 1998; Roelofs et al, 2008). Temperament and personality are heritable constructs (for a review, see Nigg, 2006), so these results are particularly interesting, as they may suggest that rumination serves as a mechanism by which biologically influenced traits established early in development assert their influence on an individual's risk for depression.

Finally, stressful experiences in childhood and adolescence have been shown to be associated with cognitive vulnerabilities to depression, including rumination. Several studies have shown that experiencing maltreatment in childhood (e.g., emotional abuse; sexual abuse) is associated with higher rumination levels in adulthood (Conway et al., 2004; Sarin & Nolen-Hoeksema, 2010; Spasojevic & Alloy, 2002). Additionally, a recent study from our group (Johnson, Carr & Whisman, 2015) found that perceptions of exposure to inter-parental conflict in childhood were associated with rumination in early adulthood controlling for parenting styles, suggesting that even stressful environmental contexts in childhood that do not have direct behavioral influences on children (as parenting styles and abuse do) may increase risk for rumination later in life. Finally, exposure to stressful events in adolescence (Michl et al., 2013), and adulthood (Michl et al., 2013; Moberly & Watkins, 2008) has been associated with subsequent rumination. Using an experience sampling design, Moberly and Watkins (2008) found that engaging in rumination partially mediated the association between negative events

and negative affect experienced over the course of a day. More recently, Michl and colleagues (2013) found that exposure to negative life events in adolescence and adulthood predicted greater rumination months after the events.

These lines of research provide a strong framework to examine risk for rumination; however, there are several limitations of the research to date that should be addressed. First, the majority of studies discussed have examined cognitive vulnerabilities to depression other than rumination. Given the specific interest in rumination as a transdiagnostic risk factor for psychopathology (Aldao, Nolen-Hoeksema, & Schweizer, 2010; Nolen-Hoeksema & Watkins, 2011) and growing evidence that rumination is linked to genetic and biological mechanisms (Johnson et al., 2014; Moore et al., 2013; Mandell et al., 2014), this literature falls short of elucidating developmental risk factors for rumination specifically.

Second, with the exception of two studies (Hilt et al., 2011; Michl et al., 2013), the existing research examining rumination has been cross-sectional in nature, requiring retrospective reports of early life experiences and prior environmental contexts. There are well-documented limitations of long-term retrospective reporting of life experiences, including rater bias and poor reliability (Monroe, 2008); thus, more prospective studies of risk factors for rumination are needed.

Third, no studies to date have examined the specificity of risk factors for rumination. It is clear that not all self-focused, repetitive thought is associated with increased risk for depression and other negative mental health outcomes. Indeed, non-analytical, experiential self-focus (Watkins & Teasdale, 2004) or "self-reflection" may be a potentially adaptive characteristic that can promote well-being. Researchers have suggested that distinguishing between rumination and other forms of self-focused, repetitive thought remains an important consideration in the field

(Watkins & Teasdale, 2004), and it remains unclear whether certain developmental factors may be associated with rumination specifically, or may simply predispose an individual to be self-reflective in general.

Finally, the vast majority of studies has examined developmental risk factors for rumination at single time points and is thus unable to examine processes of stability and change in these factors that may more specifically predict rumination. Indeed, repeated assessment of purported risk factors and examining change in these factors over time is in line with recommended practices for developmental research (e.g. Curran & Willoughby, 2003; Willet, Singer, & Martin, 1998) and could provide an important contribution to our understanding of rumination risk.

Study 2 will address these limitations by examining the influence of specific developmental factors on the risk for rumination in early adulthood using a prospective, multi-wave sample. We chose to examine theoretically-based risk factors including individual characteristics (i.e., child negative emotionality and neuroticism, parent neuroticism), and stressful life contexts and events (i.e., parenting styles, parental relationship satisfaction, family conflict and cohesion, negative life events). Based on the literature suggesting these factors are associated with other cognitive vulnerabilities to depression, and, in some instances, with rumination, we hypothesized these developmental factors would predict rumination in early adulthood. Specifically, for contextual factors, we hypothesized that rumination would be predicted by: greater relationship dissatisfaction in parents, higher ratings of strict/overcontrolling parenting and lower ratings of warm/respectful parenting, greater family conflict and lower family cohesion, and more negative life events. For individual characteristics, we hypothesized that rumination would be predicted by higher ratings of child negative

emotionality, child neuroticism, and parent neuroticism. In order to evaluate the specificity of these developmental risk pathways to rumination, we also examined the extent to which these developmental factors predicted self-reflection in early adulthood.

Examining these hypotheses in a prospective, multi-wave sample can contribute to a developmental model of risk for rumination and enhance models of risk for psychopathology. Results of this investigation may also inform prevention and intervention efforts aimed at reducing rumination and its detrimental effects. For example, identifying developmental risk factors for rumination, such as a stressful family environment, could guide intervention efforts by promoting family-based interventions as strategies for reducing rumination in parents and children (Hilt et al., 2012; Saltzman & Goldin, 2008).

Method

Study participants

Data analyses for Study 2 were conducted on data from participants enrolled in the Longitudinal Twin Study (LTS) who also participated in the Executive Function and Self Regulation (EFSR) follow-up study. The LTS is a sample of same-sex twin pairs recruited through the Colorado Department of Health born between 1986 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. Data were analyzed from the 479 families that participated in the LTS study.

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, Gonzalez, and Nolen-Hoeksema (2003). Treynor et al. (2003)

eliminated RRS items overlapping substantially with items on depression inventories and factor analyzed 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. Both factors are associated positively 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).

Second, the Rumination-Reflection Questionnaire (RRQ; Trapnell & Campbell, 1999) is a 24-item measure that is used to measure two types of self-focused thought: rumination and reflection. Reflection on this measure 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. Conversely, rumination on this scale, 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).

It is important to note that the RRQ-Re measures self-focused thought that is based in self-awareness and curiosity, not necessarily in reaction to or in the presence of 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. These measures were completed when the twins were in

adulthood (age range 21-28 years).

Negative emotionality. Two observational measures of negative emotionality were collected when the twins were 14, 20, 24, and 36 months: Negative Hedonic Tone and Frustration. Negative Hedonic Tone (Easterbrooks & Emde, 1983) is measured by rating the child's strongest negative affect during the administration of Bayley Scales of Infant Development (Bayley, 1976; four five-minute segments) and free play (15 minutes).

Frustration was assessed by examining the children's reactions to having an attractive toy removed from them or being restrained in a variety of situations. In the toy removal procedure, a toy was abruptly taken away from the child after the child was intently involved with the toy for two minutes. Children were restrained as the examiner put on an identifying vest or bib on the child and as the child was measured and instructed to lie still, typically up to 20 seconds. Restraint also was assessed while three small electrodes were attached to the child's chest for heart-rate monitoring.

Three parent report measures were used to assess child negative emotionality at 14, 20, 24, and 36 months. The mood scale from the Toddler Temperament Survey (TTS; Fullard et al., 1984) includes 13 items regarding children's moodiness or fussiness during a range of daily situations. The Differential Emotions Scale (DES; Izard et al., 1980) assesses the frequency of basic emotions with a negative hedonic valence. The Colorado Childhood Temperament Inventory (CCTI; Rowe & Plomin, 1977) emotionality scale includes 5 items regarding children's general emotionality.

Consistent with theory of temperament and personality, evidence from a recent study using these data suggests that the measures capture a single latent construct of negative emotionality that is reliable across time (Rhee et al., 2012). Given this evidence, we used a latent

variable approach when testing our hypotheses that include negative emotionality.

Personality. The Eysenck Personality Questionnaire (EPQ; Eysenck & Eyesenck, 1963). The EPQ is a well-validated self-report questionnaire that assesses three domains of personality: Psychoticism, Extraversion, and Neuroticism. Parents completed a short-form of the EPQ when the twins were age 5, and twins completed the short-form of the EPQ at age 12 and 17. The Neuroticism scales were used in the current study.

Parenting style. The Parental Attitudes Questionnaire (PAS) is a 68-item questionnaire adapted from the Parental Attitudes Toward Childrearing questionnaire (Easterbrooks & Goldberg, 1990; Goldberg & Easterbrooks, 1984; 1988;) that was completed by a parent when the twins were 5 years old. It is a self-report assessment of three dimensions of childrearing: warmth/respect, strictness/over-protectiveness, parent-child conflict and anger. Items measure assessment of attitudes, beliefs, and behaviors associated with childrearing. The reliability of subscales has been shown to be adequate in other samples (average Chronbach's α = .69; Goldberg & Easterbrooks, 1984).

Parent relationship satisfaction. The Dyadic Adjustment Scale (DAS; Spanier, 1976) was used to assess the mothers' and fathers' satisfaction with their romantic relationships. Fifteen of the 32 items focus on agreement between partners on topics such as demonstrations of affection, aims, goals, and things believed important; 15 items focus on the frequency of relationship behaviors, such as arguments and confiding in one's partner; and 2 items measure the partner's overall degree of happiness with and commitment to the relationship. The DAS is widely used and has well-established reliability and validity (e.g. Carey et al., 1993). The DAS was completed by the mother and father (separately) when the twins were ages 14, 36, and 60

months. In the current study, the items were recoded such that higher scores indicate greater dissatisfaction.

Family environment. The Family Environment Scale (FES; Moos & Moos, 1981) was used to assess family environment. The parent form of the FES was completed by parents annually when the twins were age 9 to 15 and the child form of the FES was completed by the twins annually from age 9 to 15.

Life events. The Life Events Scale for Adolescence (LESA; Graber et al., 1995) includes 54 questions about life events in the domains of family, school, and peer events and was administered to participants annually from ages 9 to 16 asking about the frequency and impact (e.g., unpleasant/pleasant) of events in the past year. Impact ratings of all 54 items were assessed across participants to identify events that were generally seen as negative by adolescents. Using a method developed by Trombello, Schoebi, and Bradbury (2011), 95% confidence intervals were calculated for each item based on the impact ratings made by the entire sample of adolescents, and items that had a confidence interval below 4 (neutral) at every time point were deemed negative. Only unpleasant, or "negative" life events were included in this analysis. Consistent with prior work from our group (Johnson et al., 2012, 2013), dependency of events was assessed by a panel of independent raters using a Likert-type scale to determine the status of an event (dependent or independent), with higher scores reflecting the event was very likely to be due, at least in part, to the individual's behavior (Hammen, 1991). With good inter-rater reliability ($\kappa = .74$), 19 negative life events were rated as dependent and 10 negative life events as independent.

Analyses

All analyses were conducted on raw data and allowed missing data. All structural

equation modeling, both phenotypic and genetic, were implemented using Mplus 7 (Muthén & Muthén, 2013). Mplus allows the analyses of a combination of ordinal and continuous data. Given that there were missing observations across time periods, the TYPE=MISSING command was used; this means that the model were estimated using the missing at random missing data method (Little & Rubin, 2002), which allows missingness to be a function of observed covariates and observed outcomes. When conducting phenotypic analyses, non-independence of the data should be considered, given that the data from the two twins in each pair are correlated. Therefore, the TYPE=COMPLEX in the ANALYSIS command was used in order to take into account non-independence of observations when computing standard errors and model fit. The MLR estimator provides maximum likelihood parameter estimates with standard errors and chi-square test statistics that are robust to non-normality. When analyses included both continuous and ordinal variables, the weighted least square mean and variance (WLSMV) estimation method was used. When WLSMV is used, Mplus uses pairwise deletion. Statistical significance of the parameters was determined by their *p* values and by chi square difference tests.. 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 greater than .95 and RMSEA less than .06 indicate good model fit (Hu & Bentler, 1998).

Examining the influence of developmental factors on the risk for rumination was completed in several steps. First, phenotypic associations between the risk factors and rumination measures were examined to determine which developmental factors should be examined in subsequent analyses. For developmental factors measured only at one time point, linear regressions were conducted to examine whether these constructs predicted rumination in

adulthood. For developmental factors with repeated measures, descriptive statistics were examined to determine which measurement method would best represent the data (e.g. composite variable, latent variable model, latent growth model). To test the specificity of these developmental pathways to rumination, the association between developmental factors and self-refection was also examined.

Results

Descriptive Statistics

Descriptive statistics for developmental factors, rumination measures, and the self-reflection measure are presented in Table 8. Several variables were not significantly associated with the rumination measures or showed modest and inconsistent associations with rumination across time points and therefore are not included in Table 8 or included in further analyses (see Appendix 5 for correlations between all study variables and rumination/self-reflection measures). The PAS warmth/respect (r = -.07 – .01, all p values > .21) and conflict/anger (r = -.02 – .02, all p values > .61) subscales were uncorrelated with rumination measures and the strict/overprotective subscale was modestly associated with RRS-B (r = .10, p = .05), but uncorrelated with other measures (r = -.04 – .05; all p values >.37). Observed negative emotionality was uncorrelated with rumination at all time points (r = -.01 – .04; all p values >.20) and parent-rated negative emotionality was modestly associated with RRS-B at the 24-month time point (r =.10, p = .01), but otherwise was not significantly associated with rumination measures (r = -.01 – .06; all p values >.17). In general, negative independent life events were not significantly associated with rumination measures. At age 13, there were significant associations between independent life events and all three rumination measures (r = .10 – .13), but independent events at all other time points were generally unassociated with

rumination and at very modest magnitudes (r = -.03 – .09; all p values >.04). Parent ratings of family cohesion (r = -.09 – .03; all p values >.20) and family conflict (r = -.09 – .00; all p values >.12) were uncorrelated with rumination measures. The adolescent ratings of family conflict were significantly correlated with RRS-B and RRQ-RU at age 15 only (r = .10 and .12) and otherwise were uncorrelated with rumination measures (r = -.04 – .07; all p values >.11).

Table 8

Means and Standard Deviations (continuous variables) and Percentage of Sample in Each Bin (ordinal variables) for Risk Factors, Rumination and Self-reflection

Panel A

Contextual Factors	9 years	10 years	11 years	12 years	13 years	14 years	15 years	16 years
Twin rated								
FES-Cohesion	15.42 (3.28)	15.52 (3.04)	15.78 (3.00)	15.04 (2.84)	15.91 (2.71)	15.78 (2.80)	15.74 (2.66)	--
NDLEs[a]				0 – 10	0 – 14	0 – 13	0 – 12	0 – 09
	0 – 11	0 – 14	0 – 13	1 – 29	1 – 34	1 – 31	1 – 28	1 – 29
	1 – 28	1 – 35	1 – 36	2 – 37	2 – 34	2 – 37	2 – 35	2 – 36
	2 – 33	2 – 32	2 – 31	3 – 24	3 – 17	3 – 20	3 – 20	3 – 25
	3 – 28	3 – 20	3 – 20	0 – 40	0 – 49	0 – 53	0 – 61	0 – 48
NILEs[b]				1 – 32	1 – 31	1 – 32	1 – 26	1 – 33
	0 – 27	0 – 35	0 – 40	2 – 28	2 – 20	2 – 15	2 – 12	2 – 19
	1 – 35	1 – 35	1 – 33					
	2 – 38	2 – 30	2 – 27					

Parent Rated	14 months	36 months	60 months
Mother DAS	37.93 (16.00)	40.40 (17.49)	41.20 (18.39)
Father DAS	37.88 (14.85)	37.70 (15.41)	36.54 (13.21)

Panel B

Individual Characteristics	14 months	36 months	60 months	12 years	17 years
Mother EPQ-N	9.96 (4.83)	9.96 (4.68)	8.99 (4.77)	--	--
Father EPQ-N	7.99 (4.50)	7.39 (4.56)	6.71 (4.25)	--	--
Child EPQ-N	--	--	--	.41 (.24)	.39 (.23)

Panel C

Rumination and Self-Reflection	Males	Females
RRS-B[c]	1.87 (.57)	2.04 (.64)
RRS-R[c]	1.95 (.68)	2.13 (.70)
RRQ-RU[c]	2.69 (.72)	2.93 (.75)
RRQ-RE	3.13 (.72)	3.10 (.77)

Note. [a]0 = 0 events; 1 = 1-2 events; 2 = 3-5 events; 3 = more than 5 events; [b]0 = 0 events; 1 = 1 event; 2 = more than 1 event; [c]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; FES = Family Environment Scale; NDLEs = Negative Dependent Life Events; NILEs = Negative Independent Life Events; DAS = Dyadic Adjustment Scale (higher scores reflect greater relationship dissatisfaction); EPQ-N = Eysenck Personality Questionnaire – Neuroticism.

Rumination measures and the self-reflection measure were normally distributed, with acceptable skewness and kurtosis values (between 1.00 and -1.00). The distribution of DAS scores was skewed, so scores were square root transformed to achieve a normal distribution for subsequent analyses. Distribution of frequency counts of life events at each time point was highly skewed; thus, both categories of life events were binned into ordinal variables, with the assumption that a continuous, normal liability distribution underlies the ordinal variables. As there is theoretical interest in the gender differences in rumination, we examined gender

differences throughout the analyses. Men had significantly lower rates of rumination on all three rumination measures.

Rumination Latent Variable

The three measures of rumination were moderately to highly correlated with each other. The self-reflection measure (RRQ-RE) was significantly correlated with measures of rumination, but these associations were modest in magnitude. We used the three rumination measures as indicators of a rumination latent variable (RLV), given the considerable overlap between them. Each indicator loaded significantly on the latent factor. There were significant gender differences ($\chi^2_{diff}[3] = 12.23$, $p = .01$) between the factor loadings for men and women; however, these gender differences were small and yielded a similar pattern, suggesting that the RLV was qualitatively similar for men and women on a phenotypic level. Nonetheless, subsequent analyses were conducted using the RLV allowing the factor loadings to differ between males and females.

Associations Between Developmental Factors, Rumination and Self-reflection

The associations between risk factors, the individual rumination measures, and self-reflection at all available time points are presented in Appendix 5. These associations provided justification to examine these developmental factors further as risk factors for rumination. The RLV was used in all subsequent analyses rather than the RRS-B, RRS-R, and RRQ-RU individually. Gender differences in the associations were examined in each association analysis by comparing a model with separate parameters for men and women to a model with the parameters fixed to be equal for men and women.

Individual Characteristics

Parent neuroticism. EPQ-N ratings completed by parents across time points were highly

correlated within rater (r = .73 – .82) and means were similar across time points, suggesting stability across time and consistent with literature on the stability of personality factors in adulthood (e.g. Costa & McRae, 1988, Viken et al., 1994). Given this evidence of stability, composite variables were created by averaging scores from mothers and fathers across time points. As seen in Table 9, mother neuroticism and father neuroticism early in the child's life were both modestly associated with offspring rumination in adulthood, and these findings did not differ by twin gender. Neither mothers' nor fathers' neuroticism significantly predicted self-reflection.

Table 9
Associations between Rumination, Self-Refection and Developmental Risk Factors

Risk Factors	With RLV		with RRQ-RE	
	Males	Females	Males	Females
Individual Characteristics				
Mother EPQ-N[a]	**.15 (.05)**	**.17 (.06)**	-.01 (.05)	-.01 (.04)
Father EPQ-N[a]	**.11 (.06)**	**.12 (.06)**	-.01 (.05)	-.01 (.05)
Twin EPQ-N age 12	**.21 (.04)**	**.23 (.04)**	**.14 (.05)[d]**	-.01 (.05)[d]
Twin EPQ-N age 17	**.29 (.04)**	**.31 (.05)**	**.12 (.05)**	**.12 (.04)**
Contextual Factors				
Mother DAS[a]	**.13 (.05)**	**.15 (.05)**	.01 (.06)	.01 (.05)
Father DAS[a]	**.18 (.06)**	**.19 (.06)**	.01 (.07)	.01 (.06)
FES Cohesion (9-13)[b]	-.01 (.07)[d]	**-.18 (.07)[d]**	.07 (.05)	.07 (.03)
FES Cohesion (14-15)[b]	**-.19 (.06)**	**-.19 (.06)**	.05 (.05)	.04 (.05)
NDLEs[c]				
Intercept (males)	.15 (.09)	--	**.22 (.09)**	--
Slope (males)	-.02 (.11)	--	-.02 (.13)	--
Intercept (females)	--	**.17 (.07)**	--	.10 (.08)
Slope 1 (females)	--	-.01 (.11)	--	.04 (.10)
Slope 2 (females)	--	**.17 (.09)**	--	.05 (.08)

Note. [a]Composite variable (average all across time points with available data); [b]latent variable (best fitting model from confirmatory factor analysis); [c]latent Growth Curve model (traditional for males, piecewise for females); [d]significant gender difference. RLV = Rumination Latent Variable; RRQ-Re = Rumination-Reflection Questionnaire-Reflection; FES = Family Environment Scale; NDLEs = Negative Dependent Life Events; DAS = Dyadic Adjustment Scale (higher scores reflect greater relationship dissatisfaction); EPQ-N = Eysenck Personality Questionnaire – Neuroticism.

Child and adolescent neuroticism. Results suggested that neuroticism at age 12 and age 17 both significantly predicted rumination in adulthood, and these associations did not differ by gender. Notably, neuroticism at age 12 (for boys only) and at age 17 (for both genders) predicted self-reflection as well, suggesting that unlike parent personality, twin personality may not be differentially related to rumination and adaptive self-focused thought.

Contextual Factors

Parent relationship satisfaction. Similar to parent EPQ-N ratings, correlations among the parent DAS assessments were moderate to high within rater and across time (r = .58 - .78), suggesting some stability of relationship satisfaction over time. However, the means suggested that mothers' relationship dissatisfaction might increase over time. In order to examine whether this increase was significant and related to the outcomes, we conducted a latent growth model. In a latent growth model, a latent Intercept and latent Slope is estimated for the variable using available data across all available assessments. The Intercept variable represents the variance that is stable with the initial levels, and the Slope represents change over time. Results suggested there was significant growth for mothers' DAS score, but this increase in relationship dissatisfaction was not associated with rumination. This result, in conjunction with the moderate to large correlations across time points, guided our decision to compute composite variables for mother and father DAS by taking the average of ratings across time points. Associations between rumination and mother and father DAS were examined in separate models. Results are presented in Table 9, and suggest relationship dissatisfaction in fathers and mothers predicted rumination in offspring. Conversely, parent relationship dissatisfaction did not predict offspring self-reflection.

Family cohesion. Adolescent-rated FES Cohesion scores were negatively correlated with rumination measures at some time points, and this was particularly true for assessments

completed during mid-adolescence (ages 14 and 15). The latent growth curve model did not fit these data well and provided no evidence that there was a significant increase or decrease in family cohesion over time. Factor analyses were then conducted and results suggested support for a two-factor model with correlated factors. The first latent factor loaded on ages 9 through 13 and reflected family cohesion in late childhood/early adolescence, and the second latent factor loaded on ratings for ages 14 and 15, and reflected family cohesion in mid-adolescence. Rumination was then included in these models to examine the extent to which these factors predicted rumination in adulthood. These models fit the data well (χ^2 [80] = 93.93; p = .14; TLI = .99; RMSEA = .02). Family cohesion in late childhood/early adolescence (for girls only; (χ^2_{diff}[1] = 3.95, p = .05) and mid-adolescence (for boys and girls) was negatively associated with later rumination, suggesting this may serve as a protective factor for the development of ruminative thinking. Self-reflection was not significantly predicted by family cohesion.

Negative dependent life events. Negative dependent life events were positively correlated with rumination measures at most time points. Results from the latent growth curve model indicated significant individual differences in change of NDLEs over time. For boys, there was an overall decrease in NDLEs across adolescence, which was adequately captured by a single latent slope (χ^2 [54] = 96.70; p < .01; TLI = .96; RMSEA = .04, for the final model including rumination). However, this model did not fit well for girls, and examining the pattern of factor loadings suggested there was evidence of two distinct trajectories of NDLEs in girls. Thus, a piecewise growth model, which includes multiple latent slopes reflecting different patterns of growth over time (Kohli & Harring, 2013), was conducted. This model fit the data well (χ^2 [55] = 85.73; p = .01; TLI = .98; RMSEA = .04, for the final model including rumination); the first slope captured a decrease in NDLEs from age 9 to age 12, and the second slope captured an

increase in NDLEs from age 12 to age 16.

In boys, rumination was not predicted by initial levels of NDLEs (latent Intercept) or by the decrease in NDLEs over time (latent Slope). In girls, initial levels of NDLEs and increases in NDLEs in mid adolescence (second latent Slope) predicted rumination in adulthood, whereas decreases in NDLEs in late childhood did not (first latent Slope). Change in NDLEs over time did not predict self-reflection in boys or girls. Initial levels of NDLEs did not predict self-reflection in girls either; however, in boys, this association was significant, suggesting higher initial levels of NDLEs predicted self-reflection in adulthood.

Discussion

This is one of the first studies to examine multiple developmental risk factors for rumination in a longitudinal, prospective sample. Several results have important implications for our understanding of the individual characteristics and contextual factors that may predispose an individual to ruminate. First, stressful environmental contexts assessed across development (infancy and toddlerhood, late childhood, and adolescence) were associated with rumination in adulthood. Specifically, parental relationship dissatisfaction early in life and dependent stressful life events in late childhood and adolescence were associated with rumination in adulthood. Additionally, perceptions of family cohesion in adolescence were negatively associated with rumination in adulthood, suggesting this environmental context may serve as a protective factor. Second, neuroticism in parents and children themselves was associated with rumination in adulthood. Third, results suggested that some developmental factors may predispose individuals to self-focused thought generally (rumination and self-reflection), whereas others increase risk for rumination specifically. Fourth, several empirically supported risk factors for other cognitive vulnerabilities to depression (e.g., negative emotionality, parenting styles) were not significantly

associated with rumination in adulthood, suggesting that unique mechanisms of risk may influence rumination.

Contextual Risk Factors for Rumination

Our findings suggest that several environmental contexts may increase (or decrease) risk for rumination in adulthood. First, offspring were more likely to ruminate as adults if their parents reported marital dissatisfaction when twins were infants and toddlers. To our knowledge, this is the first study to examine the effects of the parent relationship on offspring rumination in a longitudinal sample. These results align with the large body of research examining a related risk factor for developing ineffective coping styles and behavioral problems: exposure to inter-parental conflict (IPC). Though only one study has examined IPC as a predictor of rumination in offspring specifically (Johnson, Carr, & Whisman, 2015), a wide body of research has shown that children who are exposed to IPC in childhood are more likely to develop ineffective coping strategies, and subsequently, are at greater risk for behavioral and emotional problems (e.g. Davies & Cummings, 1994; Goeke-Morey et al., 2013; Rhoades, 2008). Though the DAS is not a measure of IPC per se, our results suggest that parents experiencing a dissatisfying relationship seem to influence their children's risk for developing an ineffective coping strategy (i.e. rumination) even 20 years later. Research suggests that children learn to engage in rumination as a way to "turn inward" to cope with environmental stress (Spasojevic & Alloy, 2002), and this could apply to witnessing parents in a dissatisfying relationship. To the extent that parents in a dissatisfying relationship express more negative emotion and display poor coping strategies and high stress reactivity in the home environment, their children are at greater risk for developing poor emotion regulation skills (for a review see Sheffield Morris et al., 2007). This interpretation is also consistent with behavior genetic studies suggesting that the influence of poor parent

relationship quality on children's internalizing symptoms is due to direct environmental influences, rather than gene-environmental interplay (e.g. Schermerhorn et al. 2011).

We also found that exposure to negative dependent life events (NDLEs) in adolescence increased risk for rumination in adulthood, particularly for girls. This finding supports those from a recent study by Michl and colleagues (2013), which reported that adolescents and adults who experienced more stressful life events reported greater rumination several months later. Our results extend this finding to suggest that exposure to NDLEs in adolescence is associated with greater rumination years after the transition into adulthood. Additionally, our study is the first to examine the effects of stability and growth in NDLEs on later rumination. Findings suggested that both initial levels of NDLEs in late childhood as well as increases in NDLEs in mid-adolescence predicted later rumination in girls. This result suggests that the effects of NDLEs on rumination are not static, but rather, rumination in adulthood may be linked to experiencing a pattern of increasing stress during the critical developmental transition from childhood to adolescence.

The association between NDLEs and rumination was not significant in boys (though the association between the NDLE intercept and rumination was marginally significant). This finding is inconsistent with the absence of gender differences in two prior longitudinal studies that examined stressful life events as a risk factor for cognitive vulnerabilities to depression (Garber & Flynn, 2001; Michl et al., 2013). However, an important consideration is the use of growth models in our study, which prior studies have not examined. The best fitting model for boys indicated a decrease in NDLEs over time; thus, it is sensible that a decrease in a potential risk factor over time was not associated with later rumination in boys. This interpretation is buttressed by the fact that there were no gender differences in the positive associations between

NDLEs and rumination when latent variables (not including growth factors) were applied to the data (r = .15 and .24 for the two NDLE latent variables). The gender difference in growth models speaks to the importance of examining rates of change in developmental risk factors, rather than treating the factors as static (e.g., Curran & Willoughby, 2003; Willet, Singer, & Martin, 1998). Our findings suggest the increase in NDLEs across adolescence is an important consideration in girls' risk for rumination, but less so for boys.

The third environmental context that was significantly associated with rumination was family cohesion, such that greater family cohesion in adolescence was negatively associated with rumination in adulthood. Prior research has generally supported a cross-sectional association between low family cohesion and psychopathology in adolescents (e.g., Prange et al., 1992) and prospective associations for later offspring psychopathology, even when controlling for parent psychopathology (Nomura et al., 2002; Pilowsky et al., 2006). Our study is the first to show that low family cohesion is associated with later rumination, suggesting that rumination may act as a mechanism by which low family cohesion affects risk for psychopathology. This interpretation is consistent with theories of the development of emotion regulation strategies that have found that family contextual factors such as the family's emotional expressiveness, whether parents encourage effective emotion regulation strategies, and the strength of parent–adolescent relationships, are associated with youth's emotion regulation (for a review see Sheffield Morris et al., 2007). Individuals may be less likely to engage in rumination —which is conceptualized as an ineffective emotion-regulation strategy — if they perceive their family environment as one in which "togetherness" and "group spirit" are encouraged and "family members really back each other up" (items from the FES-Cohesion scale). As parent ratings of family cohesion were not associated with later rumination, the protective mechanism detected in our study may reflect the

adolescent's *perception* of family cohesion. Future research extending this finding to parent report or objective measures of components of family cohesion (e.g. parent-child interactions, emotional expression and communication tasks) will be important to verify family cohesion as an environmental protective factor for rumination.

Individual Characteristics

Individual characteristics also predicted later rumination, including parent neuroticism and child neuroticism. Although several studies have shown that personality characteristics are associated (but separable) from rumination (Hankin et al., 2007), our findings suggest that parent personality also predicts offspring rumination. This effect can be interpreted in several ways. First, parents who experience high levels of neuroticism may influence children's rumination by creating environmental stressors through overcontrolling parenting, modeling ineffective emotion regulation, and expressing negative emotion towards family members. A second interpretation incorporates the well-replicated finding that neuroticism is heritable (e.g. Middeldorp et al., 2005; Viken et al., 1994). In this case, a parent may indeed shape the child's environment through "neurotic" behaviors, but also, may pass on genes to the child that also increase the child's risk for neuroticism and related behaviors, such as rumination and depression. This second interpretation is consistent with behavior genetic research on gene–environment correlation (Plomin, DeFries, & Loehlin, 1977; Scarr & McCartney, 1983), which provides a promising theoretical framework in which to explore the link between neuroticism and rumination.

Developmental Factors Not Associated with Rumination

Several theoretically-based "candidate" risk factors for rumination did not predict rumination in our study. Three prior studies have found that overcontrolling parenting predicts

rumination (Hilt et al., 2011; Johnson et al., 2015; Spasojevic & Alloy, 2002), but our measure of parenting styles was not associated with offspring rumination in adulthood. Of the sparse literature on risk for rumination, overcontrolling parenting is one of the most widely referenced risk factors for rumination, in part because of the literature suggesting it is a risk factor for other cognitive vulnerabilities to depression (Alloy et al., 2004). However, two of the three prior studies that have examined this association are potentially confounded by rater bias, as participants were asked to report on their current rumination as young adults and to retrospectively report on the parenting styles they received as children (Johnson et al., in press; Spasojevic & Alloy, 2002). The only prior study to investigate *parent*-rated parenting styles as a prospective risk factor for offspring rumination found a very modest association (r = .13; Hilt et al., 2011). In our study, parent-rated strict/over-protective parenting did not predict later rumination and warm/respectful parenting did not serve as a protective factor. Of course, our null findings may be influenced by methodological limitations of self-report measures of parenting (e.g. Holden & Edwards, 1989) or the long period of time between assessments (~20 years), but they may also suggest that overcontrolling parenting is not as robust a risk factor for rumination as it seems to be for other cognitive vulnerabilities to depression.

Negative emotionality, which has been shown to predict emotion regulation (Sheffield Morris et al., 2007) and personality and psychopathology later in life (for a review see Nigg, 2006), was not associated with rumination in adulthood. This null finding was consistent when examining empirically sound parent-rated and observational measures of negative emotionality independently at different time points, and when applying latent variable models and latent growth curve models to the parent and observational data (see Rhee et al., 2012 for details on the measures). Thus, we believe it is unlikely that this null finding can be explained by measurement

or methodological limitations. Our results suggest that negative emotionality assessed in infancy and toddlerhood is not a significant predictor of rumination later in life. It is possible that negative emotionality interacts with other factors, such as stress exposure or other ineffective coping strategies, to predict rumination and other negative outcomes.

Finally, the result that independent life events were largely unrelated to rumination in this study is an important finding. This result points to the possibility that dependent life events (those that are due in part to an individual's behavior) have a more significant impact on an individual's risk for engaging in rumination than independent life events; similar results have been found for depression (Hammen, 2005). As other studies examining life events and rumination (Michl et al., 2013; Moberly & Watkins, 2008) have not distinguished between independent and dependent life events, this is an important consideration for future research.

Strengths and Limitations

There are several strengths of the current study. First, this is the only study to date examining prospective associations between multiple developmental factors and rumination in adulthood. We included developmental factors assessed via subject's self report, parent report, and observational methods, making this the most comprehensive examination of developmental risk factors for rumination to date. Second, we examined developmental factors at multiple time points across critical stages of development (e.g. infancy, toddlerhood, late childhood, adolescence), enabling us to examine the influence of stability and change in these factors as predictors of rumination, rather than treating them as static factors. Third, we examined multiple measures of rumination and conducted analyses using a latent variable for rumination, so we are confident that our results extend beyond any particular measure of this construct. Finally, we also examined self-reflection in an effort to distinguish between predictors of general self-focused

thought and specific risk factors for rumination.

Our results should be considered in the context of some limitations. First, we only assessed rumination and self-reflection at one time point, and thus, we are unable to determine if individuals had a tendency to ruminate before experiencing certain risk factors. There is evidence that individuals may ruminate as early as childhood and certainly by adolescence (Rood et al., 2009 for a meta-analysis), so the prediction of rumination by certain risk factors in adolescence, such as stressful life events and family cohesion, could be biased if individuals were already ruminating at that time. Of course, this potential bidirectional association does not necessarily negate these environmental contexts as risk factors for rumination; however, we cannot be as confident in the directionality of these associations as we are with those that were measured (presumably) before children engage in rumination (i.e. infancy & toddlerhood). Second, our outcome measures and measures of several risk factors were completed by twins themselves. This introduces the concern of rater-bias effects or method covariance, such that individuals who engage in rumination in adulthood may simply *report* more stressors rather than *experience* more stressors in adolescence, or they may have a negative bias when reporting on family cohesion. It will be important for future research efforts to examine contextual risk factors and rumination using diverse methods, such as "objective" life event interviews (Brown & Harris, 1989), observational assessments of the family environment and, ideally, cognitive tasks to assess processes associated with rumination in addition to self-report measures.

Conclusion

Our findings suggest that stressful environmental contexts and individual characteristics measured across several developmental periods may predispose individuals to ruminate as adults. As this is one of the first studies to examine risk factors for rumination in a longitudinal, multi-

wave sample, we hope these findings serve to guide future research efforts in elucidating the environments, family relationships and characteristics that contribute to the development of a ruminative style of thinking. Insofar as rumination is a transdiagnostic risk factor for psychopathology, these research efforts will also contribute to our understanding of the nature of a range of psychological disorders.

General Conclusion

Researchers and clinicians have emphasized the critical role of rumination in the onset, course, and maintenance of depression, as evidenced by the large body of literature on rumination and the fact that rumination is targeted in several empirically based interventions for depression. Furthermore, burgeoning research suggests rumination may be a transdiagnostic risk factor, increasing risk for a number of forms of psychopathology. However, in contrast to the considerable attention garnered by the effects and alleviation of rumination, much less research examining the influences that contribute the development of a ruminative thinking style and to its associations with psychopathology has been conducted. The present studies were designed and conducted to address these gaps in the literature.

Study 1 is the first to examine the etiology of rumination as a transdiagnostic risk factor and results indicated that the associations between rumination and various forms of psychopathology (depression, eating pathology and substance dependence) have distinct genetic and environmental etiologies. That is, these results support the conceptualization of rumination as a transdiagnostic risk factor, and also suggest that unique biological and environmental mechanisms may link rumination to different disorders. Study 2 is the first longitudinal, prospective investigation of developmental risk factors for rumination in adulthood. The results of this study highlight the role of several specific environmental contexts and individual

characteristics experienced across development (infancy, childhood, and adolescence) that may increase risk for rumination in adulthood. In concert, results of these two studies lay a foundation to examine further the environmental and biological factors that increase risk for rumination and subsequent risk for several forms of psychopathology. Elucidation of the etiological influences on rumination may guide the development and refinement of interventions aimed at reducing rumination, and mitigate rumination's pervasive effects on health and well-being.

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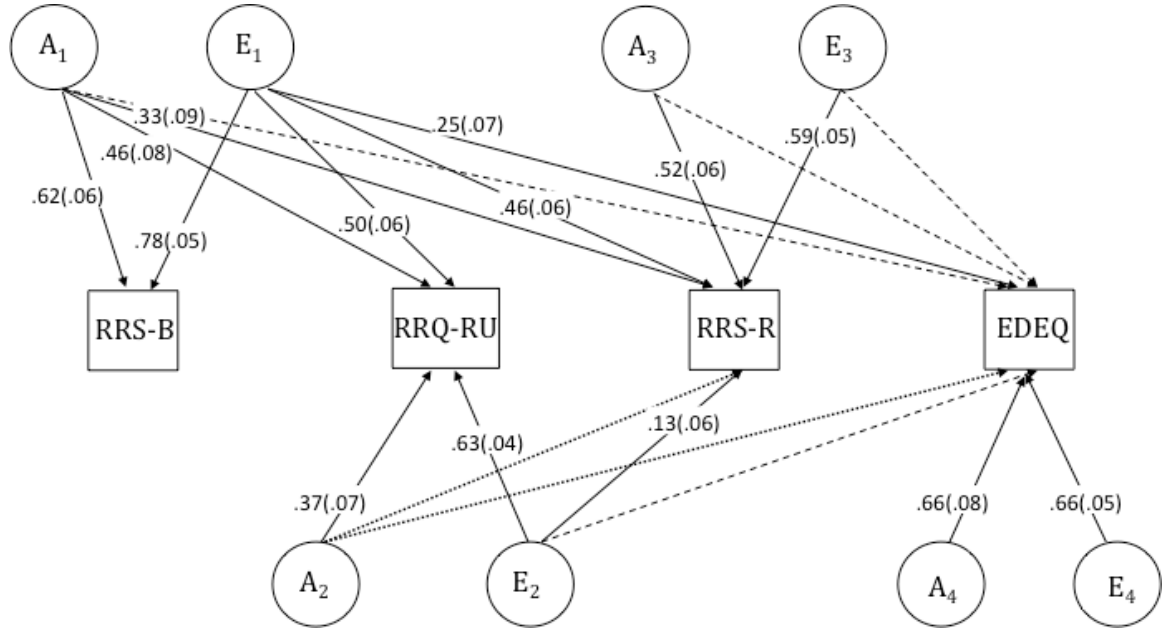
Appendix

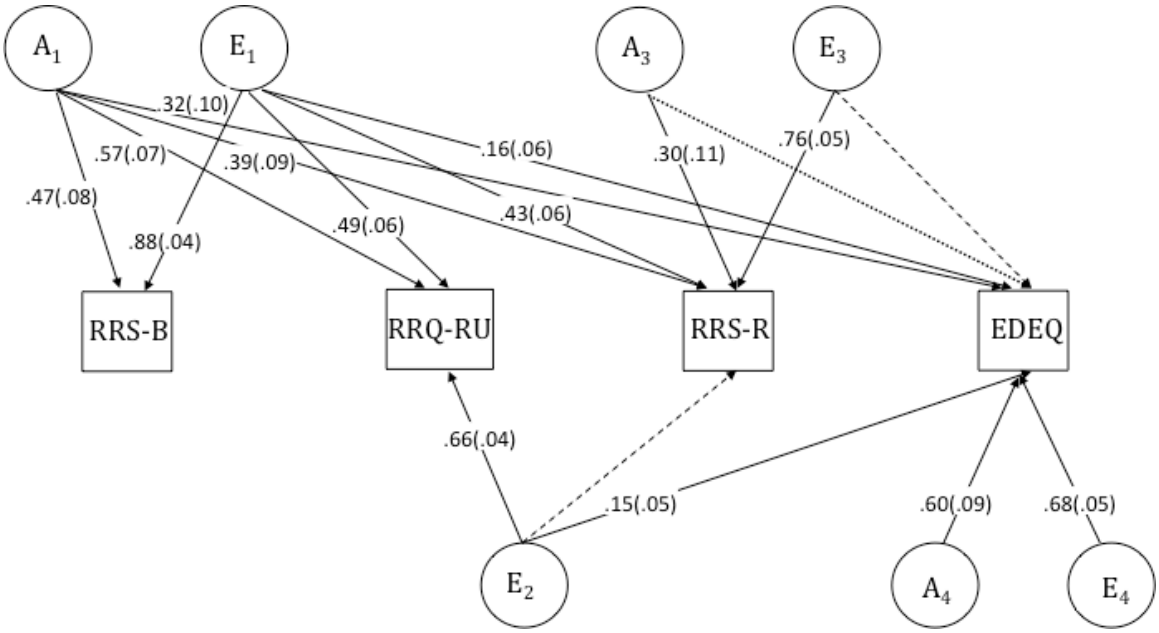
Appendix 1

Reduced Multivariate Models for Rumination Measures and EDEQ for males (Panel A) and females (Panel B)

Panel A



Panel B



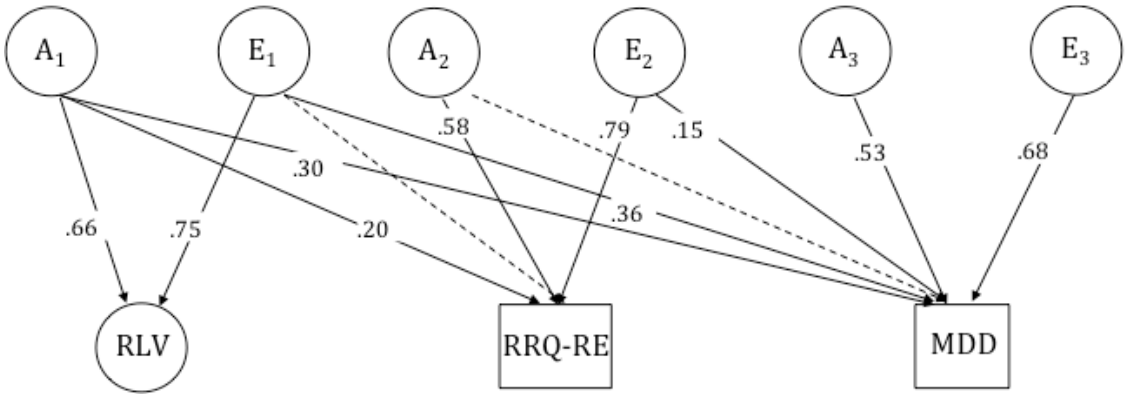
Note. The latent variable (A_2) and paths representing shared genetic variance between RRQRU,

RRSR and EDEQ were dropped from the model due to negligible loadings on the rumination measures. Standardized parameter estimates are shown. Statistically significant parameter estimates are shown and indicated by a solid line; dashed lines indicate nonsignificant parameter estimates with negligible magnitudes (0 ± .10); dotted lines indicate nonsignificant parameter estimates (<-.10 or >.10); RRS-B = Ruminative Responses Scale-Brooding; RRS-R = Ruminative Responses Scale-Reflection; RRQ-Ru = Rumination-Reflection Questionnaire-Rumination; EDEQ = Eating Disorder Examination Questionnaire; A = genetic influences; E = nonshared environmental influences.

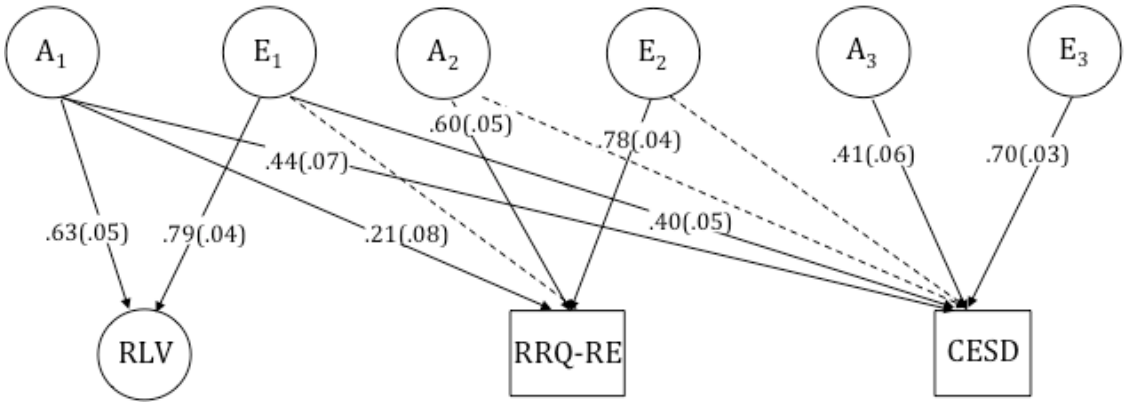
Appendix 2

Reduced Trivariate Models for Rumination, Self-reflection and CES-D (Panel A), MDD (Panel B), DV (Panel C)

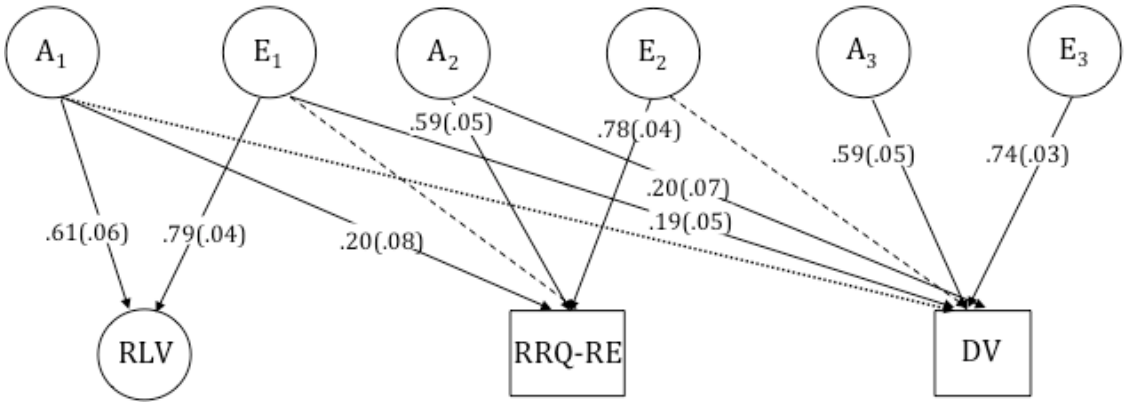
Panel A



Panel B



Panel C



Note. Standardized parameter estimates. Statistically significant parameter estimates are shown and indicated by a solid line; dashed lines indicate nonsignificant parameter estimates with

negligible magnitudes (0 ± .10); dotted lines indicate nonsignificant parameter estimates (<-.10 or >.10); 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; E = nonshared environmental influences. When using the WLSMV estimator (analyses with MDD), Mplus does not provide standard errors for standardized parameters when a covariate is included in the model.

Appendix 3

Variance in Rumination, Self-reflection, and Each Form of Psychopathology Explained by Common and Unique A, C and E Components and Phenotypic Covariance Explained by Common A, C and E Components.

	RLV	RRQ-RE		Psychopathology				Psychopathology Covariance			
	Total Variance	Common with RLV	Unique	Common with RLV	RRQ-RE[a]	Unique	with RLV	with RRQ-RE			
									Common w RLV	Unique	
CES-D											
A	.32	.10	.26	.29	.03	.00	.31		.17	-.09	
C	.07	.03	.01	.02	.01	.00	-.03		.02	-.01	
E	.61	.00	.61	.16	.00	.49	.31		.01	-.01	
Total							.59		.10		
MDD											
A	.44	.04	.15	.09	.01	.07	.20		.06	.03	
C	.00	.00	.17	.00	.00	.17	.00		.00	.00	
E	.56	.00	.62	.17	.02	.49	.27		.02	.12	
Total							.47		.23		
Sex						.02					
DV											
A	.33	.08	.26	.00	.21	.00	.00		.00	.23	
C	.04	.04	.00	.14	.04	.00	.07		-.07	.00	
E	.63	.00	.62	.04	.00	.57	.16		.01	-.05	
Total							.23		.12		

Note. The total variance due to A, C and E for each variable is similar but not identical across different models because parameters are estimated to minimize the difference between the observed and expected covariance matrices in each model. 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]Controlling for influences in common with RLV. Estimates presented in the tables may differ slightly from percentages of variance presented in the text due to rounding error.

Appendix 4

Variance in RRS-B, RRQ-RU, RRS-R and EDEQ Explained by Common and Unique A, C and E Components and Phenotypic Covariance Explained by Common A, C and E Components

	RRS-B	RRQ-RU		RRS-R			EDEQ				EDEQ Covariance with		
	Total Variance	Common with RRS-B	Unique	Common with RRS-B	Common with RRQ-RU[a]	Unique[b]	Common with RRS-B	Common with RRQ-RU[a]	Common with RRS-R[b]	Unique	RRS-B	RRQ-RU	RRS-R
Males													
A	.35	.23	.13	.13	.03	.26	.00	.05	.01	.44	.03	.11	.02
C	.03	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00
E	.62	.25	.40	.22	.02	.35	.06	.01	.00	.43	.19	.07	.10
Total											.21	.18	.12
Females													
A	.14	.23	.00	.22	.00[c]	.00	.04	.00[c]	.00[c]	.37	.08	.09	.09
C	.07	.09	.00	.00	.00	.00	.07	.00	.00	.00	.07	.08	-.01
E	.79	.25	.43	.19	.00	.59	.03	.02	.00	.46	.15	.19	.05
Total											.30	.36	.13

Note. The total variance due to A, C and E for each variable is similar but not identical across different models because parameters are estimated to minimize the difference between the observed and expected covariance matrices in each model. RRS-B = Ruminative Responses Scale-Brooding; RRS-R = Ruminative Responses Scale-Reflection; RRQ-Ru = Rumination-Reflection Questionnaire-Rumination; 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]This path represents shared genetic variance between rumination measures and EDEQ; it was fixed to zero due to negligible loadings on the rumination measures. Estimates presented in the tables may differ slightly from percentages of variance presented in the text due to rounding error.

Appendix 5

Associations between Developmental Factors and Measures of Rumination and Self-Reflection

Panel A

Family Environment Scale Cohesion	RRS-B	RRS-R	RRQ-RU	RRQ-RE
Child				
9 years	-.04	-.06	-.05	-.04
10 years	**-.09**	.01	-.04	.05
11 years	-.07	.02	-.03	.02
12 years	-.*08*	.01	-.03	.06
13 years	-.05	.02	-.06	.*08*
14 years	**-.10**	-.04	**-.12**	.00
15 years	**-.16**	-.*08*	**-.17**	.06
Parent				
9 years	-.05	-.02	-.04	.04
10 years	-.04	-.03	-.03	-.01
11 years	-.09	-.03	.00	-.01
12 years	-.04	-.01	.00	-.03
13 years	-.06	.00	.03	.00
14 years	-.05	.03	-.02	.06
15 years	-.06	.02	-.04	.12
Family Environment Scale Conflict				
Child				
9 years	.06	.00	.03	.00
10 years	.06	-.02	.02	.00
11 years	.01	-.04	.00	-.05
12 years	.07	-.04	.00	-.03
13 years	.05	-.03	.04	-.04
14 years	.05	-.04	.07	-.06
15 years	**.10**	.02	**.12**	-.08
Parent				
9 years	-.06	.01	-.03	.02
10 years	-.08	-.02	-.01	-.02
11 years	-.04	.02	-.02	-.03
12 years	-.05	-.05	-.06	-.03
13 years	-.01	-.04	-.03	-.03
14 years	-.07	-.07	-.09	-.04
15 years	-.04	-.01	-.02	-.04

Panel B

Negative Dependent Life Events	RRS-B	RRS-R	RRQ-RU	RRQ-RE
9 years	**.09**	**.08**	.07	*.08*
10 years	*.08*	.01	.03	**.11**
11 years	.07	.06	.02	.07
12 years	**.11**	*.08*	**.09**	**.16**
13 years	**.14**	.06	**.17**	**.13**
14 years	**.11**	**.11**	**.10**	**.13**
15 years	**.14**	**.13**	**.15**	**.13**
16 years	**.22**	**.16**	**.20**	**.17**
Negative Independent Life Events				
9 years	.02	-.01	.00	-.06
10 years	.06	.03	-.03	.02
11 years	**.10**	.07	.03	.04
12 years	*.09*	.07	.04	**.09**
13 years	**.13**	**.10**	**.10**	.08
14 years	**.10**	*.09*	.04	*.09*
15 years	.02	.05	.03	**.11**
16 years	.07	.05	.01	**.11**

Panel C

Negative Emotionality	RRS-B	RRS-R	RRQ-RU	RRQ-RE
Observed				
14 months	.04	.03	.05	-.06
20 months	.02	-.01	-.01	-.07
24 months	-.01	-.03	.01	-.03
Parent Rated				
14 months	.04	.04	.05	-.05
20 months	.04	.05	.07	.02
24 months	.05	.02	**.10**	.00
36 months	.00	.00	.05	.04

Panel D

Child Neuroticism	RRS-B	RRS-R	RRQ-RU	RRQ-RE
Age 12	**.17**	**.13**	**.20**	.06
Age 17	**.24**	**.19**	**.31**	**.12**

Panel E

Parenting Styles	RRS-B	RRS-R	RRQ-RU	RRQ-RE
Strict/Over-protective	**.11**	-.04	.05	-.07
Warm/Respectful	-.07	.01	-.05	.08
Conflict/Anger	.03	-.03	.02	-.04

Panel F

Parent Relationship Satisfaction	RRS-B	RRS-R	RRQ-RU	RRQ-RE
Mother				
14 months	**.14**	.03	.09	-.03
36 months	**.17**	.07	*.09*	-.01
60 months	**.12**	.07	.07	.01
Father				
14 months	**.18**	.05	.09	-.04
36 months	**.22**	**.15**	**.12**	-.02
60 months	.11	.07	.08	-.01

Panel G

Parent Neuroticism	RRS-B	RRS-R	RRQ-RU	RRQ-RE
Mother				
14 months	*.09*	.00	.08	-.03
36 months	**.13**	.04	**.12**	.07
60 months	**.15**	.06	**.13**	.01
Father				
14 months	**.12**	.05	**.11**	-.03
36 months	*.09*	-.01	.08	-.02
60 months	**.14**	.04	**.14**	.05

Note. Bold indicates $p < .05$; Italics indicates $p < .10$; RRS-B = Ruminative Responses Scale-Brooding; RRS-R = Ruminative Responses Scale-Reflection; RRQ-Ru = Rumination-Reflection Questionnaire-Rumination; RRQ-Re = Rumination-Reflection Questionnaire-Reflection