NEW RESEARCH

A Longitudinal Follow-up Study Examining Adolescent Depressive Symptoms as a Function of Prior Anxiety Treatment

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Objective: Children who are fearful and anxious are at heightened risk for developing depression in adolescence. Treating anxiety disorders in pre-/early adolescence may be one mechanism through which depressive symptoms later in adolescence can be prevented. We hypothesized that anxious youth who responded positively to cognitive-behavioral therapy (CBT) for anxiety would show reduced onset of depressive symptoms 2 years later compared to treatment nonresponders, and that this effect would be specific to youth treated with CBT compared to an active supportive comparison treatment.

Method: Participants were 80 adolescents ages 11 to 17 years who had previously completed a randomized trial comparing predictors of treatment response to CBT and child-centered therapy (CCT). Youth met *DSM-IV* criteria for generalized, separation, and/or social anxiety disorder at the time of treatment. The present study was a prospective naturalistic 2-year follow-up examining trajectories toward depression, in which participants were reassessed for depressive symptoms 2 years after anxiety treatment. Treatment response was defined as a 35% reduction in independent evaluator−rated anxiety severity on the Pediatric Anxiety Rating Scale after treatment.

Results: As hypothesized, lower levels of depressive symptoms were observed in anxious youth who responded to CBT for anxiety ($\beta = -0.807$, $p = .004$) but not CCT ($\beta = 0.254$, $p = .505$). Sensitivity analyses showed that the effects were driven by girls.

Conclusion: Findings suggest that CBT for anxiety is a promising approach to preventing adolescent depressive symptomatology, especially among girls. The results highlight the need for better early screening for anxiety and better dissemination of CBT programs targeting anxiety in youth.

Key words: cognitive-behavioral therapy, anxiety, depression, prevention

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Depression is a leading cause of worldwide disability[1,2] that increases markedly during adolescence, with one in seven adolescents experiencing an episode of depression prior to adulthood.[3,4] Depression that onsets earlier in life is associated with problems in physical health; emotional, social, and occupational functioning into adulthood; and a high likelihood of recurrence.[5-8] Yet, up to half of youth who meet criteria for depression do not receive treatment, and even among treated youth, many do not achieve full remission.[9] For these reasons, there is a critical need to identify early preventive and treatment approaches.[10]

Adolescence as a Period of Risk for Depression

The transition into adolescence may represent a sensitive period for the development and prevention of depressive symptoms. The transitional period between preadolescence and early adolescence, beginning with the onset of pubertal changes around age 9 or 10 years,[11] involves dramatic multi-systemic changes that influence social and affective functioning and learning.[12] These include rapid physical growth, changes in endocrine function and neural development, renegotiation of family and peer relationships, igniting romantic interests, advanced hypothetical thinking skills, and shifts in sleep and circadian regulation.[13-15] These changes are believed to converge in ways that amplify risk for depressive symptoms during this developmental period.[16,17] Because the preadolescent and early adolescent periods are marked by neural plasticity, this may also be an opportune time to modify risk factors in ways that could potentially have a positive impact on the life course trajectory. Providing effective interventions for vulnerable youth in pre- and early

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adolescence could curtail a lifetime of disability.[14] Research is needed to identify and test modifiable risk factors for depression that can be targeted during this developmental period.

Anxiety as a Risk Factor for Depression

Rice *et al.*[18] suggested that heightened fear and anxiety represent one pathway toward depression that could be targeted in primary preventive interventions for children. Having elevated levels of fear and/or anxiety in childhood or early adolescence dramatically increases risk for developing depression later in adolescence.[18-20] This is especially true for youth with social, separation, and generalized anxiety.[21] Although not all children with anxiety disorders go on to develop depression, about 75% of youth who do develop depression have a history of at least one anxiety disorder.[22] Genetic studies demonstrate that depressive symptoms during adolescence are correlated with earlier symptoms of anxiety, highlighting at least a partially heritable basis for this pathway.[23] Psychological treatments for anxiety are efficacious for many youth, with cognitive-behavioral therapy (CBT) treatment response rates of about 65%.[24] Notably, reported treatment response rates are higher for anxiety disorders than for depressive disorders in childhood and adolescence, regardless of whether treatment was delivered via CBT, medication, or combined CBT plus medication.[24-28] Thus, treating anxiety prior to the onset of depressive symptoms may be more efficacious and cost-effective than waiting to intervene until the emergence of depressive symptoms.

Silk *et al.*[16] suggested that one mechanism through which elevated anxiety may contribute to the onset of depressive symptoms may be through reducing reward-seeking behaviors, which in turn reduces the experience of pleasant events. Anxious youth often avoid potentially risky but also rewarding activities, such as going to a party, playing a sport, or making a new friend. This process could contribute to the social withdrawal and anhedonia that characterize—and often precede—depression.[29] The goal of CBT is to reduce avoidance by teaching youth skills to cope with the feared scenarios and then directly exposing them to these scenarios in a gradual and supportive fashion that reduces the intensity of anxiety and promotes approach behavior. Over time, successfully treated anxious youth learn to approach feared situations even if they carry some risk, and to reap the resulting rewards. Our goal, therefore, was to examine whether successful treatment of anxiety in early adolescence could reduce risk for experiencing problems with depressive symptoms later in adolescence. One previous retrospective study did not find lower depressive symptoms in adults who had been responsive to anxiety treatment, compared to those who had not been responsive to anxiety treatment, approximately 16 years prior.[30] However, Wolk *et al.*[31] reported that despite a lack of within-sample group differences, treated youth in this sample experienced lower rates of depressive disorder diagnoses over time than would have been expected based on population prevalence. No prospective studies that we are aware of have examined the possible preventative role of CBT for anxiety on depressive symptoms during adolescence, or compared CBT versus other therapies for prevention of future depressive symptoms.

Current Study

The present study examined depressive symptoms in adolescents 2 years after receiving CBT or a comparison treatment for an anxiety disorder. In the original clinical trial (Silk et al., 2018), the majority of youth in both CBT and child-centered Q2 therapy (CCT) were classified as treatment responders (CBT: 71%; CCT: 56%), but CBT showed superiority in 1-year follow-up treatment response rates, as well as full recovery rates (ie, no longer meeting diagnostic criteria) at both post-treatment and 1-year follow-up. In the present study, we focus on depressive symptoms, given evidence that depression is a dimensional construct[32] and that youth with subclinical depressive symptoms show impairment and greater likelihood of subsequent diagnosis.[33,34] This approach also afforded more power to test hypotheses, as we did not expect a large number of case-level diagnoses in our relatively small sample. We hypothesized that anxious youth who were classified as treatment responders after anxiety treatment would show lower depressive symptomatology at a 2-year follow-up compared to youth who were nonresponders, regardless of initial depressive symptoms. We also hypothesized that the benefit of treatment on future depressive symptoms would be stronger for youth who were responders to CBT compared to supportive therapy, given that CBT focuses on reducing avoidance and increasing approach behaviors, whereas CCT does not aim to directly change behavior. To determine whether any effects observed could be accounted for by acute effects on depressive symptoms, we also explored whether the link between treatment response and 2-year depressive symptoms was mediated by posttreatment depressive symptoms. Finally, we explored whether the link between treatment response and 2-year depressive symptoms was moderated by sex, pubertal status, or age at the time of treatment.

METHOD

Participants

Participants were 80 youth who met *DSM-IV*[35] criteria for a diagnosis of generalized anxiety disorder (75%), separation

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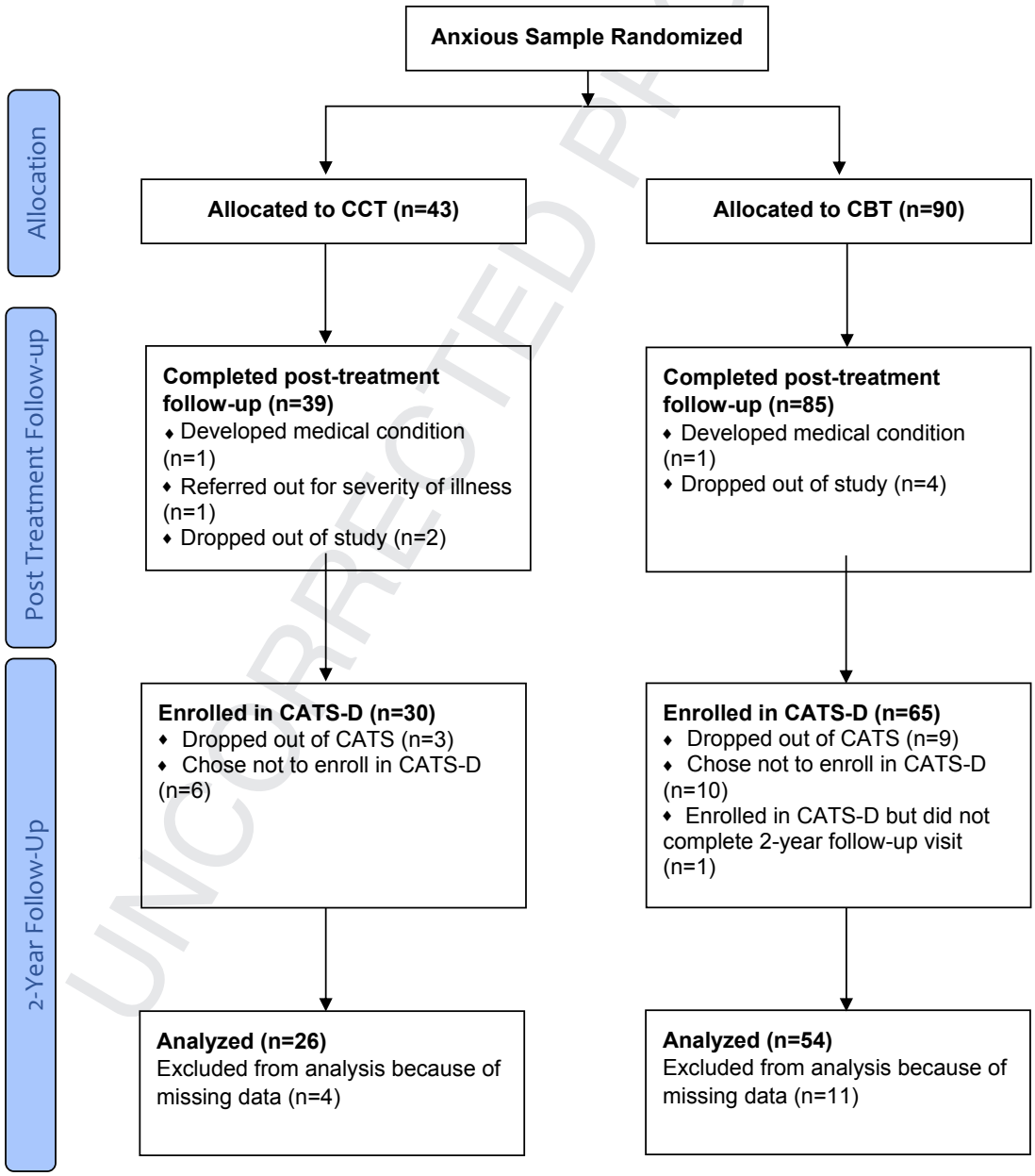
anxiety disorder (20%), and/or social anxiety disorder (24%), who previously received CBT (n = 54) or CCT (n = 26) as part of a randomized clinical trial[33] and were subsequently enrolled in a follow-up study (Figure 1). Participants were 9 to 14 years old (mean = 11.21, SD = 1.47) at the pretreatment assessment and 11 to 17 years of age (mean = 13.56, SD = 1.52) at the 2-year follow-up. Table 1 lists participant characteristics. Participants were excluded if they demonstrated an IQ below 70 as assessed by the Wechsler Abbreviated Scale of Intelligence (WASI),[36] required current ongoing treatment with psychoactive medications including anxiolytics and antidepressants, were acutely suicidal or at risk for harm to self or others, failed to meet magnetic resonance imaging (MRI) safety requirements, or had previously completed a course of CBT. Diagnostic exclusionary criteria included current primary diagnosis of major depressive disorder, current diagnosis of obsessive-compulsive disorder, posttraumatic stress disorder, conduct disorder, substance abuse or dependence, attention deficit/hyperactivity disorder combined type or predominantly

FIGURE 1 Consolidated Standards of Reporting Trials (CONSORT) Diagram



Note: *CATS-D = Child Anxiety Treatment Study–Depression Follow-up; CBT = cognitive-behavioral therapy; CCT = child-centered therapy.*

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TABLE 1 Descriptive Characteristics of Participants

	Treatment Responders (n = 56)	Treatment Nonresponders (n = 24)
Age, y	10.99 (1.6)	11.30 (1.3)
Female participants, n (%)	25 (44.6%)	17 (70.8%)
Caucasian, n (%)	50 (89.3%)	21 (87.5%)
Baseline current diagnosis [a,b], n (%)		
Separation anxiety disorder	10 (17.8%)	5 (20.8%)
Social anxiety disorder	12 (21.4%)	6 (25.0%)
Generalized anxiety disorder	42 (75.0%)	18 (75.0%)
Specific phobia	10 (17.8%)	3 (12.5%)
Major depressive disorder	1 (1.8%)	0 (0.0%)
Baseline depressive symptoms[c]	18.1 (11.4)	17.9 (9.6)
Anxiety diagnosis at 2-year follow-up[a,d], n (%)		
Separation anxiety disorder	1 (1.8%)	0 (0.0%)
Social anxiety disorder	6 (10.7%)	4 (16.7%)
Generalized anxiety disorder	10 (17.9%)	5 (20.8%)
Specific phobia	3 (5.4%)	3 (12.5%)
Depressive disorder over 2-year follow-up, n (%)		
Major depressive disorder	2 (3.5%)	1 (4.2%)
Depressive disorder not otherwise specified	2 (3.5%)	1 (4.2%)
2-Year depressive symptoms[c]	7.7 (8.1)	12.5 (11.6)

Note:
[a]*Diagnostic groups are partially overlapping because of inclusion of comorbid patients, meaning that percentages for the 3 diagnostic inclusion groups will not sum to 100.*
[b]*One participant in the treatment responder group and one participant in treatment nonresponder group met criteria for attention-deficit/hyperactivity disorder (ADHD).*
[c]*Depressive symptoms assessed using child-report on the Mood and Feelings Questionnaire.*
[d]*Among treatment responders at 2-year follow-up, 4 participants received a current diagnosis of obsessive-compulsive disorder, Tourette disorder, or posttraumatic stress disorder, and 2 participants received a diagnosis of ADHD. One participant in the treatment nonresponder group met criteria for a current ADHD diagnosis.*

hyperactive-impulsive type, or lifetime diagnosis of autism or Asperger syndrome, bipolar disorder, psychotic depression, schizophrenia, or schizoaffective disorder. Participants were originally recruited for the treatment study through community advertisements (84%) or referrals from pediatricians, school counselors, or mental health professionals (16%) from a metropolitan midwestern American city.

Procedure

Full procedures for the RCT are described in Silk *et al.*[37] (see also Supplement 1, available online). Briefly, youth were randomized to 16 sessions of psychotherapy delivered in a University of Pittsburgh clinic, with a 2:1 ratio for assignment to CBT or CCT. The CBT was delivered using the *Coping Cat* manual[38] and focused on building coping and problem-solving skills during the first half of treatment, followed by graduated exposure in the second half of treatment. CCT is a manualized nondirective, supportive therapy based on humanistic principles that was used as an active comparison treatment for the original study.[39,40] CCT places an emphasis on core nonspecific therapeutic ingredients such as active listening, reflection, accurate empathy, and encouragement to talk about feelings, but does not include directive problem solving, psychoeducation about anxiety or coping skills, or exposure. Previous research on this sample has shown that both treatments resulted in a positive treatment response for a large number of youth, with acute treatment response rates at 69% for CBT and 60% for CCT.[37]

Of the 133 youth who were originally randomized to treatment (Figure 1), 124 completed a posttreatment assessment and 96 completed a 1-year follow-up. Of these, 95 were successfully recruited into the Child Anxiety Treatment Study—Depression Follow-up (CATS-D) and were invited to return to the laboratory for assessments (conducted by interviewers blinded to treatment group) approximately 2 years after their posttreatment assessment. The goal of the CATS-D Study was to examine the impact of successful anxiety treatment on subsequent symptoms of depression and related neurobehavioral functioning in adolescents. Complete data were available for a final sample of 80 participants (Figure 1, Consolidated Standards of Reporting

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Trials [CONSORT] Diagram). There were no baseline differences in age, sex, anxiety severity, depressive severity, or treatment response between the subset of participants who comprised the final sample (n = 80) and those who participated in the larger trial that were not included in the final sample (n = 53) (all p values >.2). Data on enrollment in follow-up intervention services in the community and enrollment in a behavioral sleep intervention offered through our clinic were also collected using the Supplemental Services module from the Anxiety Disorders Interview Schedule for Children.[41]

Diagnoses were determined at pretreatment, posttreatment, and follow-up by an independent evaluator who was blinded to treatment assignment using the Schedule for Affective Disorders and Schizophrenia for School-Age Children—Present and Lifetime Version (K-SADS-PL).[42] Treatment response was defined as a 35% reduction in independent evaluator–rated anxiety severity on the Pediatric Anxiety Rating Scale (PARS)[43] from pre- to posttreatment.[44] The present sample included 56 treatment responders (70%) and 24 nonresponders (30%). One participant met criteria for a depressive disorder at the time of enrollment in the original study, but the depressive disorder was considered secondary to anxiety and did not recur over the 2-year follow-up. Six participants (7.5%) developed a depressive disorder during the 2 years after anxiety treatment (12-month prevalence, 3.75%). Depressive symptoms were assessed at all time points using youth-report on the long version of the Mood and Feelings Questionnaire (MFQ-C).[45] We focused on child-reported depressive symptoms as the primary outcome variable, given evidence that youth are typically better reporters of internalizing symptoms than are parents,[46] but we also report supplementary analyses using parent-report of depressive symptoms (MFQ-P; see Supplement 2, available online). Pubertal status was assessed at pretreatment using the Pubertal Development Scale.[47]

Analytic Plan

Analyses were conducted on all participants for whom complete data were available. Consistent with an intent-to-treat approach, we included participants who withdrew from treatment but continued to complete assessments (n = 4). Baseline demographic and clinical variables that could also contribute to risk for later depression were included as covariates, including sex, baseline depressive symptoms, and receipt of additional psychological services during the 2-year follow-up enrollment. Follow-up services were classified as enrollment in community treatment, which included psychotherapy and/or medication (yes/no), and enrollment in a supplementary sleep intervention (yes/no). Hierarchical linear regression was performed with 2-year MFQ-C scores as the outcome. In Step 1, covariates were entered. In Step 2, treatment response status (responder/nonresponder) immediately after anxiety treatment was entered. In Step 3, treatment type (CBT or CCT) and the interaction between treatment response and treatment type were entered. Exploratory analyses included the same covariates at Step 1, and examined sex and development (chronological age and pubertal status at time of treatment) and their interaction with treatment response as predictors of 2-year MFQ-C, both in the full sample and in the subsample who received CBT. Analyses were not conducted within the CCT-only subsample because of the small sample size. For interpretability, continuous variables were standardized across the full dataset, and dichotomous variables were coded as 0 and 1. We report standardized betas (with associated 95% CI), which denote the magnitude of the group difference (for dichotomous variables) or the impact of a 1-SD change in a predictor variable (for continuous variables), expressed in SD units of the DV. Q5

Finally, mediation analyses were conducted to examine whether posttreatment depressive symptoms could help to explain the link between anxiety treatment response and 2-year depressive symptoms. We used a standard three-variable path model and considered M to be a mediator of the relationship between X and Y if: (1) X is related to M (path "a"); (2) M is related to Y, controlling for X (path "b"); and (3) the effect of X on Y controlling for M is significantly different from the direct effect of X on Y (mediation path "a*b"). We tested these path coefficients for a mediation model in which X = treatment response (as defined above, based on degree of anxiety reductions [PARS]), Y = 2-year MFQ-C scores, covarying baseline (pretreatment) MFQ-C, and M = acute post-treatment depression (MFQ-C) scores. These exploratory mediation analyses were conducted on the subsample of participants (n = 66; n = 44 in the CBT subsample) for whom data were available for all measures in the model. Reported p values for each path were obtained via bootstrapping (10,000 bootstrapped samples) using the Mediation Toolbox[48] (http://wagerlab.colorado.edu/tools) implemented in Matlab (version R2016a).

RESULTS

The covariates added in Step 1 (gender, baseline depressive symptoms, and receipt of additional psychological services during the 2-year follow-up enrollment) collectively predicted 2-year depressive symptom scores ($F_{4,75} = 5.01$, $p = .001$, $R^2 = 0.211$, adjusted $R^2 = 0.169$). Specifically, gender (girls>boys: $\beta = 0.463$, 95% CI = 0.038–0.888], $p = .033$) and self-reported enrollment in community treatment during the follow-up interval (enrolled>unenrolled:

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$\beta = 0.781$, 95% CI = 0.312−1.250, $p = .001$) were statistically significant predictors of 2-year depressive symptoms, whereas enrollment in a sleep intervention ($\beta = 0.147$, 95% CI = − 0.286 to 0.580, $p = .501$) and baseline depressive symptoms ($\beta = 0.163$, 95% CI = − 0.044, 0.370, $p = .121$) did not. At Step 2, treatment response immediately after anxiety treatment was added. This predictor did not significantly predict 2-year depressive symptoms across the entire sample, although it approached significance in the expected direction ($\beta = -0.447$, 95% CI = − 0.901 to .006, $p = .053$, $\Delta R^2 = 0.039$, $\Delta F_{1,74} = 3.86$). In the final step, there was no significant main effect of treatment type ($\beta = -0.723$, 95% CI = − 1.476 to 0.030, $p = .060$); however, the interaction between treatment type and treatment response was a significant predictor ($\beta = 1.061$, 95% CI 0.138−1.985, $p = .025$, $\Delta R^2 = 0.051$, $\Delta F_{1,72} = 5.25$; final model: $F_{7,72} = 4.43$, $p < .001$, $R^2 = 0.301$, adjusted $^{R2} = 0.233$). As hypothesized, lower levels of depressive symptoms were observed in treatment responders relative to nonresponders when they had received CBT (simple effect of responder status: $\beta = -0.807$, 95% CI = − 0.1.352 to −0.263, $p = .004$) but not CCT ($\beta = 0.254$, 95% CI = −0.502 to 1.010, $p = .505$) (Figure 2). It should be noted that CBT nonresponders did not differ from CBT responders in level of pretreatment anxiety symptoms at baseline; therefore this finding is not a reflection of differences in severity of psychopathology at study entry. Supplementary analyses revealed that parent-report of depressive symptoms 2 years later was lower for treatment responders relative to nonresponders regardless of treatment type (see Supplement 2, available online).

Exploratory Analyses: Moderators of the Effects of Treatment Response on 2-Year Depressive Symptoms in Full Sample and CBT Subsample

After controlling covariates in Step 1 (as above), there was a trend toward a main effect for age ($\beta = 0.195$, 95% CI = − 0.008 to 0.398], $p = .059$), with older participants reporting higher depressive symptoms at follow-up. However, there were no main effects for pubertal status at the time of treatment ($p > .359$) or interacting effects of acute response to treatment by age ($p > .552$) or pubertal status ($p > .223$) on 2-year depression scores, in either the full sample or the CBT subsample. In the CBT sample, there was a gender-by-treatment response interaction ($\beta = -1.133$, 95% CI = − 2.148 to −0.119, $p = .029$) suggesting that female ($\beta = -1.274$, 95% CI = − 0.1.935 to −0.613, $p < .001$) but not male ($\beta = -0.141$, 95% CI = − 0.908 to 0.627, $p = .714$) CBT responders had lower levels of depressive symptoms at 2-year follow-up compared to CBT nonresponders.

Exploratory Analyses: Depressive Symptoms as a Mediator of the Effects of Treatment Response on 2-Year Depressive Symptoms in Full Sample and CBT Subsample

In the full sample with available data for the mediation analysis, mediation by posttreatment MFQ-C scores was supported, suggesting that depression symptoms immediately after treatment helped to explain the link between acute anxiety treatment response and 2-year depressive symptoms. Specifically, while covarying the effect of baseline MFQ-C in all analyses, the "a" path (treatment response predicting posttreatment MFQ-C: $b = -5.97$; $t = -2.24$; $p = .04$), "b" path (posttreatment MFQ-C predicting 2-year MFQ-C, controlling for treatment response: $b = 0.42$; $t = 2.81$; $p = .01$), and "a*b" mediation path ($b = -2.61$; $t = -1.65$; $p = .02$) were each significant. In the CBT subsample (n = 44), the mediation path was no longer significant (a*b mediation path: $b = -2.65$; $t = -10.40$; $p = .10$).

DISCUSSION

The present results indicate that anxious youth who were successfully treated with CBT for an anxiety disorder during preadolescence or early adolescence showed lower levels of depressive symptoms later in adolescence compared to anxious youth who were not successfully treated. This association between early anxiety treatment and decreased development of depressive symptoms was specific to CBT and was not found for those who responded acutely to a supportive CCT. This is the first study to demonstrate that a specific therapeutic approach (ie, CBT) may disrupt the commonly observed trajectory toward increasing depressive symptoms among adolescents with a history of anxiety. These data support recent suggestions that CBT interventions selectively targeting anxious and fearful youth could represent an efficacious and cost-effective approach to preventing depressive symptoms during this sensitive period for depression onset.[10,18,21] Although we focused only on depressive symptoms, these findings are important because higher levels of depressive symptoms place youth at heightened risk for subsequent clinical diagnoses of depression later in adolescence and adulthood.[34]

Research is needed to determine the specific mechanisms through which CBT for anxiety may reduce risk for later depressive symptoms. We used a CBT protocol that combines instruction in coping skills with graduated exposure to provide anxious youth with repeated success experiences approaching fearful situations that were previously avoided. The protocol also allows youth to test their fearful expectations and to discover that feared outcomes are not catastrophes. In contrast, the supportive therapy did not

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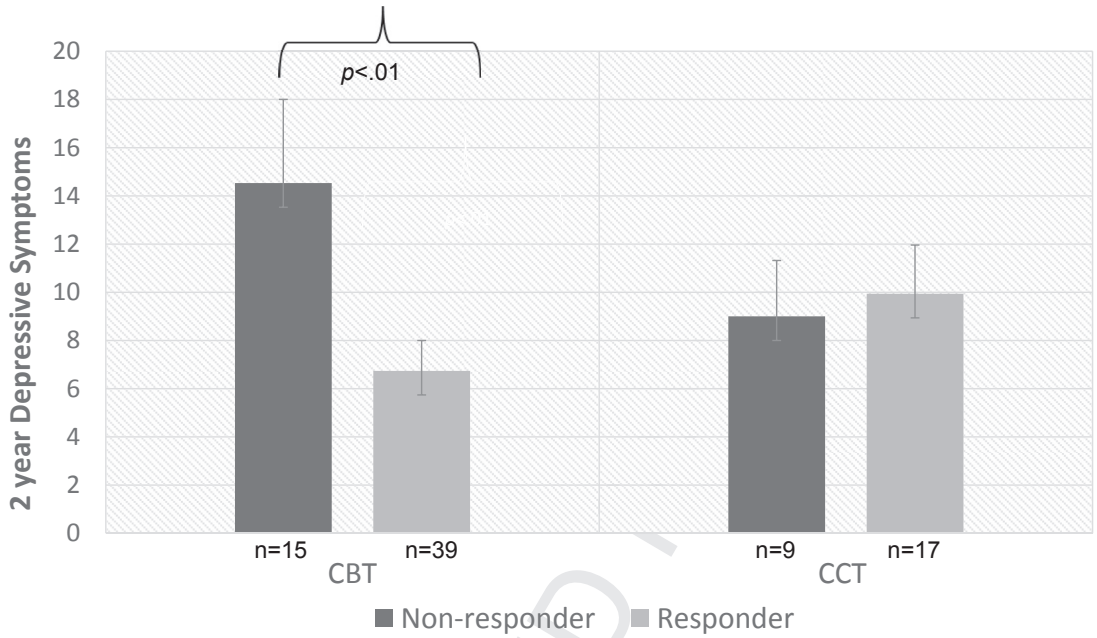
FIGURE 2 Treatment Type by Treatment Response Interaction Demonstrating Lower 2-Year Depressive Symptoms in Cognitive-Behavioral Therapy (CBT) Responders but Not Child-Centered Therapy (CCT) Responders



Note: *Error bars indicate standard error.*

include coping skills, exposure, or direct therapist encouragement to approach feared activities. Given that depression during adolescence is characterized by high levels of withdrawal and low approach, it is possible that CBT attenuates risk for depressive symptoms by reducing avoidance behaviors and/or increasing approach behavior. Alternatively, protective effects may be mediated by changes in other core characteristics shared by youth with depression and anxiety and targeted by CBT, such as levels of negative affect and associated cognitive biases.

Consistent with these possible mechanisms, exploratory analyses indicate that, within the whole sample, the association between anxiety treatment response and later depressive symptoms is at least partially accounted for by reductions in depressive symptoms that occur during the course of anxiety treatment, which could have downstream effects on approach and avoidance behaviors as well as other cognitive and affective processes. It is also important to note that mediation analyses did not hold within the CBT subsample, which is likely a result of limited power, and that our predictor (treatment response) and mediator (posttreatment depressive symptoms) were both measured at the same point of time. Future longitudinal research in larger samples with multiple posttreatment assessment points is needed to clarify the role of acute changes in depressive symptoms, as well as resulting changes in motivational, cognitive, and affective processes, in conveying the protective effect of anxiety treatment on subsequent depressive symptoms.

In terms of moderators, we did not find that treatment delivered earlier in adolescence/puberty was more or less advantageous for preventing future depressive symptoms, although longer-term follow-up in larger samples is needed before ruling out this possibility, as the study was underpowered to detect small effects, and the youngest participants in the study were only beginning to enter the period of highest risk for depression. We found that female, but not male, CBT responders had lower levels of depressive symptoms at 2-year follow-up compared to CBT nonresponders. It is not clear what mechanisms might account for this gender difference, as previous studies have typically not found gender differences in treatment response for anxiety or depression in youth.[49] It may be that anxiety and depression are more tightly linked in girls, perhaps because girls are more interpersonally sensitive, and there is a strong interpersonal component to both anxiety and depression in adolescence.[21,50] Although future research is needed to clarify mechanisms, the finding that CBT for anxiety may be especially helpful in preventing depressive symptoms for girls is important because girls are at dramatically higher risk for experiencing depression during adolescence.[51,52]

One limitation of the present investigation is the absence of an untreated sample of anxious youth, which limits our ability to infer a causal effect. However, this

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limitation is attenuated by our inclusion of an active comparison treatment to control for nonspecific aspects of intervention, such as support and attention. A related limitation inherent in the naturalistic follow-up design is that we did not preclude participants from seeking additional services during the follow-up period; thus it is possible that additional intervention services provided after the CBT treatment could have contributed to reduced depressive symptoms. However, we found that participants who sought follow-up services in the community had higher, not lower, rates of depressive symptoms at follow-up. A longer-term follow-up will be informative to determine whether benefits of early anxiety treatment on depressive symptoms persist through adolescence. In addition, the present study focused on increases in symptoms of depression, and the sample had low rates of depressive diagnoses. Future follow-up is needed to determine whether the identified benefits of early CBT for anxiety translate into lower rates of clinical diagnosis. Another limitation is that, as a result of the 2:1 randomization for CBT to CCT used in the original trial, power was much lower to detect effects within the CCT group. In particular, the cell size for CCT nonresponders was small (n = 9). For this reason, findings on the unique benefits of CBT relative to comparison need to be interpreted with caution until they can be replicated in a larger sample.

In conclusion, we found that treating anxiety with CBT during preadolescence or early adolescence appears to confer a preventive effect on adolescent depressive symptomatology, especially for girls. This finding is important because adolescent depressive symptoms are associated with serious disruptions in emotional, social, and occupational functioning into adulthood, as well as high rates of recurrence, morbidity, medical comorbidity, and lifetime disability. The present results highlight the need for screening for anxiety early in adolescence and for better availability of CBT programs targeting anxiety during this developmental stage. It would be wise for future research to examine whether briefer[53] or more dissemination-ready (eg, technology-based) interventions[54,55] might be effective in attenuating risk for depression.

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