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# Development of Sex Differences in Depressive and Co-Occurring Anxious Symptoms During Adolescence: Descriptive Trajectories and Potential Explanations in a Multiwave Prospective Study

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This study investigated psychosocial mechanisms that may account for sex differences in internalizing symptoms of depression and anxiety during adolescence using data from a prospective, multiwave study with a sample of early and middle adolescents (N=350, 6th to 10th graders; 57% female). Girls showed higher initial levels of only depressive symptoms, not anxious arousal, and increasing trajectories of depressive and anxious arousal symptoms over time compared with boys after controlling for age. Initial levels of depressive symptoms were mediated by a Rumination × Stressors interaction as well as a Negative Cognitive Style × Stressors interaction. The Negative Cognitive Style × Stressors interaction partially accounted for girls' increasing trajectories of depressive and anxious arousal symptoms over time.

One of the most well-replicated set of findings in developmental psychopathology and epidemiology is the existence of sex differences in psychopathology. Twice as many women as men are depressed and experience certain forms of anxiety, and the female preponderance in internalizing symptoms begins at different points during childhood and adolescence for anxiety and depression (see Rutter, Caspi, & Moffitt, 2003; Zahn-Waxler, Crick, Shirtcliff, & Woods, 2006, for reviews). For anxiety symptoms and disorders, the sex difference appears by middle childhood and remains throughout adolescence (Klein & Pine, 2002; Lewinsohn et al., 1998). The sex difference in depression emerges in early adolescence (ages 12-14) and diverges dramatically during middle adolescence for depressive symptoms (e.g., Ge, Lorenz, Conger, Elder, & Simons, 1994; Twenge &

Nolen-Hoeksema, 2002; Wade, Cairney, & Pevalin, 2002) and disorder (e.g., Hankin et al., 1998).

However, despite the clear descriptive developmental timeline for the unfolding of sex differences in anxiety and depression, the causes underlying the sex differences in these developmental trajectories are unclear (Rutter et al., 2003; Hankin, Wetter, & Cheely, 2008; Zahn-Waxler et al., 2006). The main purpose of the present study was to investigate potential psychosocial mechanisms that may account for the sex differences in these common internalizing symptoms during early and middle adolescence using a short-term multiwave longitudinal study of an ethnically diverse, representative community sample. Particular theoretically motivated explanatory factors, including cognitive vulnerabilities and stressful life events, based on the elaborated cognitive vulnerability-transactional stress theory of depression (Hankin & Abramson, 2001), were examined to determine whether these factors could mediate the sex difference in depressive symptoms and potentially anxiety symptoms during adolescence. Early and middle adolescence was chosen as the developmental period of particular interest as this is the time when depressive and anxious symptoms rise rapidly, especially for girls

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compared with boys. These etiological factors were investigated as putative mediating processes of the sex difference in internalizing symptoms given the strong co-occurrence of anxious and depressive symptoms (Angold, Costello, & Erkanli, 1999) and the female preponderance in forms of internalizing distress.

#### ETIOLOGICAL MECHANISMS AND SEX DIFFERENCES IN PSYCHOPATHOLOGY

Many different vulnerabilities have been proposed and examined as potential explanations for the sex difference in internalizing symptoms (for reviews, see Bell, Foster, & Mash, 2005; Hankin & Abramson, 2001; Hankin et al., 2008; Nolen-Hoeksema & Girgus, 1994; Rutter et al., 2003; Zahn-Waxler et al., 2006). The psychosocial variables and mechanisms selected for investigation in this study were theoretically based on the elaborated cognitive vulnerability-transactional stress theory of depression (Hankin & Abramson, 2001). In brief, this theory postulates that girls, compared with boys, exhibit more cognitive vulnerabilities and are exposed to more stressors starting in early adolescence and throughout adulthood. These factors predict prospective elevations in depressive symptoms, and thus the sex difference in adolescent depression may be mediated by cognitive vulnerabilities, stressors, and/or the interaction of these cognitive risks with stressors over time. As reviewed next, the cognitive vulnerabilities of a negative cognitive style (Abramson, Metalsky, & Alloy, 1989), dysfunctional attitudes (Beck, 1987), and rumination (Nolen-Hoeksema, 1991) as well as negative life events (Grant & McMahon, 2005) were examined in this study.

The main cognitive vulnerabilities to depression that have been examined with youth include a negative cognitive style from hopelessness theory (HT; Abramson et al., 1989), dysfunctional attitudes from Beck's theory (BT; Beck, 1987), and rumination from Response Styles Theory (RST; Nolen-Hoeksema, 1991). Prospective evidence shows that each of these cognitive vulnerabilities predicts future elevations of depressive symptoms (Abela & Hankin, 2008; Lakdawalla, Hankin, & Mermelstein, 2007). In particular, HT and BT are cognitive vulnerability-stress models, and the evidence clearly indicates that youth with a more negative cognitive style or dysfunctional attitudes who encounter more stressors exhibit the highest elevations in depressive symptoms over time (e.g., Hankin, 2008a; Hankin, Wetter, Cheely, & Oppeinheimer, in press; see review by Abela & Hankin, 2008). RST was not originally formulated as a vulnerability-stress model, although it has been hypothesized that rumination may interact with stressors to predict depression and potentially account for the emerging sex difference in depression (Hankin &

Abramson, 2001; Nolen-Hoeksema & Girgus, 1994). Rumination, as a main effect, predicts future elevations of depressive symptoms (e.g., Hankin, 2008b; see review by Abela & Hankin, 2008), whereas not much support has been obtained for a Rumination × Stress interaction (Nolen-Hoeksema, Larsen, & Grayson, 1999; Sarin, Abela, & Auerbach, 2005).

With respect to sex differences in these cognitive vulnerabilities, theory (Hankin & Abramson, 2001; Nolen-Hoeksema & Girgus, 1994) suggests, and some evidence shows, that a negative cognitive style (Hankin & Abramson, 2002; Mezulis, Abramson, Hyde, & Hankin, 2004) and rumination (Schwartz & Koenig, 1996; Ziegert & Kistner, 2002, but see Abela, Brozina, & Haigh, 2002) are higher among girls than boys, whereas dysfunctional attitudes may be more elevated among adult men than women (Haeffel et al., 2003). Research using a cross-sectional design shows that a negative cognitive style (Hankin & Abramson, 2002) and rumination (Schwartz & Koenig, 1996) mediated the sex difference in adolescent depression. However, the paucity of research examining cognitive theories as explanations for the sex difference in depression has been hampered by a lack of rigorous longitudinal designs and not including stressors in the analysis, given that both HT and BT are cognitive vulnerability-stress models of depression and RST has been elaborated to include a vulnerability-stress component. Moreover, none of the past research has investigated whether these cognitive factors account for the sex difference in anxiety symptoms among youth, so the specificity of these cognitive influences for explaining the sex difference in depression and co-occurring anxiety is unknown.

Finally, stressors have been shown to predict prospective increases in depressive and anxious symptoms (Grant & McMahon, 2005). The overall number of negative events increases with the transition into adolescence and parallels the emergence of the sex difference in depressive symptoms (Ge et al., 1994). Girls report more overall stressors than boys (e.g., Allgood-Merten, Lewinsohn, & Hops, 1990; Ge et al., 1994; Hankin, Mermelstein, & Roesch, 2007), and girls experience more interpersonal stressors, in particular, than boys (e.g., Hankin et al., 2007; Rudolph & Hammen, 1999). Finally, the sex difference in adolescent depression is partially mediated by girls' greater exposure to interpersonal stressors (Hankin et al., 2007; Rudolph & Hammen, 1999). These findings have been obtained regardless of whether self-reported perceived stressors (e.g., Ge et al., 1994) or objectively determined stressors (e.g., Hankin et al., 2007; Rudolph & Hammen, 1999) are assessed and examined.

In sum, the elaborated cognitive vulnerabilitytransactional stress theory of depression postulates that the sex difference in depressive symptoms, and potentially co-occurring anxiety symptoms, may be explained by these various cognitive factors, either alone or in interaction with stressors. Given the available research showing that girls exhibit higher levels of negative cognitive style and rumination and report greater stressors than do boys, additional investigation of these processes is warranted. The theory postulates a differential exposure hypothesis in that girls' greater negative cognitive style, especially in interaction with greater stressors, as well as greater rumination, either alone or in interaction with stressors, may account for girls' elevated levels of depressive, and potentially anxious, symptoms compared to boys. However, as noted next, the past work has not rigorously tested for mediating mechanisms to explain why girls are more depressed and anxious than boys, so support for these cognitive factors and stressors as theoretical accounts of the sex difference in internalizing problems is inconclusive and awaits further research.

# METHODOLOGICAL, DESIGN, AND STATISTICAL ISSUES

Rutter and colleagues (2003) delineated several important issues for assessing and examining the differential exposure hypothesis, which states that a sex difference in putative risk factors may account for sex differences in psychopathology. Several points are particularly relevant to the present. First, representative community samples, rather than psychiatric clinic groups, are needed to produce accurate estimates of the sex difference in symptoms and potential explanatory mechanisms as biases resulting from psychiatric clinical samples have been documented (Goodman et al., 1997). Second, it is essential to use appropriate statistical techniques to determine whether a factor mediates (Holmbeck, 2002) the sex difference in anxiety and depression. Yet, surprisingly, Rutter and colleagues (2003) noted that "it is all too clear that few variables have been adequately tested in relation to these rather basic minimal requirements" for establishing mediation of the sex difference (p. 1102). Even fewer studies have included multiple, theoretically motivated, mediators in the same study to account for the development of sex differences in internalizing symptoms as the majority of studies only examine one risk factor at a time. Without including several factors together in the same study, it is difficult to know precisely which factors may be responsible for accounting for the sex difference in symptoms, as many vulnerabilities to depression and anxiety overlap (Gibb & Coles, 2005). Finally, it is crucial to examine patterns of comorbidity. It is well known that co-occurrence of disorders is ubiquitous (Angold et al., 1999), yet little research has focused on

sex differences in co-occurring symptoms (Rutter et al., 2003). Little is known with respect to whether the factors and processes that may explain the sex difference in depression also account for the sex difference in anxiety or whether these influences differentially explain girls' greater depressive versus anxiety symptoms.

# THE CURRENT STUDY

Descriptive epidemiological data clearly demonstrate a sex difference in the development of symptoms of anxiety and depression, yet the reasons for and the mechanisms contributing to the female preponderance of internalizing problems remain elusive. Although many different factors could be examined to account for this phenomenon (Hankin et al., 2008; Zahn-Waxler et al., 2006), the present study was theoretically motivated by and focused on particular psychosocial processes derived from the elaborated cognitive vulnerability-transactional stress theory of depression (Hankin & Abramson, 2001). Cognitive vulnerabilities, including a negative cognitive style, rumination, and dysfunctional attitudes, as well as greater exposure to stressors were examined in a short-term multiwave prospective study of early and middle adolescents from a general community sample. It was hypothesized that the main effects of cognitive vulnerabilities, specifically rumination and negative cognitive style, as well as stressors and the interaction of these cognitive vulnerabilities (i.e., negative cognitive style and rumination) with stressors would explain why girls exhibit more depressive symptoms (both initial levels and increasing trajectories of depression over time) than boys. In addition, these explanatory factors were tested to see whether they would mediate the sex difference in anxious symptoms given the well-known co-occurrence among these internalizing symptoms.

#### METHOD

#### Participants

Participants were youth who were recruited from five Chicago area schools. Selected schools included one inner-city private middle school, one affluent private middle school, and three public schools (one middle schools) serving predominantly and two high middle-class neighborhoods. There were 467 students available in the appropriate grades (6-10) from these selected schools and invited to participate. Parents of 390 youth (83.5%) provided active consent; all 390 youth were willing to participate. 356 youth (91%) completed the baseline questionnaire. The 34 students who were willing to participate but did not complete the baseline visit were sick or absent from school and were unable to reschedule. There were no significant differences in demographic characteristics (age, sex, ethnicity) between the number of available youth in schools (N=467), those who provided consent (N=390), and those who participated (N=356). Data were examined from 350 youth who provided complete data (symptoms and risk factors) at baseline. Rates of participation in the study decreased slightly at each wave of follow-up: Wave 2 (N=303), Wave 3 (N=308), and Wave 4 (N=345). Age ranged from 11 to 17 (M=14.5, SD=1.40); 57% were female, 13% were Latino, 6% were Asian or Pacific Islander, 21% African American, 53% White, and 7% bi- or multiracial.

#### Procedures

Students participated in this study with active parental consent. Permission to conduct this investigation was provided by the school districts and their Institutional Review Boards, school principals, the individual classroom teachers, and university Institutional Review Board. Trained research personnel visited classrooms in the schools and briefly described the study to youth, and letters describing the study were sent home to parents. Specifically, students and parents were told that this study was about adolescent mood and experiences and participation would require completion of questionnaires at four different time points. Students who agreed to participate and returned active parental consent read and signed a child assent form after asking any questions about the study. Youth completed a battery of questionnaires during class time and were debriefed at the end of the study. Participants completed questionnaire packets at four time points over a 5-month period, with approximately 5 weeks between each time point. The spacing for the follow-up intervals was chosen to provide enhanced, accurate recall of symptoms (see Costello, Erkanli, & Angold, 2006, for evidence that shorter time frames provide more accurate, less biased findings). Youth were compensated \$10 for their participation at each wave in the study, for a possible total of \$40 for completing all four assessments.

#### Measures

Dependent variable: Children's Depression Inventory (CDI; Kovacs, 1985). The CDI is a self-report measure that assesses depression in children and adolescents using 27 items. Each item is rated on a scale from 0 to 2, and scores range from 0 to 57. Higher scores indicate more depression. The CDI has been shown to have good reliability (internal consistency, test-retest reliability) and validity (e.g., associations with other depressive symptom measures and diagnostic interviews of clinical depression) as a measure of depression in children and adolescents (Klein, Dougherty, & Olino, 2005). Internal consistency of the CDI at each time point was strong (see Table 1). The range of CDI scores from this sample was comparable to published norms (Kovacs, 1981). Youth completed the CDI at all four time points.

Dependent variable: Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995). The MASQ for this study was modified from the original MASQ, which contains 90 items to assess the general distress and specific anxiety and depressive symptoms based on the tripartite theory of anxiety and depression (Clark & Watson, 1991). For this study, only the Anxious Arousal (ANXAR) subscale was used to assess relatively specific physiological arousal symptoms that are not overly saturated with general negative affect. Youth responded to 10 ANXAR items on a Likert scale from 1 to 5, and reported scores are the average item scores of all items (range = 1-5). The 10 items with the highest factor loadings out of the original MASO ANXAR (Watson et al., 1995) scale were used given the time constraints of having youth complete the questionnaire packets in the classroom. Reliability (internal consistency, test-retest reliability, factor structure) and validity (concurrent, discriminant, and construct validity) of the MASQ has been demonstrated in previous studies with adolescents (Hankin, 2008b; Hankin et al., 2008; Watson et al., 1995). The MASQ was given at all four time points. Internal consistency of the MASQ at each time point was good (see Table 1).

Mediating variable: Children's Response Style Questionnaire (CRSQ; Abela et al., 2002). The CRSQ, a measure of the constructs featured in RST, is based on the Response Styles Questionnaire (Nolen-Hoeksema & Morrow, 1991). The CRSQ uses 25 items clustered into the three general response styles of rumination, distraction, and problem solving. Children are asked to rate how frequently they respond to a sad mood with the particular response. The 13-item subscale of Rumination was used in this study. Scores on the Rumination scale range from 0 to 39. A higher score indicates a more frequent use of that response style. There is evidence for the scale's validity and it posses good internal consistency and reasonable test-retest reliability (Abela et al., 2002; Hankin, 2008a, in press-c). It was given at Time 1.

Mediating variable: Adolescent Cognitive Style Questionnaire (ACSQ; Hankin & Abramson, 2002). The ACSQ measures the inferential styles about cause, consequence, and self, as featured in HT. The ACSQ presents the adolescent with negative hypothetical events in achievement and interpersonal domains and asks the youth to make inferences about the cause (stable–unstable, and global–specific), consequences, and characteristics about the self based on the hypothetical event. Each item dimension is rated from 1 to 7. Average item-scores on the total ACSQ range from 1 to 7 with higher scores indicating more negative inferential styles. The ACSQ has demonstrated excellent internal consistency reliability, good test–retest reliability, and factor structure (Hankin, 2008c; Hankin & Abramson, 2002) as well as validity (concurrent, discriminant, predictive, and construct validity; Hankin, 2008b). It was given at Time 1.

Mediating variable: Dysfunctional Attitudes Scale (Lewinsohn, Joiner, & Rohde, 2001). The Dysfunctional Attitudes Scale is a nine-item scale assessing propensity to endorse dysfunctional attitudes, the cognitive vulnerability emphasized in Beck's (1987) theory. Moderate internal consistency and test-retest reliability along with good validity has been reported (Hankin, 2008c; Hankin et al., 2008; Lewinsohn et al., 2001). Adolescents rated the items on a 5-point Likert scale, with higher scores indicating greater levels of dysfunctional attitudes. It was given at Time 1.

Mediating variable: Adolescent Life Events Questionnaire (ALEQ; Hankin & Abramson, 2002). The ALEQ assesses a broad range of life events that typically occur among adolescents, including school/achievement problems, friendship and romantic difficulties, and family problems. Examples of items from the ALEQ include "got a bad report card" to assess school events, "had an argument with a close friend" for friendship events, "boyfriend/girlfriend broke up with you but you still want to go out with them" for romantic events, and "your parents grounded you" for family events. It consists of 57 different negative life events. Youth were asked to indicate whether these negative events had occurred to them, so scores are counts of total stressors. The ALEQ was given at Time 1 to assess baseline stressors and at Time 4 to assess stressors that occurred over the prospective follow-up. It has good validity (concurrent and predictive) in that it has predicted prospective increases in depressive and anxious symptoms (Hankin, 2008b).

#### RESULTS

#### Preliminary Analyses

Descriptive statistics and intercorrelations for the main variables at baseline are presented in Table 1. The cognitive vulnerabilities and stressors correlated with the symptoms of depression and anxious arousal, and negative events were associated with both depressive and anxious symptoms. Depressive and anxious arousal

Descriptives, Intercorrelations, and Sex Differences Among Main Measures												
1	2	3	4	5	6	7	8	9	10	11	12	13
.34												
.23	.22											
.42	.31	.26										
.41	.23	.08	.63									
.42	.22	.23	.74	.56								
.42	.15	.21	.61	.56	.83							
.40	.17	.19	.67	.55	.69	.57						
.36	.13	.14	.58	.58	.63	.57	.87					
.33	.23	.17	.70	.56	.68	.52	.70	.64				
.39	.17	.14	.64	.56	.64	.53	.65	.63	.85			
.23	.21	.00	.33	.43	.29	.28	.22	.26	.41	.41		
.25	.15	.13	.47	.39	.44	.37	.42	.41	.66	.69	.25	
3.17	14.0	24.4	12.81	2.20	12.05	2.2	12.26	2.21	15.22	2.22	.54	.46
1.12	5.88	5.8	8.6	.75	9.2	.73	9.3	.72	12.8	.76	.21	.26
.95	.80	.70	.90	.86	.91	.85	.91	.83	.90	.85		
3.44	16.0	23.64	15.03	2.3	13.5	2.25	13.44	2.31	18.35	2.43	.57	.48
1.23	5.55	5.94	8.82	.82	10.55	.78	10.52	.81	15.0	.86	.21	.26
2.81	14.67	25.32	12.53	2.1	10.36	2.15	10.85	2.09	11.46	1.97	.51	.42
1.06	4.61	5.5	8.43	.64	6.85	.64	7.27	.58	7.97	.52	.21	.26
2.87**	2.45*	2.72**	2.61**	2.39*	3.17**	1.22	2.62**	2.84**	5.19***	5.82***	$2.2^{*}$	3.04***
.55	.25	29	.30	.27	.35	.07	.28	.28	.57	.57	.23	.28
	<i>1</i> .34 .23 .42 .41 .42 .40 .36 .33 .25 3.17 1.12 .95 3.44 1.23 2.81 1.06 2.87** .55	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	Descriptives, Intercorr1234.34.23.22.42.31.26.41.23.08.63.42.22.23.74.42.15.21.61.40.17.19.67.36.13.14.58.33.23.17.70.39.17.14.64.23.21.00.33.25.15.13.473.1714.024.412.811.125.885.88.6.95.80.70.903.4416.023.6415.031.235.555.948.822.8114.6725.3212.531.064.615.58.432.87**2.45*2.72**2.61**.55.2529.30	Descriptives, Intercorrelations,12345.34.23.22.42.31.26.41.23.08.63.42.22.23.74.56.42.15.21.61.56.40.17.19.67.55.36.13.14.58.58.33.23.17.70.56.23.21.00.33.43.25.15.13.47.393.1714.024.412.812.201.125.885.88.6.75.95.80.70.90.863.4416.023.6415.032.31.235.555.948.82.822.8114.6725.3212.532.11.064.615.58.43.642.87**2.45*2.72**2.61**2.39*.55.2529.30.27	Descriptives, Intercorrelations, and Sex D123456.34.23.22.42.31.26.41.23.08.63.42.22.23.74.56.42.15.21.61.56.83.40.17.19.67.55.69.36.13.14.58.58.63.33.23.17.70.56.68.39.17.14.64.56.64.23.21.00.33.43.29.25.15.13.47.39.443.1714.024.412.812.2012.051.125.885.88.6.759.2.95.80.70.90.86.913.4416.023.6415.032.313.51.235.555.948.82.8210.552.8114.6725.3212.532.110.361.064.615.58.43.646.852.87**2.45*2.72**2.61**2.39*3.17**.55.2529.30.27.35	Descriptives, Intercorrelations, and Sex Difference1234567.34.23.22.42.31.26.41.23.08.63.42.22.23.74.56.42.15.21.61.56.83.40.17.19.67.55.69.57.36.13.14.58.58.63.57.33.23.17.70.56.68.52.39.17.14.64.56.64.53.23.21.00.33.43.29.28.25.15.13.47.39.44.373.1714.024.412.812.2012.052.21.125.885.88.6.759.2.73.95.80.70.90.86.91.853.4416.023.6415.032.313.52.251.235.555.948.82.8210.55.782.8114.6725.3212.532.110.362.151.064.615.58.43.646.85.642.87**2.45*2.72**2.61**2.39*3.17**1.22.55.2529.30.27.35.07	Descriptives, Intercorrelations, and Sex Differences Among12345678.34.23.22.42.31.26.41.23.08.63.42.22.23.74.56.42.15.21.61.56.83.40.17.19.67.55.69.57.36.13.14.58.58.63.57.87.33.23.17.70.56.64.53.65.23.21.00.33.43.29.28.22.25.15.13.47.39.44.37.423.1714.024.412.812.2012.052.212.261.125.885.88.6.759.2.739.3.95.80.70.90.86.91.85.913.4416.023.6415.032.313.52.2513.441.235.555.948.82.8210.55.7810.522.8114.6725.3212.532.110.362.1510.851.064.615.58.43.646.85.647.272.87**2.45*2.72**2.61**2.39*3.17**1.222.62**.55.2529.30.27.35.07.28	Descriptives, Intercorrelations, and Sex Differences Among Main Mea123456789.34.23.22.42.31.26.41.23.08.63.42.22.23.74.56.42.15.21.61.56.83.40.17.19.67.55.69.57.36.13.14.58.58.63.57.87.33.23.17.70.56.68.52.70.64.39.17.14.64.56.64.53.65.63.23.21.00.33.43.29.28.22.26.25.15.13.47.39.44.37.42.41 $3.17$ 14.024.412.812.2012.052.212.262.211.125.885.88.6.759.2.739.3.72.95.80.70.90.86.91.85.91.833.4416.023.6415.032.313.52.2513.442.311.235.555.948.82.82.8210.55.7810.52.812.8114.6725.3212.532.110.362.1510.852.091.064.615.58.43.646.85.647.27.58	Descriptives, Intercorrelations, and Sex Differences Among Main Measures12345678910.34.23.22.42.31.26.41.23.08.63.42.22.23.74.56.42.15.21.61.56.83.40.17.19.67.55.69.57.36.13.14.58.58.63.57.87.33.23.17.70.56.68.52.70.64.39.17.14.64.56.64.53.65.63.85.23.21.00.33.43.29.28.22.26.41.25.15.13.47.39.44.37.42.41.663.1714.024.412.812.2012.052.212.262.2115.221.125.885.88.6.759.2.739.3.7212.8.95.80.70.90.86.91.85.91.83.903.4416.023.6415.032.313.52.2513.442.3118.351.235.555.948.82.8210.55.7810.52.8115.02.8114.6725.3212.532.1110.362.1510.852.0911.4	1  2  3  4  5  6  7  8  9  10  11    .34  .23  .22  .31  .26	1  2  3  4  5  6  7  8  9  10  11  12    .34  .23  .22  .22  .42  .31  .26  .41  .23  .08  .63    .42  .22  .23  .74  .56  .42  .15  .21  .61  .56  .83    .40  .17  .19  .67  .55  .69  .57

TABLE 1 Descriptives, Intercorrelations, and Sex Differences Among Main Measures

*Note*: All correlations above .10 are significant at p < .05, correlations above .13 are significant at p < .01, and correlations above .16 are significant at p < .001. ACSQ = Negative Inferential Style; RUM = Ruminative Response Styles subscale; DAS = Children's Dysfunctional Attitudes Scale; CDI = Children's Depression Inventory; ANX = Anxious Arousal; ALEQ = Adolescent Life Events Questionnaire.

symptoms were associated with each other concurrently and prospectively across waves of data as expected. There were no ethnic or racial differences (White vs. non-White in one analysis, and White vs. African American in a second analysis) in any of the variables (all ts < 1.50). Moreover, no significant Sex × Ethnicity results were found, so these interactions were not considered further. Last, Table 1 reports results from independent t tests and the accompanying effect sizes (ES), in terms of Cohen's d (Cohen, 1988; small = .2; medium = .5; large = .8), that were used to investigate whether there were sex differences in variables at baseline and over time. As seen in Table 1, several significant sex differences were observed. Compared to boys, girls reported a more negative cognitive style (medium ES), more rumination (small ES), greater exposure to stressors (small ES), as well as more depressive and anxious symptoms (mostly small ES at the different time points). Compared to girls, boys exhibited more dysfunctional attitudes than girls (small ES).

## Sex Differences in Internalizing Symptoms

Hierarchical linear modeling (HLM 5.04; Raudenbush, Bryk, Cheong, & Congdon, 2001) was used to investigate whether there were sex differences in depressive and anxious arousal symptom trajectories over the four waves of data. This was accomplished in HLM by constructing Level 1 and 2 equations. At Level 1, regression equations modeled separately the variation in the repeated measures (e.g., depressive symptoms) as a function of time (i.e., the four waves of data). Each equation includes various parameters to capture features of an individual youth's level of symptoms (e.g., depression or anxiety) over time, including an intercept that describes an individual's initial level on the variable and a *slope* that describes an individual's average amount of linear change on the variable across time. Only linear change is reported because additional data

points (i.e., at least five waves) are preferable to model reliably and adequately possible quadratic effects for depressive and anxious symptoms over time (Hedeker & Gibbons, 2006). At Level 2, the between-subjects' variables of sex, age, and a Sex  $\times$  Age interaction were used to capture individual differences in the Level 1 parameters. HLM can flexibly handle cases with missing data, so participants with missing data were not eliminated from the data set.

As shown in Table 2, significant sex differences and age effects in depressive and anxious symptoms emerged. Effect sizes, in terms of r (Rosenthal & Rosnow, 1991), are included and can be interpreted as a small ES of .10, medium ES of .30, and large ES of .50. The Sex  $\times$  Age interaction was not significant for intercepts or slopes, so these interactions were not considered further. After controlling for the influence of age, girls reported more initial depressive symptoms than boys (i.e., significant sex difference in depression intercept), and this was a small ES. But boys and girls did not differ on anxiety intercept after controlling for age in the model. Also, sex differences in slopes of depression (medium ES) and anxiety (small ES) were found. Girls reported significantly greater increases in depressive and anxious symptoms over time compared with boys. Significant differences for age were found such that older adolescents exhibited more depressive (small ES) and anxious (small ES) symptoms for intercepts than younger youth. In sum and consistent with hypotheses, significant sex differences in internalizing symptoms were found that could be investigated in mediation analyses.

# **Mediational Analyses**

Mediation analyses were conducted to test whether cognitive vulnerabilities, stressors, and the cognitive vulnerability-stress interactions could account for girls' greater levels of depressive symptom intercepts,

			Intere	cept	Slope						
Predictor	В	SE	t	df	ES (r)	b	SE	t	df	ES (r)	
Depressive Symptoms											
Sex	.15	.047	3.18	1, 346**	.17	6.13	.89	6.19	1, 346***	.31	
Age	1.5	.32	4.63	1, 346***	.24	.29	.30	.99	1, 346	.05	
$Sex \times Age$	.46	.48	.94	1, 346	.05	.80	.58	1.38	1, 346	.07	
Anxious Arousal Symptoms											
Sex	.07	.07	1.02	1, 346	.05	.26	.07	3.69	1, 346***	.19	
Age	.12	.02	5.24	1, 346***	.27	.03	.02	1.36	1, 346	.07	
Sex  imes Age	.03	.05	.62	1, 346	.03	.098	.055	1.77	1, 346	.09	

TABLE 2 Sex and Age Effects on the Trajectories of Symptoms of Depression and Anxious Arousal

*Note*: ES = effect size.

p < .01. p < .001.

depressive symptom slopes, and anxiety symptom slopes (Holmbeck, 2002). To demonstrate mediation, the following conditions must be met: (a) sex differences in symptoms must exist, (b) sex differences in vulnerabilities or stressors must be present, (c) vulnerabilities or stressors must predict symptoms while the effect of sex is included in the model, and (d) the significant association between sex and symptoms must be reduced (i.e., be nonsignificant and have smaller ES) once the vulnerabilities or stressors are included in the analysis. The findings just reported demonstrated the first and second conditions in that there were significant sex differences in symptoms, stressors, and the cognitive vulnerabilities of negative cognitive style and rumination. No mediation analyses could be conducted on anxiety intercepts because there was no significant sex difference in this outcome. Also, because boys exhibited more dysfunctional attitudes than girls, neither dysfunctional atti-Dysfunctional Attitudes × Stress tudes nor the interaction was included in the mediation analyses because these processes cannot explain why girls report more depressive and anxious symptoms than boys.

To test the third and fourth conditions, HLM analyses were conducted to account for the sex difference in depressive symptoms intercepts and slopes as well as anxiety slopes. In particular, one model tested HT and examined the Negative Cognitive Style × Stress interaction, a second model tested the original RST and investigated the main effect influences of rumination and stressors, a third model tested the extended RST hypothesis for a Rumination × Stress interaction, and a final model integrated HT and original RST and included all of these factors (Negative Cognitive Style  $\times$ Stress and Rumination). This final model was constructed to evaluate how much the inclusion of factors from both cognitive theories of HT and RST could account for the sex difference in depression and anxiety. An integrated HT and extended RST model, which included the Negative Cognitive Style × Stress interaction and the Rumination × Stress interaction would not converge, likely because of collinearity between these two interaction terms included in the same model, so that model is not reported.

For Model 1 (Negative Cognitive Style  $\times$  Stressors), all necessary between-subjects variables (i.e., main effects of sex, age, negative cognitive style, stressors, and the interaction of Negative Cognitive Style  $\times$  Stress) were entered in Level 2 to predict both intercepts and slopes for depression and anxiety slopes at Level 1. Similarly for Model 2 (Rumination and Stressors), the required between-subjects variables (i.e., main effects of sex, age, rumination, and stressors) were entered in Level 2 to predict intercepts and slopes of depression and anxiety slopes at Level 1. Next, for Model 3 (Rumination  $\times$  Stressors), all necessary between-subjects

variables (i.e., main effects of sex, age, rumination, stressors, and the interaction of Rumination × Stress) were entered in Level 2 to predict both intercepts and slopes for depression and anxiety slopes at Level 1. Finally Model 4 (Negative Cognitive Style × Stressors and Rumination) included all necessary main effects and the Negative Cognitive Style × Stress interaction at Level 2 to predict intercepts and trajectories of depression and anxiety slopes at Level 1. In Table 3, the findings for mediation of sex differences in depression intercepts and slopes are presented first and anxiety slopes second.

For the third criterion of mediation for the sex difference in depressive symptoms, results indicated that the Negative Cognitive Style × Stress interaction predicted intercepts and trajectories of depressive symptoms over time in Model 1. Analyses that decomposed the Negative Cognitive Style × Stress interaction revealed that youth with a more negative cognitive style and reported more stressors exhibited the greatest elevation in depressive symptoms. Rumination and stressors were both associated with intercepts, but not trajectories, of depressive symptoms in Model 2. In Model 3, the Rumination × Stress interaction predicted intercepts and trajectories of depressive symptoms such that youth with a more ruminative response style who reported more stressors experienced higher elevations in depressive symptoms. Finally, in Model 4, both rumination and the Negative Cognitive Style × Stress interaction predicted intercepts for depressive symptom elevations, whereas only the Negative Cognitive Style × Stress interaction predicted trajectories of depression over time. The ES for the Negative Cognitive Style × Stress interaction predicting depression intercept was small and medium for predicting slopes. The ES for rumination predicting intercepts was small, and the ES for Rumination × Stress interaction was medium for intercepts and slopes.

For the fourth criterion of mediation, the findings showed that the sex difference in depressive symptoms intercepts was no longer significant for any of the models when the mediating variables were included. The sex difference in depression trajectories was reduced slightly, but remained significant, in all models. To provide an estimate of the effect size of these mediators accounting for the sex difference in depressive symptoms, the percentage of the association between sex and depressive symptoms that was explained by these mediators was examined (Holmbeck, 2002). For the sex difference in intercepts of depressive symptoms, the association between sex and depression intercept was reduced substantially (94% of the association was explained by mediators in Model 1, 76% for Model 2, 48% for Model 3, and 82% for Model 4). The link between sex and depressive symptom trajectories was diminished

TABLE 3										
Mediational Analyses to Account for Sex Differences in the	Trajectories of Depressive and Anxious Arousal Symptoms									

			ept	Slope						
Predictor	b	SE	t	df	ES(r)	b	SE	t	df	ES(r)
Depressive Symptoms										
Negative Cognitive Style × Stress										
Sex	08	72	11	1 344	005	4 50	78	5 54	1 344***	29
Δα	20	.72	.11	1, 344	.005	 03	31	12	1, 344	.29
	.20	5.23	.70	1, 344	.04	12.18	3.01	4.04	1, 344	.000
Stress	2 7 2	2.00	1 31	1, 344	.00	58	60	4.04	1, 344	.21
$\Delta CSO \times Stress$	5.80	2.09	2.45	1, 344	13	5.82	.00	6.31	1, 344	.05
Pumination and Stross	5.80	2.30	2.43	1, 544	.15	5.62	.92	0.51	1, 544	.52
Sev	60	78	78	1 345	04	5 3 2	88	5 00	1 3/15***	31
	1.58	.78	5.81	1, 345	30	002	.00	01	1, 345	.51
Age	20	.27	5 72	1, 345	20	.002	.51	.01	1, 345	.00
Stress	2 21	.00	1 10	1, 345	.29	1.01	.07	3 /1	1, 345	.05
Dumination × Stress	2.21	.49	4.49	1, 545	.23	1.91	.50	5.41	1, 545	.10
Sev	04	02	1.65	1 344	00	17	03	5 4 5	1 3//***	20
Ago	.04	.02	5.25	1, 344	.09	.17	.03	01	1, 344	.29
Age	.05	.01	5.25	1, 344	.27	.01	.01	.91	1, 344	.05
Kulli	.002	.005	.05	1, 344	.04	.02	.004	0.15	1, 344	.52
Suress	.02	.02	1.12	1, 344	.00	.002	.02	.10	1, 344	.005
Rum × Stress	.009	.001	0.11	1, 344	.32	.01	.001	7.42	1, 344	.37
Negative Cognitive Style × Stress and Kunniation	26	72	51	1 242	0.2	4 42	70	5 (2	1 242***	20
Sex	.30	.72	.51	1, 343	.03	4.45	./8	3.62	1, 343	.28
Age	.39	.29	2.01	1, 343	.11	.15	.32	.40	1, 343	.02
ACSQ	.81	4.95	.10	1, 343	.01	11.82	3.00	3.93	1, 343	.21
Rum	.30	.08	3.6/	1, 343	.19	.09	.08	1.04	1, 343	.05
Stress	2.82	1.93	1.46	1, 343	.08	.48	.59	.81	1, 343	.04
$ACSQ \times Stress$	5.47	2.16	2.53	1, 343**	.14	5.85	.92	6.34	1, 343***	.32
Anxious Arousal Symptoms										
Negative Cognitive Style × Stress										
Sex	.04	.06	.66	1, 344	.03	.18	.06	2.85	1, 344**	.15
Age	.05	.03	1.64	1, 344	.09	.05	.03	1.85	1, 344†	.09
ACSQ	.48	.31	1.57	1, 344	.08	1.39	.18	7.55	1, 344***	.37
Stress	.11	.13	.89	1, 344	.05	.01	.04	.32	1, 344	.01
$ACSQ \times Stress$	.18	.14	1.24	1, 344	.07	.52	.06	8.87	1, 344***	.43
Rumination and Stress										
Sex	.003	.06	.06	1, 345	.003	.27	.07	3.76	1, 345***	.19
Age	.13	.02	6.03	1, 345***	.31	.03	.02	1.36	1, 345	.07
Rum	.023	.005	4.01	1, 345**	.21	.00	.006	.89	1, 345	.05
Stress	.29	.04	6.99	1, 345***	.35	.03	.04	.81	1, 345	.04
Rumination $\times$ Stress										
Sex	.03	.06	.44	1, 344	.02	.21	.07	3.09	1, 344***	.16
Age	.13	.02	5.59	1, 344***	.28	.01	.02	.62	1, 344	.03
Rum	.001	.008	.12	1, 344	.005	.05	.009	5.11	1, 344***	.27
Stress	.21	.04	4.47	1, 344	.23	.18	.05	3.58	1, 344***	.19
$Rum \times Stress$	.01	.003	3.75	1, 344**	.19	.02	.003	5.98	1, 344***	.31
Negative Cognitive Style $\times$ Stress and Rumination										
Sex	.02	.06	.42	1, 343	.02	.18	.06	2.95	1, 343**	.15
Age	.06	.02	2.37	1, 343*	.12	.05	.03	1.61	1, 343	.09
ACSQ	.44	.29	1.49	1, 343	.08	1.36	.18	7.55	1, 343***	.37
Rum	.01	.006	2.63	1, 343**	.14	.00	.005	.77	1, 343	.04
Stress	.11	.13	.87	1, 343	.04	.23	.05	4.82	1, 343***	.25
ACSO × Stress	.16	.13	1.20	1, 343	.02	.52	.06	8.96	1, 343***	.43

*Note.* Rum = Ruminative Response Styles subscale; ACSQ = Adolescent Cognitive Style Questionnaire; Stress = negative life events. <sup>†</sup> <math>p < .07. <sup>\*</sup>p < .05. <sup>\*\*</sup>p < .01. <sup>\*\*\*</sup>p < .001.

mildly (7%) of the association was explained by mediators in Model 1, 0% for Model 2, 12% for Model 3, and 10% for Model 4). Taken together, these findings

suggest that most of the sex difference in initial levels of adolescent depressive symptoms can be explained by girls' experiencing more stressors and interpreting these stressors in a negative manner (i.e., the Negative Cognitive Style  $\times$  Stress interaction) and by girls' greater levels of rumination interacting with more stressors (i.e., the Rumination  $\times$  Stress interaction) compared with boys. Further, the combination of girls' encountering more stressors and interpreting these events in a more pessimistic way (i.e., the Negative Cognitive Style  $\times$  Stress interaction) as well as ruminating on the affect that may result from these events (i.e., the Rumination  $\times$  Stress interaction) partly explained why girls' levels of depressive symptoms increase over time more so compared to boys.

For the third criterion of mediation for the sex difference in anxious symptom slopes, the Negative Cognitive Style  $\times$  Stress interaction and the Rumination  $\times$  Stress interaction both predicted trajectories of anxious arousal symptoms over time (medium ES). Although mediation of the sex difference in anxiety intercepts could not be formally examined, results of the HLM analyses for anxiety intercepts indicated that the main effect of rumination in Model 2 and the Rumination × Stress interaction in Model 3 predicted initial levels of anxious arousal. For the fourth criterion of mediation, the sex difference in anxious symptom trajectories was reduced somewhat, but remained significant, in the models that included the Negative Cognitive Style × Stress interaction (models 1 and 4) and Model 3 in which the Rumination × Stress interaction was included. The link between sex and anxious symptom trajectories was diminished moderately (21% of association explained by mediators in Model 1, 0% for Model 2, 16% for Model 3, and 21% for Model 4). Taken together, these findings suggest that girls' increasing trajectories of anxious symptoms were partially accounted for by encountering more stressors and interpreting these negative events in a more pessimistic way or by ruminating on the affect generated by these events.

### DISCUSSION

It is well documented that girls are more likely than boys to experience symptoms of anxiety and depression, yet the reasons and mechanisms underlying these sex difference patterns in internalizing psychopathology have remained elusive. The results of the present study show that some of the sex differences in these internalizing symptoms can be accounted for by particular cognitive vulnerabilities and stressors as risk mechanisms, in particular the Negative Cognitive Style × Stress interaction and the Rumination × Stressors interaction. These cognitive processes and exposure to stressors explained most of the sex difference in initial level of depressive symptoms and some of the sex difference in prospective trajectories of depressive and anxious symptoms.

In particular, girls, compared with boys, reported higher initial levels of depressive symptoms only, but not anxious arousal symptoms, and increasing trajectories of both depressive and anxious symptoms across the four waves of data after controlling for age. The independent t tests, in which age was not controlled, showed significant sex differences in anxious arousal (three out of four waves) and depressive symptoms (all four waves), and these findings appear consistent with the past epidemiological literature. This suggests that girls exhibit more anxious arousal and depressive symptoms than boys when age influences are not controlled, but the sex difference in initial anxious arousal symptoms are not maintained after removing the effect of age in this sample of early to middle adolescents. The sex difference in initial depressive symptoms was observed regardless of whether age was controlled. Also, specific anxious arousal symptoms were assessed, rather than general anxiety symptoms or other particular manifestations of anxiety (e.g., social anxiety, worry, etc.). Future research needs to replicate these findings as past research has not investigated sex differences in anxious arousal symptoms among adolescence (see Watson et al., 1995, for evidence of sex differences in anxious arousal among adults).

The sex difference in initial levels of depressive symptoms was accounted for by cognitive vulnerabilities (a higher ruminative response style and a negative cognitive style) interacting with stressors. These findings are consistent with the elaborated cognitive vulnerabilitytransactional stress model (Hankin & Abramson, 2001) that postulated that girls would exhibit higher levels of cognitive vulnerabilities and exposure to stressors, and the interactive combination of these factors would account for girls reporting more depressive symptoms than boys. It is interesting to note that the interaction of a negative cognitive style and stressors partially explained the sex difference in depressive symptoms slope as well as intercepts, whereas rumination as a main effect, as originally postulated by RST, only accounted for the sex difference in intercepts of depressive symptoms and not slopes. No support was found for dysfunctional attitudes as a mediator of the sex difference in either anxiety or depressive symptoms because boys exhibited more dysfunctional attitudes than girls, so this precluded any further mediation analyses of the sex difference in internalizing symptoms (see also Haeffel et al., 2003, for evidence showing that adult men report more dysfunctional attitudes than women). Overall, these findings provide support for particular cognitive vulnerabilities (i.e., negative cognitive style and rumination) interacting with stressors as an explanation of the sex difference in both depression intercepts and slopes.

These findings add new information to and have implications for a developmental psychopathological

account of how cognitive processes, alone and in interaction with stressors, can explain why girls become more depressed than boys over time during adolescence. First, the limited past research to investigate mediation of the sex difference in adolescent depression has focused solely on main effects, such as the independent mediating role of a negative cognitive style (Hankin & Abramson, 2002), rumination (Schwartz & Koenig, 1996), or stressors (e.g., Hankin et al., 2007; Rudolph, 2002), so it was not known how these different processes operate in conjunction to account for girls' elevated levels of depression in adolescence. The present study examined the influence of both of the core hypothesized mechanisms from HT and RST. First, separate models were analyzed to investigate the independent contributions from HT, the original and extended RST, and then factors from both models were entered together in one model to provide a more comprehensive understanding of the additive and interactive effects that may account for the sex difference in adolescent depression and anxiety. By including the core etiological processes, based on these prominent cognitive theories of depression, this study allowed for a stronger investigation of how much of the female preponderance in depressive and anxious symptoms could be explained by these cognitive processes, both as independent main effects and in interaction with stressors.

Second, this study separately examined how these cognitive factors and stressors predicted initial levels of depressive symptoms (i.e., intercepts) as well as trajectories over time (i.e., slopes). Past research examining cognitive vulnerabilities and stressors as mediators of the sex difference in internalizing symptoms has not used multi-wave data and separately investigated intercepts from slopes. No information was available in the literature concerning whether and how these different cognitive factors and stressors differentially account for the initial level and the increasing trajectory of girls' greater depressive symptom levels compared with boys. The findings that the main effects of rumination and stress from the original RST, Rumination × Stress interaction from the extended RST, and the Negative Cognitive Style × Stress interaction from HT all explained almost all of the sex difference in depression intercept, but only the cognitive vulnerabilities of negative cognitive style and rumination interacting with stress partially mediated the sex difference in depression trajectory provide important new information for understanding why more girls are depressed than boys and can inform theoretical models of the sex difference in adolescent depression.

None of the extant theories of the development of the sex difference in depression has postulated specific factors or processes that would differentially account for girls' greater initial levels of depression in contrast to their increasing trajectories over time than boys. The present findings, in which certain factors accounted for only initial levels, and particular influences explained both intercepts and trajectories, suggest that future empirical and theoretical research is needed to consider which explanatory factors and processes contribute differentially to girls' higher initial levels as well as trajectories of depressive symptoms over time. With statistical and methodological advances (e.g., Curran & Willoughby, 2003) that can take appropriate advantage of multi-wave data to separately analyze initial levels from trajectories of symptoms for girls and boys, it is hoped that more refined theories with increasing sophistication will follow to improve understanding of why more girls become increasingly depressed than boys starting in early adolescence and expanding throughout middle adolescence into young adulthood (Hankin et al., 1998).

For anxious symptoms, mediation analyses could only be conducted for the sex difference in anxiety trajectories because girls did not differ from boys on initial levels of anxiety after controlling for age. The Negative Cognitive Style × Stress interaction and the Rumination × Stress interaction both accounted partially for girls' increasing trajectories of anxious symptoms over time. Explanations for the sex difference in anxiety symptoms was examined in this study given the finding that girls are more anxious than boys and that anxiety commonly co-occurs with depression. It was of interest to examine the specificity of mediating influences that would account for the sex difference in depression as well as anxiety. Rumination, as a main effect, explained why girls reported more initial levels of depressive symptoms, but not anxiety symptom trajectories. The cognitive vulnerabilities (i.e., negative cognitive style and rumination) interacted with stressors to partially mediate the sex difference in both trajectories of depression and anxiety. Thus, these results indicate that some cognitive processes explained aspects of the sex difference in both depression and anxiety, whereas other cognitive mechanisms accounted for the sex difference in depression only. These results of differential prediction and mediation of the sex difference in internalizing symptoms underscore the importance of assessing multiple etiological processes as well as symptoms over multiple waves of data because the same set of factors and mechanisms do not explain the sex difference in both intercepts as well as trajectories of internalizing symptoms over time.

#### Strengths and Limitations

The findings from the present research should be interpreted with particular limitations in mind. First, the data on symptoms and psychosocial factors were all self-reported by the youth. Replication of these results with multiple methods and informants should be undertaken. Second, diagnostic clinical levels of anxiety and depression were not assessed in this study with structured diagnostic interviews, so it is unknown whether the findings will generalize to these problems at the disorder level. Although research suggests that internalizing syndromes are represented and conceptualized best as dimensional continua, rather than discrete categories (e.g., Hankin, Fraley, Lahey, & Waldman, 2005), use of structured diagnostic interviews in future research can address this issue. Third, the timing of the prospective intervals (i.e., every 5 weeks over 5 months) was fairly short. Future multiwave research can build on these findings and investigate longer time spans between intervals. Finally, negative life events were examined only at Times 1 and 4, so it cannot be determined precisely that stressors assessed at Time 4 preceded changes in depressive or anxious symptoms at Times 2 or 3. Other multiwave research with stressors and symptoms assessed and analyzed at multiple waves suggests that stressors do, indeed, precede and predict prospective change in internalizing symptoms (Hankin, 2008b; Hankin et al., 2008), although additional multiwave research is needed in the investigation of sex differences in the development of psychopathology.

On the other hand, several strengths of the present study enhance confidence in the findings. First, as noted in greater detail previously, the multiwave prospective design enabled a more rigorous examination of mechanisms explaining the sex differences in both initial levels and changes over time in symptoms. Second, data analytic methods best suited for multiwave longitudinal repeated measures data were used, and mediation analyses to address mechanisms underlying the sex difference in anxiety and depressive symptoms were employed. Third, a modestly large sample of early and middle adolescents was used, so there was sufficient power to detect even small effect sizes in sex differences in symptoms and the mediators of these outcomes. Related, examination of early and middle adolescence is appropriate given the developmental epidemiological literature suggesting this is the time when the sex differences in internalizing symptoms dramatically diverge. Last, a community-based sample was used, so more accurate and less biased estimates and inferences could be made. Also, the sample was relatively racially and ethnically diverse and represented a relatively wide socioeconomic range, as opposed to predominantly White, middle-class samples used in most past research. As no Sex × Ethnicity effects were found, the results suggest that the mechanisms explaining the sex differences in symptoms may apply across ethnicities, although future research with larger samples with

greater representation of ethnic groups will be needed to verify this.

#### Implications for Research, Policy, and Practice

Practically, these findings suggest that girls' greater initial levels of depression and their increasing trajectories of depressive and anxious symptoms can be explained, to some degree, by certain cognitive processes and stressors. Several empirically supported interventions exist that target these risk factors. For example, cognitive behavioral treatments are efficacious at reducing depression (Weisz, McCarty, & Valeri, 2006), and these interventions target cognitive vulnerabilities, such as a negative cognitive style, rumination, and stress levels. With the clear global burden associated with depression (Murray & Lopez, 1996), especially for girls and women, the present results have potential implications for policy and practice by identifying some psychosocial factors that may be amenable to intervention.

These findings also have implications for future research. One particularly intriguing potential is integrating the psychosocial mechanisms examined in this study with biological, hormonal, and genetic influences that may account for the sex differences in internalizing symptoms (e.g., Cyranowski, Frank, Young, & Shear, 2000; Hankin & Abramson, 2001). Increasingly more research is focusing on gene-environment interactions and correlations, such as the well-replicated finding that variations in the serotonin transporter gene interact with stressful experiences to predict depression (e.g., Rutter, Moffitt, & Caspi, 2006). Molecular genetic research suggests that girls with this genetic variant are more prone to depression, whereas boys may be protected (Sjoberg et al., 2006). Other recent genetic research showed that rumination mediated the link between another molecular genetic risk to depression (brain derived neurotrophic factor) and depressive symptoms among adolescent girls and their mothers (Hilt, Sander, Nolen-Hoeksema, & Simen, 2007). Behavioral genetic research suggests that postpubertal adolescent girls have a greater latent genetic liability to depression and to experience more stressful life events than boys (Hankin & Abramson, 2001). Although most research has not found biological or hormonal factors to account for the sex difference in adolescent depression (Hankin et al., 2008), such burgeoning molecular and behavioral genetic research suggests new possible avenues for mechanisms that may explain why girls experience more depression than boys. A fuller account of the development of the sex difference in depression and anxiety will likely be obtained by integrating genetic and biological risks with psychosocial factors (e.g., Hankin & Abramson, 2001; Rutter et al., 2003).

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