Predicting the Transition from Anxiety to Depressive Symptoms in Early Adolescence: Negative Anxiety Response Style as a Moderator of Sequential Comorbidity

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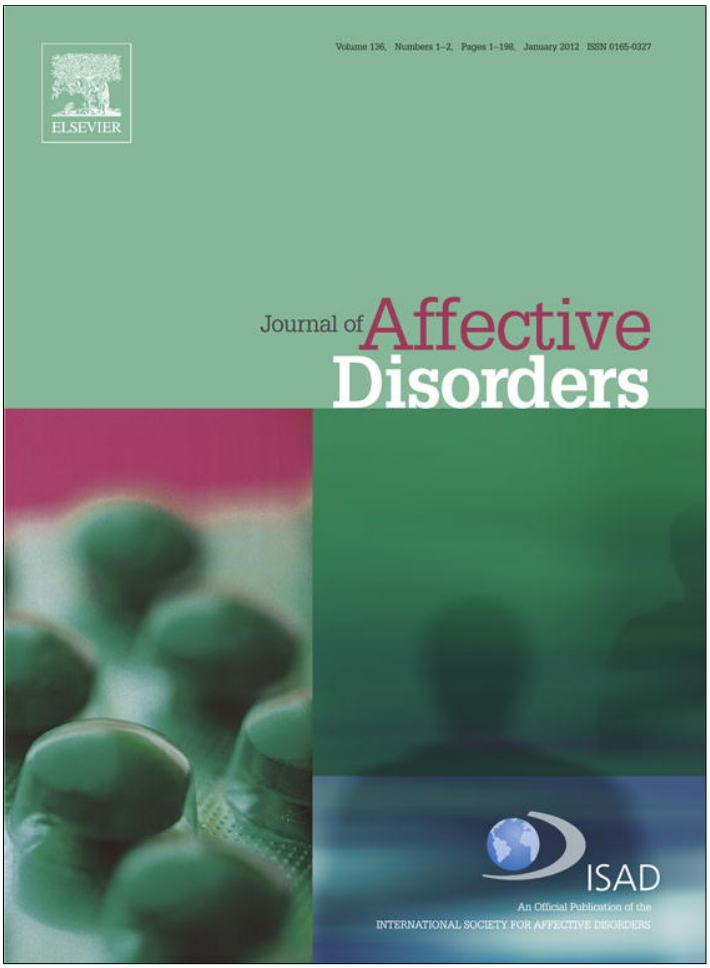

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Research report

Predicting the transition from anxiety to depressive symptoms in early adolescence: Negative anxiety response style as a moderator of sequential comorbidity

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ABSTRACT

Background: Anxiety often precedes depression. The anxiety response styles theory of comorbidity suggests anxious individuals with a tendency to ruminate or make hopeless attributions about anxiety symptoms (negative anxiety response styles [NARS]) are more vulnerable to subsequent depressive symptoms. However, this theory has never been tested in adolescence, when the anxiety-depression transition may frequently occur, or using an extended (one-year) follow-up period.
Method: 128 early adolescent girls (M=12.39 years) participated with caregivers in a one-year longitudinal study. At baseline and follow-up, participants completed diagnostic interviews and self-report measures assessing child NARS and brooding rumination.
Results: T1 NARS predicted longitudinal elevations in depressive symptoms and increased associations between T1 anxiety and T2 depressive symptoms.
Limitations: This study examines anxiety and depression comorbidity using a community sample. The sample is relatively low on sociodemographic diversity.
Conclusions: Results support the anxiety response styles theory, with potential implications for early identification of anxious youth at risk for later development of comorbid depression.

1. Introduction

Internalizing disorders typically do not occur in isolation of each other; anxiety disorders and depression frequently co-occur across the lifespan, both concurrently and sequentially (Cummings et al., 2014). Although researchers have long recognized the challenges that comorbidity poses for research and treatment (e.g., Kendall and Clarkin (1992)), the mechanisms that underlie anxiety-depression co-occurrence remain poorly understood. Among those with lifetime comorbidity, anxiety disorders are likely to precede depression, a pattern applying to both disorders (e.g., Essau (2003), Kovacs et al. (1988), Orvaschel et al. (1995), and Wittchen et al. (2000); reviewed by Cummings et al. (2014)) and symptoms within disorders (Starr and Davila, 2012c). The "transition" from pure anxiety to comorbid depression may frequently occur during early adolescence, as most anxiety disorders typically emerge in childhood, whereas depressive symptoms and disorders begin to surge in adolescence (especially among girls; Gotlib and Hammen, 1992; Nolen-Hoeksema and Girgus, 1994). As depression during adolescence sets the stage for persistent, potentially lifelong difficulties (Colman et al., 2007; Glied and Pine, 2002), understanding why and for whom this transition occurs may prove critical to prevention efforts.

Although anxiety does not exclusively precede depression, because this pattern occurs in the majority of comorbid cases, models of anxiety-depression co-occurrence that account for the temporal antecedence of anxiety have the potential to explain comorbidity across a large portion of comorbid individuals (Cummings et al., 2014; Hamilton et al., 2015; Moffitt et al., 2007). One possible interpretation of the temporal antecedence of anxiety over depression is that anxiety acts as a causal risk factor for later depression. Evidence has recently emerged in support of this hypothesis. For example, a longitudinal analysis of the Oregon Adolescent Depression Project revealed that an anxiety-primary causal model (i.e., anxiety causing later depression) provided superior data fit than shared risk factors or depression-primary

Abbreviations: NARS, negative anxiety response styles; T1 and T2, time 1 and 2 respectively; RAQ, response to anxiety questionnaire

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models (Mathew et al., 2011). Other studies suggest that individuals with co-occurring symptoms tend to perceive their anxiety as causing depressive symptoms (Frewen et al., 2012, 2013). Recent investigations have begun to identify possible mediators of the anxiety-depression causal chain, including interpersonal problems, behavioral avoidance, and rumination (Grant et al., 2007, 2014; Jacobson and Newman, 2014; Jose and Weir, 2013; Moitra et al., 2008; Starr et al., 2014).

However, less is known about specific conditions that make anxiety more likely to spur depression during adolescence. As noted by Starr and Davila (2012a, b), persistent anxiety is a difficult experience and may function akin to a chronic stressor; as with stress, pre-existing risk factors may moderate the impact of anxiety on depression. Cohen et al. (2014) termed this approach to understanding comorbidity "diathesis-anxiety," implying that, as with diathesis-stress models (Monroe and Simons, 1991), pre-existing vulnerabilities interact with the "stress" of anxiety to produce depressive symptoms. The current study builds on the *anxiety response style theory of comorbidity* (Starr and Davila, 2012b), which proposes one possible diathesis: negative anxiety response styles.

1.1. Overview of anxiety response styles theory

The anxiety response styles theory of comorbidity is based upon the notion that individuals differ in how they respond cognitively to anxiety symptoms, and these differences may have implications for depression risk. *Negative anxiety response style* (NARS) is defined as the tendency to have ruminative or hopeless cognitions about anxiety symptoms, including thoughts focusing on the unpleasant nature of anxiety symptoms, perceived inability to control or stop anxiety, negative self-implications of being anxious, and potential negative consequences of anxiety (Starr and Davila, 2012b). Building upon research on rumination, hopelessness, and cognitive vulnerability (Alloy and Clements, 1998; Nolen-Hoeksema et al., 2008), NARS reflects the extent to which individuals respond to anxiety symptoms with several cognitive processes with established links to depressive symptoms. NARS is related to, but conceptually and empirically distinct from, several existing constructs that also reflect symptom response styles. For example, research on rumination typically focuses on responses to depressed mood (i.e., depressive rumination; Nolen-Hoeksema et al., 2008). Extending this to anxiety, NARS reflects thoughts about the causes and implications of anxiety symptoms and encompasses what could be described as anxious rumination. Although correlated, NARS and depressive rumination show distinct patterns of associations to anxiety and depression (e.g., with NARS more consistently predicting both anxiety and depression and depressive rumination showing greater specificity to depression; Rector et al., 2008; Starr and Davila, 2012b), supporting divergent validity. NARS can also be distinguished from anxiety sensitivity, which reflects fearful (as opposed to ruminative and hopeless) responses to physiological anxiety symptoms (Reiss et al., 1986), and from other related constructs, including distress tolerance, negative attributional style, and worry (Starr and Davila, 2012b).

The anxiety response style theory suggests that NARS influences likelihood that anxiety transitions into depression (Starr and Davila, 2012b). NARS could transform the experience of anxiety symptoms from a relatively innocuous occurrence into an acutely painful event that damages self-esteem and instills negative future expectations. Like depressive rumination, ruminative responses to anxiety (when combined with high anxiety levels) may disrupt problem-solving and evoke negative autobiographical memories (Lyubomirsky et al., 1998; Lyubomirsky and Nolen-Hoeksema, 1995; Raes et al., 2005). In turn, these processes could lead to increasing depressive symptoms. Although research on the anxiety response style theory remains limited, a series of studies by Starr and Davila has generated support for the role of NARS as a diathesis for comorbidity. Among college students, NARS predicted stronger cross-sectional associations between anxiety and depressive symptoms, beyond related constructs (Starr and Davila, 2012b, Study 2; c.f., Study 1). In a sample of adults with generalized anxiety disorder (GAD), NARS moderated the association between baseline anxiety and depressive symptoms four weeks later, with anxiety predicting increased depression for those with high NARS, but reduced depression among those with low NARS (Starr and Davila, 2012b, Study 3). In a daily diary study conducted with the same GAD sample, daily reports of anxious (but not depressive) rumination predicted stronger concurrent associations between daily anxious mood and daily depressed mood, as did daily negative attributions about anxiety (i.e., appraisals of daily anxiety symptoms as uncontrollable, incessant, and negatively impactful; Starr and Davila, 2012b). Importantly, these results appear to be specific to negative responses to anxiety symptoms as opposed to depressive rumination. Although these findings provide a foundation for anxiety response style theory, more research is needed. No studies have examined the extended consequences of NARS (beyond four weeks). Moreover, research has been limited to adult samples, and it may be informative to study this phenomenon during adolescence, when the initial anxiety-depression transition may occur.

Although research on anxiety response styles theory remains limited, the extensive literature on depressive rumination provides general support for the contribution of perseverative cognitive responses to anxiety-depression comorbidity. Depressive rumination has been identified as a transdiagnostic feature of depression and anxiety disorders, and is particularly elevated among those with mixed anxiety-depression (Jose and Weir, 2013; McEvoy et al., 2013; McLaughlin and Nolen-Hoeksema, 2011; Nolen-Hoeksema, 2000). However, little research has explored depressive rumination's role in comorbidity from a "diathesis-anxiety" perspective. In an exception, Cohen et al. (2014) showed that depressive rumination interacts with anxiety symptoms to predict later depressive symptoms in a longitudinal sample of community youth, and that the diathesis-anxiety approach outperformed alternative models of sequential comorbidity. It is possible that NARS better accounts for this finding, as the tendency to dwell on and make negative attributions about anxiety (rather than depressive) symptoms may be more relevant to consequences of anxiety disorders.

1.2. The current study

We tested several hypotheses related to NARS in a community sample of early adolescent girls followed over one-year. As girls are more vulnerable than boys to anxiety, depression, and rumination (Lewinsohn et al., 1993; Nolen-Hoeksema et al., 1999), the use of an all-female sample helps boost statistical power by increasing variability on constructs of interest. First, aligning with prior research, we anticipated that anxiety would predict later depressive symptoms but not vice versa. Second, we expected NARS to predict increasing depressive symptoms over time. Finally, we predicted NARS would moderate sequential comorbidity, with a stronger predictive relationship between anxiety and later depressive symptoms for youth with more negative anxiety response styles, and that this would be specific to NARS compared to brooding rumination. Brooding is an aspect of depressive rumination that is particularly tied to depressive symptoms and potentially most relevant to comorbidity models (Jose and Weir, 2013; Lopez et al., 2012; Treynor et al., 2003).

2. Method

2.1. Participants and procedure

Participants were recruited from two counties in New England through local schools (76.6%), word-of-mouth (13.1%), and advertisements or flyers (10.3%). Eligible adolescents were female, in sixth or seventh grade, and accompanied by a participating primary caregiver. At Time 1 (T1) participation included interviews conducted during a laboratory visit and online questionnaires completed from home. Approximately one-year later, 85% ($n=109$) participated in a follow-up (T2), including phone interviews and online questionnaires. Most analyses utilized available data from the full sample ($N=128$), with missing data estimated as described in the data analytic approach section. T2 attrition was not predicted by age, family income, or any T1 study variables ($ps>.10$).

Mean age was 12.39 years ($SD=.76$) for adolescents. Family income distribution was: ≤$20,000 (10.2%); $21,000–$40,000 (10.2%); $41,000–$60,000 (18.8%); $61,000–$100,000 (25.8%); >$100,000 (35.2%), suggesting a slightly higher family income than that of the two sampled counties (median incomes: $48,450 and $50,221). Most girls (82.0%) were Caucasian, demographically consistent with the two sampled counties (94% and 97% Caucasian). All procedures were approved by the [BLINDED] Institutional Review Board.

2.2. Measures

2.2.1. Anxiety disorder and depressive symptoms

At T1 and T2, adolescents were interviewed with the Schedule for Affective Disorders and Schizophrenia for School-Aged Children-Present and Lifetime version (Kaufman et al., 1997), a widely-used semi-structured diagnostic interview with well-established validity (Kaufman et al., 1997). Symptoms of depressive (major depression, dysthymia) and anxiety (GAD, obsessive compulsive disorder, specific phobia, social phobia, posttraumatic stress disorder) disorders were rated: 0=no symptoms; 1=mild symptoms; 2=moderate, sub-threshold symptoms; 3=DSM-IV criteria. T1 ratings reflect lifetime history of symptoms, and T2 ratings reflect symptoms since T1. To form depressive and anxiety disorder composites, we took the maximum of those ratings across the two depressive disorders (depression composite) and five anxiety disorders (anxiety composite) at each time point. We chose to create composite variables to increase variance for our predictor and outcome variables, and because the anxiety response styles theory was designed to examine co-occurrence between depression and general anxiety symptoms, and not to account for specific anxiety disorder comorbidities. Similar approaches have been applied in other studies of comorbidity (e.g., Starr et al. (2014)). At T1 and T2 respectively, 14.1% and 11.0% reported mild depressive symptoms; 8.6% and 9.2% reported moderate, subthreshold symptoms; and 6.3% and 5.5% met depression diagnostic criteria. For anxiety at T1 and T2 respectively, 35.9% and 36.7% endorsed mild symptoms; 32.8% and 35.8% reported moderate symptoms; and 16.4% and 11% met criteria for an anxiety disorder. Of those reporting anxiety symptoms, the majority (61.3%) reported symptoms of or met criteria for more than one anxiety disorder. Specific phobia was the most commonly endorsed disorder (with 9.1% meeting full criteria, 28.0% reporting moderate and 40.2% mild symptoms), followed by social phobia (1.5% full, 10.6% moderate, and 30.3% mild), GAD (3.8% full, 7.6% moderate, and 17.4% mild), PTSD (3% full, 1.5% moderate, and 8.3% mild), and OCD (1.5% full, 3.8% moderate, and 6.8% mild). Inter-rater reliability was assessed using audio-recordings of 27% of interviews, with intra-class correlations ranging from .80 to 1.00.

2.2.2. NARS

Adolescents completed the 32-item Responses to Anxiety Questionnaire (RAQ; Starr and Davila, 2012b). Minor wording adjustments were made to increase developmental appropriateness for adolescents (e.g., replacing “my job” with “my school work”). For each item (e.g., “Think that your anxiety will keep you from getting what you want”), participants were asked to indicate how often they “think or do each one” when feeling “anxious, nervous or worried” using a 4-point scale from 1 (almost never) to 4 (almost always); responses were averaged for a total score. Previous research supports the RAQ's reliability and convergent and discriminant validity (Starr and Davila, 2012b), and here Cronbach's alpha was .96.

2.2.3. Brooding

At T1, girls completed the 5-item brooding subscale of the Ruminative Responses Scale (Nolen-Hoeksema and Morrow, 1991; Treynor et al., 2003), with directions modified for adolescents (Burwell and Shirk, 2007; Hilt and Pollack, 2013), consistent with numerous recent studies and with current conceptualizations of rumination (see Burwell and Shirk (2007), Hilt et al. (2014), Hilt and Pollack (2013), Nolen-Hoeksema et al. (2008), Stroud et al. (2015)). Specifically, for each item (e.g., “Think ‘Why do I always react this way?’”), participants indicated how often they “think or do each one” when they “feel stressed or upset” using a scale from 1 (almost never) to 4 (almost always); items were averaged for a total score. The brooding subscale predicts adolescent anxiety and depressive symptoms more strongly than do other rumination components, and studies support psychometric properties (Burwell and Shirk, 2007; Jose and Weir, 2013; Treynor et al., 2003); here, $\alpha=.80$.

2.3. Data analytic approach

Hypotheses were tested with path analysis in M*plus* 7.3 (Muthén and Muthén, 1998–2012) using maximum likelihood estimation with robust standard errors. An application of the general linear model, path analysis allows for parsimonious examination of hypothesized causal pathways across multiple correlated variables. Using path analysis over standard regression procedures also allows for missing data estimation procedures. Little's MCAR indicated that the data were missing completely at random ($\chi^2[20]=14.89$, $p=.783$). Thus, missing data was estimated with full maximum information likelihood, consistent with best-practice recommendations (Enders, 2010). All path models included manifest variables only and all but one (as noted), were fully saturated; thus, fit indices were not applicable (as saturated models show perfect fit). Significant interactions were decomposed by conducting simple slope tests in M*plus* at high ($M+1SD$) and low ($M-1SD$) levels of the moderator.

As a preliminary step, we examined whether demographic variables (family income, child age at T1, and ethnicity [Caucasian vs. non-Caucasian]) significantly predicted study outcome variables (T2 anxiety and T2 depression). No demographic variables were predicted with T2 anxiety, but family income ($\beta=-.22$, $p=.026$) and child age ($\beta=.20$, $p=.041$) each predicted T2 depression. Because of this, we examined whether the pattern of significance was altered when age and family income were included as covariates in all models.

3. Results

Bivariate intercorrelations between major study variables are provided in Table 1 along with descriptive statistics.

Table 1
Bivariate correlations between study variables.

	1.	2.	3.	4.	5.	6.	7.	*M*	*SD*
1. T1 Anxiety Disorder Sx	–							1.48	.96
2. T1 Depressive Symptoms	.39**	–						.49	.90
3. T1 RAQ	.30**	.27**	–					1.65	.54
4. T1 Brooding	.20*	.11	.72**	–				2.03	.65
5. T2 Anxiety Disorder Sx	.50**	.14	.29**	.25*	–			1.40	.88
6. T2 Depressive Symptoms	.39**	.55**	.38**	.28**	.37**	–		.45	.88
7. T2 RAQ	.34**	.37**	.59**	.49**	.40**	.67**	–	1.53	.51

* $p < .05$.
** $p < .01$.

3.1. Longitudinal associations between anxiety and depressive symptoms and NARS

To examine temporal comorbidity patterns between anxiety disorder and depression symptoms within this sample, we constructed a two-wave panel model with cross-lagged effects, with T1 anxiety and depressive symptoms each predicting both anxiety and depressive symptoms at T2, and T1 variables and T2 disturbances allowed to covary. Fig. 1a illustrates this model and displays standardized coefficients for all pathways. Anxiety and depressive symptoms showed significant stability, with each T1 variable predicting itself at T2 ($ps < .001$). Consistent with prior research, T1 anxiety predicted increases in T2 depressive symptoms ($p = .012$), but T1 depressive symptoms did not predict T2 anxiety symptoms ($p = .613$).

We next explored NARS as a predictor of prospective increases in symptoms by using the same two-wave panel model described above (with T1 anxiety and depression as predictors and T2 anxiety and depressive symptoms as outcomes), trimming the non-significant pathway from T1 depression to T2 anxiety, and adding T1 RAQ as a predictor of both T2 anxiety and T2 depression (allowing it to correlate with T1 symptoms variables). Fig. 1b presents this model with standardized coefficients. Model fit was excellent (Hu and Bentler, 1998), $\chi^2[1] = .90$, $p = .343$, Root Mean Squared Error of Approximation [RMSEA] = .000, Comparative Fit Index [CFI] = 1.000; note that these perfect RMSEA and CFI values are expected in cases where, as here, $\chi^2 < df$. The RAQ predicted significant increases in both depressive symptoms ($p = .035$) and anxiety ($p = .025$). All findings held when age and family income were included as covariates.

Table 2
Results of path analysis models testing moderating effect of negative anxiety response styles on association between T1 anxiety disorder symptoms and T2 depressive symptoms.

Predictor	*β*	95% C.I.	*b*	*SE*	*p*
Model $R^2 = .38$					
Intercept	.25	.11, .39	.22	.07	.001
T1 Depressive Symptoms	.39	.17, .61	.38	.11	.001
T1 Anxiety Disorder Sx	.17	.02, .37	.16	.07	.030
T1 RAQ	.20	.02, .37	.32	.15	.026
T1 Anxiety Disorder Sx × RAQ	.17	.03, .32	.31	.13	.019

Notes. Confidence interval values are standardized.

3.2. Moderation of sequential comorbidity between anxiety and depression by NARS

To examine whether NARS increases the degree to which anxiety is associated with later depressive symptoms, we estimated a path model where T2 depression is predicted by main effects of T1 RAQ and T1 anxiety symptoms (both centered) and their interaction, with T1 depressive symptoms included as a covariate. Full results are reported in Table 2. Notably, the interaction term was significant. As illustrated in Fig. 2, T1 anxiety predicted increases

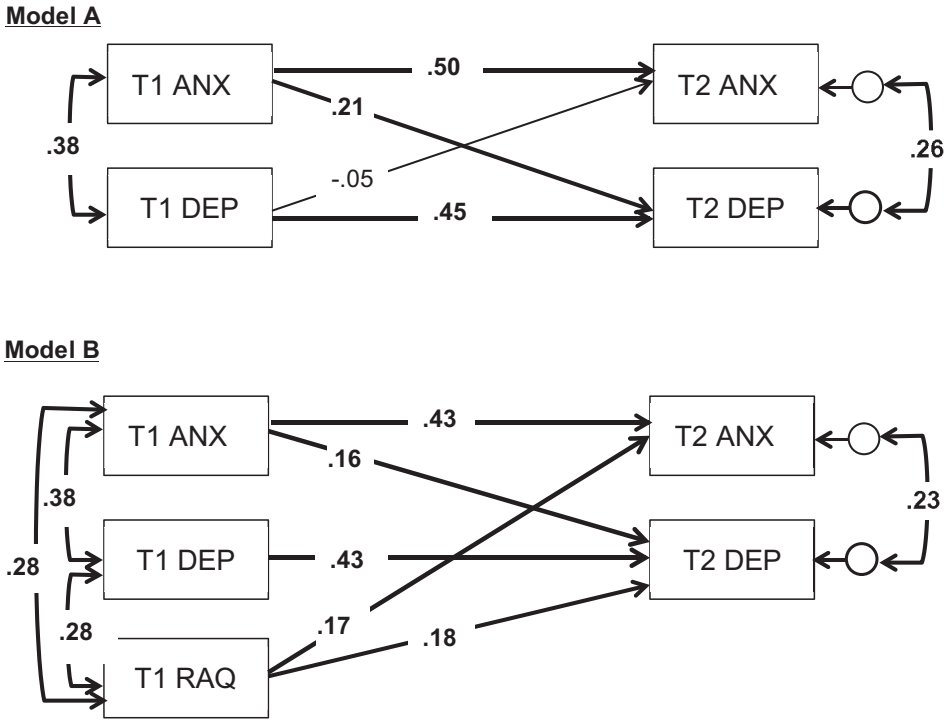

Fig. 1. Statistical diagrams illustrating models for preliminary path analyses, with standardized coefficients. Model A tests temporal associations between anxiety and depressive symptoms at T1 and T2. Model B examines negative anxiety response style (assessed using RAQ) as a longitudinal predictor of anxiety and depressive symptoms. Significant pathways ($p < .05$) are bolded.

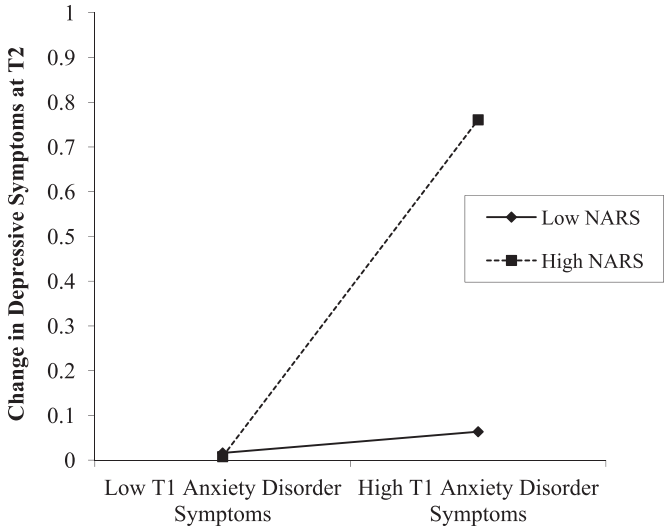

Fig. 2. Graphical depiction of interaction between T1 anxiety disorder symptoms and negative anxiety response styles (RAQ), predicting T2 depressive symptoms. T1 depressive symptoms were included in this model as a covariate, so *Y* axis refers to change in depression from T1 to T2. High and low levels of anxiety disorder symptoms and RAQ reflect one standard deviation above and below the mean of each variable.

in T2 depressive symptoms for girls high on the RAQ ($b=.32$, 95%C. I.=[.09, .54], $SE=.12$, $p=.007$), but not for girls with low RAQ scores ($b=-.01$ [−.17,.56], $SE=.12$, $p=.905$).

To evaluate specificity of findings, we examined possible effects of brooding. First, controlling for brooding did not modify the significance of the interaction between T1 RAQ and T1 anxiety predicting increases in depression at T2. Second, we examined whether brooding moderated the association between T1 anxiety and T2 depressive symptoms (controlling for T1 depressive symptoms); the interaction was not significant ($p=.271$).

Finally, although not predicted by our model, to examine the possibility that NARS also moderates the association between T1 depression and increases in anxiety at T2, we tested a model that included main effects of RAQ and T1 depression, plus T1 anxiety as a covariate, with T2 anxiety as the outcome. The interaction was non-significant ($p=.785$).

All findings held when age and family income were included as covariates.

4. Discussion

In a developmentally informed extension of previous research, the current study provides novel evidence for the anxiety response styles theory of comorbidity (Starr and Davila, 2012b), which proposes that the tendency to respond to anxiety symptoms with ruminative and hopeless cognitions contributes to the development of secondary depressive symptoms. This study was the first to examine the construct of NARS in adolescence (a developmental period in which the transition from anxiety to depression often occurs) and to explore the consequences of NARS using an extended longitudinal design. Results supported several important tenets of anxiety response styles theory.

First, NARS prospectively predicted increases in depressive and anxiety symptoms, even controlling for co-occurring symptoms. This provides evidence that anxiety-focused rumination and hopelessness act as a general diathesis for the development of internalizing symptoms, although the main effect on depression was subsequently qualified by an interaction and should be interpreted with caution. Although further research is needed to explore how NARS contributes to increasing internalizing symptoms, it may involve some of the same mechanisms through which rumination is presumed to influence depression, such as by increasing negative autobiographical memories and impairing problem solving (Lyubomirsky et al., 1998; Lyubomirsky and Nolen-Hoeksema, 1995). Interestingly, although the anxiety response styles theory was devised to explain the development of comorbid depression, results suggest that NARS also amplifies anxiety symptoms, a finding that warrants further examination. Perhaps NARS elevates attunement to anxiety symptoms, leading them to become more pronounced overtime.

Second, NARS moderated the association between anxiety and later depressive symptoms, such that adolescents with more depressogenic anxiety response styles showed a stronger association between anxiety and subsequent depression. This is consistent with a “diathesis-anxiety” model, and suggests we can identify subsets of anxious youths at highest risk for later depressive symptoms. Anxious adolescents who tend to fret about the causes and consequences of anxiety or view anxiety as difficult to overcome and likely to impede goals may come to develop negative beliefs about themselves and the future, setting the cognitive stage for depression (Lyubomirsky and Nolen-Hoeksema, 1995; Nolen-Hoeksema et al., 2008). As these youth enter adolescence, encountering unfamiliar developmental contexts and new expectations (e.g., romantic involvement, increased familial autonomy, peer challenges; Steinberg, 1987), they may become increasingly convinced that their anxiety will prevent them from achieving desired outcomes, culminating in dysphoric emotions.

Importantly, brooding did not moderate the link between T1 anxiety and T2 depressive symptoms, suggesting that NARS plays a relatively specific role in predicting the anxiety-depression transition in early adolescence (although it should be noted that reliability for the RAQ measure was higher than for the brooding scale, and while reliability for both scales were in the good-to-excellent range, this may have influenced results). NARS is conceptually descended from depressive rumination, and the two constructs overlap; however, current findings add to existing evidence suggesting that styles of responding to anxiety symptoms specifically (versus styles of responding to other forms of distress) have unique implications for sequential comorbidity (Starr and Davila, 2012b). Notably, previous studies have suggested a role of depressive rumination in contributing to anxiety-depression co-occurrence as a mediator and moderator (Cohen et al., 2014; McLaughlin and Nolen-Hoeksema, 2011; Nolen-Hoeksema, 2000). Although both factors may contribute, anxiety-focused response style may more precisely account for some previous moderation findings.

Preliminary analyses revealed that anxiety predicted increases in later depressive symptoms, but not vice versa, consistent with the modal pattern of anxiety preceding depression that has been established in numerous studies (e.g., Essau (2003), Starr et al. (2014) and Wittchen et al. (2000)). However, multiple trajectories of anxiety-depression comorbidity likely exist (Cummings et al., 2014), for which NARS may or may not be relevant. For example, previous research suggests a more complex relationship between anxiety and depression, with a subset of individuals for whom depression precedes anxiety (Avenevoli et al., 2001; Hamilton et al., 2015; Kouros et al., 2013; Lamers et al., 2011; Moffitt et al., 2007). Perhaps youth who experience depression before anxiety develop a negative cognitive style that is later directed towards anxiety symptoms, leading to maintenance and worsening of both depressive and anxious symptoms. Some evidence also suggests anxiety and depression may be reciprocally related (Cummings et al., 2014; Garber and Weersing, 2010; McLaughlin and Nolen-Hoeksema, 2011); more work is needed to understand how the interplay of cognitive factors contributes to this possible cyclical relationship.

Importantly, the anxiety response styles theory of comorbidity was never intended to fully explain anxiety-depression co-occurrence, which likely exists for many reasons (Starr and Davila, 2012b). Most prominently, numerous researchers have attributed comorbidity to imprecisely drawn diagnostic boundaries and shared symptom structure (e.g., Krueger and Markon (2006), Watson (2005) and Watson et al. (1995)). The idea that how people respond to anxiety symptoms has implications for sequential comorbidity is not mutually exclusive with structural comorbidity models; indeed, as a factor that is linked to both depression and anxiety, NARS likely contributes to (or perhaps represents a facet of) broadband internalizing pathology. Future research should explore how anxiety response styles influence porous structural boundaries between anxiety and depression. Further, it would be interesting to examine whether anxiety response style influences somatic symptoms, such as sleep disturbances, and whether this may translate into depressive symptoms (Gregory et al., 2005; Zawadzki et al., 2013). Moreover, other factors may also predict the anxiety-depression transition during adolescence. For example, researchers have recently proposed roles for reward processing and social evaluative threat (Silk et al., 2012) and interpersonal dysfunction (Starr et al., 2014), and studies should examine whether these factors and NARS influence symptom co-occurrence independently or operate in concert.

4.1. Limitations and future directions

Results should be interpreted within the context of study limitations. First, our sample was drawn from the community, and correspondingly most pathology was subthreshold, and rates of depression were low (although consistent with other studies of this age group; e.g., Rohde et al. (2009)). Unselected samples enable identification of early predictors of psychopathology, and subthreshold symptoms are in themselves impairing and predictive of later disorder onset (Judd et al., 1998; Shankman et al., 2009). That said, it is unclear whether identical mechanisms govern both symptom co-occurrence within normative samples and disorder comorbidity in more severe populations. Future research should examine NARS in clinically-referred adolescent samples to ensure that results generalize to clinically significant pathology and better inform treatment. Studies should also replicate findings in more sociodemographically diverse samples, especially given the importance of cultural factors in the presentation of anxiety and depression (Kirmayer, 2001). Future research should also follow mixed-gender adolescent samples over a longer period to explore the developmental progression of NARS and potential implications for gender differences in anxiety and depression. Finally, the relatively small sample and low variation in depressive symptoms may have led to truncated power in our analyses, and results should be replicated in larger samples.

Despite these limitations, our findings provide an important step in understanding the developmental mechanisms through which anxiety and depression co-occur and lay a foundation for future research on comorbidity in youth. Future studies should probe the construct boundaries and factor structure of NARS. Based on previous findings supporting a single-factor structure (Starr and Davila, 2012b), NARS was conceptualized as unidimensional, encompassing elements of both ruminative and hopeless anxiety-focused response styles. However, further examination might reveal distinct subfactors, or identify other forms of depressogenic anxiety-focused thinking that should be incorporated into NARS. The present study also did not distinguish between different forms of anxiety. Although it makes sense for initial studies to approach NARS from a transdiagnostic perspective, future research should examine whether symptoms of different anxiety disorders interact with varying diatheses to predict subsequent depression.

Results have important implications for treatment and prevention research. Despite a thriving literature on effective interventions for anxious youth, research has not, to our knowledge, examined whether specific treatment protocols are effective at buffering against later development of comorbid depression. For example, clinical interventions and prevention programs that specifically target maladaptive anxiety response styles in children and early adolescents could potentially prevent spillover of anxiety symptoms into later depressive outcomes. Although numerous programs exist that target negative thought patterns linked to anxiety (Cartwright-Hatton et al., 2004; Norton and Price, 2007; Roemer et al., 2009), results suggest that modifying NARS, including cognitions directly focused on the nature, causes, and consequences of the anxiety symptoms, may be particularly beneficial for preventing comorbid depression. In addition, treatment programs incorporating mindfulness and acceptance-based techniques may be especially congruent with this model (e.g., Eifert and Forsyth, 2005; Hayes et al., 2006), as these strategies focus on maintaining a nonjudgmental stance and minimizing experientially avoidant responses to symptoms. The current findings suggest that, if intervention techniques can effectively reduce the tendency to engage in depressogenic thought patterns in response to anxiety symptoms, the transition from anxiety to depression during adolescence may be preventable.

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