MENTAL HEALTH OUTCOMES IN YOUNG ADULTS 16 YEARS AFTER RECEIVING TREATMENT FOR CHILD ANXIETY

A Dissertation

Submitted

to the Temple University Graduate Board

In Partial Fulfillment

of the Requirements for the Degree of

DOCTOR OF PHILOSOPHY

By

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August, 2012

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ABSTRACT

Childhood anxiety disorders are often considered gateway disorders: having an anxiety disorder in youth is associated with a higher likelihood of developing a related psychological disorder in adulthood. Successfully treating youth with anxiety disorders may reduce the likelihood of subsequent anxiety, depressive, and substance use disorders later in life. This study evaluates follow-up outcomes associated with treatment for childhood anxiety by comparing successfully and unsuccessfully treated participants 16 years after the completion of treatment. A sample of 66 youth (ages 7 to 14 at time of initial study treatment, ages 18 to 32 at present follow-up) who had been diagnosed with an anxiety disorder and randomized to treatment in a randomized clinical trial on average 16.24 (SD = 3.56) years prior participated in the present follow-up evaluation that included self-report measures and a diagnostic interview conducted to assess anxiety, depression, and substance misuse. Results indicate that, relative to those who respond successfully to CBT intervention for an anxiety disorder in childhood, those who were less responsive to CBT for childhood anxiety had higher rates of panic disorder, alcohol dependence, and drug abuse in adulthood. The present study is the first to assess the 16-year follow-up effects of CBT treatment for an anxiety disorder in youth on anxiety, depression, and substance abuse through the period of young adulthood when these disorders are often seen.

ACKNOWLEDGEMENTS

I would like to thank the following individuals who contributed to making this project possible: Brenda Benjamin, Christopher Benjamin, Emily Benjamin, Nicholas Wolk, Philip Kendall, Lauren Alloy, Michael McCloskey, Bradley Conner, Julie Harrison, Cara Settipani, Douglas Brodman, Molly Brozell, and the National Institutes of Health (MH086954).

This study is dedicated to my mother.

TABLE OF CONTENTS

PAGE

LIST OF TABLES

Table Page

CHAPTER 1

MANUSCRIPT IN JOURNAL ARTICLE FORM

Mental Health Outcomes in Young Adults 16 Years after Receiving Treatment for Child Anxiety

Separation Anxiety Disorder (SAD), Generalized Anxiety Disorder (GAD), and Social Phobia (SP) are typically treated similarly and researched collectively in children and adolescents (e.g., Kendall et al., 1997; Walkup et al., 2008). These disorders are highly comorbid with one another (Beidel, Turner, & Morris, 1999; Essau, Conradt, & Petermann, 1999; Kendall et al, 2010; Wittchen, Stein, & Kessler, 1999) and have been conceptualized as sharing an underlying anxiety construct (e.g., Bell-Dolan & Brazeal, 1993; Pine & Grun, 1998). Anxiety disorders are common in youth (e.g., Bernstein & Borchardt, 1991) and in adults, both in 12-month prevalence estimates from the general population (Kessler, Chui, Demler, & Walters, 2005) and across the lifespan (28.8%; Kessler, Berglund, Demler, Jin, & Walters, 2005). Research indicates that approximately 10 to 20% of children in the general population and primary care settings report clinically significant levels of anxiety (Chavira, Stein, Bailey, & Stein, 2004; Costello, Mustillo, Keeler & Angold, 2004).

Anxiety disorders have a negative effect on functioning. The behavior of anxious children is characterized by avoidance and this avoidance can result in significant distress for the children themselves as well as caregivers. Anxious children commonly avoid age-appropriate situations and social interactions necessary for healthy development and are rated as less well liked by peers (Verduin & Kendall, 2008). Difficulties in social relations are common (e.g., Greco & Morris, 2005; Hartup, 1983; Strauss, Forehand, Smith, & Frame, 1986), as are impaired academic achievement (e.g., King & Ollendick, 1989; Van Amerigen, Mancini, & Farvolden, 2003), and future emotional well-being

(e.g., Beidel, 1991; Feehan, McGee, & Williams, 1993). Research suggests that most anxiety disorders do not abate over time (e.g., Keller et al., 1992; Pine, Cohen, Gurley, Brook, & Ma, 1998). Retrospective reports demonstrate that adults with anxiety disorders report having experienced significant anxiety as children (e.g., Ost, 1987; Sheehan, Sheehan & Minichiello, 1981). Anxiety disorders in childhood are associated with later depression (Angold, Costello, & Erkanli, 1999; Strauss, Lease, Last, & Francis, 1988), suicidal attempts and ideation (Brent et al., 1986; Rudd, Joiner, & Rumzek, 2004), and substance use (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Lopez, Turner, & Saavadra, 2005). Evidence also suggests anxiety disorders in youth may temporally precede the emergence of depressive disorders (e.g., Alpert, Maddocks, Rosenbaum, & Fava, 1994; Biederman, Faraone, Mick, & Lelon, 1995).

Anxiety disorders in youth can have important long-term implications. For instance, as previously noted, adults with anxiety disorders often report having suffered from anxiety as youth (e.g., Last, Hersen, Kazdin, Francis, & Grubb, 1987; Weissman, Leckman, Merikangas, Gammon, & Prosoff, 1974). Higher levels of anxiety and depression are reported in older youth than in younger children suggesting anxiety symptoms, when untreated, worsen over time. As age increases, the presence of an anxiety disorder also has an associated increase in the likelihood of a depressive disorder (Beesdo et al., 2007, Brady & Kendall, 1992; Pine et al., 1998), and childhood anxiety disorders typically precede the onset of substance use disorders (Merikangas et al., 1998). Given the high lifetime co-occurrence of anxiety and substance use disorders (35% to 45%; Kessler et al., 1996) and the deleterious outcomes associated with substance dependence (Toumbourou et al., 2007), this primacy warrants further investigation.

Indeed, there is evidence that effective short-term treatment for adolescent depression is associated with reduced substance use disorders (Curry et al., 2012). Initial data suggested that CBT for child anxiety may allay later substance use (e.g., Kendall, Safford, Flannery-Schroeder, & Webb, 2004) but that evaluation was undertaken before the participants reached the age of greatest risk for the associated disorders.

Several studies have examined the efficacy of CBT for childhood anxiety disorders and reviews of the literature support its utility (Kazdin & Weisz, 1998; Ollendick, King, & Chorpita 2006). CBT has been found efficacious for specific problems and diagnosed disorders (e.g., Beidel et al., 2007; Walkup et al., 2008). Literature reviews (e.g., Kazdin & Weisz, 1998; Ollendick & King, 1998) have offered conclusions that, judged against the criteria proposed for the determination of an efficacious treatment (Chambless & Hollon, 1998), CBT can be considered an efficacious treatment.

Several RCTs have demonstrated the efficacy of one version of CBT for anxiety disordered youth, the *Coping Cat* program. A preliminary RCT included 47 children with an anxiety disorder. Results demonstrated that 64% of the treated youth no longer presented with their pretreatment principal anxiety disorder at posttreatment and gains were maintained at one-year (Kendall, 1994) and 3.35-year follow-up (Kendall & Southam-Gerow, 1996). A second RCT with 118 anxiety-disordered youth found that 55% of treated cases no longer met diagnostic criteria for their principal anxiety disorders at posttreatment (Kendall et al., 1997). Eighty-six (91.5%) of the original 94 treated participants were followed-up 7.4 years after treatment. The majority of the successfully treated children maintained gains at 7.4-year follow-up and those who were successfully

treated showed reduced substance use compared to youth who were less successfully treated (Kendall et al., 2004; Puleo, Conner, Benjamin, & Kendall, 2011). Although this 7.4 year follow-up provided information regarding the sequelae of childhood anxiety in mid-late adolescence, it is likely that most of the participants in that study were assessed prior to the age at which they would be at the highest risk for substance misuse and depressive symptoms. A third RCT examined the efficacy of CBT for GAD, SP, and/or SAD (Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008) with 161 children (aged 7-14 years) referred from multiple community sources. Of the randomized cases, 55 were assigned to Individual CBT (ICBT), 56 to Family CBT (FCBT), and 50 to a family education/support/attention (FESA) control condition. Following treatment, 57%, 55%, and 37% no longer met criteria for their primary diagnosis in the ICBT, FCBT, and FESA conditions, respectively. Significantly more children in the two CBT conditions no longer met diagnostic criteria compared to the FESA condition. The relative and combined efficacy of CBT and sertraline was recently examined in a large, multisite RCT (i.e., the Child/Adolescent Anxiety Multimodal Study [CAMS]; Walkup et al., 2008) with 488 children who met diagnostic criteria for SAD, GAD, and/or SP and results suggest that the combined treatment, and each of the monotherapies, can be successful in reducing distressing anxiety in youth. Overall, these studies provide positive evaluations of the efficacy of the *Coping Cat* program for anxiety-disordered youth, and similar efficacy has been demonstrated for other operationalizations of CBT for child anxiety (e.g., Silverman, Pina, & Viswesvaran, 2008).

Multiple research groups have made advances in the treatment of anxiety disorders in children (see also Barrett, Dadds, & Rapee, 1996; Beidel et al., 2007;

Silverman et al., 2008). Despite these advances, limited evidence exists with respect to sequelae at the time in adulthood when the disorders are likely to emerge. Several one-year follow-ups have been conducted (e.g., Kazdin, Siegel, & Bass, 1992; Kendall et al., 1997; Kolko, Loar, & Sturnick, 1990) and one 6-year follow-up was reported (Barrett, Duffy, Dadds, & Rapee, 2001). Longer follow-ups are uncommon and those that are available lack appropriate methodological rigor (e.g., do not follow a RCT). One of the longest follow-up studies completed with anxious youth (7.4-year follow-up; Kendall et al., 2004), was described earlier. A review by Nevo and Manassis (2009) highlighted the dearth of long-term outcome studies of anxious youth and the lack of evidence for the role early anxiety treatment might play in healthy development into adulthood (see also Hirshfeld-Becker, Micco, Simoes, & Henin, 2008).

The present study evaluated outcomes associated with treatment for childhood anxiety by comparing (a) successfully treated and (b) unsuccessfully treated participants 16 years after the completion of treatment. The primary aim examined whether participants treated successfully during childhood differ from those for whom treatment was not as successful on symptoms and diagnoses of (a) adult anxiety, (b) depression, and (c) substance use disorders in young adulthood. A second aim was whether the two treatment outcome groups differed from the general population on symptoms or diagnoses of anxiety, depression, and substance use disorders in early adulthood. It was hypothesized that effective treatment for an anxiety disorder in childhood would reduce the occurrences of anxiety and related disorders in young adulthood. Further, it was hypothesized that successfully treated participants would be no more likely to meet criteria for an anxiety, mood, or substance use disorder diagnosis than individuals in the

community. Unsuccessfully treated participants were hypothesized to be more likely to meet criteria for an anxiety, mood, or substance use disorder diagnosis than individuals in the community.

Method

All participants (now adults) received treatment at the Child and Adolescent Anxiety Disorders Clinic (CAADC), Temple University. One sample came from an RCT reported in 1997 (Kendall et al) and others came from an RCT reported in 2008 (Kendall et al). Both RCTs had participants randomized to treatment condition and to therapist, and were conducted at the same location.

Participants

Participants from Kendall et al. (1997). The participants were children, aged 9-13 years at the time of intake, referred from multiple community sources, and diagnosed with a DSM principal anxiety disorder. All received CBT, although the study design required that 34 of the children be assigned to a wait-list. At the end of the wait-list duration (8 weeks), if the cases continued to meet criteria for an anxiety disorder, children who had served on the waiting list were randomized to a therapist and given CBT.

For the present study, we attempted to contact all participants who were in the intent-to-treat sample (118). No additional exclusion criteria were used. Note, however, that the original 118 children were selected using the following criteria: (a) children had to have a DSM anxiety disorder (GAD, SP, SAD) as a principal diagnosis; (b) children whose principal diagnosis was simple phobia (or phobias) were not included, but children with diagnosable specific phobias as secondary

problems were included; (c) children were excluded if they displayed psychotic symptoms; or (d) if they were using anti-anxiety or anti-depressant medications at the time of intake.

Demographics. The following provides a detailed description of the participants who completed treatment via the Kendall et al. (1997) study and whom we attempted to contact for follow-up. Thirty-eight percent of the original treated sample were female and 85% were Caucasian. Fifty-two percent were 9-10 years old at the time of treatment, and 48% were 11-13 years old. Family income was below $20,000 for 6.4%, $40,000 for 27.7%, $60,000 for 33%, $80,000 for 23.5% and above $80,000 for 6.4% of the sample. Participants received principal anxiety disorder diagnoses (GAD/OAD, n = 55; SAD, n = 22; SP/AD, n = 17) at the time of intake to the CAADC on the basis of structured clinical interviews conducted separately with both the parents and child. When parent and child differed, diagnoses were based on parental reports. Forty-eight percent of the 94 children were comorbid with simple phobia at the time of admission to the CAADC; 14% with ADHD; 8% with oppositional defiant disorder; 6% with major depression; and 1% with conduct disorder.

Treatment outcome was defined in the reported studies in two ways. Type 1 responders (a more conservative outcome measure) were participants whose principal disorder at pretreatment was no longer present at posttreatment. Fifty-five percent of participants were Type 1 responders. Of those 55%, approximately 12% still met criteria for an anxiety disorder. In other words, 11 participants continued to have their secondary or tertiary anxiety disorder at posttreatment, but were still considered treatment successes. Type 2 responders were participants whose principal disorder at pretreatment

was no longer principal at posttreatment. Seventy-eight percent of participants were Type 2 responders. Of those 78% (22 individuals), approximately 37% continued to meet criteria for another anxiety disorder at posttreatment.

We also attempted to contact participants who withdrew from treatment post-randomization; 54% of this portion of the sample was female and 67% were Caucasian. Fifty-six percent were 9-10 years old, and 44% were 11-13 years old. Family income was below $20,000 for 19.2%, $40,000 for 38.5%, $60,000 for 23.1%, $80,000 for 3.8% and above $80,000 for 15.4%. These participants had a principal anxiety disorder (GAD/OAD, n = 12; SP/AD, n = 5; SAD, n = 11) based on structured interviews conducted separately with both the parent(s) and child. Sixty-one percent of the 28 children were comorbid with simple phobia at the time of admission to the CAADC; 15.3% with ADHD; 3.6% with major depression; and 11.7% with oppositional defiant disorder.

Participants from Kendall et al. (2008). Additional participants were recruited from former patients who were randomized to the same CBT (individual CBT; Kendall et al, 2008) as provided in the other RCT. The participants were children, aged 7-14 years at the time of intake, also referred from multiple community sources, and diagnosed with a DSM principal anxiety disorder. A total of 55 of the 161 children were randomized to individual CBT, including 5 subjects who withdrew prior to completing all study time points (others were randomized to either Family CBT [FCBT] or a family education/support/attention [FESA] control condition). Regarding the initial RCT outcomes, chi-square analyses indicated that 64%, 64%, and 42% of youth no longer retained their pretreatment principal diagnosis as principal at posttreatment for the ICBT,

FCBT, and FESA conditions, respectively. Principal diagnoses were no longer present for 57%, 55%, and 37% of youth in the ICBT, FCBT, and FESA conditions, respectively, at posttreatment. When outcomes were examined by condition for both definitions of treatment response, the differences between ICBT and FESA were significant, as were differences between FCBT and FESA. Primary outcomes were not statistically different when comparing ICBT to FCBT. Treatment gains were maintained at one year follow-up. For the present study, all participants who were randomized into the ICBT condition of the RCT and at the time of the present follow-up were ≥ 18 years of age were sought to be included. Despite the general lack of differential outcomes between ICBT and FCBT, only those who were randomized into ICBT were included in the current follow-up to maintain continuity of treatment with that received in Kendall et al. (1997).

For the present study, we attempted to contact all participants who were (a) in the intent-to-treat sample, (b) randomized to ICBT, and (c) at least 18 years of age at the time of the present follow-up resulting in a possible 32 participants. Attempts to contact participants continued until the desired N of 66 was achieved (based on pre-study power analyses indicating a sample size of 66 needed to test the hypotheses). No additional exclusion criteria were used. Note, however, that the original sample was selected using the following criteria: (a) children had to have a DSM anxiety disorder (GAD, SP, SAD) as a principal diagnosis; (b) children whose principal diagnosis was simple phobia (or phobias) were not included, but children with diagnosable specific phobias as secondary problems were included; (c) children were required to have at least one English speaking parent; (d) children were excluded if they displayed psychotic symptoms; (e) had a disabling medication condition; (f) mental retardation; (g) if they were using anti-anxiety

or anti-depressant medications at the time of intake; or (h) were participating in concurrent treatment. Participants who withdrew from the treatment study post-randomization to ICBT were eligible to participate and considered as non-responders in the present analyses.

Demographics. The following provides a detailed description of the participants who completed treatment via the Kendall et al. (2008) study. Forty-four percent of the original treated sample were female and 85% were Caucasian. Sixty-three percent were 7-10 years old at the time of treatment, and 37% were 11-14 years old, 9% were African American, 3% were Hispanic, and 3% were self-identified as mixed race or "other." Family income was reported as below $20,000 (3%), $40,000 (13%), $60,000 (22%), $80,000 (26%) and above $80,000 (36%). Parental education was reported as follows: fathers and mothers did not complete high school (4% and 1%, respectively), were high school graduates without college (30% and 26%), had some college education (19% and 24%), completed a 4-year college education (23% and 31%), obtained graduate school training (23% and 16%), or self-identified their education as "other" (0% and 1%). Eighty-eight children were diagnosed with a *principal* diagnosis of GAD, 63 with SP, and 47 with SAD based on structured interviews. Composite diagnoses were computed using the "or" rule (child or parent reported the diagnosis and the clinician subsequently assigned a clinician severity rating (CSR) of 4 or greater. Twenty-four percent of the children were comorbid with GAD, 37% with SP, 32% with SAD, 53% with specific phobia, 32% with attention-deficit/hyperactivity disorder (ADHD), 14% with oppositional defiant disorder (ODD), 6% with Dysthymia and 5% with major depressive

disorder (MDD). Other comorbid diagnoses were low (e.g., 1 child met for conduct disorder).

Participant Demographics for the Present Follow-Up Study. Participants for the present study were a mean age of 27.23 (SD=3.54) and completed treatment for anxiety in childhood a mean 16.24 (SD=3.56) years prior to their participation in the present follow-up study. Participants were 51.5% female and predominantly Caucasian (84.8%). The majority was employed (69.7%) and household incomes were variable. Over half of participants had never been married (56.1%). Pretreatment primary anxiety diagnoses were as follows: 56.1% had a primary diagnosis of GAD/OAD, 27.3% SP/AV, and 16.7% SAD. At posttreatment, 71.2% had their primary diagnosis as no longer primary in their diagnostic profile and 60.6% no longer met diagnostic criteria for their primary pretreatment diagnosis. See Table 1.

Of the 150 individuals who were eligible to participate in the present study, 66 were located and participated, 16 were located and declined participation, another 2 initially were reached and agreed to participate and then became unreachable prior to completion of study participation, and 36 could not be located despite all efforts (i.e., all leads were exhausted). For the remaining 30 individuals, possible contact information was identified however the individuals and/or their parents were never reached directly despite multiple attempts.

Assessment Instruments

The World Mental Health Composite International Diagnostic Interview (CIDI), the interview used in the NCS-R (Kessler et al., 2005), was chosen because it allowed us to make comparisons between the sample of previously treated anxious youth and the

community sample assessed in the NCS-R (Kessler et al., 2005). Self-report measures were also gathered for comparisons between the successfully treated and unsuccessfully treated participants.

Structured Diagnostic Interview.

World Mental Health (WMH) Survey Initiative Version of the World Health Organization (WHO) Composite International Diagnostic Interview (CIDI; Robins, Wing, Wittchen, Helzer, Babor, Burke, et al.,1988; Kessler & Ustun, 2004). The CIDI is a fully structured comprehensive lifetime interview designed to obtain information on mood disorders, anxiety disorders, substance use disorders, childhood disorders and other relevant disorders (e.g., eating disorders). Diagnoses are based on and consistent with DSM-IV (APA, 1994) and ICD-10 (WHO, 1992). This interview assesses demographic variables (e.g., marital and occupational status, income) and subsequent treatment. CIDI diagnoses are significantly related to independent clinical diagnoses, and test-retest reliability is high (Kessler & Ustun, 2004).

Participant's Self-Report Measures.

Drug Use Screening Inventory-Revised (DUSI-R; Tarter, 1990). The DUSI-R is a self-report 159-item questionnaire that assesses severity of problems and consequences over the past week and past year in 10 domains for adolescents and adults: Substance abuse (including involvement with a variety of drugs), psychiatric disorders, behavior problems, school adjustment, health status, work adjustment, peer relations, social competency, family adjustment, and leisure/recreation. It also contains a 10-item lie scale to estimate validity of responses. Two profiles are generated, one being a profile indexing absolute problem density of disorder (0-100%) for each of the 10 domains whereas the

other profile is a relative problem index ranking problem severity in the 10 domains. The summary problem index represents the overall severity of problems. The DUSI-R is internally consistent (internal reliability coefficients: .74 for males and .78 for females; split half reliability correlations: .70 for males and .67 for females) across the 10 domains. It is also temporally stable, with the mean retest (one week) coefficients being .95 for males and .88 for females. Concurrent validity for the substance use (.72) and psychiatric disorder (.65) domains of the DUSI-R were established in relation to the K-SADS (as cited in Kirisci, Mezzich, & Tarter, 1995).

Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988). The BAI is a 21-item self-report questionnaire that assesses the frequency and severity of anxiety symptomatology over one week. Each item is rated on a 4-point scale: 0 (*not at all*) to 3 (*severely, I could barely stand it*). The BAI is comprised of two main dimensions of anxiety: cognitive and physiological (or somatic) symptoms. The BAI has been shown to have good internal consistency, retest reliability, and convergent and discriminant validity (Beck et al., 1988; Beck & Steer, 1991; Hewitt & Norton, 1993). The BAI has demonstrated distinct items loading onto anxiety, separate from depression, among a variety of samples, including undergraduate student and psychiatric populations (Creamer, Foran, & Bell, 1995; Osman, Kopper, Barrios, Osman, & Wade, 1997).

Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996). The BDI-II is a 21-item measure of depressive symptoms experienced during the past two weeks. Each item has four statements that reflect varying degrees of symptom severity. Participants are instructed to indicate the number of the statement (ranging from 0 to 3, with increasing severity) that best corresponds with their symptom severity. A total BDI-II

score is calculated by taking a sum of all items and can range from 0 to 63. Similar to the BAI, the BDI-II loads onto two dimensions of depression: cognitive and somatic symptoms. The BDI-II has demonstrated excellent internal consistency, factorial validity, criterion validity, convergent and discriminant validity, and internal reliability in numerous samples with varying ethnicity and psychopathology (Arnau, Meagher, Norris, & Bramson, 2001; Carmody, 2005; Contreras, Fernandez, Malcarne, Ingram, & Vaccarino, 2004; Dozois, Dobson, & Ahnberg, 1998; Grothe et al., 2005; Storch, Roberti, & Roth, 2004; Weeks & Heimberg, 2005).

Sheehan Disability Scale (SDS; Sheehan, 1983). The SDS is one of the most frequently used self-report disability measures. It consists of four items measuring current impaired functioning across three domains in individuals with anxiety disorders: Social Life/Leisure Activities, Work, Family Life/Home Responsibilities, and Work and Social Disability change of symptoms. Each item is rated on a scale from 0 (*Not at all*) to 10 (*Very severely – I never do these*) of increasing impairment due to the person's problems, except for the fourth item which is rated on a scale from 1 (*No complaints, normal activity*) to 5 (*Symptoms radically change/prevent normal work or social activities*). The SDS has been found to have adequate to high internal consistency, satisfactory construct validity, and criterion-related validity as demonstrated in multiple psychiatric populations and primary care patients (Hambrick, Turk, Heimberg, Schneier, & Liebowitz, 2004; Leon, Olfson, Portera, Farber, & Sheehan, 1997; Leon, Shear, Portera, & Klerman, 1992; Placchi, 1997; Rubin et al., 2000).

Quality of Life Inventory (QOLI; Frisch, Cornell, Villanueva, & Retzlaff, 1992). The QOLI is a brief, 32-item, self-report questionnaire that assesses life

satisfaction across 16 domains (e.g., love, work, health, friends, family, self-esteem) and is weighted by the relative importance of each domain to the individual (Frisch, 2004). The QOLI was found to be sensitive to treatment-related change in two clinical samples and demonstrated predictive validity for academic retention in clinical samples (Frisch et al., 2005). Additionally, the QOLI demonstrated moderate internal consistency and retest reliability among 3 clinical and 3 nonclinical samples (Frisch et al., 1992). The QOLI was also found to have adequate item-total correlations, strong convergent and discriminant validity, and high criterion validity between clinical and nonclinical samples (Frisch et al., 1992).

Clinician Rated Measure.

Participant Additional Treatment (PAT). A rating scale was developed to quantify additional treatment obtained since initial study participation. After reviewing services reported on the CIDI interview for each participant, one of two experienced diagnosticians at the CAADC independently rated the degree to which the participant "received additional services" on a 0 to 4 likert scale. The principal investigator served as the expert judge of additional treatment ratings and the other raters were required to match her ratings on three sample interviews prior to rating study participants at a kappa level of .80 or above. Raters were also be subject to a random check during the rating period to ensure that the reliability ratings of additional treatment remained above .80. This level of reliability was maintained.

Study Procedures

Participants were attempted to be reached using the detailed contact information provided at the start of the initial RCTs and/or updated at previous follow-up. During

initial participation, the child provided written assent and their parent(s) provided written consent prior to participating in the diagnostic interviews and completing written questionnaires. Participants who were able to be reached directly were asked to participate following a description of the project. If a participant was currently living separately from his or her family and the contact sheet provided information about the parent's residence, we described the project to the parent. We then asked the parent to contact their child to ask permission for the parent to provide us with the child's direct contact information. We also attempted to arrange a time to call the parent back to inquire if the child consented for their contact information to be released.

As some participants had moved out of the immediate region or had schedules that did not readily permit travel for an in-person interview, those who expressed difficulty coming to the research lab were invited to participate via phone and internet. When possible, local participants were invited to participate in one 3-4 hour evaluation. Nine participants participated in person, while 57 completed phone interviews. Prior to any participation, participants were asked to sign informed consent. Compensation in the form of a gift card was provided to participants for their time. All interviewers completed a group didactic training session and reviewed study materials on their own.

Results

Preliminary analyses compared long-term follow-up study participants with nonparticipants (those unable to be contacted or unwilling to participate in the present study) to examine differences on initial variables such as referral source, demographics (e.g., age, gender, ethnicity, family income), therapist factors (e.g., experience), pre- and

post-treatment dependent measures (i.e., questionnaire measures and diagnostic status), and ratings of the child-therapist relationship.

Participants and nonparticipants from the Kendall et al., 1997 sample were compared. Participation was not significantly associated with referral source χ^2 (2) = 3.77, p = .15, gender χ^2 (1) = 3.73, p = .054, race χ^2 (4) = 4.43, p = .35, or family income category χ^2 (8) = 15.18, p = .056. One-way analysis of variance comparing participants versus nonparticipants on years of therapist experience was also non-significant F (1, 93) = 1.06, p = .31. A significant difference between participants and nonparticipants on child age was observed F (1, 95) = 4.08, $p < .05$ such that children who were younger at time of initial treatment participated in the present follow up at higher rates than older children. Chi-square analyses comparing participants versus nonparticipants by pretreatment primary diagnosis were non-significant for both parent and child reported diagnoses. One-way analyses of variance comparing participants versus nonparticipants on mean pretreatment Multidimensional Anxiety Scale for Children (MASC) score were also non-significant for both parent and child reported MASC total scores.

Additional analyses compared participants from Kendall et al., 1997 with participants from Kendall et al., 2008. Participation was not significantly associated with gender χ^2 (1) = .57, p = .45 or race χ^2 (4) = 1.80, p = .77. A significant difference between participants and nonparticipants on child age was observed F (1, 64) = 249.34, $p < .01$ such that participants from the Kendall et al., 1997 were significantly older than participants from Kendall et al., 2008. Chi-square analyses comparing participants from Kendall et al., 1997 with participants from Kendall et al., 2008 on pretreatment primary

diagnosis indicated a significant differences between samples χ^2 (2) = 8.18, $p < .05$ (see Table 1).

Additionally, participants who completed phone interviews were compared with those who participated in person on several key variables. Mode of participation was not significantly associated with gender χ^2 (1) = 1.38, $p = .24$ or race χ^2 (4) = 5.55, $p = .24$. Child age was also not significantly associated with mode of participation F (1, 64) = .66, $p = .42$. Finally, chi-square analysis comparing mode of participation on pretreatment primary diagnosis were also non-significant χ^2 (2) = 5.04, $p = .08$.

Results from the diagnostic interview administered during the present follow-up study indicate that 43.9% of individuals met DSM-IV diagnostic criteria for one or more anxiety disorder with onset of age 18 or older, or with onset in childhood and duration persisting into adulthood. Anxiety disorders included are as follows: GAD, SP, SAD, Panic Disorder, Agoraphobia, Obsessive-Compulsive Disorder, and/or Specific Phobia. Additionally, 27.3% met criteria for one or more depressive disorder, including Major Depressive Disorder, a Major Depressive Episode, and/or Dysthymic Disorder. Finally, 42.4% met criteria for one or more substance use disorder including Alcohol Abuse, Alcohol Dependence, Drug Abuse, Drug Dependence, and/or Nicotine Dependence. See Table 2. Rates are also reported by type of treatment response in Table 3. Treatment response is defined in two ways: (a) the absence of the principal anxiety disorder at posttreatment and (b) the principal anxiety disorder no longer principal at posttreatment.

Participants completed ratings of current mood, anxiety, and substance use symptomology as well as quality of life and overall disability/impairment. Overall mean ratings for each measure were within normal (i.e., nonclinical) limits and as follows:

BDI-II mean total score = 10.62 (SD = 11.63), BAI mean total score = 9.62 (SD = 9.62), SDS Overall Disability Score mean = 20.3 (SD = 1.12), QOLI Total Score mean = 24.72 (SD = 24.89), and DUSI-R Overall Problem Density Index mean = 20.58 (SD = 13.99).

Primary Analyses: Effects of Treatment on Sequelae of Anxiety

To test the effect of previous treatment outcome on the sequelae of anxiety, logistic regressions examined whether outcome status (successful/unsuccessful as indicated by posttreatment diagnostic status) predicts adult anxiety, depressive disorder and subsequent substance use disorders (i.e., if participants meet diagnostic criteria for any anxiety disorder(s) on the CIDI interview with an onset age ≥ 18 years or onset in childhood persisting into adulthood, they were considered to have an adult anxiety disorder).

When successful treatment is defined as the principal anxiety disorder no longer primary at posttreatment, unsuccessful treatment significantly predicted the presence of adult Panic Disorder, demonstrating an odds ratio of 6.00. Additionally, unsuccessful treatment significantly predicted the presence of adult Alcohol Dependence, demonstrating an odds ratio of 16.43. All other logistic regressions predicting anxiety disorders, depressive diagnoses, and substance use disorders were non-significant. See Table 4.

When successful treatment is defined as the principal anxiety disorder no longer present at posttreatment, unsuccessful treatment again significantly predicted the presence of adult Panic Disorder, demonstrating an odds ratio of 9.29. Additionally, unsuccessful treatment, defined as the principal anxiety disorder remaining present at posttreatment, significantly predicted the presence of adult Alcohol Dependence,

demonstrating an odds ratio of 9.29, as well as Drug Abuse, with an odds ratio of 7.00. All other logistic regressions predicting anxiety disorders, depressive diagnoses, and substance use disorders were non-significant. See Table 5.

Follow-up tests examined if the significant relationships between treatment response and adult disorders reported above hold when we include (a) primary diagnosis at time of additional treatment presentation or (b) additional treatment in the models as covariates. When successful treatment is defined as the principal anxiety disorder no longer primary at posttreatment, treatment outcome continued to significantly predict the presence of adult Panic Disorder when pretreatment primary diagnosis (B (SE) = 3.19 (1.24), odds ratio = 11.27, $p < .01$) and additional treatment (B (SE) = 1.85 (.93), odds ratio = 6.38, $p = .05$) were included in the models as covariates. Additionally, when successful treatment is defined as the principal anxiety disorder no longer primary at posttreatment, treatment outcome also continued to significantly predict the presence of adult Alcohol Dependence when pretreatment primary diagnosis (B (SE) = 2.71 (1.38), odds ratio = 14.99, $p = .05$) and additional treatment (B (SE) = 2.89 (1.16), odds ratio = 18.05, $p = .05$) were included in the models as covariates.

When successful treatment is defined as the principal anxiety disorder no longer present at posttreatment, treatment outcome continued to significantly predict the presence of adult Panic Disorder when pretreatment primary diagnosis (B (SE) = 3.18 (1.36), odds ratio = 23.93, $p = .02$) and additional treatment (B (SE) = 2.25 (1.13), odds ratio = 9.47, $p = .05$) were included in the models as covariates. In contrast, treatment outcome no longer significantly predict the presence of adult Alcohol Dependence when pretreatment primary diagnosis was included in the model (B (SE) = 2.02 (1.24), odds

ratio = 7.52, p = .10). Treatment outcome did continue to predict Alcohol Dependence when additional treatment (B (SE) = 2.25 (1.14), odds ratio = 9.47, p = .05) was included in the model as a covariate. Finally, when successful treatment is defined as the principal anxiety disorder no longer present at posttreatment, treatment outcome continued to significantly predict the presence of adult Drug Abuse when pretreatment primary diagnosis (B (SE) = 1.87 (.90), odds ratio = 6.46, p = .04) and additional treatment (B (SE) = 1.95 (.85), odds ratio = 7.05, p = .02) were included in the models as covariates. Analyses examined (a) age and (b) time since treatment relationships with outcome: No significant relationships with outcome at follow-up were observed.

One-way analyses of variance comparing responder status on mean self-report form scores were non-significant for the BDI-II, BAI, SDS, QOLI, and DUSI-R indicating that successful treatment response was not associated with ratings of adult state depression, state anxiety, disability/impairment, quality of life, or self-report ratings of substance misuse symptomology. This held for both definitions of treatment outcome status. See Table 6.

Effects of Posttreatment Diagnostic Status on Additional Treatment. Additional therapeutic services after initial study treatment were examined dichotomously (i.e., presence or absence of additional treatment) as well as continuously (Participant Additional Treatment [PAT] clinician rated measure). When additional treatment is defined as presence or absence of additional treatment, 93.9% (n = 62) of participants endorsed receiving at least some additional services. When additional services were coded by experienced diagnosticians, 4 (6.1%) individuals received no additional services, 16 (24.2%) obtained some additional services, 20 (30.3%) obtained a

moderate amount of additional services, 18 (27.3%) received a great deal of additional services, and 8 (12.1%) were rated as receiving services more often than not since initial treatment completion.

When additional services are examined dichotomously using logistic regression, neither posttreatment diagnostic status defined as the principal anxiety disorder no longer primary at posttreatment (B (SE) = .97 (1.04), Odds Ratio = 2.65, $p = .35$) nor the principal anxiety disorder no longer present at posttreatment significantly predicted additional services (B (SE) = 1.63 (1.18), Odds Ratio = 5.09, $p = .17$).

When additional services are examined continuously (PAT data) using linear regression, neither posttreatment diagnostic status defined as the principal anxiety disorder no longer primary at posttreatment ($\beta = -.03$, $p = .83$, $R^2 < .01$) nor the principal anxiety disorder no longer present at posttreatment significantly predicted additional services ($\beta = .02$, $p = .99$, $R^2 < .01$).

Secondary Analyses: Normative Comparisons

Successfully treated and unsuccessfully treated participants are compared to same-age community participants from the NCS-R (Kessler et al., 2005) on diagnostic status (disorder present or absent in past 12 months) for anxiety, mood, and substance use disorders. Logistic models are used to test these effects. Included in the model are three groups: successfully treated participants, unsuccessfully treated participants, and the comparison group (NCS-R; Kessler et al., 2005). Follow-up tests according to Dunnett's criterion are conducted comparing the treatment groups with the comparison group providing an overall effect is seen. Age is included as a stratification variable (covariate) to allow participants to be compared to same-aged peers in the NCS-R (Kessler et al.,

2005) study. See Table 7 for frequency of 12-month disorders in the present follow-up sample.

When successful treatment is defined as the principal anxiety disorder no longer primary at posttreatment, group status (successfully treated participants, unsuccessfully treated participants, and the comparison group) significantly predicted GAD and Panic Disorder. Follow-up tests indicate the unsuccessfully treated participants significantly differed from the normative comparison group (GAD $p < .05$; Panic Disorder $p < .01$) in that the unsuccessfully treated group had higher rates of GAD and Panic Disorder than the normative comparison group. Successfully treated participants did not significantly differ from the normative comparison group for either GAD or Panic Disorder. See Table 8.

Group status did not significantly predict any depressive disorders when successful treatment is defined as the principal anxiety disorder no longer primary at posttreatment. See Table 9. When successful treatment is defined as the principal anxiety disorder no longer primary at posttreatment, group status significantly predicted Alcohol Abuse, Alcohol Dependence, and Nicotine Dependence. Follow-up tests indicate the unsuccessfully treated participants significantly differed from the normative comparison group (Alcohol Abuse $p < .01$; Alcohol Dependence $p < .01$) such that the unsuccessfully treated group had higher rates of Alcohol Abuse and Alcohol Dependence than the normative comparison group. Successfully treated participants did not significantly differ from the normative comparison group for either Alcohol Abuse or Alcohol Dependence. Regarding Nicotine Dependence, unsuccessfully treated participants did not significantly differ from the normative comparison group. Successfully treated participants

significantly differed from the normative comparison group (Nicotine Dependence $p <$ .01) and the successfully treated group had higher rates of Nicotine Dependence than the normative comparison group. See Table 10.

When successful treatment is defined as the principal anxiety disorder no longer present at posttreatment, group status (successfully treated participants, unsuccessfully treated participants, and the comparison group) significantly predicted GAD, Panic Disorder, and Agoraphobia. Follow-up tests indicate the successfully treated participants significantly differed from the normative comparison group (GAD $p = .01$; Panic Disorder $p < .01$, Agoraphobia $p < .05$) in that they demonstrated higher rates of these disorders than the normative comparison group. Unsuccessfully treated participants did not significantly differ from the normative comparison group for GAD, Panic Disorder, or Agoraphobia. See Table 11.

Group status did not significantly predict any depressive disorders when successful treatment is defined as the principal anxiety disorder no longer present at posttreatment. See Table 12. When successful treatment is defined as the principal anxiety disorder no longer present at posttreatment, group status significantly predicted Alcohol Abuse, Alcohol Dependence, and Nicotine Dependence. Follow-up tests indicate the successfully treated participants significantly differed from the normative comparison group (Alcohol Abuse $p < .01$; Alcohol Dependence $p < .01$) in that successfully treated participants had higher rates of Alcohol Abuse and Alcohol Dependence than the normative comparison group. Unsuccessfully treated participants did not significantly differ from the normative comparison group for either Alcohol Abuse or Alcohol Dependence. Regarding Nicotine Dependence, successfully treated participants did not

significantly differ from the normative comparison group. Unsuccessfully treated participants significantly differed from the normative comparison group (Nicotine Dependence $p < .01$) such that they exhibited higher rates of Nicotine Dependence than the normative comparison group. See Table 13.

Discussion

The present results indicate that, relative to those who respond successfully to CBT intervention for an anxiety disorder in childhood, those who were less responsive to CBT for childhood anxiety had higher rates of panic disorder, alcohol dependence, and drug abuse in adulthood. These findings were mostly consistent when treatment response was defined in two different ways, as well as when potentially important factors such as pretreatment primary anxiety diagnosis and subsequent treatment were included in the models.

The present study is the first to examine 16-year follow-up of CBT for childhood anxiety disorders on anxiety, depression, and substance misuse in young adulthood when these disorders are likely to be evident. Knowledge about advantageous effects of an anxiety intervention on general mental health offers important insight regarding prevention of secondary disorders by treating a primary disorder (Kessler & Price, 1993). By examining treated cases from an RCT, we examined the degree to which receiving treatment can prevent the development of additional psychopathology and associated symptoms later in life.

As previously noted, having an anxiety disorder in youth has been associated with higher rates of other disorders in adulthood (e.g., Pine et al., 1998), and childhood anxiety disorders typically precede the onset of substance use disorders (Merikangas et al., 1998).

Given the high lifetime co-occurrence of anxiety and substance use disorders (35% to 45%; Kessler et al., 1996) and the deleterious outcomes associated with substance dependence (Toumbourou et al., 2007), this indication that childhood anxiety disorders may serve as a gateway disorder for later substance misuse is compelling and warrants further investigation. Treatment of acute substance dependence has yielded mixed results and 40-60% of those treated return to active use within 12 months following treatment (Finney & Moos, 1992; Hubbard, Craddock, Flynn, Anderson, & Etheridge, 1997). Opportunities for preventing disorders that, if left to develop in adolescence and adulthood, are often difficult to treat and associated with deleterious outcomes, exist. The present results suggest that successfully treating anxiety disorders early (between ages 7 and 14) may protect against the development of later associated disorders. Evidence suggests that as much as 60% of adult substance dependence may be prevented by early treatment of disorders in youth (Kendall & Kessler, 2002; Kessler et al., 2001), and CBT in particular holds promise as a tool for preventing the development of secondary substance use comorbidities in youth with anxiety. Given that CBT for child anxiety is a cost-effective and generally feasible and acceptable method of intervention, it's utility in preventing the development of substance misuse and panic disorder in adulthood warrants further investigation.

As the literature suggests that childhood anxiety disorders predict adult anxiety, depressive and substance use disorders, the present results were unexpected in that no depressive disorders were predicted by unsuccessful treatment and panic disorder was the only anxiety disorder predicted by initial study treatment outcome. This suggests that the

risk factors for untreated childhood anxiety and unsuccessfully treated child anxiety may vary).

Given that this was not a prospective study, it is difficult to know for certain whether the successful treatment itself is related to better outcomes or if treatment outcome is a proxy for other factors. For example, it may be that individuals whose parents are more involved, motivated, and/or proactive about obtaining and engaging in treatment for their children have children who are more likely to be treatment responders, and that these factors contribute to the relationship between treatment outcome and long-term diagnostic status. Other possible risk and protective factors, such as child intellect and motivation, psychosocial stressors, and duration of illness at initial treatment presentation could potentially contribute to long-term outcomes. Future studies that adopt a developmental psychopathology perspective to examine these and other important risk and protective factors, particularly within the context of a longitudinal, prospective study, are needed.

The current study also compared successfully and unsuccessfully treated participants 16 years after treatment to the general population (i.e., NCS-R sample; Kessler et al., 2005) on rates of anxiety, mood, and substance use disorders and symptoms. When successful treatment was defined more liberally as the principal anxiety disorder no longer primary at posttreatment, the unsuccessfully treated group had higher rates of GAD, Panic Disorder, Alcohol Abuse, and Alcohol Dependence than the normative comparison group and the successfully treated group had higher rates of Nicotine Dependence than the normative comparison group. However, when treatment outcome was defined more conservatively, as the principal anxiety disorder no longer

present at posttreatment, the successfully treated participants had higher rates of GAD, Panic Disorder, Agoraphobia, Alcohol Abuse, and Alcohol Dependence than the normative comparison group and unsuccessfully treated participants exhibited higher rates of Nicotine Dependence than the normative comparison group. This inconsistency was unexpected. Subsequent examination of the data indicated that small fluctuations in frequencies of disorders by definition of treatment outcome resulted in these inconsistencies. For example, when successful treatment was defined as the principal anxiety disorder no longer primary at posttreatment, 3 individuals who were successfully treated endorsed a diagnosis of Alcohol Abuse in the past 12 months compared with 4 individuals who were unsuccessfully treated. However, when treatment outcome was defined as the principal anxiety disorder no longer present at posttreatment, 5 individuals who were successfully treated endorsed a diagnosis of Alcohol Abuse in the past 12 months compared with 2 individuals who were unsuccessfully treated. This suggested that any findings between treatment outcome and the normative comparison group were spurious, and post-hoc power analyses confirmed that these analyses were overpowered to detect significant effects. A larger sample of follow-up participants relative to the normative comparison sample would be necessary to reliably examine relationships between how successfully and unsuccessfully treated participants compare to their same-aged community peers.

Limitations include that the present sample was predominantly Caucasian, middle to high SES, and college educated. Future treatment outcome studies, particularly those with longitudinal designs and long-term follow-ups, would benefit from including diverse samples. Participants who completed the CAMS trial (Walkup et al., 2008) are the largest

and most diverse sample of youth treated for child anxiety disorders to date. The currently in-progress follow-up of this sample (i.e., the Child/Adolescent Anxiety Multimodal Extended Long-term Study [CAMELS]) has the potential to address some of the limitations of the existing CBT for child anxiety long-term follow-up studies.

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Table 1

Demographic Data: Frequency Data

Variable	Overall Sample (N=66)	RCT-2 (n=54)	RCT-3 (n=12)
	n (%)	n (%)	n (%)
Sex			
Male	32 (48.5%)	25 (46.3%)	7 (58.3%)
Female	34 (51.5%)	29 (53.7%)	5 (41.7%)
Race			
Caucasian	56 (84.8%)	45 (83.3%)	11 (91.7%)
African American	5 (7.6%)	4 (7.4%)	1 (8.3%)
Asian/Pacific Islander	2 (3.0%)	2 (3.7%)	0
Other/Biracial/Multiracial	3 (4.5%)	3 (5.6%)	0
Employment Status			
Employed/Self-Employed	46 (69.7%)	40 (74.1%)	6 (50.0%)
Unemployed	14 (21.2%)	11 (20.5%)	3 (25.0%)
Other/Student	5 (7.6)	2 (3.7%)	3 (25.5%)
Income			
$0	9 (13.6%)	5 (9.3%)	4 (33.3%)
$1-24,999	26 (39.4%)	19 (35.2%)	7 (58.3%)
$25,000-49,999	19 (28.8%)	13 (24.1%)	1 (8.3%)
$50,000-74,999	8 (12.1%)	8 (14.8%)	0
$75,000-99,999	2 (3.0%)	2 (3.7%)	0
≥$100,000	2 (3.0%)	2 (3.7%)	0
Marital Status			
Married/Partnered	26 (39.2%)	25 (46.3%)	1 (8.3%)
Never Married	37 (56.1%)	26 (48.1%)	11 (91.7%)
Separated/Divorced	3 (4.5%)	3 (5.6%)	0
Pretreatment Primary Diagnosis			
GAD/OAD	37 (56.1%)	32 (59.3%)	5 (41.7%)
SP/AV	18 (27.3%)	11 (20.4%)	7 (58.3%)
SAD	11 (16.7%)	11 (20.4%)	0
Posttreatment Response			
Primary No Longer Primary	47 (71.2%)	39 (72.2%)	8 (66.7%)
Primary No Longer Present	40 (60.6%)	32 (59.3%)	8 (66.7%)

Note. RCT-2=Subjects previously randomized to treatment in Kendall et al., 1997. RCT-3= Subjects previously randomized to treatment in Kendall et al., 2008. GAD/OAD=Generalized Anxiety Disorder/Overanxious Disorder of Childhood. SP/AV=Social Phobia/Avoidant Disorder. SAD=Separation Anxiety Disorder. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment.

Table 2

Adult Diagnostic Status

Diagnosis	Overall Sample (N=66)	RCT-2 (n=54)	RCT-3 (n=12)
	n (%)	n (%)	n (%)
Any Anxiety Disorder	29 (43.9%)	24 (44.4%)	5 (41.7%)
Generalized Anxiety Disorder	11 (16.7%)	9 (16.7%)	2 (16.7%)
Social Phobia	17 (25.8%)	14 (25.9%)	3 (25.0%)
Separation Anxiety Disorder	5 (7.6%)	5 (9.3%)	0
Panic Disorder	6 (9.1%)	5 (9.3%)	1 (.3%)
Agoraphobia	4 (6.1%)	4 (7.4%)	0
Obsessive-Compulsive Disorder	5 (7.6%)	5 (9.3%)	0
Specific Phobia	12 (18.2%)	10 (18.5%)	2 (16.7%)
Any Depressive Disorder	18 (27.3%)	16 (29.6%)	2 (16.7%)
Major Depressive Disorder	17 (25.8%)	15 (27.8%)	2 (16.7%)
Major Depressive Episode	18 (27.3%)	16 (29.6%)	2 (16.7%)
Dysthymic Disorder	2 (3.0%)	2 (3.7%)	0
Any Substance Use Disorder	28 (42.4%)	23 (42.6%)	5 (41.7%)
Alcohol Abuse	16 (24.2%)	14 (25.9%)	2 (16.7%)
Alcohol Dependence	6 (9.1%)	5 (9.3%)	1 (8.3%)
Drug Abuse	9 (13.6%)	7 (13.0%)	2 (16.7%)
Drug Dependence	2 (3.0%)	2 (3.7%)	0
Nicotine Dependence	18 (27.3%)	14 (25.9%)	4 (33.3%)

Note. RCT-2=Subjects previously randomized to treatment in Kendall et al., 1997. RCT-3= Subjects previously randomized to treatment in Kendall et al., 2008. Diagnoses based on CIDI interview. Diagnoses were met if subject endorsed the presence of a lifetime diagnosis with onset at ≥ age 18 or onset in childhood with persistence into adulthood.

Table 3
Adult Diagnostic Status by Posttreatment Response

Diagnosis	Primary No Longer Primary (N=47)	Primary Still Primary (N=19)	Primary No Longer Present (N=40)	Primary Still Present (N=26)
	n (%)	n (%)	n (%)	n (%)
Any Anxiety Disorder	19 (40.4%)	10 (52.6%)	15 (37.5%)	14 (53.8%)
Generalized Anxiety Disorder	8 (17.0)	3 (15.8%)	6 (15.0%)	5 (19.2%)
Social Phobia	10 (21.3%)	7 (36.8%)	7 (17.5%)	10 (38.5%)
Separation Anxiety Disorder	3 (6.4%)	2 (10.5%)	2 (5.0%)	3 (11.5%)
Panic Disorder	2 (4.3%)	4 (21.1%)	1 (2.5%)	5 (19.2%)
Agoraphobia	3 (6.4%)	1 (5.3%)	2 (5.0%)	2 (7.7%)
Obsessive-Compulsive Disorder	3 (6.4%)	2 (10.5%)	1 (2.5%)	4 (15.4%)
Specific Phobia	8 (17.0%)	4 (21.1%)	7 (17.5%)	5 (19.2%)
Any Depressive Disorder	12 (25.5%)	6 (31.6%)	10 (25%)	8 (30.8%)
Major Depressive Disorder	11 (23.4%)	6 (31.6%)	10 (25%)	7 (26.9%)
Major Depressive Episode	12 (25.5%)	6 (31.6%)	10 (25%)	8 (30.8%)
Dysthymic Disorder	1 (2.1%)	1 (5.3%)	1 (2.5%)	1 (3.8%)
Any Substance Use Disorder	19 (40.4%)	9 (47.4%)	16 (40.0%)	12(46.2%)
Alcohol Abuse	10 (21.3%)	6 (31.6%)	8 (20.0%)	8 (30.8%)
Alcohol Dependence	1 (2.1%)	5 (26.3%)	1 (2.5%)	5 (19.2%)
Drug Abuse	5 (10.6%)	4 (21.1%)	2 (5.0%)	7 (26.9%)
Drug Dependence	0	2 (10.5%)	0	2 (7.7%)
Nicotine Dependence	14 (29.8%)	4 (21.1%)	12 (30.0%)	6 (23.1%)

Note. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary Still Primary=Pretreatment primary diagnosis remained primary diagnosis at posttreatment. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Primary Still Present=Pretreatment primary diagnosis was no longer primary but remained in diagnostic profile at posttreatment.

Table 4

Logistic Regressions Examining Primary Diagnosis No Longer Primary at Posttreatment as a Predictor of DSM-IV Disorders at 16-Year Follow-Up

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*P*
Generalized Anxiety Disorder					
Constant	1.67 (.63)				
Primary No Longer Primary	-.09 (.74)	.22	.91	3.89	.90
Social Phobia					
Constant	.54 (.48)				
Primary No Longer Primary	.77 (.59)	.67	2.16	6.92	.20
Separation Anxiety Disorder					
Constant	2.14 (.75)				
Primary No Longer Primary	.55 (.96)	.27	1.73	11.25	.57
Panic Disorder[1]					
Constant	1.32 (.56)				
Primary No Longer Primary	1.79 (.92)	1.00	6.00	36.12	.05
Agoraphobia					
Constant	2.89 (1.03)				
Primary No Longer Primary	-.21 (1.19)	.08	.82	8.36	.86
Obsessive-Compulsive Disorder					
Constant	2.14 (.75)				
Primary No Longer Primary	.55 (.96)	.27	1.73	11.25	.57
Specific Phobia					
Constant	1.32 (.56)				
Primary No Longer Primary	.26 (.68)	.34	1.30	4.96	.70
Major Depressive Disorder					
Constant	.77 (.49)				
Primary No Longer Primary	.41 (.60)	.46	1.51	4.91	.49
Major Depressive Episode					
Constant	.77 (.49)				
Primary No Longer Primary	.30 (.60)	.42	1.35	4.33	.62
Dysthymic Disorder					
Constant	2.89 (1.03)				
Primary No Longer Primary	.94 (1.44)	.15	2.56	43.08	.52
Alcohol Abuse					
Constant	.77 (.49)				
Primary No Longer Primary	.54 (.61)	.52	1.71	5.63	.38
Alcohol Dependence[2]					
Constant	1.03 (.52)				
Primary No Longer Primary	2.80 (1.14)	1.77	16.43	152.60	.01
Drug Abuse					
Constant	1.32 (.56)				
Primary No Longer Primary	.81 (.74)	.53	2.24	9.46	.27
Drug Dependence					
Constant	2.14 (.75)				
Primary No Longer Primary	19.06 (5862.75)	.00	1.90[8]	-	1.00
Nicotine Dependence					
Constant	1.32 (.56)				
Primary No Longer Primary	-.46 (.65)	.18	.63	2.23	.47

[1] R^2=.06 (Cox & Snell), .13 (Nagelkerke). Model $\chi^2(1)=4.11$, $p < .05$

[2] R^2=.12 (Cox & Snell), .27 (Nagelkerke). Model $\chi^2(1)=8.63$, $p < .01$

Table 5

Logistic Regressions Examining Primary Diagnosis No Longer Present at Posttreatment as a Predictor of DSM-IV Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Generalized Anxiety Disorder					
Constant	1.44 (.50)				
Primary No Longer Primary	.30 (.67)	.37	1.35	4.98	.65
Social Phobia					
Constant	.47 (.40)				
Primary No Longer Primary	1.08 (.58)	.95	2.95	9.17	.06
Separation Anxiety Disorder					
Constant	2.04 (.61)				
Primary No Longer Primary	.91 (.95)	.39	2.48	15.96	.34
Panic Disorder[1]					
Constant	1.44 (.50)				
Primary No Longer Primary	2.23 (1.13)	1.01	9.29	84.78	.05
Agoraphobia					
Constant	2.49 (.74)				
Primary No Longer Primary	.46 (1.03)	.21	1.58	12.02	.66
Obsessive-Compulsive Disorder					
Constant	1.71 (.54)				
Primary No Longer Primary	1.96 (1.15)	.75	7.09	67.46	.09
Specific Phobia					
Constant	1.44 (.50)				
Primary No Longer Primary	.12 (.65)	.32	1.12	4.00	.86
Major Depressive Disorder					
Constant	1.00 (.44)				
Primary No Longer Primary	.10 (.57)	.36	1.11	3.40	.86
Major Depressive Episode					
Constant	.81 (.43)				
Primary No Longer Primary	.29 (.56)	.45	1.33	4.00	.61
Dysthymic Disorder					
Constant	3.22 (1.02)				
Primary No Longer Primary	.45 (1.44)	.09	1.56	26.09	.76
Alcohol Abuse					
Constant	.81 (.43)				
Primary No Longer Primary	.58 (.58)	.57	1.78	5.55	.32
Alcohol Dependence[2]					
Constant	1.44 (.50)				
Primary No Longer Primary	2.23 (1.13)	1.02	9.29	84.78	.05
Drug Abuse[3]					
Constant	1.00 (.44)				
Primary No Longer Primary	1.95 (.85)	1.32	7.00	37.01	.02
Drug Dependence					
Constant	2.49 (.74)				
Primary No Longer Primary	18.72 (6355.07)	.00	1.35[8]	-	1.00
Nicotine Dependence					
Constant	1.20 (.47)				
Primary No Longer Primary	-.36 (.58)	.23	.70	2.18	.54

[1] R^2=.08 (Cox & Snell), .17 (Nagelkerke). Model $\chi^2(1)=5.40$, $p < .05$

[2] R^2=.08 (Cox & Snell), .17 (Nagelkerke). Model $\chi^2(1)=5.40$, $p < .05$.

[3] R^2=.09 (Cox & Snell), .17 (Nagelkerke). Model $\chi^2(1)=6.41$, $p < .05$.

Table 6

ANOVAs Comparing Mean Self-Report Form Scores at 16-Year Follow-Up by Posttreatment Response

	Oneway Analysis of Variance		
	df	*F*	*P*
Primary No Longer Primary			
BDI-II	(1, 61)	.47	.50
BAI	(1, 61)	.47	.49
SDS	(1, 61)	1.16	.29
QOLI	(1, 59)	.02	.90
DUSI-R	(1, 59)	2.13	.15
Primary No Longer Present			
BDI-II	(1, 61)	1.49	.23
BAI	(1, 61)	1.76	.19
SDS	(1, 61)	.91	.35
QOLI	(1, 59)	.63	.43
DUSI-R	(1, 59)	1.89	.18

Note. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. BDI-II= Beck Depression Inventory-II Total Score; BAI=Beck Anxiety Inventory Total Score; SDS=Sheehan Disability Scale Overall Disability Score; QOLI=Quality of Life Inventory Total Score; DUSI-R=Drug Use Screening Inventory-Revised Overall Problem Density Index.

Table 7

Adult 12-Month Diagnostic Status by Posttreatment Response

Diagnosis	Primary No Longer Primary (N=47)	Primary Still Primary (N=19)	Primary No Longer Present (N=40)	Primary Still Present (N=26)
	n (%)	n (%)	n (%)	n (%)
Anxiety Disorders				
Generalized Anxiety Disorder	3 (6.38%)	3 (15.79%)	4 (10.00%)	2 (7.69%)
Social Phobia	6 (12.77%)	2 (10.53%)	4 (10.00%)	4 (15.38%)
Separation Anxiety Disorder	0	1 (5.26%)	1 (2.50%)	0
Panic Disorder	2 (4.26%)	4 (21.05%)	5 (12.5%)	1 (3.85%)
Agoraphobia	2 (4.26%)	1 (5.26%)	2 (5.00%)	1 (3.85%)
Specific Phobia	4 (8.51%)	3 (15.79%)	4 (10.00%)	3 (11.54%)
Depressive Disorders				
Major Depressive Disorder	3 (6.38%)	4 (21.05%)	4 (10.00%)	3 (11.54%)
Major Depressive Episode	4 (8.51%)	4 (21.05%)	5 (12.5%)	3 (11.54%)
Dysthymic Disorder	1 (2.12%)	1 (5.26%)	1 (2.50%)	1 (3.85%)
Substance Use Disorders				
Alcohol Abuse	3 (6.38%)	4 (21.05%)	5 (12.5%)	2 (7.69%)
Alcohol Dependence	1 (2.12%)	3 (15.79%)	3 (7.50%)	1 (3.85%)
Drug Abuse	1 (2.12%)	1 (5.26%)	1 (2.50%)	1 (3.85%)
Drug Dependence	0	1 (5.26%)	1 (2.50%)	0
Nicotine Dependence	7 (14.89%)	2 (10.53%)	2 (5.00%)	7 (26.92%)

Note. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary Still Primary=Pretreatment primary diagnosis remained primary diagnosis at posttreatment. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Primary Still Present=Pretreatment primary diagnosis was no longer primary but remained in diagnostic profile at posttreatment.

Table 8

Logistic Regressions Comparing Successful Treatment Response (Defined as Primary No Longer Primary), Unsuccessful Treatment Response, and Same-Age Community Participants on 12-Month DSM-IV Anxiety Disorder Diagnoses Controlling for Age

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*P*
Generalized Anxiety Disorder[1]					
Constant	2.88 (.14)				
Age	.01 (.01)	1.00	1.01	1.01	.07
Normative Comparison Group					.09
Primary Still Primary	-1.35 (.63)	.08	.26	.90	.03
Primary No Longer Primary	-.34 (.60)	.22	.71	2.31	.57
Social Phobia					
Constant	1.76 (.11)				
Age	.02 (.00)	1.02	1.02	1.03	<.01
Normative Comparison Group					.69
Primary Still Primary	-.14 (.75)	.20	.87	3.79	.85
Primary No Longer Primary	-.37 (.44)	.29	.69	1.64	.40
Separation Anxiety Disorder					
Constant	2.01 (.23)				
Age	.05 (.01)	1.04	1.05	1.07	<.01
Normative Comparison Group					.87
Primary Still Primary	-.55 (1.03)	.08	.58	4.36	.59
Primary No Longer Primary	17.71 (5851.74)	.00	49236947.98		1.00
Panic Disorder[2]					
Constant	2.85 (.17)				
Age	.02 (.00)	1.01	1.02	1.02	<.01
Normative Comparison Group					<.01
Primary Still Primary	-1.96 (.57)	.05	.14	.43	<.01
Primary No Longer Primary	-.18 (.73)	.20	.84	3.48	.81
Agoraphobia					
Constant	3.66 (.23)				
Age	.01 (.01)	1.00	1.01	1.02	.02
Normative Comparison Group					.28
Primary Still Primary	-1.10 (1.03)	.04	.33	2.52	.29
Primary No Longer Primary	-.89 (.73)	.10	.41	1.73	.23
Specific Phobia					
Constant	1.77 (.10)				
Age	.01 (.00)	1.01	1.01	1.02	<.01
Normative Comparison Group					.70
Primary Still Primary	-.42 (.63)	.19	.66	2.26	.50
Primary No Longer Primary	.29 (.53)	.47	1.31	3.66	.61

Note. Normative Comparison Group=Participants from the National Comorbidity Survey-Replication study (NCS-R; Kessler et al., 2005). Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary Still Primary=Pretreatment primary diagnosis remained primary diagnosis at posttreatment.

[1]*Dunnetts t* comparing Normative Comparison Group and Primary Still Primary= $p < .05$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Primary= $p = .72$

[2]*Dunnetts t* comparing Normative Comparison Group and Primary Still Primary= $p < .01$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Primary= $p = .80$

Table 9

Logistic Regressions Comparing Successful Treatment Response (Defined as Primary No Longer Primary), Unsuccessful Treatment Response, and Same-Age Community Participants on 12-Month DSM-IV Depressive Disorder Diagnoses Controlling for Age

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Major Depressive Disorder					
Constant	1.82 (.11)				
Age	.02 (.00)	1.01	1.02	1.02	<.01
Normative Comparison Group					.19
Primary Still Primary	-.97 (.57)	.13	.38	1.15	.09
Primary No Longer Primary	.38 (.60)	.45	1.46	4.73	.53
Major Depressive Episode					
Constant	1.53 (.10)				
Age	.02 (.00)	1.02	1.02	1.02	<.01
Normative Comparison Group					.37
Primary Still Primary	-.73 (.57)	.16	.48	1.47	.20
Primary No Longer Primary	.31 (.53)	.49	1.37	3.83	.55
Dysthymic Disorder					
Constant	3.42 (.18)				
Age	.01 (.01)	1.00	1.01	1.01	.12
Normative Comparison Group					.77
Primary Still Primary	-.70 (1.03)	.07	.50	3.77	.50
Primary No Longer Primary	.24 (1.02)	.17	1.27	9.28	.81

Note. Normative Comparison Group=Participants from the National Comorbidity Survey-Replication study (Kessler et al., 2005). Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary Still Primary=Pretreatment primary diagnosis remained primary diagnosis at posttreatment.

Table 10

Logistic Regressions Comparing Successful Treatment Response (Defined as Primary No Longer Primary), Unsuccessful Treatment Response, and Same-Age Community Participants on 12-Month DSM-IV Substance Use Disorder Diagnoses Controlling for Age

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Alcohol Abuse[1]					
Constant	1.46 (.20)				
Age	.06 (.01)	1.05	1.06	1.08	<.01
Normative Comparison Group					<.01
Primary Still Primary	-1.76 (.57)	.06	.17	.53	<.01
Primary No Longer Primary	-.44 (.60)	.20	.65	2.11	.47
Alcohol Dependence[2]					
Constant	2.59 (.27)				
Age	.05 (.01)	1.03	1.05	1.07	<.01
Normative Comparison Group					<.01
Primary Still Primary	-2.20 (.64)	.03	.11	.39	<.01
Primary No Longer Primary	-.08 (1.02)	.13	.92	6.77	.94
Drug Abuse					
Constant	1.68 (.30)				
Age	.08 (.01)	1.06	1.08	1.10	<.01
Normative Comparison Group					.72
Primary Still Primary	-.85 (1.04)	.06	.43	3.27	.41
Primary No Longer Primary	.02 (1.02)	.14	1.02	7.48	.99
Drug Dependence					
Constant	2. 85 (.49)				
Age	.08 (.02)	1.05	1.08	1.11	<.01
Normative Comparison Group					.19
Primary Still Primary	-1.92 (1.05)	.02	.15	1.14	.07
Primary No Longer Primary	16.32 (5840.98)	.00	12230532.57		1.00
Nicotine Dependence[3]					
Constant	2.61 (.16)				
Age	.02 (.00)	1.01	1.02	1.03	<.01
Normative Comparison Group					<.01
Primary Still Primary	-.94 (.75)	.09	.39	1.70	.21
Primary No Longer Primary	-1.35 (.42)	.11	.26	.59	<.01

Note. Normative Comparison Group=Participants from the National Comorbidity Survey-Replication study (Kessler et al., 2005). Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary Still Primary=Pretreatment primary diagnosis remained primary diagnosis at posttreatment.

[1]*Dunnetts t* comparing Normative Comparison Group and Primary Still Primary= $p < .01$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Primary= $p = .12$

[2]*Dunnetts t* comparing Normative Comparison Group and Primary Still Primary= $p < .01$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Primary= $p = .78$

[3]*Dunnetts t* comparing Normative Comparison Group and Primary Still Primary= $p = .16$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Primary= $p < .01$

Table 11

Logistic Regressions Comparing Successful Treatment Response (Defined as Primary No Longer Present), Unsuccessful Treatment Response, and Same-Age Community Participants on 12-Month DSM-IV Anxiety Disorder Diagnoses Controlling for Age

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Generalized Anxiety Disorder[1]					
Constant	2.87 (.14)				
Age	.01 (.00)	1.00	1.01	1.01	.07
Normative Comparison Group					.06
Primary Still Present	-1.32 (.55)	.09	.27	.78	.02
Primary No Longer Present	-.08 (.73)	.22	.92	3.85	.91
Social Phobia					
Constant	1.76(.11)				
Age	.02 (.00)	1.02	1.02	1.03	<.01
Normative Comparison Group					.56
Primary Still Present	-.58 (.55)	.19	.56	1.63	.29
Primary No Longer Present	-.09 (.53)	.32	.91	2.58	.86
Separation Anxiety Disorder					
Constant	2.06 (.23)				
Age	.05 (.01)	1.04	1.05	1.077	<.01
Normative Comparison Group					.97
Primary Still Present	-.25 (1.03)	.10	.78	5.80	.81
Primary No Longer Present	17.72 (6342.85)	.00	49759573.98		1.00
Panic Disorder[2]					
Constant	2.85 (.17)				
Age	.02 (.00)	1.01	1.02	1.02	<.01
Normative Comparison Group					<.01
Primary Still Present	-1.85 (.50)	.06	.16	.42	<.01
Primary No Longer Present	.37 (1.02)	.20	1.45	10.65	.71
Agoraphobia[3]					
Constant	3.66 (.23)				
Age	.01 (.01)	1.00	1.01	1.02	.02
Normative Comparison Group					.12
Primary Still Present	-1.51 (.75)	.05	.22	.95	.04
Primary No Longer Present	-.34 (1.02)	.10	.72	5.27	.74
Specific Phobia					
Constant	1.77 (.10)				
Age	.01 (.00)	1.01	1.01	1.02	<.01
Normative Comparison Group					.61
Primary Still Present	-.40 (.55)	.23	.67	1.96	.47
Primary No Longer Present	.41 (.60)	.46	1.50	4.90	.50

Note. Normative Comparison Group=Participants from the National Comorbidity Survey-Replication study (NCS-R; Kessler et al., 2005). Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Primary Still Present=Pretreatment primary diagnosis was no longer primary but remained in diagnostic profile at posttreatment.

[1]*Dunnetts t* comparing Normative Comparison Group and Primary Still Present= $p = .97$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Present = $p = .01$

[2]*Dunnetts t* comparing Normative Comparison Group and Primary Still Present= $p = .99$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Present = $p < .01$

[3]*Dunnetts t* comparing Normative Comparison Group and Primary Still Present= $p = .84$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Present = $p < .05$

Table 12

Logistic Regressions Comparing Successful Treatment Response (Defined as Primary No Longer Present), Unsuccessful Treatment Response, and Same-Age Community Participants on 12-Month DSM-IV Depressive Disorder Diagnoses Controlling for Age

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Major Depressive Disorder					
Constant	1.82 (.11)				
Age	.02 (.00)	1.01	1.02	1.02	<.01
Normative Comparison Group					.52
Primary Still Present	-.60 (.55)	.19	.55	1.61	.28
Primary No Longer Present	.21 (.60)	.38	1.23	4.02	.73
Major Depressive Episode					
Constant	1.53 (.10)				
Age	.02 (.00)	1.02	1.02	1.02	<.01
Normative Comparison Group					.35
Primary Still Present	-.62 (.50)	.20	.54	1.43	.21
Primary No Longer Present	.45 (.60)	.48	1.58	5.13	.45
Dysthymic Disorder					
Constant	3.42 (.18)				
Age	.01 (.00)	1.00	1.01	1.01	.12
Normative Comparison Group					.93
Primary Still Present	-.37 (1.02)	.09	.69	5.14	.72
Primary No Longer Present	.08 (1.02)	.15	1.08	7.91	.94

Note. Normative Comparison Group=Participants from the National Comorbidity Survey-Replication study (Kessler et al., 2005). Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Primary Still Present=Pretreatment primary diagnosis was no longer primary but remained in diagnostic profile at posttreatment.

Table 13

Logistic Regressions Comparing Successful Treatment Response (Defined as Primary No Longer Present), Unsuccessful Treatment Response, and Same-Age Community Participants on 12-Month DSM-IV Substance Use Disorder Diagnoses Controlling for Age

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Alcohol Abuse[1]					
Constant	1.46 (.20)				
Age	.06 (.01)	1.05	1.06	1.08	<.01
Normative Comparison Group					<.01
Primary Still Present	-1.67 (.51)	.07	.19	.51	<.01
Primary No Longer Present	-.16 (.73)	.20	.85	3.56	.82
Alcohol Dependence[2]					
Constant	2.59 (.27)				
Age	.05 (.01)	1.03	1.05	1.07	<.01
Normative Comparison Group					.01
Primary Still Present	-1.86 (.63)	.05	.16	.53	<.01
Primary No Longer Present	-.24 (1.02)	.11	.79	5.82	.82
Drug Abuse					
Constant	1.68 (.30)				
Age	.08 (.01)	1.06	1.08	1.10	<.01
Normative Comparison Group					.86
Primary Still Present	-.56 (1.03)	.08	.57	4.27	.59
Primary No Longer Present	-.13 (1.02)	.12	.88	6.48	.90
Drug Dependence					
Constant	2.85 (.49)				
Age	.08 (.02)	1.05	1.08	1.11	<.01
Normative Comparison Group					.29
Primary Still Present	-1.63 (1.04)	.03	.20	1.49	.12
Primary No Longer Present	16.34 (6328.92)	.00	12429871.81		1.00
Nicotine Dependence[3]					
Constant	2.61 (.16)				
Age	.02 (.00)	1.01	1.02	1.03	<.01
Normative Comparison Group					<.01
Primary Still Present	-.60 (.74)	.13	.55	2.33	.41
Primary No Longer Present	-1.54 (.42)	.09	.21	.49	<.01

Note. Normative Comparison Group=Participants from the National Comorbidity Survey-Replication study (Kessler et al., 2005). Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Primary Still Present=Pretreatment primary diagnosis was no longer primary but remained in diagnostic profile at posttreatment.

[1]*Dunnetts t* comparing Normative Comparison Group and Primary Still Present= $p = .45$
Dunnetts t comparing Normative Comparison Group and Primary No Longer Present = $p < .01$

[2]*Dunnetts t* comparing Normative Comparison Group and Primary Still Present= $p = .67$
Dunnetts t comparing Normative Comparison Group and Primary No Longer Present = $p < .01$

[3]*Dunnetts t* comparing Normative Comparison Group and Primary Still Present= $p = < .01$
Dunnetts t comparing Normative Comparison Group and Primary No Longer Present = $p = .40$

CHAPTER 2

LITERATURE REVIEW

Child Anxiety Disorders and their Comorbidities

Anxiety disorders commonly occur in children, impacting approximately 10 to 20% of youth (Chavira, Stein, Bailey, & Stein, 2004; Costello, Mustillo, Keeler & Angold, 2004). Anxious youth often avoid developmentally-appropriate interactions and situations, and anxiety disorders in youth are associated with negative consequences including being less well liked by peers (Verduin & Kendall, 2008), poor social relations (e.g., Hartup, 1983; Greco & Morris, 2005; Strauss, Forehand, Smith, & Frame, 1986), and impaired academic achievement (e.g., King & Ollendick, 1989; Van Amerigen, Mancini, & Farvolden, 2003). Anxiety disorders are often chronic (e.g., Keller et al., 1992; Pine, Cohen, Gurley, Brook, & Ma, 1998) and, if untreated, associated with anxiety disorders in adulthood (e.g., Ost, 1987; Sheehan, Sheehan & Minichiello, 1981) as well as later depression (Angold, Costello, & Erkanli, 1999; Strauss, Lease, Last, & Francis, 1988), suicidality (Brent et al., 1986; Rudd, Joiner, & Rumzek, 2004), and substance use (Costello, Mustillo, Erkanli, Keeler, & Angold, 2003; Lopez, Turner, & Saavadra, 2005; Merikangas et al., 1998).

Research on comorbidity has advanced significantly over the past 20 years (Angold et al., 1999). Most initial disorder onsets occur in childhood, and later disorders are generally temporally secondary comorbidities (Kessler & Wang, 2008). A better understanding of comorbidity is important for several reasons (Hall, Degenhardt, & Teeson, 2009). First, comorbid conditions are common, impacting 27.7 to 35 percent of individuals who meet lifetime diagnostic criteria for one disorder (Bourdon, Rae, Locke, Narrow, & Regier, 1992; Kessler, Berglund et al., 2005). Second, having one or more comorbid disorder has been associated with more severe psychopathology (e.g., suicide

attempts, violence, psychosis), poorer treatment response (e.g., among adolescents presenting for inpatient treatment with comorbid externalizing and substance use disorders), and longer illness course (Kavanagh, Mueser, & Baker, 2003; Kessler, 1995; Kessler, Berglund et al., 2005; Kessler & Wang, 2008; Tomlinson, Brown, & Abrantes, 2004). Third, understanding comorbidity can inform diagnostic classification (Angold et al., 1999). Finally, a better understanding of comorbidity may suggest possibilities for prevention (i.e., prevention of secondary disorders by treating a primary disorder). Several hypotheses have been proposed to explain comorbidity (Cerda, Sagdeo, & Galea, 2008; Hall et al., 2009; Kessler & Wang, 2008), including: (a) one disorder may directly produce another (Raimo & Schuckit, 1998), (b) comorbid disorders may be the result of a shared underlying cause (Jessor & Jessor, 1977; Reinherz, Giaconia, Carmola Hauf, Wasserman, & Paradis, 2000), (c) some disorders increase risk for later disorders (Kessler, 1995), and (d) artifactual or methodological explanations (e.g., symptom overlap; Drabick & Kendall, in press).

Understanding comorbidity among anxiety disorders and between anxiety disorders and other disorders has been a challenge. Studies vary in terms of which anxiety disorders they include, with disorders associated with trauma (i.e., posttraumatic stress disorder; PTSD) often "lumped together" with phobic disorders, panic disorder, etc. to form a single anxiety construct (e.g., early reports from the Oregon Adolescent Depression Project; Lewinsohn, Gotlib, Lewinsohn, Seeley, & Allen, 1998). There has also been a lack of consensus about which comorbid disorders to control for when conducting analyses of associations, and researchers have not always been clear as to whether comorbid disorders are occurring concurrently or sequentially (Costello, Egger,

& Angold, 2005). There has historically been an overreliance on cross-sectional and retrospective studies of comorbidity (Angold et al., 1999). Are temporal relationships between anxiety disorders in youth and later comorbid disorders reliably observed? Do anxiety disorders in youth serve as "gateway" disorders for later comorbidity? The present review critically evaluates the research on child anxiety comorbidity, with a focus on longitudinal prospective studies. First, the specific symptoms, prevalence, assessment, impairment, and sequelae of childhood anxiety disorders are discussed. Next, research regarding the comorbidities commonly associated with child anxiety disorders is reviewed, including studies of comorbid anxiety disorders, depressive disorders, substance misuse, and externalizing disorders. The possibilities for secondary disorder prevention will be discussed. Finally, cognitive-behavioral therapy (CBT) for child anxiety disorders will be discussed as a promising tool for preventing secondary comorbidities by treating a primary disorder in youth.

Anxiety Disorders in Youth

DSM-IV identifies several anxiety disorders in youth. The present review has an eye toward (a) separation anxiety disorder (SAD), (b) generalized anxiety disorder (GAD), and (c) social phobia (SP; Avoidant Disorder in DSM-III-R) as SAD, GAD, and SP are typically treated similarly and researched collectively in children and adolescents (Crawley, Beidas, Benjamin, Martin & Kendall, 2008; Kendall et al., 1997; Walkup et al., 2008). These disorders are highly comorbid with one another (e.g., in the CAMS multisite trial, 78.44% of youth had one or more anxiety comorbidity [including SAD, GAD, and SP], Beidel, Turner, & Morris, 1999; Essau, Conradt, & Petermann, 1999; Kendall et al., 2010; Wittchen, Stein, & Kessler, 1999) and have been conceptualized as

sharing an underlying anxiety construct (e.g., Bell-Dolan & Brazeal, 1993; Pine & Grun, 1998). Children with SAD are characterized by inappropriate and excessive anxiety regarding separation from home or a caretaker. Children with SAD may exhibit somatic symptoms such as stomachaches, fear that either they or their caretakers will experience illness or an accident, fear being lost, avoid staying alone, and/or have nightmares involving the content of separation fears. GAD is characterized by excessive and persistent anxiety and worry about a number of activities or events. Children with GAD are unable to or have difficulty controlling their worries and the anxiety and worry are accompanied by physical symptoms such as fatigue or restlessness. SP is characterized by a marked and persistent fear of performance or social situations that may result in embarrassment. Unlike adults, children with SP may or may not recognize that their fear is unreasonable or excessive. They often appear shy or withdrawn in social exchanges with peers and adults and most often avoid social or performance situations.

SAD, GAD, and SP exhibit strong covariation with each other cross-sectionally and over time and evidence suggests that having one of these three disorders in particular heightens the risk for a range of later anxiety disorders (Hirshfeld-Becker, Micco, Simoes, & Henin, 2008). SAD, GAD, and SP infrequently occur as the sole disorder present (Kendall & Brady, 1995), and show similar familial relationships with adult anxiety and depression (e.g., Fyer, Mannuzza, Chapman, Martin, & Klein, 1995; Last, Hersen, Kazdin, Orvaschel, & Perrin, 1991). Childhood anxiety disorders predict a range of psychiatric disorders in adolescence and young adulthood including SAD, overanxious disorder (OAD), SP, panic attacks, conduct disorder (CD), depression, oppositional defiant disorder (ODD), and attention-deficit/hyperactivity disorder (ADHD; Bittner et

al., 2007; Foley, Pickles, Maes, Silberg, & Eaves, 2004) though consistent unique pathways from specific childhood anxiety disorders to later disorders have not been demonstrated. Additionally, though variations in treatment response by specific disorder have occasionally been found, most studies have failed to find significant differences in treatment response by disorder for SAD, GAD, and SP, despite the high comorbidity among the disorders and regardless of which disorder is primary in the child's diagnostic profile (e.g., Kendall et al., 1997).

Prevalence

Anxiety disorders are common in youth (e.g., Bernstein & Borchardt, 1991) and in adults, both in 12-month prevalence estimates from the general population (Kessler, Chiu, Demler, & Walters, 2005) and across the lifespan (28.8%; Kessler, Berglund et al., 2005). Research indicates that approximately 10 to 20% of children in the general population and primary care settings report clinically significant levels of anxiety (Chavira et al., 2004; Costello et al., 2004). Epidemiological studies indicate that anxiety disorders are the most common lifetime disorders, occurring more often than mood, impulse control, and substance use disorders (Kessler, Berglund et al., 2005). In addition to being more common, anxiety disorders have a younger age of onset, often first occurring in childhood, and temporally precede the development of later comorbid mood and substance use disorders (e.g., Christie et al., 1988).

Assessment

When assessing youth for a possible anxiety disorder, several areas should be emphasized: the onset, development, and context of symptoms, a developmental history, as well as children's medical, family, social, and academic history (Bernstein, Borchardt,

& Perwein, 1996). Keeping these factors in mind, the assessment instruments used to evaluate anxiety in youth should reliably and validly measure symptoms across time and multiple domains (e.g., cognitive, behavioral, and psychological channels), discriminate between disorders, assess severity, include multiple observations (e.g., parent and child report), and allow for measurement of therapeutic change (Kendall & Flannery-Schroeder, 1998; Stallings & March, 1995).

Interviews are a common method for assessing anxiety in youth and these interviews can range from highly structured to unstructured. Agreement between youth and parent reports on questions asked within interviews varies, even when using structured interviews (e.g., Choudhury, Pimentel, & Kendall, 2003; Comer & Kendall, 2004; Edelbrock, Costello, Dulcan, Conover, & Kala, 1986; Edelbrock, Costello, Dulcan, Kalas, & Conover, 1985; Grills & Ollendick, 2003) and there is additional variability associated with the child's age (i.e., older youth are more reliable self-reporters of their own anxiety whereas parental reliability decreases as children get older; Edelbrock et al., 1985).

Self-report measures are the most common method for assessing anxiety and while they provide important information about the youth's subjective anxious experience, are quick to administer, and generally inexpensive, they have limitations. For example, self-report measures should not be used as a sole diagnostic tool because they may be poor at discriminating anxiety disorders from other disorders (Perrin & Last, 1992). Additionally, self-reports can fail to capture information about the child's specific anxieties, and this specific information is very valuable for individualizing treatment (Kendall & Suveg, 2006). Finally, self-reports may lack normative data for children of

various developmental levels and may not be appropriate for children at diverse stages of cognitive maturity.

Behavioral observation throughout the assessment process can provide clinicians with very useful information (e.g., fingernail biting, avoidance of eye contact, quiet speech). Parent and teacher ratings provide additional reports of the child's behavior in various contexts. These unstructured observations can provide important information though they may be limited by observer bias and, especially in the case of parent and teacher reports, may be impacted by the reporter's lack of appropriate training regarding anxiety. Additionally, parents and teachers may not be aware of the nature of the child's internal experience of anxiety (e.g., Kendall & Flannery-Schroeder, 1998; Schniering, Hudson, & Rapee, 2000). Given that one individual assessment tool may not capture the child's anxiety symptoms, multimethod assessment is generally recommended.

Differential diagnosis is also an important consideration. Ideally, as already noted, diagnoses are based on a thorough multi-method and multi-informant assessment. When symptom criteria for multiple disorders are met, it is appropriate for multiple diagnoses to be assigned. Differential diagnosis and assessment of comorbidity is important both within the anxiety disorders and between anxiety disorders and other disorders. For example, because the worries of GAD youth occur in multiple domains, it is possible that the content of a child's worries resembles fears or concerns often seen in other childhood anxiety disorders (e. g., SAD or SP). Additionally, physical symptoms associated with anxiety may appear similar to symptoms of ADHD or to depression (e.g., difficulty paying attention, relaxing, and sitting still). To assure methodological biases do not attribute to inflated rates of comorbidity, researchers should be mindful in taking steps to

ensure that disorder co-occurrence is not a function of a particular assessment strategy (e.g., structured interviews, self-report forms), particular informants (parent, child, teacher), samples (e.g., clinical samples), or study design (e.g., cross-sectional; Drabick & Kendall, in press). Because of concerns about symptom overlap attributing to elevated rates of comorbidity, some have examined rates of disorder co-occurrence with and without the overlapping symptoms (e.g., Gadow & Nolan, 2002). While this modifies the defined disorder, it can be useful in minimizing concerns that high levels of comorbidity are solely attributable to symptom overlap (Drabick & Kendall, in press).

Impairment

Anxiety disorders have a negative effect on functioning. The behavior of anxious children is characterized by avoidance and this avoidance can result in significant distress for the children themselves as well as caregivers. Anxious children commonly avoid age-appropriate situations and social interactions necessary for healthy development and are rated as less well liked by peers (Verduin & Kendall, 2008). Difficulties in social relations are common (e.g., Greco & Morris, 2005; Hartup, 1983; Langley, Bergman, McCracken, & Piacentini, 2004; Strauss et al., 1986), as are impaired academic achievement (e.g., King & Ollendick, 1989; Van Amerigen et al., 2003), and future emotional well-being (e.g., Beidel, 1991; Feehan, McGee, & Williams, 1993). Research suggests that most anxiety disorders do not abate over time (e.g., Keller et al., 1992; Pine et al., 1998).

Retrospective reports demonstrate that adults with anxiety disorders report having experienced notable anxiety as children (e.g., Ost, 1987; Sheehan et al., 1981). Anxiety disorders in childhood are associated with later depression (Angold et al., 1999; Strauss

et al., 1988), suicidal attempts and ideation (Brent et al., 1986; Rudd et al., 2004), and substance use (Costello et al., 2003; Lopez et al., 2005; Merikangas et al., 1998). Evidence also suggests anxiety disorders in youth may temporally precede the emergence of depressive disorders (e.g., Alpert, Maddocks, Rosenbaum, & Fava, 1994; Biederman, Faraone, Mick, & Lelon, 1995). Other potential sequelae of anxiety disorders in childhood include an increased probability for the later development of substance use problems, and researchers have highlighted the need to better understand the pathways between these disorders (Clark & Winters, 2002).

Long-Term Implications

Anxiety disorders in youth have important long-term implications. For instance, as previously noted, a significant number of adults with anxiety disorders report having suffered from anxiety as youth (e.g., Last, Hersen, Kazdin, Francis, & Grubb, 1987; Weissman, Leckman, Merikangas, Gammon, & Prosoff, 1964). Higher levels of anxiety and depression are reported in older youth than in younger children suggesting anxiety symptoms, when untreated, worsen over time. Additionally, an anxiety disorder in youth has been associated with higher rates of other disorders in adulthood (e.g., substance abuse problems; Pine et al., 1998). As age increases, the presence of an anxiety disorder also has an associated increase in the likelihood of a depressive disorder (Beesdo et al., 2007, Brady & Kendall, 1992; Pine et al., 1998), and childhood anxiety disorders typically precede the onset of substance use disorders (Merikangas et al., 1998). Given the high lifetime co-occurrence of anxiety and substance use disorders (35% to 45%; Kessler et al., 1996) and the deleterious outcomes associated with substance dependence (Toumbourou et al., 2007), this primacy is compelling and warrants further investigation.

Treatment of acute substance dependence has yielded mixed results and 40-60% of those treated return to active use within 12 months following treatment (Finney & Moos, 1992; Hubbard, Craddock, Flynn, Anderson, & Etheridge, 1997). Thus, substance dependence has increasingly been viewed as a chronic condition that requires continuous long-term intervention (McLellan, Lewis, O'Brien, & Kleber, 2000) and heightened prevention efforts. The indication that CBT for child anxiety may allay later substance use (e.g., Kendall, Safford, Flannery-Schroeder, & Webb, 2004) has not been adequately studied. Most investigations of child anxiety treatment do not include substance use measures and lack follow-ups of sufficient length to address questions of potential secondary benefits for substance use disorder risk reduction or prevention (Compton, Burns, Egger, & Robertson, 2002), thus further investigation is warranted.

Comorbidity

Comorbidity regarding child and adolescent psychiatric disorders has been a topic of interest since the late 1980s (Angold et al., 1999). Interest in understanding and treating comorbidity has increased considerably over the years. In particular, the manner in which comorbidity may increase our understanding of the development of psychopathology has been an increasing area of interest, though considerable questions remain unanswered. More recently, the utility of a developmental psychopathology perspective in examining diagnoses more generally and comorbidity in particular has been emphasized (e.g., Drabick & Kendall, in press).

The developmental psychopathology perspective emphasizes the importance of considering normative development as the comparison for determining whether the behavior of children and adolescents is problematic, or atypical (e.g., Drabick, 2009;

Kendall & Comer, 2010; Steinberg, 2002). There is also the need to consider both categorical and dimensional diagnostic models to identify youth with subthreshold, but nevertheless impairing, symptoms and those at-risk for disorder development (e.g., Bubier & Drabick, 2008; Maser et al., 2009). Additionally, the importance of considering contextual factors (e.g., family, neighborhood), which can be useful in predicting illness course, symptom severity, and treatment outcome (e.g., Bubier, Drabick, & Breiner, 2009; Holmbeck & Kendall, 2002) is emphasized from a developmental psychopathology perspective. Principles of multifinality and equifinality are especially pertinent to discussions of comorbidity. Multifinality, the anticipation that a single risk factor can lead to various outcomes depending on contextual factors, is important in considering why contextual factors do not always have a uniform effect on individuals (Cicchetti & Rogosch, 1996). Equifinality, the notion that different processes can promote the same outcome, is pertinent to the consideration of comorbidity. That is, youth may meet criteria for multiple diagnoses and the means by which they arrive at these diagnoses are variable and consistent with a variety of explanations for disorder co-occurrence (Cicchetti & Rogosch, 1996; Drabick & Kendall, in press).

Why Study Comorbidity?

Many individuals who have had a mental disorder in their lifetime report having their first disorder onset in childhood, and later disorders are generally temporally secondary comorbidities (i.e., half of all lifetime cases have an age of onset by age 14 and three fourths by age 24; Kessler, Berglund, et al., 2005; Kessler & Wang, 2008). The study of comorbidity is important for several reasons (Hall et al., 2009). First, the presence of comorbid conditions is common. Epidemiological data suggests that 27.7 to

35 percent of individuals who meet lifetime diagnostic criteria for one disorder will also have one or more comorbid diagnoses in their lifetime (Bourdon et al., 1992; Kessler, Berglund et al., 2005). Some disorders, such as disorders of substance use and depression, have even higher rates of comorbidity. For example, a recent review of studies examining adolescent substance use disorders found that 60% of these youth had one or more comorbid diagnosis (Armstrong & Costello, 2002). Failure to attend to comorbidity may result in the misappraisal of symptoms or associated characteristics to a single disorder, when these symptoms may in fact be associated with one or more of the comorbidities present (Kessler, 1995; Kessler & Wang, 2008).

Second, the presence of comorbidity has been associated with poorer treatment response, the need for more treatment, and more severe and longer illness course (Kavanagh et al., 2003; Kessler, 1995; Kessler, Berglund et al., 2005; Tomlinson et al., 2004). Disorder severity is strongly related to high comorbidity, even though the initial temporally primary disorder may be relatively mild (Kessler & Wang, 2008). Individuals with comorbid conditions generally are more impaired, are more socially disabled, have lower work productivity, and are more costly at a societal level (e.g., Wittchen, Nelson, & Lachner, 1998). They also utilize a greater number of mental health resources (Rohde, Lewinsohn, & Seeley, 1996; Wittchen et al., 1998). The greater number of comorbid disorders in adolescents has been shown to linearly increase the risk for the development of later disorders (Woodward & Fergusson, 2001). Comorbidity may be overlooked in treatment, which may explain the poorer treatment response of individuals with comorbidity (Hall et al., 2009). For example, if an individual presents with depression and alcohol dependence, it is important to understand if the alcohol dependence is the

cause of the depression (in which case treatment of the substance dependence would be indicated as the primary intervention course) or if the individual is using alcohol in an effort to manage depressive symptoms (in which case primary treatment of the depression may be indicated; Schuckit et al., 1997a, b).

Third, the co-occurrence of disorders can inform diagnostic classification (Angold et al., 1999). Comorbidity may reflect a meaningful association between underlying disorders, but it may also imply there is a problem with the classification system. High comorbidities between particular disorders has led some to suggest that a dimensional approach to disorder classification may be more appropriate than the current categorical system of the DSM (Schoevers, Deeg, van Tilburg, & Beekman, 2005). Epidemiological studies of comorbidity are generally more useful than clinic-based studies where comorbidity may reflect referral biases rather than representative rates of co-occurring disorders (Angold et al., 1999). Finally, a more thorough understanding of comorbidity may suggest possibilities for prevention of secondary disorders by treatment of the primary disorder.

Hypotheses of Comorbidity

Several hypotheses have been proposed to explain psychiatric comorbidity (Cerda et al., 2008; Hall et al., 2009; Kessler & Wang, 2008). Those most commonly asserted to explain the comorbidity between anxiety disorders and other disorders include: (a) one disorder may directly produce another (e.g., alcohol dependence can produce depression; Raimo & Schuckit, 1998), (b) comorbid disorders may arise from a shared underlying cause (Jessor & Jessor, 1977; Reinherz et al., 2000), (c) some disorders increase risk for later disorders (e.g., some individuals with anxiety may use alcohol to "self-medicate"

their symptoms; Kessler, 1995), and (d) artifactual or methodological explanations (e.g., symptom overlap; Drabick & Kendall, in press). A more thorough review of comorbidities associated with child anxiety can help elucidate if any of these hypotheses are supported by the literature for child anxiety disorders and their common comorbidities. A more thorough discussion of the hypotheses that have been most commonly discussed and that have received at least some empirical support follows.

One disorder may directly produce another. Research suggests that, in some instances, one disorder may directly lead to the development of a secondary disorder. This may occur due to toxic or pharmacological effects of drugs on neurotransmitter functioning or metabolism, or the effects of drugs on general psychological functioning (Brook, Cohen, & Brook, 1998). There is some evidence, while sparse, to support the negative impact of substance use on emotional, physiological, and/or psychological functioning. For example, there is evidence that, for individuals who are alcohol dependant, heavy alcohol use may produce depressive symptoms (Raimo & Schuckit, 1998). While this potential explanation for the co-occurrence of disorders is important to consider in individuals with a history of substance misuse presenting with comorbid symptoms, it is of little explanatory value in informing our understanding of disorders that are temporally secondary to anxiety.

Comorbid disorders may arise from common causes. Comorbidity may occur as the result of common causes, including shared risk factors or etiological processes (Angold, 1999, Drabick & Kendall, in press). Psychosocial risk factors are commonly observed across a number of disorders (Brook et al., 1998), including child temperament (Muris & Ollendick, 2005), parental psychopathology (Loeber, Farrington, Stouthamer-

Loeber, & Van Kammen, 1998), peer rejection (Daeter-Deckard, 2001), parenting behaviors (Garber & Weersing, in press), and parent-child conflict (Loeber et al., 1998). For example, family circumstances, poverty, and abusive environments may increase the likelihood of development of delinquency, substance use, risky sexual behavior, and poor academic performance (Jessor & Jessor, 1977; Reinherz et al., 2000).

Regarding the comorbidity between anxiety and depressive disorders specifically, it has been suggested that some of the symptom overlap is likely due to an underlying risk factor of general negative affectivity (Barlow, 2000; Clark & Watson, 1991). Negative affectivity is related to the construct of neuroticism and represents the extent to which one feels distress (e.g., guilty, angry, upset, worried) and not relaxed or calm (Garber & Weersing, in press). The tripartite model (Clark & Watson, 1991) posits that high negative affectivity characterizes both anxiety and depression. Low positive affect and loss of interest or pleasure are unique characteristics of depression and somatic tension and elevated physiological arousal are unique characteristics of anxiety. By removing the shared variance attributable to negative affectivity, the correlation between anxiety and depression is reduced and their unique features are more easily discriminated (Garber & Weersing, in press). Support for the tripartite model has been demonstrated in clinical and nonclinical samples of youth (Laurent & Ettelson, 2001), though studies up to this point have mainly focused on developing and validating scales to assess tripartite constructs in youth. Future studies would benefit from including measures of negative affectivity and the tripartite model more globally in prospective longitudinal and epidemiological samples.

Predisposing genetic or biological factors, including disorders of neurotransmitter dysfunction, metabolic problems (Brook et al., 1998), information processing biases (Garber & Weersing, in press), and autonomic functioning (Beauchaine, 2001) may also conjure risk for a variety of disorders. For example, anxious and depressed youth have been shown to demonstrate greater amygdala activation when viewing fearful faces than controls (Beesdo et al., 2009). Regarding genetics, preliminary evidence suggests that youth with a first-degree relative with depression are more likely to develop depression themselves, youth with a panic-disordered proband are at increased risk of developing SAD, and for youth of depression plus panic disorder or agoraphobia probands, additional risk for developing a depressive and anxiety disorder is conferred (Weissman et al., 1984). The aforementioned processes may serve as shared risk factors for multiple disorders, which may underlie comorbidity (Drabick & Kendall, in press). For example, a common genetic diathesis may result in anxiety or depression depending on the timing of occurrence of environmental stressors; in genetically vulnerable children stressors may produce anxiety whereas stressors in adolescence may lead to depression Garber & Weersing, in press). These shared processes are likely moderated by additional risk and protective factors, highlighting the need to examine comorbidity and these processes throughout development.

Some disorders may indirectly increase risk for the development of other disorders. Some disorders may serve as "gateway" disorders for the development of later disorders, increasing risk for the development of particular comorbidity. When considered from a developmental psychopathology perspective, this explanation is appealing because it considers developmental pathways, contextual factors including

transactional relationships between individuals and their environment, and risk and resiliency factors (Drabick & Kendall, in press). Comorbidity may result from the primary psychological condition (e.g., GAD) leading to difficulties in interpersonal functioning, educational achievement, etc., and these difficulties may contribute to the development of a secondary disorder (e.g., depression).

One early hypothesis consistent with the notion that a particular disorder may serve as a risk factor for a later disorder is Klein's (1964) "separation anxiety hypothesis" which posits that SAD and disrupted personal relationships in childhood are precursors to adult panic disorder and agoraphobia. Inconsistent support has been found for the separation anxiety hypothesis with most confirmatory support relying on retrospective data, while prospective studies have generally failed to provide affirmative evidence (e.g., Aschenbrand, Kendall, Webb, Safford, & Flannery-Schroeder, 2003).

Child anxiety may frequently temporally precede depression through a variety of indirect causal mechanisms (Garber & Weersing, in press). For example, SP likely increases attempts to avoid negative evaluation (e.g., Alden & Taylor, 2004; Leary & Kowalski, 1995). This may lead to dysfunctional social behaviors that are intended to be protective, but in fact increase rejection (e.g., social withdrawal can lead to peer rejection; Gazelle & Ladd, 2003), resulting in loneliness, sadness, and decreased self-worth. Avoidance of expressed emotion has also been shown to mediate the relationship between SP and depressive symptoms (Grant, Beck, Farrow, & Davila, 2007). Additionally, negative appraisals about social events have been identified as cognitive vulnerabilities to depression (Abramson, Metalsky, & Alloy, 1989).

Kessler (1995) suggests that individuals with anxiety or affective disorders may misuse substances as a method of managing their distress. Alcohol, for example, may improve mood in the short term, however over time alcohol dependence may develop. It has also been suggested that the presence of psychiatric disorders may lead to substance misuse through various mechanisms, such as disrupted parent-child relationships, decreased parental monitoring, or deviant peer relationships (e.g., the presence of anxiety and its associated behavioral inhibition leads to decreased parental monitoring which in turn leads to increased opportunity for experimentation with substances; Brook et al., 1998). This hypothesis also has implications for externalizing disorders. Children with CD may start using substances at an early age due to their increased tendency to engage in risky behaviors. Early substance use as well as early associations with substance-using peers has been associated with increased risk for the development of substance use disorders at a younger age (Cerda et al., 2008; Fergusson & Horwood, 1997).

Artifactual/methodological explanations. Comorbidity has been suggested to reflect chance disorder co-occurrence, sampling bias, population stratification, symptom overlap, and the possibility that the comorbid disorder is a variant or an atypical expression of one disorder (Drabick & Kendall, in press). One possibility is that comorbidity of two disorders occurs by chance because if the occurrence of each disorder is high, their co-occurrence will also be high. Another concern is that comorbid conditions are often observed in clinic samples because individuals with more than one disorder may be more likely to seek treatment (Angold, 1999; Klein & Riso, 1993). Data from epidemiological studies generally reduces concerns about chance comorbidity and

sampling bias because these studies address issues regarding base rates and chance and are population representative.

An additional artifactual concern has to do with population stratification (Drabick & Kendall, in press). This refers to the possibility that two disorders share separate underlying risk factors that commonly occur in the same subgroup of the population, resulting in inflated and artifactual observations of comorbidity. Drabick and Kendall (in press) illustrate this with maternal depression. Childhood depression has been linked to maternal depression (e.g., Frye & Garber, 2005), and conduct problems are related to family discord which is commonly observed in families with parental depression (e.g., Patterson, 1993). Therefore, childhood depression and conduct problems may be observed to co-occur in samples with high rates of maternal depression because mothers with depression are more likely to experience marital problems. The utilization of a developmental psychopathology approach that attends to issues of selection, generalizability, and contextual factors will be useful in minimizing concerns about population stratification in future studies.

Symptom overlap across diagnostic categories has also been raised as a possible explanation for high rates of comorbidity between some disorders (e.g., anxiety disorders and depressive disorders). For example, items on self-report scales of anxiety and depression are often rather similar (Garber & Weersing, in press). Additionally, some symptoms are present in multiple disorders (e.g., inattention in ADHD, anxiety, depression) and therefore individuals presenting with these overlapping symptoms may be more likely to meet criteria for more than one disorder when these symptoms appear in multiple diagnostic categories (Drabick & Kendall, in press). This explanation does not

appear to satisfactorily explain comorbidity, as multiple investigators have conducted comorbidity analyses excluding overlapping items and still observed high rates of comorbidity (e.g., Cole, Truglio, & Peeke, 1997; Stark & Laurent, 2001). Additionally, proposed revisions for DSM-V eliminate some specific symptom overlap (e.g., GAD symptoms that overlap with MDD symptoms), which may help to reduce artifactual comorbidity, though these new symptom criteria have yet to be validated.

Finally, some have suggested that, when disorders co-occur, the comorbid disorder may represent a variant or atypical form of one condition (Klein & Riso, 1993). Specifically, alternative symptom expression may occur because disorders are not manifested homogeneously and because overlapping symptoms do occur. For example, regarding the high comorbidity among anxiety and depressive disorders, one might argue that depression in childhood is not manifested in the same way as it is in adulthood, and anxiety may be the manifestation of the negative affect experienced by depressed youth. Drabick and Kendall (in press) suggest that this explanation can be evaluated by comparing individuals with comorbid conditions to individuals with only one of the conditions on etiological processes, risk and protective factors, developmental pathways, and outcomes to examine whether the comorbid condition is more similar to one of the conditions alone, and whether these similarities change with development.

Comorbidity among Anxiety Disorders

Understanding comorbidity among anxiety disorders has been a challenge for several reasons: studies vary in terms of which anxiety disorders they include, there is a lack of consensus about which comorbid disorders to control for when examining associations, and there has been inconsistent distinction in the research regarding whether

comorbid disorders are occurring concurrently or sequentially (Costello et al., 2005). Many studies lump anxiety disorders together, causing difficulty in both understanding comorbidity among anxiety disorders as well and comorbidities between anxiety and other disorders.

Anxious and withdrawn behaviors in young children (age 8) are associated with elevated risk of SP, specific phobia, and panic disorder/agoraphobia (Goodwin, Fergusson, & Horwood, 2004b). Additionally, behavioral inhibition in childhood has been shown to predict later SP and early anxiety sensitivity predicts later panic disorder (Hirshfeld-Becker et al., 2008). Negative affectivity predicts a range of later psychopathology, including anxiety (Hirshfeld-Becker et al., 2008). Adolescent specific phobia and SP predict adult specific phobia and SP, respectively (Pine et al., 1998).

Among adults with anxiety disorders, follow-back analyses using data from a prospective longitudinal birth cohort study suggest that a third had anxiety disorders as children and the psychiatric history of adults with anxiety disorders were largely nonspecific, though histories of anxiety and depression were most common (Gregory et al., 2007). Some specificity has also been observed: adults with panic disorder do not tend to have histories of childhood disorders, adults with PTSD have higher rates of CD in youth, and those with specific phobias in adulthood have juvenile histories of phobias but not other anxiety disorders. Females may be more likely to have multiple comorbid anxiety disorders than males (Lewinsohn, Zinbarg, Seeley, Lewinsohn, & Sack, 1997).

Evidence exists for high levels of comorbidity among phobias, including specific phobias, SP, and agoraphobia as well as between OAD and SP (Costello et al., 2005). Supporting the high level of comorbidity among phobias, specific familial aggregation of

the phobias has been observed in families of probands with these disorders and no other anxiety comorbidity, and the majority of familial phobia cases occur without anxiety comorbidity (Fyer et al., 1995; Low, Cui, & Merikangas, 2008).

There is also some evidence that early SAD and GAD predict later anxiety disorders (Hirshfeld-Becker et al., 2008), though other studies suggest SAD is likely to remit over time (with 59% of youth with SAD being free of any anxiety disorder at 18-month follow-up; Foley, et al., 2004). Among those for whom SAD did not remit at follow-up (20% of SAD cases were persistent), higher rates of OAD were observed. Several studies have also examined concurrent comorbidity between SAD and panic, but most have failed to find a significant association (Costello et al., 2005). Continuity between certain anxiety disorders, namely PTSD and panic disorder, over time is much stronger than for other anxiety disorders (i.e., specific phobia and GAD; Costello et al. 2003). Rates of SP and panic disorder have been shown to increase over time, whereas SAD prevalence decreases over time (Costello et al. 2003).

Comorbidity between Anxiety and Depression

Anxiety disorders and depressive disorders commonly co-occur in youth (e.g., Costello et al., 2003; Rohde, Lewinsohn, & Seeley, 1991). An early review of anxiety and depression in youth suggests that these disorders are highly comorbid, and while highly correlated measurement of anxiety and depression may explain some of these findings (Brady & Kendall, 1992), meaningful differences exist between anxious, depressed, and concurrently anxious and depressed youth. Despite some symptom overlap, studies have shown that most individuals with comorbid anxiety and depressive disorders retain these diagnoses even when overlapping symptoms are taken out of

consideration (Angold et al., 1999). While depressed youth tend to score high on both measures of depression and anxiety, purely anxious youth score high on anxiety measures but lower on measures of depression symptomology. Young children characterized by high (but not disordered) levels of anxious and withdrawn behavior are at increased risk for the development of major depressive disorder (MDD) during adolescence and young adulthood (Goodwin et al., 2004b). Youth with comorbid anxiety and depression tend to show more severe and diverse anxious and depressive symptomology and they tend to be older than depressed or anxious only youth (Bernstein & Borchardt, 1991). Among high-risk African American youth, those who self-report consistently higher depression symptoms also report higher levels of co-occurring anxiety, and females endorsing more depressive symptoms report more anxiety symptoms than males (Repetto, Caldwell, & Zimmerman, 2004). Because anxiety and depressive disorders predict one another developmentally, this has led some to treat them as part of the same syndrome, however; there is some evidence that the link between anxiety and depression only applies to some anxiety disorders (Costello et al., 2005). A closer examination of the trajectories of anxiety and depressive disorders is important to understand if these disorders should be treated and researched collectively or as separate constructs.

In adults assessed prospectively over time, comorbid anxiety and depression tends to be more persistent and stable than either disorder alone, and those with anxiety only earlier in adulthood tend to develop either depression alone or comorbid anxiety and depression (though depression alone tends to be more stable; Merikangas et al., 2003). The anxiety symptoms tend to precede the onset of depression symptoms in the few studies that allow for the examination of temporal relationships and in longitudinal

studies of adults approximately half of individuals with anxiety also will have depression at some point in time (Murphy et al., 2004). Another longitudinal prospective study with adults, modeling depressive and anxious symptoms in Swedish twin pairs, found anxiety symptoms predicted depressive symptoms, but not vice versa, and anxiety symptoms were more stable than depressive symptoms (Wetherell, Gatz, & Pedersen, 2001).

One longitudinal prospective study with adults suggests the sex difference in rates of MDD in adulthood (i.e., approximately twice as high in females) can largely be explained by anxiety (Breslau, Schultz, & Peterson, 1995). Breslau & colleagues demonstrated that prior anxiety disorders predicted subsequent MDD for both males and females, and history and number of previous anxiety disorders accounted for a large proportion of the sex differences in MDD, such that by controlling for prior anxiety, the association between gender and MDD is reduced by more than 50%. Thus, it may be that higher rates of anxiety in females compared to males may partially explain the higher rates of MDD in adult women compared to men.

Retrospective reports of adults with MDD indicate high rates of comorbid lifetime Axis I disorders, including SP and specific phobias (Alpert et al., 1994). In two-thirds of individuals reporting comorbid anxiety and MDD, the anxiety disorders temporally precede the onset of MDD. Additional retrospective data regarding number of episodes and severity and duration of episodes suggests that these may be impacted by comorbid anxiety (Wilhelm, Parker, Dewhurst-Savellis, & Asghari, 1999) in that those with two or more lifetime depressive episodes are more likely to have met lifetime criteria for an anxiety disorder and more likely to have met criteria for multiple anxiety disorders over their lifetime. Among adolescents and young adults endorsing a lifetime diagnosis of SP

with a comorbid depressive disorder, the anxiety disorder preceded the onset of the depressive disorder in 81.6% of cases (Wittchen et al., 1999). While these results provide interesting preliminary evidence regarding the comorbidity of anxiety and depression, the problems with relying on retrospective reports has been well articulated in the literature and prospective longitudinal studies are needed to replicate these results and increase confidence in their findings.

Among adolescents referred for treatment that meet criteria for MDD, these youth differ significantly from normal controls in that they have higher rates of OAD, agoraphobia, SP, SAD, obsessive-compulsive disorder (OCD), and multiple anxiety disorders (diagnoses determined using the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Epidemiologic Version [K-SADS-E; Orvaschel & Puig-Antich, 1987]; Biederman et al., 1995). In a study of German adolescents, 29.4% of those who met diagnostic criteria for SP also had a depressive disorder (Essau et al., 1999). Additionally, the presence of a childhood anxiety disorder or a childhood depressive disorder is associated with increased suicidal behavior (Rudd et al., 2004).

Prospective longitudinal studies of youth. The majority of studies demonstrate a temporal precedence for early anxiety disorders being predictive of later depressive disorders. Those who are comorbid for anxiety and depression are more likely than those with depression alone to have their depressive disorders persist over time or reoccur, to have more severe symptoms during a depressive episode, and to be at increased risk for suicidal ideation and suicide attempts (Stein et al., 2001). Data from the Great Smokey Mountains Study (GSMS), which followed youth ages 9 to 13 assessed annually to age 16 with the Child and Adolescent Psychiatric Assessment (CAPA; Angold et al., 1995)

interview, demonstrated strong concurrent comorbidity between anxiety and depression. Heterotypic continuity from anxiety to depression (odds ratio [OR] = 3.0), as well as from depression to anxiety (OR = 5.7) was observed and this finding was stronger for girls than boys (Costello et al., 2003). Similar results were observed with a New Zealand birth cohort longitudinal sample in that those with both depression and anxiety disorders at age 15 were at high risk to continue to have these disorders at age 18 (Feehan, McGee, & Williams, 1993). While these studies provide support for a relationship between anxiety and depression, they collapsed across anxiety disorders. Examining relationships between specific anxiety disorders and the depressive disorders is more informative.

In a longitudinal study of high school students (from The Oregon Adolescent Depression Project) assessed on average 13.8 months between time 1 and time 2, time 1 assessment utilized a version of the Schedule for Affective Disorders and Schizophrenia for School-Aged Children (K-SADS) that combined features of the epidemiological version (K-SADS-E) and the present episode version (K-SADS-P). At time 2, participants were interviewed with The Longitudinal Interval Follow-up Evaluation for adolescents (A-LIFE; Keller et al., 1987), which illicit information since the initial K-SADS interview. MDD was the most common comorbidity among those with anxiety disorders, and it was more common than comorbid anxiety disorders (Lewinsohn et al., 1997). A temporal precedence for anxiety and later MDD was demonstrated, and this was observed to be most true for the following anxiety disorders: specific phobia, SAD, OAD, and SP but it was less true for panic disorder and OCD.

Broad associations between OAD, GAD, panic, and MDD were observed in an epidemiological sample ages 9 to 18 followed at three time points across a 9-year time

span (Pine et al., 1998) and assessed with the Diagnostic Interview Schedule for Children (DISC; Costello, Edelbroch, & Costello, 1985). For example, the relationship between adolescent OAD and adult depression (OR = 2.92 from time 1 to adulthood, 2.23 from time 2 to adulthood) was similar in size to the relationship between adolescent and adult depression (OR = 4.36 from time 1 and 2.17 from time 2) and the relationship between adolescent depression and adult GAD was also strong (OR = 3.22 from time 1 and 10.87 from time 2). Overall, the result of this study indicated that while many adolescent disorders remediate by adulthood, disorders that were present in adulthood were generally preceded by an adolescent internalizing disorder.

Follow-up analyses from the Great Smokey Mountains Study found that, at age 19, childhood OAD, but not GAD, predicted later depression (OR = 6.4 for OAD, 0.6 for GAD; Bittner et al., 2007). Further analyses with this sample, when followed to age 21 revealed no evidence of homotypic prediction from adolescent depression to young adult depression as the effect of adolescent depression and adult depression was entirely accounted for by comorbidity of adolescent depression with GAD, ODD, and substance use (adjusted OR = 0.9; Copeland, Shanahan, Costello, & Angold, 2009). GAD and depression continued to cross-predict one another (OR = 7.4 for GAD to depression, OR = 5.5 for depression to GAD). Further examination revealed developmental gradations such that only childhood depression predicted young adult GAD (OR = 3.7) and only adolescent GAD predicted adult depression (OR = 7.4). Adolescent OAD more strongly predicted later depression in males (OR = 37.1) than a GAD diagnosis in adolescence did. These additional findings suggest that, while GAD and MDD are closely related constructs, distinctions do exist in their course as childhood GAD and MDD predict

different adult disorders and young adult GAD and MDD were predicted by different childhood and adolescent disorders.

In a prospective, longitudinal sample of German adolescents and young adults from the Early Developmental Stages of Psychopathology Study (EDSP; ages 14 to 24 at baseline) assessed with the *DSM-IV* Munich-Composite International Diagnostic Interview (M-CIDI; Wittchen, Lachner, Wunderlich, & Pfister, 1998), SP was found to be associated with increased risk for subsequent depression, independent of age of onset of SP (OR = 3.12; Beesdo et al., 2007). Socially anxious children with increased behavioral inhibition appeared to be at highest risk for the development of later depressive disorders (hazard ratio [HR] = 1.34). Additional data from the EDSP sample found that those comorbid with SP and depression at baseline had a worse prognosis than those without comorbidity at baseline (disorder persistence/recurrence OR = 2.3, attempted suicide OR = 6.1; Stein et al., 2001). Another interesting finding from this sample was that SP severity was only partially related to later depression and this partial relationship was explained by the presence of panic-like symptoms in SP youth (Beesdo et al., 2007). This partial failure to demonstrate a dose-response relationship between SP severity and risk for later depression suggests that intervention for panic attacks in SP youth may reduce the risk for later depression. Thus, these results indicate that targeted early intervention for SP youth with panic symptoms and high behavioral inhibition may be the most cost-effective way to reduce the risk for later depressive disorders in SP youngsters.

In a community subsample of 8 to 17 year old twins with SAD at time 1 from the Virginia Twin Study for Adolescent Behavioral Development, those with persistent SAD

at 18-month follow-up showed higher rates of new MDD when assessed with the CAPA. Elevated risk of new depressive disorders (OR = 3.32) was the only factor that distinguished persistent SAD from transient SAD when comorbidity was controlled for (Foley et al., 2004).

A longitudinal prospective study of youth whose mothers either had a history of depression or were lifetime free of psychiatric disorders demonstrated gender differences in risk for depression (Gallerani, Garber, & Martin, 2010). Youth were assessed at baseline (6^{th} grade) with The Schedule for Affective Disorders and Schizophrenia for School-aged Children – Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997) and then annually through 12^{th} grade with the A-LIFE. Among girls, rates of depression were high regardless of previous anxiety while, for boys, subthreshold anxiety predicted later subthreshold depression at a rate of 1.5 times that observed for boys without a history of anxiety. This highlights the importance of examining gender differences in studies of anxiety and depression comorbidity.

Among young girls in the Pittsburg Girls Study (ages 5 to 8 at initial study contact, followed to ages 11 to 13; Keenan, Feng, Hipwell, & Klostermann, 2009), symptoms of GAD and SP at ages 7, 8, and 10, and symptoms of SAD at age 9 only, assessed by parent report using the Screen for Child Anxiety and Related Emotional Disorders (SCARED; Birmaher et al., 1997), predicted later depressive disorders (assessed via the Child Symptom Inventory; CSI; Gadow & Sprafkin, 1994). Depressive symptoms, while predictive of later depressive disorders, were not predictive of anxiety. A significant limitation of this study; however, is the reliance on parent-report forms and

the absence of a diagnostic interview. Depressive disorder diagnoses were generated by applying symptom threshold criteria to data obtained from the CSI.

Not all studies have supported the temporal relationship between anxiety and later depressive disorders. Results from a prospective longitudinal cohort study of New Zealand youth followed for anxiety and depression from ages 11 to 32 assessed with the DISC indicates that the relationship between GAD and MDD may not follow the temporal precedence usually reported in studies (Moffitt et al., 2007). GAD began concurrently of before MDD in 37% of cases, but MDD began before of concurrently in 32% of cases. In those comorbid for anxiety and depression, GAD onset occurred first for one third of participants, MDD onset occurred first in one third, and the disorders began concurrently for one third. There are several possibilities for why these results may vary from the frequent reports of anxiety preceding depression. One possibility is that the retrospective reports commonly used to support a temporal precedence are inaccurate. Another explanation may be that the relationship between GAD and MDD is different than between other anxiety disorders and depression, since many previous studies have lumped anxiety disorders together.

Comorbidity between Anxiety and Substance Misuse

Substance use disorders are highly comorbid with other Axis I disorders and, while the most commonly observed comorbidities are CD and ODD, some studies have demonstrated a link between anxiety and substance use disorders (Armstrong & Costello, 2002; Rohde et al., 1996).

Longitudinal studies of adults generally indicate a temporal precedence for anxiety disorders, with substance use disorders occurring subsequently (Merikangas et

al., 1998), though some studies in adults support a reciprocal causal relationship over time, with anxiety disorders leading to alcohol dependence and vice versa at comparable rates (Kushner, Sher, & Erickson, 1999). Primary anxiety disorder diagnoses have been shown to predict a more persistent course of alcohol dependence in adults (Kessler et al., 1996). Internalizing symptoms in general have been associated with increased negative consequence from substance use, regardless of use level (Stice, Barrera, & Chassin, 1998).

There is also some evidence that the presence of an anxiety disorder may actually reduce the association between externalizing disorders and substance use disorders (Hofmann, Richey, Kashdan, & McKnight, 2009). This was true for a number of anxiety disorder (when PTSD was included with other anxiety disorders) and the relationship between externalizing problems and alcohol abuse, though only PTSD significantly mediated the link between externalizing problems and alcohol dependence (OR = 9.2). Low prevalence rates of anxiety disorders in some samples of comorbidity and risk for substance use have also limited the ability to examine relationships between anxiety and later substance use (e.g., Clark et al., 1997). Similarly, in a sample of 11-17 year olds assessed at a single time point with the DISC, anxiety disorders did not increase risk for substance use disorder, though substance dependence was more likely to be associated with multiple comorbidities including anxiety (Roberts, Roberts, & Xing, 2007).

Among adolescents and young adults retrospectively endorsing a lifetime diagnosis of SP, 85.2% of those with comorbid substance use disorders reported that their anxiety preceded the onset of the substance use disorder (Wittchen et al., 1999), though the link between anxiety and substance use disorders is unclear. This is largely because

the majority of studies have been retrospective or cross-sectional in design (Cerda et al., 2005). Additionally, there has been an overreliance on dimensional, as opposed to diagnostic measures of anxiety. Finally, the retrospective reports of those who are actively using or withdrawing from substances may not be accurate.

Among adolescents presenting for inpatient psychiatric treatment and at community mental health centers, 33% met criteria for both anxiety and substance use disorders while among youth presenting at an inpatient substance abuse treatment facility, the prevalence of these comorbidities was much higher (67%; Deas-Nesmith, Brady, & Campbell, 1998). Alcohol and marijuana were the most frequently used substances while SP and PTSD were the most commonly reported anxiety disorders in these comorbid youth. In older adolescent girls, there is a trend for increased alcohol use to be associated with anxiety disorders (Rohde et al., 1996). In another cross-sectional study, frequency of alcohol, cigarette, and illicit substance use was associated with increased risk for an anxiety disorder, with the frequency of comorbid anxiety increasing with more frequent substance use (Kandel et al., 1997). Of adolescents who met diagnostic criteria for SP, 23.5% also had a substance use disorder and a large proportion met the criteria for alcohol abuse (Essau et al., 1999). While these results are interesting, they do not inform the temporal precedence of the disorders present. Additionally, as noted earlier, the assessment of anxiety disorders in individuals who may be actively withdrawing from substances is challenging, and it can be unclear if the anxiety symptoms reflect true anxiety disorders or rather are symptoms of withdrawal.

Prospective longitudinal studies of youth. The existing prospective, diagnostic studies have yielded mixed results. While limited, there is some evidence that substance

misuse may increase risk for later anxiety disorders. For example, cigarette smoking during adolescents may increase the risk for later anxiety disorders (Johnson et al., 2000). Heavy cigarette smoking in adolescents (mean age 16; heavy smoking defined as ≥ 20 cigarettes per day, interviewed with the DISC) has been associated with higher risk for the development of agoraphobia (OR = 6.79), GAD (OR = 5.53), and panic disorder (OR = 15.58) when controlling for a number of comorbidities and risk factors while, in the same sample, anxiety disorders in adolescents did not predict later cigarette smoking. One possible explanation for these findings is that cigarette smoking may contribute to the development of anxiety because of factors including impaired respiration and the posited anxiogenic effects of nicotine (Breslau & Klein, 1999; Dilsaver, 1987; West & Hajek, 1997). Interestingly, some research has demonstrated an association between impaired respiration and agoraphobia, GAD, and panic disorder but not other anxiety disorders (Biber & Alkin, 1999; Pine et al., 2000; Klein, 1994; Perna, Bertani, Arancio, Ronchi, & Bellodi, 1995).

In addition to evidence that early nicotine dependence may increase risk for later anxiety disorders, there is also evidence that early and frequent cannabis use is associated with increased symptoms of anxiety (Hayatbakhsh et al., 2007) such that those who used cannabis more frequently were more likely to show anxiety symptoms on the Youth Self-Report (YSR; Achenbach, 1991; OR = 3.4) and this outcome was more pronounced for earlier onset of cannabis use (OR = 2.5 for those who began using before age 15, OR = 1.8 for later onset). Symptoms of anxiety did not predict use of cannabis in this Australian birth cohort followed to age 21. These associations were independent of individual and family background factors (i.e., sex, family income, maternal marital

status and psychopathology, and other substance use), suggesting the relationship between cannabis use and anxiety was not the product of these underlying risk factors. It should be noted that a significant limitation of this study was that anxiety symptoms were self-reported at ages 14 and 21, and cannabis initiation was reported retrospectively at age 21. Thus it is possible that anxiety symptoms developed in the interim between assessment time points and could have preceded the onset of cannabis use. If these results could be replicated entirely prospectively with shorter intervals between assessments and more rigorous assessment procedures, several possible explanations have been proposed that may explain the association between early cannabis use and later anxiety including: use of cannabis may have impacts on neurotransmitters in a manner that produces anxiety symptoms, frequent cannabis use may have psychosocial consequences associated with mental health impairment, and early use of cannabis may predict other illicit drug use that increases risk for mental health problems (though this particular explanation was not supported by these results).

While the above two studies found a temporal relationship between nicotine and cannabis use and later anxiety, the majority of studies show the opposite temporal patter (i.e., anxiety disorders increase risk for later substance misuse) or no relationship between anxiety and substance misuse. Anxiety more generally in young girls (but not boys) has been shown to predict the later development of substance use disorders at ages 16 and 19 in the GSMS (Bittner et al., 2007; Costello et al., 2003). Many studies have largely lumped anxiety disorders into a single construct for the purpose of evaluating risk for later substance use. There is some evidence to suggest that variations in risk for later substance use occur among the various anxiety disorders of childhood. For example, SP

may serve as a unique risk factor for later substance use; SP in particular has been associated with increased risk for later nicotine dependence (OR = 2.53; Sonntag, Wittchen, Hofler, Kessler, & Stein, 2000). Social anxiety in adolescence was also shown to uniquely predict the later development of alcohol (OR = 4.5) and cannabis (OR = 6.5) dependence in adulthood (Buckner, et al., 2008). This relationship remained after controlling for other anxiety and mood disorders and no other anxiety or mood disorders predicted later alcohol or cannabis use in this longitudinal sample from the Oregon Adolescent Depression Project.

Another study by Zimmermann et al. (2003) from the EDSP study supports the specificity of SP in predicting later substance misuse. In this 4-year prospective longitudinal study of adolescents and young adults (ages 14 to 24 at baseline) in Munich assessed with the M-CIDI, SP at baseline was predictive of onset of regular alcohol use (OR = 1.4) and the persistence of alcohol dependence (OR = 2.0). Additionally, panic attacks also predicted alcohol use (OR = 4.2) and persistence of dependence (OR = 6.2), and panic disorder predicted the persistence of use/abuse (OR = 2.6). The baseline disorders were predictive whether or not they had remitted, suggesting participants were largely not using alcohol to manage anxiety symptoms. Other anxiety disorders were not predictive of the onset or course of alcohol use and alcohol disorders and these results held when controlling for other anxiety and mood disorders, illegal drug use disorders, antisocial behavior, and demographic variables.

Kaplow, Curran, Angold, and Costello (2001) observed that youth in the GSMS (assessed at ages 9, 11, and 13with the CAPA) with GAD were at higher risk for initiation of alcohol use (OR = 1.14), while youth with early SAD were at decreased risk

(OR = 0.71). These results held when controlling for comorbid depressive symptomology. The authors proposed several possible explanations for these findings: as GAD may involve worries about social competence and acceptance, youth with GAD may use alcohol to "fit in" with peers; youth with GAD may be more likely to use alcohol to alleviate their anxiety symptoms in social situations, perhaps because of worries of impression management that have been documented in individuals with GAD; youth with SAD may be more behaviorally inhibited than GAD youth, which may be associated with decreased engagement in risky behavior; and SAD youth may be less likely to participate in socialization with peers decreasing access to substances by virtue of the difficulty separating from caregivers that is a hallmark symptom of their disorder.

Another examination from the EDSP study in Munich (included a sample of adolescents ages 14 to 17 assessed with the M-CIDI) found associations between panic and SAD with cannabis use (panic OR = 5.2, SAD OR = 2.3) and cannabis use disorders (panic OR = 4.7, SAD OR = 2.9) but when examined in a predictive model, only panic predicted cannabis use (OR = 1.5) but not cannabis use disorder (Wittchen et al., 2007). Additionally, histories of GAD and specific phobias were not associated with cannabis use and cannabis use disorders cross-sectionally, but prospectively both GAD and phobias predicted cannabis use disorders (GAD OR = 3.9, phobias OR = 1.8) but not general cannabis use. Only the association between panic and cannabis use was consistently observed across time points. The relationship between panic and use but not disordered levels of use, may indicate that panic plays a role only in the initiation of use, but not maintenance. These studies lend support to the notion that early anxiety disorders may serve as gateway disorders for the development of later substance misuse, perhaps

because substance use may serve as a socially acceptable anxiety management strategy (e.g., Sonntag et al., 2000).

Early anxiety may also be a protective factor against nicotine use (Costello, Erkanli, Federman, & Angold, 1999). Using the GSMS sample, Costello and colleagues found that boys and girls with anxiety began smoking significantly later than nonanxious youth when controlling for other comorbidity. Perhaps the behavioral inhibition common to anxiety disorders may explain this delayed nicotine use, or it is possible that the anxious cognitions commonly observed in anxious youth are associated with increased influence of public pressures not to engage in smoking behavior. Note that, for this study, nicotine use was defined as smoking, on average, one or more cigarettes per day for 3 months. It may be that, while initial smoking initiation is delayed, nicotine dependence is more likely to develop at an earlier age. In another report from this sample (Kaplow et al., 2001), girls with anxiety disorders, but not boys, were more likely to be at increased risk for later substance use disorders.

Finally, several studies have failed to find a significant relationship between anxiety and substance misuse. In one such study, youth were followed longitudinally from ages 1 to 10 at intake into young adulthood (mean age = 22.05 years; Brook et al., 1998). For these youth, no early psychiatric disorders (assessed by a supplemented version of the Diagnostic Interview Schedule for Children Version 1 [DISC -1]; Costello, Edelbrock, Kalas, Kessler, & Klaric, 1982) predicted later substance use though earlier drug use predicted later depressive and disruptive disorders.

For boys in particular (7^{th} grade at initial study recruitment), higher levels of anxiety in early adolescence were shown to predict lower levels of alcohol use disorder

symptoms and alcohol dependence in young adulthood (relative risk ratio [RRR] = .674; Pardini, White, & Stouthamer-Loeber, 2007) though this study, from the Pittsburg Youth Study, utilized only parent, teacher, and child self reported anxiety symptoms, not diagnostic assessments of anxiety, and anxiety scores were calculated by selecting the highest rater (i.e., parent, teacher, or child) and summing anxiety items from that informant to create a score, a significant limitation.

Paternal substance use disorders was shown to increase risk for later disorders of negative affect (assessed via the K-SADS) in boys (mean age 11.4 at baseline followed up at mean ages 13.4 and 16.1) in one study using survival analysis, however; in this study early disorders of negative affect did not increase risk for later substance use disorders regardless of paternal substance use (Clark, Parker, & Lynch, 1999). Because these youth were only followed though a mean age of 16.1, it is possible that disorders of negative affect are initially protective, though with time may confer increased risk.

In a birth-cohort longitudinal study of 1000 New Zealand individuals assessed for substance use and anxiety at ages 16 to18 and again at ages 18 to 21 with the CIDI, those with anxiety disorders had odds of substance dependence between 1.3 and 3.9 times higher than those without anxiety disorders (Goodwin, Fergusson, & Horwood, 2004a). These associations were largely explained by covariate factors including childhood and family factors (i.e., exposure to family adversity, parental psychopathology, child abuse, personality factors, and childhood behavioral adjustment), prior substance dependence, comorbid depression, and deviant peer affiliations. When these factors were controlled for, anxiety disorders no longer increased risk for substance dependence. These results support a non-causal relationship between anxiety and substance dependence, suggesting

that the presence of a series of other factors may increase risk for both anxiety and substance dependence.

Comorbidity between Anxiety and Externalizing Disorders

Anxiety disorders have been shown to be highly comorbid with externalizing disorders, and comorbid rates of anxiety with ADHD and ODD/CD are approximately three times higher than would be expected by chance (Angold et al., 1999; Costello et al., 2005; Ollendick, Jarrett, Grills-Taquechel, Hovey, & Wolff, 2008). It has been suggested that anxiety disorders may increase risk for the development of externalizing disorders, possibly because anxiety may lower the threshold for expression of disruptive behavior disorders (Bubier & Drabick, 2009). Alternatively, disruptive behavior disorders may confer risk for anxiety disorders (e.g., Nock, Kazdin, Hiripi, & Kessler, 2007). Previous research has demonstrated support for both explanations.

Among 8 to 17 year old twin children participating in the Virginia Twin Study for Adolescent Behavioral Development, of those with persistent SAD, 15% had also developed ODD at their follow-up assessment (follow-up interval ranged from 7 to 55 months; Foley et al., 2004). This was a nonsignificant effect for boys, though girls with persistent SAD had significantly higher levels of ODD symptoms at follow-up. In a clinical sample of anxious youth aged 5 to 18 assessed with the K-SADS-P, 7.1% of youth developed ODD or CD at 12-month follow-up (Last, Perrin, Hersen, & Kazdin, 1996). Data from the GSMS found that youth with GAD had significantly higher rates of CD at follow-up (OR = 3.6; Bittner et al., 2007).

In contrast to the above studies which found increased risk for externalizing disorders among youth with anxiety, several studies have suggested disruptive behavior

disorders confer greater risk for later anxiety disorders. In a sample of preschool aged children 2 to 5 years old followed for 48 to 72 months, those with ODD (assessed with the Diagnostic Interview for Children and Adolescents [DICA]; Reich, Wiener, & Herjanic, 1990 administered to parents and children over 7) at their initial assessment were at increased risk for developing a later anxiety disorder (maximum marginal likelihood [MML] estimate = 2.01; Lavigne et al., 2001). Among boys aged 7 to 12 followed annually to age 18 with the DISC, ODD and ADHD were predictive of later OAD (incidence rate ratio [IRR] = 1.05 for ODD, IRR = 1.03 for ADHD) and OAD in childhood was not related to increased risk for later ODD (Burke, Loeber, Lahey, & Rathouz, 2005). In another sample of boys (aged 4.5 to 5 years assessed with the DISC) with ODD, at 2-year follow-up 7.6% of those diagnosed with either ODD, ADHD, or ODD and ADHD at intake had a comorbid anxiety disorder (Speltz, McClellan, DeKlyen, & Jones, 1999). Data from the GSMS sample demonstrated that adolescent (aged 13-16) ODD predicted adult (aged 19 to 21) GAD for males and females (OR = 9.2) and panic disorder only in males (OR = 5.3; Copeland et al., 2009). Finally, elevated levels of parent-reported externalizing problems on the Child Behavior Checklist (CBCL; Achenbach & Edelbrock, 1991) among 4 to 16 year olds at intake were related to significantly increased rates of anxiety disorders (HR = 1.8) on the CIDI at 14-year follow-up in a Dutch epidemiological sample (Roza, Hofstra, van der Ende, & Verhulst, 2003).

Finally, several studies have failed to find associations between anxiety disorders and disruptive behavior disorders. Among an epidemiological sample of high school students assessed with the K-SADS, the disruptive behavior disorders (including ADHD,

ODD, and CD) were not associated with anxiety disorders when aggregated (Lewinsohn et al., 1997), though the relatively short follow-up period (mean interval 13.8 months, SD = 2.3) and older age of participants at first assessment (9^{th} through 12^{th} grades) may have limited the ability to find associations. Similarly, Mannuzza and colleagues (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998) found that boys with ADHD (determined by parent and teacher report and clinical judgment; mean age 7.3 years) were not at increased risk for anxiety disorders in adulthood (determined by a DSM-III semistructured interview).

Discussion of Child Anxiety Comorbidities

Depressive disorders demonstrate the most reliable comorbidity pattern with anxiety disorders and the majority of the evidence supports the notion that anxiety disorders often temporally precede the onset of depressive disorders. At the present time it remains unclear why this temporal pattern exists, though it seems reasonable to assume that anxiety disorders do not directly lead to depressive disorders as there lacks an underlying theoretical explanation for how this may occur. Additionally, while preliminary research suggests artifactual and methodological explanations do not fully account for the high comorbidity between anxiety and mood disorders, it will be important for future studies to continue to be mindful of these potential issues. Do anxiety and depressive disorders share an underlying predisposing risk factor (e.g., negative affectivity; Kendall & Watson, 1989)? Or do anxiety disorders indirectly increase risk for later depressive disorders? These questions remain unanswered and warrant further investigation. Additionally, for some, depressive disorders preceded the onset of anxiety disorders suggesting multiple trajectories from early anxiety and

depressive disorders to later comorbidity. Future epidemiological and longitudinal prospective studies would benefit from employing a developmental psychopathology perspective that includes assessment of risk and protective factors as well as contextual variables. By examining such constructs, it is likely that we will be able to better understand trajectories of early anxiety and depressive disorders which will be useful in informing intervention and prevention efforts. The notion that underlying negative affectivity may explain at least some of the comorbidity between anxiety and depressive disorders has received support in cross-sectional studies of clinical and non-clinical youth. Assessment of negative affectivity over time in large epidemiological samples may also inform the developmental trajectories of these disorders and explain anxiety's frequent temporal precedence to depressive disorders.

The results are much less clear for the relationships between anxiety disorders and comorbid substance misuse and externalizing disorders. Prospective investigations of the temporal relationships between these disorders and anxiety disorders have yielded mixed results, suggesting less reliable and consistent associations than for the depressive disorders and anxiety. Additionally, methodological limitations (e.g., short follow-up intervals), limit confidence in some of these studies. There is at least preliminary evidence to suggest that, for some, anxiety disorders increase risk for later substance misuse (though the opposite temporal pattern may be true in other cases). This suggests multiple potential explanations for patterns of comorbidity between anxiety and substance misuse. The concepts of equifinality, the notion that different processes can promote the same outcome, and multifinality, the notion that a single risk factor can lead to various outcomes depending on contextual factors, are important when considering the

varied outcomes of youth regarding anxiety and substance use trajectories (Cicchetti & Rogosch, 1996). To date, little research has examined the contextual and risk and protective factors that contribute to (or protect against) anxiety and comorbid substance use. A better understanding of these developmental psychopathology processes will offer important information in our understanding of why some with anxiety disorders appear to be at increased risk for substance misuse while others appear to be protected from it.

Regarding anxiety and externalizing disorders, the emerging evidence, while generally limited in length of follow-up, may indicate a shared underlying predisposing factor for both anxiety and disruptive behavior disorders (e.g., poor emotion regulation) as these disorders both tend to emerge at young ages in the available studies. Again, the utilization of a developmental psychopathology perspective that includes assessments of more environmental and contextual factors and assessment of risk and resiliency variables over time will be informative. Future studies utilizing sound methodological instruments and longer follow-up intervals will help elucidate some of these relationships. Additionally, future studies will benefit from parsing out the various anxiety disorders as some may lead to increased risk for substance misuse and externalizing disorders, while others may show the opposite temporal pattern or no reliable relationship.

Prevention of Comorbidities: The Role of Early Intervention

There is some evidence that youth with comorbid internalizing disorders and substance use disorders are less likely to use substances following treatment (Tomlinson et al., 2004). Additionally, while youth assessed prior to initiation of group CBT for depression who had anxiety comorbid with depression reported higher depression

measure scores at intake, they showed greater decreases in depression scores posttreatment than participants without comorbid anxiety at intake (Rohde, Clarke, Lewinsohn, Seeley, & Kaufman, 2001). Studies such as these support that early intervention for childhood disorders may have potential secondary benefits for comorbid disorders (Glantz, 2002). There is evidence that as much as 60% of adult substance dependence may be prevented by early treatment of disorders in youth (Kendall & Kessler, 2002; Kessler et al., 2001).

Kessler and Price (1993) note that prevention trials with diagnosed clinical samples at risk for developing secondary disorders have the potential to increase the social demand for early preventative intervention. From a financial and policy perspective, it may be easier to make the case for intervening upon individuals already suffering from a diagnosed disorder than to obtain support for more global prevention efforts. Additionally, as secondary comorbidities have been shown to exacerbate the primary disorders known to accompany the secondary disorders, intervention to prevent secondary disorders may have the added benefit of secondary prevention of exacerbated primary disorders. Intervention approaches for primary disorders aimed at prevention of secondary disorders are feasible (and likely more feasible than other methods of prevention, such as school-wide substance prevention programs) because empirically-supported treatment programs already exist for disorders known to increase risk for later comorbid disorders. CBT for child anxiety offers promise as one early intervention that may be beneficial in the prevention of later disorders. These treatments could be used as stand-alone prevention efforts or modified to include teaching adaptive coping strategies that apply for both the primary disorder being targeted in treatment as well as common

secondary comorbid conditions. For example, CBT for child anxiety, which has demonstrated efficacy, could be modified to include social skills training for youth with SP which may have the potential to decrease the likelihood of engagement in substance misuse in social situations to manage anxiety.

Cognitive-Behavioral Treatment of Childhood Anxiety

Cognitive and behavioral theories have influenced the development of efficacious treatments for child anxiety. Following from respondent conditioning theories (e.g., Watson & Rayner, 1920; Wolpe & Lazarus, 1966), CBT incorporates extinction, habituation, and counterconditioning to decrease anxious responses. Social learning theory (Bandura, 1977), which emphasizes the role children's self-efficacy plays in anxious responses and effective coping, spurred the inclusion of modeling, and cognitive models (e.g., Beck, 1985) prompted an increased emphasis on cognitive restructuring. Consistent with the tripartite view of anxiety (Barlow, 2000), the treatment of anxiety focuses on multiple areas depending on the child's unique vulnerabilities including developing coping responses, mastery, and a sense of self-control. For a more detailed discussion of the theoretical underpinnings of CBT for youth anxiety, see Gosch, Flannery-Schroeder, Mauro, and Compton (2006).

One version of CBT for anxious youth, the *Coping Cat* program, combines cognitive (e.g., identifying and challenging anxious self-talk, problem-solving) and behavioral (e.g., modeling, relaxation, exposure tasks, and contingency management) strategies to help children cope with anxiety. The *Coping Cat* program is a 16-session manualized treatment (Kendall & Hedtke, 2006a) developed for youth ages 7 to 13 that uses a client workbook (Kendall & Hedtke, 2006b). The manual guides treatment,

whereas the workbook contains client tasks for use in session and as homework, which correspond with the treatment.

The *Coping Cat* program is designed to teach youth to recognize signs of anxious arousal and to apply strategies to effectively cope with anxiety provoking situations. An overarching goal of the program is to help children learn to identify, regulate, and cope with their anxiety. The *Coping Cat* has six components: psychoeducation, relaxation/somatic management, cognitive coping/restructuring, problem-solving, exposure to anxiety provoking situations, and relapse prevention. Throughout, the therapist serves as a "coach," teaching and modeling skills and guiding the child to practice these skills. The first eight sessions are dedicated to anxiety management strategies that are presented to the child as a tool set. These strategies are taught to the child through collaborative discussion and fun activities and are presented in a sequence that allows for building skill upon skill. After learning the anxiety management skills, children proceed to the second phase of treatment where they practice applying the skills during anxiety-provoking situations. A graduated hierarchy of anxiety-provoking situations is developed based on the child's specific anxieties. The underlying concepts of the *Coping Cat* program overlap with other cognitive-behavioral interventions for youth anxiety.

Efficacy of CBT. Studies have examined the efficacy of CBT for child anxiety disorders and literature reviews support its utility (Kazdin & Weisz, 1998; Ollendick, King, & Chorpita, 2006; Silverman, Pina, & Viswesvaran, 2008). CBT has been found efficacious for specific problems and diagnosed disorders (e.g., Beidel et al., 2007). For example, CBT produced favorable gains when addressing specific problems such as

nighttime fears (e.g., Ollendick, Hagopian, & Huntzinger, 1991), school refusal (Chorpita, Albano, Heimberg, & Barlow, 1996; Kearney & Albano, 2000), and overanxiousness (Eisen & Silverman, 1993; Kane & Kendall, 1989). Literature reviews (e.g., Ollendick & King, 1998; Silverman, et al., 2008) have offered conclusions that, judged against the criteria proposed for an efficacious treatment (Chambless & Hollon, 1998), CBT is considered "probably efficacious" for diagnosed cases.

RCTs have demonstrated the efficacy of the *Coping Cat* program. A preliminary RCT included 47 children with an anxiety disorder. Results demonstrated that 64% of the treated youth no longer presented with their pretreatment principal anxiety disorder at posttreatment and gains were maintained at one-year (Kendall, 1994) and 3.35-year follow-up (Kendall & Southam-Gerow, 1996). A second RCT with 118 anxiety-disordered youth found that 55% of treated cases no longer met diagnostic criteria for their principal anxiety disorders at posttreatment (Kendall et al., 1997). Children were aged 9-13 years at the time of intake, referred from multiple community sources, and diagnosed with a DSM principal anxiety disorder (GAD/OAD, n = 55; SAD, n = 22; SP/AD, n = 17) at the time of intake based on structured clinical interviews conducted separately with both the parents and child. A total of 94 of the 118 children were treated in this sample using CBT. All children received CBT treatment, although the study design required that 34 of the children be assigned to a wait-list. At the end of the wait-list duration (8 weeks), if the cases continued to meet criteria for an anxiety disorder, children who had served on the waiting list were randomized to a therapist and given CBT. Eighty-six (91.5%) of the original 94 treated participants were followed-up 7.4 years after treatment. The majority of the successfully treated children maintained gains

at 7.4-year follow-up and those who were successfully treated showed reduced substance use compared to youth who were not successfully treated (Kendall et al., 2004). A recent reevaluation of the impact of CBT for child anxiety on the later development of substance use disorders in the same sample considered established predictors of substance use disorders (e.g., family substance abuse history) and treatment outcome (e.g., severity of internalizing psychopathology). Results demonstrated the robustness of the initial findings (Puleo, Conner, Benjamin, & Kendall, 2011). Although this 7.4 year follow-up provided information regarding the sequelae of childhood anxiety in mid-late adolescence, it is likely that most of the participants in that study were assessed prior to the age in which they would be at the highest risk for substance misuse and depressive symptoms as the mean age of the sample was 19.3 years. A third RCT compared individual CBT (ICBT), family CBT (FCBT), and an active family-based education/support/attention (FESA) condition (Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008) with 161 SAD, GAD, and/or SP children. Following treatment, 57%, 55%, and 37% no longer met criteria for their primary diagnosis in the ICBT, FCBT, and FESA conditions respectively. Significantly more children in the two CBT conditions no longer met diagnostic criteria compared to the FESA condition. Overall, these studies provide positive evaluations of the efficacy of CBT for anxiety-disordered youth. The relative and combined efficacy of CBT and sertraline was recently examined in a large, multisite RCT (Walkup et al., 2008) with 488 children who met diagnostic criteria for SAD, GAD, and/or SP and results suggest that the combined treatment, and each of the monotherapies, can be successful in reducing distressing anxiety in youth.

Beidel and Turner have developed and evaluated a treatment for SP in youth (i.e., Social Effectiveness Training for Children) with beneficial gains. RCTs evaluating the efficacy of CBT for specific phobias in children have also been conducted (see Silverman et al., 1999; Ost, 1998) and indicate phobias can be treated with exposure. Multiple research groups have made advances in the treatment of anxiety disorders in children (see also Barrett et al., 1996; Silverman, Pina, & Viswesvaran, 2008) though limited evidence exists with respect to long-term sequelae. Predictors of long-term gains, maintenance, and preventive effects have not been fully explored.

Long-term effects of CBT. Follow-up evaluations are a marker of methodological rigor and have been deemed as necessary in the demonstration of treatment efficacy (Chambless & Hollon, 1998; Nich & Carrol, 1997; Ollendick, 1986). To demonstrate long-term maintenance of gains, a treatment must have produced results at the follow-up assessment that demonstrate improvement from pretreatment and an absence of detrimental change since posttreatment or shorter-term follow-up. Although many studies support the immediate efficacy of psychosocial treatment, few studies address the long-term outcomes (see Kazdin, 1993). Several one-year follow-ups have been conducted (e.g., Kazdin, Siegel, & Bass, 1992; Kendall et al., 1997; Kolko, Loar, & Sturnick, 1990) and one 6-year follow-up was reported (Barrett, Duffy, Dadds, & Rapee, 2001). Longer term follow-ups are uncommon and many lack appropriate methodological rigor (e.g., do not follow a RCT, fail to use similar assessment measures at baseline and follow-up). One of the longest follow-up studies completed with anxious youth (7.4-year follow-up; Kendall et al., 2004), was described earlier. A recent review by Nevo & Manassis (2009) highlights the dearth of long-term outcome studies of anxious youth and

the lack of evidence for the role early anxiety treatment might play in healthy development into adulthood. More long-term studies are needed (Hirshfeld-Becker et al., 2008) and it will be important for these studies to include measures of substance use in their assessment batteries as these have been omitted from most outcome studies of anxiety treatment (Compton et al., 2002). Inclusion of outcome measures to assess possible secondary disorders over time will be important for gleaning if the early intervention reduces risk for onset of secondary disorders known to cluster with the primary disorders (Kessler & Price, 1993). If evidence is obtained to support the intervention's benefit in reducing risk for secondary disorders, this would provide important information about the causal pathways of common comorbidities and allow researchers to begin to answer some of the unanswered questions about anxiety comorbidity. Is the effect of early intervention on reduced risk for secondary disorders mediated by remission of the primary disorder targeted in treatment? Does the intervention modify common pathways to different disorders? Does duration of early treatment or type of treatment modify effects of primary disorder predicting secondary disorders? Data from long-term follow-up studies with strong methodologies will help us better understand the nature of comorbidity and improve prevention efforts.

Conclusions

Anxiety disorders in youth are common and associated with both short-term and long-term impairment. Comorbidity among the anxiety disorders is commonly observed, as well as comorbidity between anxiety disorders and depressive disorders, substance misuse, and externalizing disorders. A better understanding of why comorbidity occurs is important because comorbid disorders increase risk for a variety of negative sequelae and

are associated with poorer prognosis. At the present time, research supports a temporal precedence between anxiety disorders and later depressive disorders while the relationships between anxiety and substance use and anxiety and externalizing disorders are less clear.

Research is needed to identify the factors that contribute to various trajectories from childhood anxiety disorders to comorbid disorders later in development, and research in this area is likely to be informed by a developmental psychopathology perspective. Additionally, opportunities for prevention exist. Treating anxiety disorders early, before secondary comorbidities develop, may protect against the development of later associated disorders. CBT for child anxiety holds promise as a tool for preventing the development of secondary comorbidities in youth with anxiety. Longer-term follow-up studies that track youth treated for anxiety at an early age through the developmental periods when secondary comorbidities are more likely to develop are necessary for examining the preventative potential of CBT for youth anxiety. Last, it is important for future studies to use methodologically sound assessment instruments and include measures of comorbid conditions and contextual factors, particularly those that have been infrequently included in past studies (e.g., substance use outcome measures). If CBT for child anxiety is shown to be efficacious at allaying risk for later disorders, it will offer a cost-effective and likely feasible and acceptable method of preventing disorders that, if left to develop in adolescence and adulthood, are often difficult to treat and associated with deleterious outcomes.

CHAPTER 3

RESULTS

Results

Participants for the present study were a mean age of 27.23 (SD=3.54) and completed treatment for anxiety in childhood a mean 16.24 (SD=3.56) years prior to their participation in the present follow-up study. See Table 1.

Table 1
Demographic Data: Mean (SD) Age and Time since Treatment

	Age	Time Since Treatment
Sample	*M* (SD)	*M* (SD)
Overall Sample (N=66)	27.23 (3.54)	16.24 (3.56)
RCT-2 (n=54)	28.70 (1.55)	17.86 (.73)
RCT-3 (n=12)	20.58 (1.88)	8.96 (1.25)

Note. RCT-2=Subjects previously randomized to treatment in Kendall et al., 1997. RCT-3= Subjects previously randomized to treatment in Kendall et al., 2008.

Participants were 51.5% female and predominantly Caucasian (84.8%). The majority were employed (69.7%) and household incomes were variable. Over half of participants had never been married (56.1%). Pretreatment primary anxiety diagnoses were as follows: 56.1% had a primary diagnosis of GAD/OAD, 27.3% SP/AV, and 16.7% SAD. At posttreatment, 71.2% had their primary diagnosis as no longer primary in their diagnostic profile and 60.6% no longer met diagnostic criteria for their primary pretreatment diagnosis. See Table 2.

Table 2

Demographic Data: Frequency Data

Variable	Overall Sample (N=66)	RCT-2 (n=54)	RCT-3 (n=12)
	n (%)	n (%)	n (%)
Sex			
Male	32 (48.5%)	25 (46.3%)	7 (58.3%)
Female	34 (51.5%)	29 (53.7%)	5 (41.7%)
Race			
Caucasian	56 (84.8%)	45 (83.3%)	11 (91.7%)
African American	5 (7.6%)	4 (7.4%)	1 (8.3%)
Asian/Pacific Islander	2 (3.0%)	2 (3.7%)	0
Other/Biracial/Multiracial	3 (4.5%)	3 (5.6%)	0
Employment Status			
Employed/Self-Employed	46 (69.7%)	40 (74.1%)	6 (50.0%)
Unemployed	14 (21.2%)	11 (20.5%)	3 (25.0%)
Other/Student	5 (7.6)	2 (3.7%)	3 (25.5%)
Income			
$0	9 (13.6%)	5 (9.3%)	4 (33.3%)
$1-24,999	26 (39.4%)	19 (35.2%)	7 (58.3%)
$25,000-49,999	19 (28.8%)	13 (24.1%)	1 (8.3%)
$50,000-74,999	8 (12.1%)	8 (14.8%)	0
$75,000-99,999	2 (3.0%)	2 (3.7%)	0
≥$100,000	2 (3.0%)	2 (3.7%)	0
Marital Status			
Married/Partnered	26 (39.2%)	25 (46.3%)	1 (8.3%)
Never Married	37 (56.1%)	26 (48.1%)	11 (91.7%)
Separated/Divorced	3 (4.5%)	3 (5.6%)	0
Pretreatment Primary Diagnosis			
GAD/OAD	37 (56.1%)	32 (59.3%)	5 (41.7%)
SP/AV	18 (27.3%)	11 (20.4%)	7 (58.3%)
SAD	11 (16.7%)	11 (20.4%)	0
Posttreatment Response			
Primary No Longer Primary	47 (71.2%)	39 (72.2%)	8 (66.7%)
Primary No Longer Present	40 (60.6%)	32 (59.3%)	8 (66.7%)

Note. RCT-2=Subjects previously randomized to treatment in Kendall et al., 1997. RCT-3= Subjects previously randomized to treatment in Kendall et al., 2008. GAD/OAD=Generalized Anxiety Disorder/Overanxious Disorder of Childhood. SP/AV=Social Phobia/Avoidant Disorder. SAD=Separation Anxiety Disorder. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment.

Preliminary analyses compared long-term follow-up study participants with nonparticipants (those unable to be contacted or unwilling to participate in the present study) to examine differences on variables such as referral source, demographics (e.g., age, gender, ethnicity, family income), therapist factors (e.g., experience), pre- and post-treatment dependent measures (i.e., questionnaire measures and diagnostic status), and ratings of the child-therapist relationship. Participants and nonparticipants from the Kendall et al., 1997 sample were compared with one another. Participation was not significantly associated with referral source χ^2 (2) = 3.77, p = .15, gender χ^2 (1) = 3.73, p = .054, race χ^2 (4) = 4.43, p = .35, or family income category χ^2 (8) = 15.18, p = .056. One-way analysis of variance comparing participants versus nonparticipants on years of therapist experience was also non-significant F (1, 93) = 1.06, p = .31. A significant difference between participants and nonparticipants on child age was observed F (1, 95) = 4.08, $p < .05$ such that children who were younger at time of initial treatment participated in the present follow up at higher rates than older children. Chi-square analyses comparing participants versus nonparticipants by pretreatment primary diagnosis were non-significant for both parent and child reported diagnoses. One-way analyses of variance comparing participants versus nonparticipants on mean pretreatment Multidimensional Anxiety Scale for Children (MASC) score were also non-significant for both parent and child reported MASC total scores.

Additional analyses compared participants from Kendall et al., 1997 with participants from Kendall et al., 2008. Participation was not significantly associated with gender χ^2 (1) = .57, p = .45 or race χ^2 (4) = 1.80, p = .77. A significant difference between participants and nonparticipants on child age was observed F (1, 64) = 249.34, $p < .01$

such that participants from the Kendall et al., 1997 were significantly older than participants from Kendall et al., 2008. Chi-square analyses comparing participants from Kendall et al., 1997 with participants from Kendall et al., 2008 on pretreatment primary diagnosis indicated a significant differences between samples χ^2 (2) = 8.18, $p < .05$ (see Table 2).

Results from the diagnostic interview administered during the present follow-up study indicate that 43.9% of individuals met DSM-IV diagnostic criteria for one or more anxiety disorder with onset of age 18 or older, or with onset in childhood and duration persisting into adulthood. Anxiety disorders included are as follows: GAD, SP, SAD, Panic Disorder, Agoraphobia, Obsessive-Compulsive Disorder, and/or Specific Phobia. Additionally, 27.3% met criteria for one or more depressive disorder, including Major Depressive Disorder, a Major Depressive Episode, and/or Dysthymic Disorder. Finally, 42.4% met criteria for one or more substance use disorder including Alcohol Abuse, Alcohol Dependence, Drug Abuse, Drug Dependence, and/or Nicotine Dependence. See Table 3 for rates for specific disorders and breakdown by initial treatment sample. Rates are also reported by type of treatment response in Table 4. Treatment response is defined in two ways: (a) the absence of the principal anxiety disorder at posttreatment and (b) the principal anxiety disorder no longer principal at posttreatment.

Table 3
Adult Diagnostic Status

Diagnosis	Overall Sample (N=66)	RCT-2 (n=54)	RCT-3 (n=12)
	n (%)	n (%)	n (%)
Any Anxiety Disorder	29 (43.9%)	24 (44.4%)	5 (41.7%)
Generalized Anxiety Disorder	11 (16.7%)	9 (16.7%)	2 (16.7%)
Social Phobia	17 (25.8%)	14 (25.9%)	3 (25.0%)
Separation Anxiety Disorder	5 (7.6%)	5 (9.3%)	0
Panic Disorder	6 (9.1%)	5 (9.3%)	1 (.3%)
Agoraphobia	4 (6.1%)	4 (7.4%)	0
Obsessive-Compulsive Disorder	5 (7.6%)	5 (9.3%)	0
Specific Phobia	12 (18.2%)	10 (18.5%)	2 (16.7%)
Any Depressive Disorder	18 (27.3%)	16 (29.6%)	2 (16.7%)
Major Depressive Disorder	17 (25.8%)	15 (27.8%)	2 (16.7%)
Major Depressive Episode	18 (27.3%)	16 (29.6%)	2 (16.7%)
Dysthymic Disorder	2 (3.0%)	2 (3.7%)	0
Any Substance Use Disorder	28 (42.4%)	23 (42.6%)	5 (41.7%)
Alcohol Abuse	16 (24.2%)	14 (25.9%)	2 (16.7%)
Alcohol Dependence	6 (9.1%)	5 (9.3%)	1 (8.3%)
Drug Abuse	9 (13.6%)	7 (13.0%)	2 (16.7%)
Drug Dependence	2 (3.0%)	2 (3.7%)	0
Nicotine Dependence	18 (27.3%)	14 (25.9%)	4 (33.3%)

Note. RCT-2=Subjects previously randomized to treatment in Kendall et al., 1997. RCT-3= Subjects previously randomized to treatment in Kendall et al., 2008. Diagnoses based on CIDI interview. Diagnoses were met if subject endorsed the presence of a lifetime diagnosis with onset at ≥ age 18 or onset in childhood with persistence into adulthood.

Table 4
Adult Diagnostic Status by Posttreatment Response

Diagnosis	Primary No Longer Primary (N=47)	Primary Still Primary (N=19)	Primary No Longer Present (N=40)	Primary Still Present (N=26)
	n (%)	n (%)	n (%)	n (%)
Any Anxiety Disorder	19 (40.4%)	10 (52.6%)	15 (37.5%)	14 (53.8%)
Generalized Anxiety Disorder	8 (17.0)	3 (15.8%)	6 (15.0%)	5 (19.2%)
Social Phobia	10 (21.3%)	7 (36.8%)	7 (17.5%)	10 (38.5%)
Separation Anxiety Disorder	3 (6.4%)	2 (10.5%)	2 (5.0%)	3 (11.5%)
Panic Disorder	2 (4.3%)	4 (21.1%)	1 (2.5%)	5 (19.2%)
Agoraphobia	3 (6.4%)	1 (5.3%)	2 (5.0%)	2 (7.7%)
Obsessive-Compulsive Disorder	3 (6.4%)	2 (10.5%)	1 (2.5%)	4 (15.4%)
Specific Phobia	8 (17.0%)	4 (21.1%)	7 (17.5%)	5 (19.2%)
Any Depressive Disorder	12 (25.5%)	6 (31.6%)	10 (25%)	8 (30.8%)
Major Depressive Disorder	11 (23.4%)	6 (31.6%)	10 (25%)	7 (26.9%)
Major Depressive Episode	12 (25.5%)	6 (31.6%)	10 (25%)	8 (30.8%)
Dysthymic Disorder	1 (2.1%)	1 (5.3%)	1 (2.5%)	1 (3.8%)
Any Substance Use Disorder	19 (40.4%)	9 (47.4%)	16 (40.0%)	12(46.2%)
Alcohol Abuse	10 (21.3%)	6 (31.6%)	8 (20.0%)	8 (30.8%)
Alcohol Dependence	1 (2.1%)	5 (26.3%)	1 (2.5%)	5 (19.2%)
Drug Abuse	5 (10.6%)	4 (21.1%)	2 (5.0%)	7 (26.9%)
Drug Dependence	0	2 (10.5%)	0	2 (7.7%)
Nicotine Dependence	14 (29.8%)	4 (21.1%)	12 (30.0%)	6 (23.1%)

Note. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary Still Primary=Pretreatment primary diagnosis remained primary diagnosis at posttreatment. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Primary Still Present=Pretreatment primary diagnosis was no longer primary but remained in diagnostic profile at posttreatment.

Participants completed ratings of current mood, anxiety, and substance use symptomology as well as quality of life and overall disability/impairment. Overall mean ratings for each measure were within normal (i.e., nonclinical) limits (see Table 5).

Table 5

Self-Report Forms at 16-Year Follow-Up

Measure	*M* (SD)
Beck Depression Inventory-II (BDI-II)	10.62 (11.63)
Primary No Longer Primary	9.95 (11.43)
Primary Still Primary	12.16 (12.25)
Primary No Longer Present	9.00 (11.26)
Primary Still Present	12.93 (11.98)
Beck Anxiety Inventory (BAI)	9.62 (9.23)
Primary No Longer Primary	9.09 (9.08)
Primary Still Primary	10.84 (9.71)
Primary No Longer Present	8.43 (8.90)
Primary Still Present	11.31 (9.59)
Sheehan Disability Scale (SDS)	2.03 (1.12)
Primary No Longer Primary	1.93 (1.09)
Primary Still Primary	2.26 (1.20)
Primary No Longer Present	1.92 (1.09)
Primary Still Present	2.19 (1.17)
Quality of Life Inventory (QOLI)	24.72 (24.89)
Primary No Longer Primary	24.47 (23.68)
Primary Still Primary	25.33 (28.27)
Primary No Longer Present	26.83 (23.21)
Primary Still Present	21.68 (27.34)
Drug Use Screening Inventory- Revised (DUSI-R)	20.58 (13.99)
Primary No Longer Primary	18.90 (11.35)
Primary Still Primary	24.59 (18.68)
Primary No Longer Present	18.54 (11.28)
Primary Still Present	23.51 (16.99)

Note. BDI-II= Total Score; BAI=Total Score; SDS=Overall Disability Score; QOLI=Total Score; DUSI-R=Overall Problem Density Index. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary Still Primary=Pretreatment primary diagnosis remained primary diagnosis at posttreatment. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Primary Still Present=Pretreatment primary diagnosis was no longer primary but remained in diagnostic profile at posttreatment.

Primary Analyses: Effects of Treatment on Sequelae of Anxiety

To test the effect of previous treatment outcome on the sequelae of anxiety, logistic regressions examined whether outcome status (successful/unsuccessful as indicated by posttreatment diagnostic status) predicts adult anxiety, depressive disorder and subsequent substance use disorders (i.e., if participants meet diagnostic criteria for any anxiety disorder(s) on the CIDI interview with an onset age ≥ 18 years or onset in childhood persisting into adulthood, they were considered to have an adult anxiety disorder). Logistic regression serves as the primary test of this hypothesis with outcome status as the main effect of interest.

Successful treatment is defined in two ways[1]: (a) the principal anxiety disorder no longer primary at posttreatment and (b) the absence of the principal anxiety disorder at posttreatment. Separate analyses are conducted using each definition of successful and unsuccessful treatment, as consistent with the published report of the original treatment outcome study and the published 7.4 year follow up report.

When successful treatment is defined as the principal anxiety disorder no longer primary at posttreatment, treatment outcome did not significantly predict adult diagnostic status when diagnoses were collapsed into broad categories of anxiety, depressive, and substance use disorders. See Table 6.

[1] A third definition of treatment response, where successful treatment was defined as the absence of any anxiety disorder diagnoses at posttreatment, was evaluated. This definition did not predict the presence of any adult disorders.

Table 6

Logistic Regressions Examining Primary Diagnosis No Longer Primary at Posttreatment as a Predictor of Any Anxiety, Depressive, and Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Any Anxiety Disorder					
Constant	-.11 (.46)				
Primary No Longer Primary	.49 (.55)	.56	1.64	4.79	.37
Any Depressive Disorder					
Constant	.77 (.49)				
Primary No Longer Primary	.30 (.60)	.42	1.35	4.33	.62
Any Substance Use Disorder					
Constant	.11 (.46)				
Primary No Longer Primary	.28 (.55)	.45	1.33	3.88	.61

Additional logistic regression analyses examined treatment response as a predictor of individual DSM-IV diagnoses in adulthood. Unsuccessful treatment, defined as the principal anxiety disorder remaining primary at posttreatment, significantly predicted the presence of adult Panic Disorder, demonstrating an odds ratio of 6.00. All other logistic regressions predicting anxiety disorders were non-significant. See Table 7.

Table 7

Logistic Regressions Examining Primary Diagnosis No Longer Primary at Posttreatment as a Predictor of DSM-IV Anxiety Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*P*
		Lower	Odds Ratio	Upper	
Generalized Anxiety Disorder					
Constant	1.67 (.63)				
Primary No Longer Primary	-.09 (.74)	.22	.91	3.89	.90
Social Phobia					
Constant	.54 (.48)				
Primary No Longer Primary	.77 (.59)	.67	2.16	6.92	.20
Separation Anxiety Disorder					
Constant	2.14 (.75)				
Primary No Longer Primary	.55 (.96)	.27	1.73	11.25	.57
Panic Disorder[1]					
Constant	1.32 (.56)				
Primary No Longer Primary	1.79 (.92)	1.00	6.00	36.12	.05
Agoraphobia					
Constant	2.89 (1.03)				
Primary No Longer Primary	-.21 (1.19)	.08	.82	8.36	.86
Obsessive-Compulsive Disorder					
Constant	2.14 (.75)				
Primary No Longer Primary	.55 (.96)	.27	1.73	11.25	.57
Specific Phobia					
Constant	1.32 (.56)				
Primary No Longer Primary	.26 (.68)	.34	1.30	4.96	.70

[1] R^2=.06 (Cox & Snell), .13 (Nagelkerke). Model $\chi^2(1)=4.11$, $p < .05$

Additionally, logistic regressions examining the principal anxiety disorder remaining primary at posttreatment predicting individual depressive diagnoses yielded non-significant findings. See Table 8.

Table 8

Logistic Regressions Examining Primary Diagnosis No Longer Primary at Posttreatment as a Predictor of DSM-IV Depressive Disorders at 16-Year Follow-Up

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*P*
Major Depressive Disorder					
Constant	.77 (.49)				
Primary No Longer Primary	.41 (.60)	.46	1.51	4.91	.49
Major Depressive Episode					
Constant	.77 (.49)				
Primary No Longer Primary	.30 (.60)	.42	1.35	4.33	.62
Dysthymic Disorder					
Constant	2.89 (1.03)				
Primary No Longer Primary	.94 (1.44)	.15	2.56	43.08	.52

Unsuccessful treatment, defined as the principal anxiety disorder remaining primary at posttreatment, significantly predicted the presence of adult Alcohol Dependence, demonstrating an odds ratio of 16.43. All other logistic regressions predicting substance use disorders were non-significant. See Table 9.

Table 9

Logistic Regressions Examining Primary Diagnosis No Longer Primary at Posttreatment as a Predictor of DSM-IV Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*P*
		Lower	Odds Ratio	Upper	
Alcohol Abuse					
Constant	.77 (.49)				
Primary No Longer Primary	.54 (.61)	.52	1.71	5.63	.38
Alcohol Dependence[1]					
Constant	1.03 (.52)				
Primary No Longer Primary	2.80 (1.14)	1.77	16.43	152.60	.01
Drug Abuse					
Constant	1.32 (.56)				
Primary No Longer Primary	.81 (.74)	.53	2.24	9.46	.27
Drug Dependence					
Constant	2.14 (.75)				
Primary No Longer Primary	19.06 (5862.75)	.00	1.90[8]	-	1.00
Nicotine Dependence					
Constant	1.32 (.56)				
Primary No Longer Primary	-.46 (.65)	.18	.63	2.23	.47

[1] R^2=.12 (Cox & Snell), .27 (Nagelkerke). Model $\chi^2(1)=8.63$, $p < .01$

When successful treatment is defined as the principal anxiety disorder no longer present at posttreatment, treatment outcome did not significantly predict adult diagnostic status when diagnoses were collapsed into broad categories of anxiety, depressive, and substance use disorders. See Table 10.

Table 10

Logistic Regressions Examining Primary Diagnosis No Longer Present at Posttreatment as a Predictor of Any Anxiety, Depressive, and Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*P*
		Lower	Odds Ratio	Upper	
Any Anxiety Disorder					
Constant	-.15 (.39)				
Primary No Longer Primary	.67 (.51)	.71	1.94	5.30	.19
Any Depressive Disorder					
Constant	.81 (.43)				
Primary No Longer Primary	.29 (.56)	.45	1.33	4.00	.61
Any Substance Use Disorder					
Constant	.15 (.39)				
Primary No Longer Primary	.25 (.51)	.47	1.29	3.49	.62

Additional logistic regression analyses examined treatment response as a predictor of individual DSM-IV diagnoses in adulthood. Unsuccessful treatment, defined as the principal anxiety disorder remaining present at posttreatment, significantly predicted the presence of adult Panic Disorder, demonstrating an odds ratio of 9.29. All other logistic regressions predicting anxiety disorders were non-significant. See Table 11.

Table 11

Logistic Regressions Examining Primary Diagnosis No Longer Present at Posttreatment as a Predictor of DSM-IV Anxiety Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Generalized Anxiety Disorder					
Constant	1.44 (.50)				
Primary No Longer Primary	.30 (.67)	.37	1.35	4.98	.65
Social Phobia					
Constant	.47 (.40)				
Primary No Longer Primary	1.08 (.58)	.95	2.95	9.17	.06
Separation Anxiety Disorder					
Constant	2.04 (.61)				
Primary No Longer Primary	.91 (.95)	.39	2.48	15.96	.34
Panic Disorder[1]					
Constant	1.44 (.50)				
Primary No Longer Primary	2.23 (1.13)	1.01	9.29	84.78	.05
Agoraphobia					
Constant	2.49 (.74)				
Primary No Longer Primary	.46 (1.03)	.21	1.58	12.02	.66
Obsessive-Compulsive Disorder					
Constant	1.71 (.54)				
Primary No Longer Primary	1.96 (1.15)	.75	7.09	67.46	.09
Specific Phobia					
Constant	1.44 (.50)				
Primary No Longer Primary	.12 (.65)	.32	1.12	4.00	.86

[1] R^2=.08 (Cox & Snell), .17 (Nagelkerke). Model $\chi^2(1)=5.40$, $p < .05$

Additionally, logistic regressions examining the principal anxiety disorder remaining primary at posttreatment predicting individual depressive diagnoses yielded non-significant findings. See Table 12.

Table 12
Logistic Regressions Examining Primary Diagnosis No Longer Present at Posttreatment as a Predictor of DSM-IV Depressive Disorders at 16-Year Follow-Up

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Major Depressive Disorder					
Constant	1.00 (.44)				
Primary No Longer Primary	.10 (.57)	.36	1.11	3.40	.86
Major Depressive Episode					
Constant	.81 (.43)				
Primary No Longer Primary	.29 (.56)	.45	1.33	4.00	.61
Dysthymic Disorder					
Constant	3.22 (1.02)				
Primary No Longer Primary	.45 (1.44)	.09	1.56	26.09	.76

Unsuccessful treatment, defined as the principal anxiety disorder remaining primary at posttreatment, significantly predicted the presence of adult Alcohol Dependence, demonstrating an odds ratio of 9.29, as well as Drug Abuse, with an odds ratio of 7.00. All other logistic regressions predicting substance use disorders were non-significant. See Table 13.

Table 13

Logistic Regressions Examining Primary Diagnosis No Longer Present at Posttreatment as a Predictor of DSM-IV Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Alcohol Abuse					
Constant	.81 (.43)				
Primary No Longer Primary	.58 (.58)	.57	1.78	5.55	.32
Alcohol Dependence[1]					
Constant	1.44 (.50)				
Primary No Longer Primary	2.23 (1.13)	1.02	9.29	84.78	.05
Drug Abuse[2]					
Constant	1.00 (.44)				
Primary No Longer Primary	1.95 (.85)	1.32	7.00	37.01	.02
Drug Dependence					
Constant	2.49 (.74)				
Primary No Longer Primary	18.72 (6355.07)	.00	1.35[8]	-	1.00
Nicotine Dependence					
Constant	1.20 (.47)				
Primary No Longer Primary	-.36 (.58)	.23	.70	2.18	.54

[1] R^2=.08 (Cox & Snell), .17 (Nagelkerke). Model $\chi^2(1)=5.40$, $p < .05$.
[2] R^2=.09 (Cox & Snell), .17 (Nagelkerke). Model $\chi^2(1)=6.41$, $p < .05$.

Follow-up tests determine if the significant relationships between treatment response and adult disorders reported above hold when we include (a) primary diagnosis at time of additional treatment presentation or (b) additional treatment in the models as covariates. When successful treatment is defined as the principal anxiety disorder no longer primary at posttreatment, treatment outcome continued to significantly predict the presence of adult Panic Disorder when pretreatment primary diagnosis (odds ratio = 11.27) and additional treatment (odds ratio = 6.38) were included in the models as covariates (see Table 14).

Table 14

Follow-Up Logistic Regressions Examining Primary Diagnosis No Longer Primary at Posttreatment as a Predictor of DSM-IV Panic Disorder at 16-Year Follow-Up Controlling for Prettreatment Primary Diagnosis and Additional Treatment

	B (SE)	95% CI for Odds Ratio			*P*
		Lower	Odds Ratio	Upper	
Panic Disorder[1]					
Constant	1.32 (.56)				
Primary No Longer Primary	1.79 (.92)	1.00	6.00	36.12	.05
Controlling for Pre Diagnosis[2]					
Constant	2.42 (1.04)				
SP/AV					.11
SAD	-3.64 (1.72)	.00	.03	.76	.03
GAD	-1.91 (1.31)	.01	.15	1.92	.14
Primary No Longer Primary	3.19 (1.24)	2.14	11.27	273.06	.01
Controlling for Additional Tx[3]					
Constant	2.19 (1.16)				
PAT Ratings	-.39 (.43)	.29	.68	1.58	.39
Primary No Longer Primary	1.85 (.93)	1.03	6.38	39.42	.05

Note. Controlling for Pre Diagnosis=Controlling for pretreatment primary diagnosis. SP/AV=Social Phobia/Avoidant Disorder. SAD=Separation Anxiety Disorder. GAD/OAD=Generalized Anxiety Disorder/Overanxious Disorder of Childhood. Controlling for Additional Tx=Controlling for additional treatment between posttreatment and 16-year follow-up. PAT Ratings= Participant Additional Treatment ratings.

[1] R^2=.06 (Cox & Snell), .13 (Nagelkerke). Model $\chi^2(1)=4.11$, $p < .05$

[2] R^2=.14 (Cox & Snell), .30 (Nagelkerke). $\chi^2(3)=.16$, $p = .98$ (Hosmer & Lameshow), Model $\chi^2(3)=9.62$, $p < .05$

[3] R^2=.07 (Cox & Snell), .16 (Nagelkerke). $\chi^2(4)=5.40$, $p = .25$ (Hosmer & Lameshow), Model $\chi^2(2)=4.97$, $p = .08$

When successful treatment is defined as the principal anxiety disorder no longer primary at posttreatment, treatment outcome continued to significantly predict the presence of adult Alcohol Dependence when pretreatment primary diagnosis (odds ratio = 14.99) and additional treatment (odds ratio = 18.05) were included in the models as covariates (see Table 15).

Table 15
Follow-Up Logistic Regressions Examining Primary Diagnosis No Longer Primary at Posttreatment as a Predictor of DSM-IV Alcohol Dependence at 16-Year Follow-Up Controlling for Prettreatment Primary Diagnosis and Additional Treatment

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Alcohol Dependence[1]					
Constant	1.03 (.52)				
Primary No Longer Primary	2.80 (1.14)	1.77	16.43	152.60	.01
Controlling for Pre Diagnosis[2]					
Constant	.76 (.61)				
SP/AV					.46
SAD	-.61 (1.55)	.03	.55	11.28	.70
GAD	1.23 (1.25)	.29	3.42	39.82	.33
Primary No Longer Primary	2.71 (1.38)	1.00	14.99	224.50	.05
Controlling for Additional Tx[3]					
Constant	2.03 (1.20)				
PAT Ratings	-.45 (.46)	.26	.64	1.58	.33
Primary No Longer Primary	2.89 (1.16)	1.87	18.05	173.93	.01

Note. Controlling for Pre Diagnosis=Controlling for pretreatment primary diagnosis. SP/AV=Social Phobia/Avoidant Disorder. SAD=Separation Anxiety Disorder. GAD/OAD=Generalized Anxiety Disorder/Overanxious Disorder of Childhood. Controlling for Additional Tx=Controlling for additional treatment between posttreatment and 16-year follow-up. PAT Ratings= Participant Additional Treatment ratings.
[1] R^2=.12 (Cox & Snell), .27 (Nagelkerke). Model $\chi^2(1)=8.63$, $p < .01$
[2] R^2=.15 (Cox & Snell), .32 (Nagelkerke). $\chi^2(3)= 1.04$, $p = .79$ (Hosmer & Lameshow), Model $\chi^2(3)=10.40$, $p < .05$
[3] R^2=.14 (Cox & Snell), .30 (Nagelkerke). $\chi^2(4)= 5.33$, $p = .26$ (Hosmer & Lameshow), Model $\chi^2(2)=9.64$, $p < .01$

When successful treatment is defined as the principal anxiety disorder no longer present at posttreatment, treatment outcome continued to significantly predict the presence of adult Panic Disorder when pretreatment primary diagnosis (odds ratio = 23.93) and additional treatment (odds ratio = 9.47) were included in the models as covariates (see Table 16).

Table 16
Follow-Up Logistic Regressions Examining Primary Diagnosis No Longer Present at Posttreatment as a Predictor of DSM-IV Panic Disorder at 16-Year Follow-Up Controlling for Prettreatment Primary Diagnosis and Additional Treatment

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Panic Disorder[1]					
Constant	1.44 (.50)				
Primary No Longer Present	2.23 (1.13)	1.01	9.29	84.78	.05
Controlling for Pre Diagnosis[2]					
Constant	2.42 (1.04)				
SP/AV					.15
SAD	-3.09 (1.61)	.00	.05	1.07	.06
GAD	-1.19 (1.23)	.03	.31	3.43	.34
Primary No Longer Present	3.18 (1.36)	1.67	23.93	342.52	.02
Controlling for Additional Tx[3]					
Constant	2.22 (1.14)				
PAT Ratings	-.35 (.42)	.31	.71	1.62	.41
Primary No Longer Present	2.25 (1.13)	1.03	9.47	87.34	.05

Note. Controlling for Pre Diagnosis=Controlling for pretreatment primary diagnosis. SP/AV=Social Phobia/Avoidant Disorder. SAD=Separation Anxiety Disorder. GAD/OAD=Generalized Anxiety Disorder/Overanxious Disorder of Childhood. Controlling for Additional Tx=Controlling for additional treatment between posttreatment and 16-year follow-up. PAT Ratings= Participant Additional Treatment ratings.
[1] R^2=.06 (Cox & Snell), .13 (Nagelkerke). Model $\chi^2(1)=4.11$, $p < .05$
[2] R^2=.14 (Cox & Snell), .30 (Nagelkerke). $\chi^2(3)= .77$, $p = .94$ (Hosmer & Lameshow), Model $\chi^2(3)=9.73$, $p < .05$
[3] R^2=.09 (Cox & Snell), .19 (Nagelkerke). $\chi^2(4)= 4.59$, $p = .47$ (Hosmer & Lameshow), Model $\chi^2(2)=6.11$, $p < .05$

When successful treatment is defined as the principal anxiety disorder no longer present at posttreatment, treatment outcome no longer significantly predict the presence of adult Alcohol Dependence when pretreatment primary diagnosis was included in the model. Treatment outcome did continue to predict Alcohol Dependence when additional treatment (odds ratio = 9.47) was included in the model as a covariate (see Table 17).

Table 17

Follow-Up Logistic Regressions Examining Primary Diagnosis No Longer Present at Posttreatment as a Predictor of DSM-IV Alcohol Dependence at 16-Year Follow-Up Controlling for Prettreatment Primary Diagnosis and Additional Treatment

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Alcohol Dependence[1]					
Constant	1.44 (.50)				
Primary No Longer Present	2.23 (1.13)	1.02	9.29	84.78	.05
Controlling for Pre Diagnosis[2]					
Constant	.82 (.61)				
SP/AV					.28
SAD	.13 (1.35)	.08	1.14	15.87	.93
GAD/OAD	1.86 (1.20)	.61	6.39	66.57	.12
Primary No Longer Present	2.02 (1.24)	.67	7.52	84.53	.10
Controlling for Additional Tx[3]					
Constant	2.22 (1.14)				
PAT Ratings	-.35 (.42)	.31	.71	1.62	.41
Primary No Longer Present	2.25 (1.14)	1.03	9.47	87.34	.05

Note. Controlling for Pre Diagnosis=Controlling for pretreatment primary diagnosis. SP/AV=Social Phobia/Avoidant Disorder. SAD=Separation Anxiety Disorder. GAD/OAD=Generalized Anxiety Disorder/Overanxious Disorder of Childhood. Controlling for Additional Tx=Controlling for additional treatment between posttreatment and 16-year follow-up. PAT Ratings= Participant Additional Treatment ratings.

[1] R^2=.12 (Cox & Snell), .27 (Nagelkerke). Model $\chi^2(1)=8.63$, $p < .01$

[2] R^2=.12 (Cox & Snell), .27 (Nagelkerke). $\chi^2(3)= 2.21$, $p = .70$ (Hosmer & Lameshow), Model $\chi^2(3)=8.66$, $p < .05$

[3] R^2=.09 (Cox & Snell), .19 (Nagelkerke). $\chi^2(4)= 7.40$, $p = .19$ (Hosmer & Lameshow), Model $\chi^2(2)=6.11$, $p< .05$

Finally, when successful treatment is defined as the principal anxiety disorder no longer present at posttreatment, treatment outcome continued to significantly predict the presence of adult Drug Abuse when pretreatment primary diagnosis (odds ratio = 6.46) and additional treatment (odds ratio = 7.05) were included in the models as covariates (see Table 18).

Table 18

Follow-Up Logistic Regressions Examining Primary Diagnosis No Longer Present at Posttreatment as a Predictor of DSM-IV Drug Abuse at 16-Year Follow-Up Controlling for Prettreatment Primary Diagnosis and Additional Treatment

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Drug Abuse[1]					
Constant	1.00 (.44)				
Primary No Longer Present	1.95 (.85)	1.32	7.00	37.01	.02
Controlling for Pre Diagnosis[2]					
Constant	.84 (.60)				
SP/AV					.93
SAD	.19 (1.30)	.10	1.21	15.33	.88
GAD/OAD	.34 (.83)	.27	1.39	7.07	.69
Primary No Longer Present	1.87 (.90)	1.12	6.46	37.34	.04
Controlling for Additional Tx[3]					
Constant	1.41 (.90)				
PAT Ratings	-.19 (.34)	.42	.83	1.63	.59
Primary No Longer Present	1.95 (.85)	1.33	7.05	37.44	.02

Note. Controlling for Pre Diagnosis=Controlling for pretreatment primary diagnosis. SP/AV=Social Phobia/Avoidant Disorder. SAD=Separation Anxiety Disorder. GAD/OAD=Generalized Anxiety Disorder/Overanxious Disorder of Childhood. Controlling for Additional Tx=Controlling for additional treatment between posttreatment and 16-year follow-up. PAT Ratings= Participant Additional Treatment ratings.

[1] R^2=.09 (Cox & Snell), .17 (Nagelkerke). Model $\chi^2(1)=6.41$, $p < .05$

[2] R^2=.10 (Cox & Snell), .17 (Nagelkerke). $\chi^2(3)= 1.83$, $p = .77$ (Hosmer & Lameshow), Model $\chi^2(3)=6.56$, $p = .08$

[3] R^2=.10 (Cox & Snell), .18 (Nagelkerke). $\chi^2(4)= 6.61$, $p = .25$ (Hosmer & Lameshow), Model $\chi^2(2)=6.71$, $p< .05$

One-way analyses of variance comparing responder status on mean self-report form scores were non-significant for the BDI-II, BAI, SDS, QOLI, and DUSI-R indicating that successful treatment response was not associated with ratings of adult state depression, state anxiety, disability/impairment, quality of life, or self-report ratings of substance misuse symptomology. This held for both definitions of treatment outcome status. See Table 19.

Table 19
ANOVAs Comparing Mean Self-Report Form Scores at 16-Year Follow-Up by Posttreatment Response

	Oneway Analysis of Variance		
	df	*F*	*P*
Primary No Longer Primary			
BDI-II	(1, 61)	.47	.50
BAI	(1, 61)	.47	.49
SDS	(1, 61)	1.16	.29
QOLI	(1, 59)	.02	.90
DUSI-R	(1, 59)	2.13	.15
Primary No Longer Present			
BDI-II	(1, 61)	1.49	.23
BAI	(1, 61)	1.76	.19
SDS	(1, 61)	.91	.35
QOLI	(1, 59)	.63	.43
DUSI-R	(1, 59)	1.89	.18

Note. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. BDI-II= Beck Depression Inventory-II Total Score; BAI=Beck Anxiety Inventory Total Score; SDS=Sheehan Disability Scale Overall Disability Score; QOLI=Quality of Life Inventory Total Score; DUSI-R=Drug Use Screening Inventory-Revised Overall Problem Density Index.

Effects of Posttreatment Diagnostic Status on Additional Treatment. Additional therapeutic services after initial study treatment are examined dichotomously (i.e., presence or absence of additional treatment) as well as continuously (PAT data). When additional treatment is defined as presence or absence of additional treatment, 93.9% (n = 62) of participants endorsed receiving at least some additional services. When additional services were coded by experienced diagnosticians, 4 (6.1%) of individuals received no additional services, 16 (24.2%) obtained some additional services, 20 (30.3%) obtained a moderate amount of additional services, 18 (27.3%) received a great deal of additional services, and 8 (12.1%) were rated as receiving services more often than not since initial treatment completion.

When additional services are examined dichotomously using logistic regression, neither posttreatment diagnostic status defined as the principal anxiety disorder no longer primary at posttreatment (B (SE) = .97 (1.04), Odds Ratio = 2.65, $p = .35$) nor the principal anxiety disorder no longer present at posttreatment significantly predicted additional services (B (SE) = 1.63 (1.18), Odds Ratio = 5.09, $p = .17$).

When additional services are examined continuously (PAT data) using linear regression, neither posttreatment diagnostic status defined as the principal anxiety disorder no longer primary at posttreatment ($\beta = -.03$, $p = .83$, $R^2 < .01$) nor the principal anxiety disorder no longer present at posttreatment significantly predicted additional services ($\beta = .02$, $p = .99$, $R^2 < .01$).

Secondary Analyses: Normative Comparisons

Successfully treated and unsuccessfully treated participants are compared to same-age community participants from the NCS-R (Kessler et al., 2005) on diagnostic status (disorder present or absent in past 12 months) for anxiety, mood, and substance use disorders. Logistic models are used to test these effects. Included in the model are three groups: successfully treated participants, unsuccessfully treated participants, and the comparison group (NCS-R; Kessler et al., 2005). Follow-up tests according to Dunnett's criterion are conducted comparing the treatment groups with the comparison group providing an overall effect is seen. Age is included as a stratification variable (covariate) to allow participants to be compared to same-aged peers in the NCS-R (Kessler et al., 2005) study[2]. See Table 20 for frequency of 12-month disorders in the present follow-up sample.

[2] Additional stratification variables were considered (e.g., gender, SES) however there was not consistent support in the child anxiety literature to suggest a theoretical reason for including such additional variables in the present models.

Table 20
Adult 12-Month Diagnostic Status by Posttreatment Response

Diagnosis	Primary No Longer Primary (N=47)	Primary Still Primary (N=19)	Primary No Longer Present (N=40)	Primary Still Present (N=26)
	n (%)	n (%)	n (%)	n (%)
Anxiety Disorders				
Generalized Anxiety Disorder	3 (6.38%)	3 (15.79%)	4 (10.00%)	2 (7.69%)
Social Phobia	6 (12.77%)	2 (10.53%)	4 (10.00%)	4 (15.38%)
Separation Anxiety Disorder	0	1 (5.26%)	1 (2.50%)	0
Panic Disorder	2 (4.26%)	4 (21.05%)	5 (12.5%)	1 (3.85%)
Agoraphobia	2 (4.26%)	1 (5.26%)	2 (5.00%)	1 (3.85%)
Specific Phobia	4 (8.51%)	3 (15.79%)	4 (10.00%)	3 (11.54%)
Depressive Disorders				
Major Depressive Disorder	3 (6.38%)	4 (21.05%)	4 (10.00%)	3 (11.54%)
Major Depressive Episode	4 (8.51%)	4 (21.05%)	5 (12.5%)	3 (11.54%)
Dysthymic Disorder	1 (2.12%)	1 (5.26%)	1 (2.50%)	1 (3.85%)
Substance Use Disorders				
Alcohol Abuse	3 (6.38%)	4 (21.05%)	5 (12.5%)	2 (7.69%)
Alcohol Dependence	1 (2.12%)	3 (15.79%)	3 (7.50%)	1 (3.85%)
Drug Abuse	1 (2.12%)	1 (5.26%)	1 (2.50%)	1 (3.85%)
Drug Dependence	0	1 (5.26%)	1 (2.50%)	0
Nicotine Dependence	7 (14.89%)	2 (10.53%)	2 (5.00%)	7 (26.92%)

Note. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary Still Primary=Pretreatment primary diagnosis remained primary diagnosis at posttreatment. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Primary Still Present=Pretreatment primary diagnosis was no longer primary but remained in diagnostic profile at posttreatment.

When successful treatment is defined as the principal anxiety disorder no longer primary at posttreatment, group status (successfully treated participants, unsuccessfully treated participants, and the comparison group) significantly predicted GAD and Panic Disorder. Follow-up tests indicate the unsuccessfully treated participants significantly differed from the normative comparison group (GAD $p < .05$; Panic Disorder $p < .01$) in that the unsuccessfully treated group had higher rates of GAD and Panic Disorder than the normative comparison group. Successfully treated participants did not significantly differ from the normative comparison group for either GAD or Panic Disorder. See Table 21.

Table 21

Logistic Regressions Comparing Successful Treatment Response (Defined as Primary No Longer Primary), Unsuccessful Treatment Response, and Same-Age Community Participants on 12-Month DSM-IV Anxiety Disorder Diagnoses Controlling for Age

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*P*
Generalized Anxiety Disorder[1]					
Constant	2.88 (.14)				
Age	.01 (.01)	1.00	1.01	1.01	.07
Normative Comparison Group					.09
Primary Still Primary	-1.35 (.63)	.08	.26	.90	.03
Primary No Longer Primary	-.34 (.60)	.22	.71	2.31	.57
Social Phobia					
Constant	1.76 (.11)				
Age	.02 (.00)	1.02	1.02	1.03	<.01
Normative Comparison Group					.69
Primary Still Primary	-.14 (.75)	.20	.87	3.79	.85
Primary No Longer Primary	-.37 (.44)	.29	.69	1.64	.40
Separation Anxiety Disorder					
Constant	2.01 (.23)				
Age	.05 (.01)	1.04	1.05	1.07	<.01
Normative Comparison Group					.87
Primary Still Primary	-.55 (1.03)	.08	.58	4.36	.59
Primary No Longer Primary	17.71 (5851.74)	.00	49236947.98		1.00
Panic Disorder[2]					
Constant	2.85 (.17)				
Age	.02 (.00)	1.01	1.02	1.02	<.01
Normative Comparison Group					<.01
Primary Still Primary	-1.96 (.57)	.05	.14	.43	<.01
Primary No Longer Primary	-.18 (.73)	.20	.84	3.48	.81
Agoraphobia					
Constant	3.66 (.23)				
Age	.01 (.01)	1.00	1.01	1.02	.02
Normative Comparison Group					.28
Primary Still Primary	-1.10 (1.03)	.04	.33	2.52	.29
Primary No Longer Primary	-.89 (.73)	.10	.41	1.73	.23
Specific Phobia					
Constant	1.77 (.10)				
Age	.01 (.00)	1.01	1.01	1.02	<.01
Normative Comparison Group					.70
Primary Still Primary	-.42 (.63)	.19	.66	2.26	.50
Primary No Longer Primary	.29 (.53)	.47	1.31	3.66	.61

Note. Normative Comparison Group=Participants from the National Comorbidity Survey-Replication study (NCS-R; Kessler et al., 2005). Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary Still Primary=Pretreatment primary diagnosis remained primary diagnosis at posttreatment.

[1]*Dunnetts t* comparing Normative Comparison Group and Primary Still Primary= $p < .05$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Primary= $p = .72$

[2]*Dunnetts t* comparing Normative Comparison Group and Primary Still Primary= $p < .01$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Primary= $p = .80$

Group status did not significantly predict any depressive disorders when successful treatment is defined as the principal anxiety disorder no longer primary at posttreatment. See Table 22.

Table 22
Logistic Regressions Comparing Successful Treatment Response (Defined as Primary No Longer Primary), Unsuccessful Treatment Response, and Same-Age Community Participants on 12-Month DSM-IV Depressive Disorder Diagnoses Controlling for Age

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Major Depressive Disorder					
Constant	1.82 (.11)				
Age	.02 (.00)	1.01	1.02	1.02	<.01
Normative Comparison Group					.19
Primary Still Primary	-.97 (.57)	.13	.38	1.15	.09
Primary No Longer Primary	.38 (.60)	.45	1.46	4.73	.53
Major Depressive Episode					
Constant	1.53 (.10)				
Age	.02 (.00)	1.02	1.02	1.02	<.01
Normative Comparison Group					.37
Primary Still Primary	-.73 (.57)	.16	.48	1.47	.20
Primary No Longer Primary	.31 (.53)	.49	1.37	3.83	.55
Dysthymic Disorder					
Constant	3.42 (.18)				
Age	.01 (.01)	1.00	1.01	1.01	.12
Normative Comparison Group					.77
Primary Still Primary	-.70 (1.03)	.07	.50	3.77	.50
Primary No Longer Primary	.24 (1.02)	.17	1.27	9.28	.81

Note. Normative Comparison Group=Participants from the National Comorbidity Survey-Replication study (Kessler et al., 2005). Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary Still Primary=Pretreatment primary diagnosis remained primary diagnosis at posttreatment.

When successful treatment is defined as the principal anxiety disorder no longer primary at posttreatment, group status significantly predicted Alcohol Abuse, Alcohol Dependence, and Nicotine Dependence. Follow-up tests indicate the unsuccessfully treated participants significantly differed from the normative comparison group (Alcohol Abuse $p < .01$; Alcohol Dependence $p < .01$) such that the unsuccessfully treated group had higher rates of Alcohol Abuse and Alcohol Dependence than the normative comparison group. Successfully treated participants did not significantly differ from the normative comparison group for either Alcohol Abuse or Alcohol Dependence. Regarding Nicotine Dependence, unsuccessfully treated participants did not significantly differ from the normative comparison group. Successfully treated participants significantly differed from the normative comparison group (Nicotine Dependence $p < .01$) and the successfully treated group had higher rates of Nicotine Dependence than the normative comparison group. See Table 23.

Table 23

Logistic Regressions Comparing Successful Treatment Response (Defined as Primary No Longer Primary), Unsuccessful Treatment Response, and Same-Age Community Participants on 12-Month DSM-IV Substance Use Disorder Diagnoses Controlling for Age

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Alcohol Abuse[1]					
Constant	1.46 (.20)				
Age	.06 (.01)	1.05	1.06	1.08	<.01
Normative Comparison Group					<.01
Primary Still Primary	-1.76 (.57)	.06	.17	.53	<.01
Primary No Longer Primary	-.44 (.60)	.20	.65	2.11	.47
Alcohol Dependence[2]					
Constant	2.59 (.27)				
Age	.05 (.01)	1.03	1.05	1.07	<.01
Normative Comparison Group					<.01
Primary Still Primary	-2.20 (.64)	.03	.11	.39	<.01
Primary No Longer Primary	-.08 (1.02)	.13	.92	6.77	.94
Drug Abuse					
Constant	1.68 (.30)				
Age	.08 (.01)	1.06	1.08	1.10	<.01
Normative Comparison Group					.72
Primary Still Primary	-.85 (1.04)	.06	.43	3.27	.41
Primary No Longer Primary	.02 (1.02)	.14	1.02	7.48	.99
Drug Dependence					
Constant	2. 85 (.49)				
Age	.08 (.02)	1.05	1.08	1.11	<.01
Normative Comparison Group					.19
Primary Still Primary	-1.92 (1.05)	.02	.15	1.14	.07
Primary No Longer Primary	16.32 (5840.98)	.00	12230532.57		1.00
Nicotine Dependence[3]					
Constant	2.61 (.16)				
Age	.02 (.00)	1.01	1.02	1.03	<.01
Normative Comparison Group					<.01
Primary Still Primary	-.94 (.75)	.09	.39	1.70	.21
Primary No Longer Primary	-1.35 (.42)	.11	.26	.59	<.01

Note. Normative Comparison Group=Participants from the National Comorbidity Survey-Replication study (Kessler et al., 2005). Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment. Primary Still Primary=Pretreatment primary diagnosis remained primary diagnosis at posttreatment.

[1]*Dunnetts t* comparing Normative Comparison Group and Primary Still Primary= $p < .01$
Dunnetts t comparing Normative Comparison Group and Primary No Longer Primary= $p = .12$

[2]*Dunnetts t* comparing Normative Comparison Group and Primary Still Primary= $p < .01$
Dunnetts t comparing Normative Comparison Group and Primary No Longer Primary= $p = .78$

[3]*Dunnetts t* comparing Normative Comparison Group and Primary Still Primary= $p = .16$
Dunnetts t comparing Normative Comparison Group and Primary No Longer Primary= $p < .01$

When successful treatment is defined as the principal anxiety disorder no longer present at posttreatment, group status (successfully treated participants, unsuccessfully treated participants, and the comparison group) significantly predicted GAD, Panic Disorder, and Agoraphobia. Follow-up tests indicate the successfully treated participants significantly differed from the normative comparison group (GAD $p = .01$; Panic Disorder $p < .01$, Agoraphobia $p < .05$) in that they demonstrated higher rates of these disorders than the normative comparison group. Unsuccessfully treated participants did not significantly differ from the normative comparison group for GAD, Panic Disorder, or Agoraphobia. See Table 24.

Table 24

Logistic Regressions Comparing Successful Treatment Response (Defined as Primary No Longer Present), Unsuccessful Treatment Response, and Same-Age Community Participants on 12-Month DSM-IV Anxiety Disorder Diagnoses Controlling for Age

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Generalized Anxiety Disorder[1]					
Constant	2.87 (.14)				
Age	.01 (.00)	1.00	1.01	1.01	.07
Normative Comparison Group					.06
Primary Still Present	-1.32 (.55)	.09	.27	.78	.02
Primary No Longer Present	-.08 (.73)	.22	.92	3.85	.91
Social Phobia					
Constant	1.76(.11)				
Age	.02 (.00)	1.02	1.02	1.03	<.01
Normative Comparison Group					.56
Primary Still Present	-.58 (.55)	.19	.56	1.63	.29
Primary No Longer Present	-.09 (.53)	.32	.91	2.58	.86
Separation Anxiety Disorder					
Constant	2.06 (.23)				
Age	.05 (.01)	1.04	1.05	1.077	<.01
Normative Comparison Group					.97
Primary Still Present	-.25 (1.03)	.10	.78	5.80	.81
Primary No Longer Present	17.72 (6342.85)	.00	49759573.98		1.00
Panic Disorder[2]					
Constant	2.85 (.17)				
Age	.02 (.00)	1.01	1.02	1.02	<.01
Normative Comparison Group					<.01
Primary Still Present	-1.85 (.50)	.06	.16	.42	<.01
Primary No Longer Present	.37 (1.02)	.20	1.45	10.65	.71
Agoraphobia[3]					
Constant	3.66 (.23)				
Age	.01 (.01)	1.00	1.01	1.02	.02
Normative Comparison Group					.12
Primary Still Present	-1.51 (.75)	.05	.22	.95	.04
Primary No Longer Present	-.34 (1.02)	.10	.72	5.27	.74
Specific Phobia					
Constant	1.77 (.10)				
Age	.01 (.00)	1.01	1.01	1.02	<.01
Normative Comparison Group					.61
Primary Still Present	-.40 (.55)	.23	.67	1.96	.47
Primary No Longer Present	.41 (.60)	.46	1.50	4.90	.50

Note. Normative Comparison Group=Participants from the National Comorbidity Survey-Replication study (NCS-R; Kessler et al., 2005). Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Primary Still Present=Pretreatment primary diagnosis was no longer primary but remained in diagnostic profile at posttreatment.

[1]*Dunnetts t* comparing Normative Comparison Group and Primary Still Present= $p = .97$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Present = $p = .01$

[2]*Dunnetts t* comparing Normative Comparison Group and Primary Still Present= $p = .99$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Present = $p < .01$

[3]*Dunnetts t* comparing Normative Comparison Group and Primary Still Present= $p = .84$

Dunnetts t comparing Normative Comparison Group and Primary No Longer Present = $p < .05$

Group status did not significantly predict any depressive disorders when successful treatment is defined as the principal anxiety disorder no longer present at posttreatment. See Table 25.

Table 25
Logistic Regressions Comparing Successful Treatment Response (Defined as Primary No Longer Present), Unsuccessful Treatment Response, and Same-Age Community Participants on 12-Month DSM-IV Depressive Disorder Diagnoses Controlling for Age

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Major Depressive Disorder					
Constant	1.82 (.11)				
Age	.02 (.00)	1.01	1.02	1.02	<.01
Normative Comparison Group					.52
Primary Still Present	-.60 (.55)	.19	.55	1.61	.28
Primary No Longer Present	.21 (.60)	.38	1.23	4.02	.73
Major Depressive Episode					
Constant	1.53 (.10)				
Age	.02 (.00)	1.02	1.02	1.02	<.01
Normative Comparison Group					.35
Primary Still Present	-.62 (.50)	.20	.54	1.43	.21
Primary No Longer Present	.45 (.60)	.48	1.58	5.13	.45
Dysthymic Disorder					
Constant	3.42 (.18)				
Age	.01 (.00)	1.00	1.01	1.01	.12
Normative Comparison Group					.93
Primary Still Present	-.37 (1.02)	.09	.69	5.14	.72
Primary No Longer Present	.08 (1.02)	.15	1.08	7.91	.94

Note. Normative Comparison Group=Participants from the National Comorbidity Survey-Replication study (Kessler et al., 2005). Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Primary Still Present=Pretreatment primary diagnosis was no longer primary but remained in diagnostic profile at posttreatment.

When successful treatment is defined as the principal anxiety disorder no longer present at posttreatment, group status significantly predicted Alcohol Abuse, Alcohol Dependence, and Nicotine Dependence. Follow-up tests indicate the successfully treated participants significantly differed from the normative comparison group (Alcohol Abuse $p < .01$; Alcohol Dependence $p < .01$) in that significantly treated participants had higher rates of Alcohol Abuse and Alcohol Dependence than the normative comparison group. Unsuccessfully treated participants did not significantly differ from the normative comparison group for either Alcohol Abuse or Alcohol Dependence. Regarding Nicotine Dependence, successfully treated participants did not significantly differ from the normative comparison group. Unsuccessfully treated participants significantly differed from the normative comparison group (Nicotine Dependence $p < .01$) such that they exhibited higher rates of Nicotine Dependence than the normative comparison group. See Table 26.

Table 26

Logistic Regressions Comparing Successful Treatment Response (Defined as Primary No Longer Present), Unsuccessful Treatment Response, and Same-Age Community Participants on 12-Month DSM-IV Substance Use Disorder Diagnoses Controlling for Age

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Alcohol Abuse[1]					
Constant	1.46 (.20)				
Age	.06 (.01)	1.05	1.06	1.08	<.01
Normative Comparison Group					<.01
Primary Still Present	-1.67 (.51)	.07	.19	.51	<.01
Primary No Longer Present	-.16 (.73)	.20	.85	3.56	.82
Alcohol Dependence[2]					
Constant	2.59 (.27)				
Age	.05 (.01)	1.03	1.05	1.07	<.01
Normative Comparison Group					.01
Primary Still Present	-1.86 (.63)	.05	.16	.53	<.01
Primary No Longer Present	-.24 (1.02)	.11	.79	5.82	.82
Drug Abuse					
Constant	1.68 (.30)				
Age	.08 (.01)	1.06	1.08	1.10	<.01
Normative Comparison Group					.86
Primary Still Present	-.56 (1.03)	.08	.57	4.27	.59
Primary No Longer Present	-.13 (1.02)	.12	.88	6.48	.90
Drug Dependence					
Constant	2.85 (.49)				
Age	.08 (.02)	1.05	1.08	1.11	<.01
Normative Comparison Group					.29
Primary Still Present	-1.63 (1.04)	.03	.20	1.49	.12
Primary No Longer Present	16.34 (6328.92)	.00	12429871.81		1.00
Nicotine Dependence[3]					
Constant	2.61 (.16)				
Age	.02 (.00)	1.01	1.02	1.03	<.01
Normative Comparison Group					<.01
Primary Still Present	-.60 (.74)	.13	.55	2.33	.41
Primary No Longer Present	-1.54 (.42)	.09	.21	.49	<.01

Note. Normative Comparison Group=Participants from the National Comorbidity Survey-Replication study (Kessler et al., 2005). Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Primary Still Present=Pretreatment primary diagnosis was no longer primary but remained in diagnostic profile at posttreatment.

[1]*Dunnetts t* comparing Normative Comparison Group and Primary Still Present= $p = .45$
Dunnetts t comparing Normative Comparison Group and Primary No Longer Present = $p < .01$

[2]*Dunnetts t* comparing Normative Comparison Group and Primary Still Present= $p = .67$
Dunnetts t comparing Normative Comparison Group and Primary No Longer Present = $p < .01$

[3]*Dunnetts t* comparing Normative Comparison Group and Primary Still Present= $p = < .01$
Dunnetts t comparing Normative Comparison Group and Primary No Longer Present = $p = .40$

Exploratory Analyses

Exploratory analyses examined relationships between specific childhood anxiety disorders and outcome at long-term follow-up.

Pretreatment primary diagnosis did not significantly predict adult diagnostic status when diagnoses were collapsed into broad categories of anxiety, depressive, and substance use disorders. See Table 27. Additional logistic regression analyses examined primary diagnosis as a predictor of individual DSM-IV diagnoses in adulthood. Logistic regressions examining pretreatment primary diagnosis predicting anxiety disorders were all non-significant (see Table 28) as were logistic regressions predicting individual depressive diagnoses (see Table 29). Pretreatment primary diagnosis significantly predicted the presence of adult Alcohol Dependence, demonstrating an odds ratio of 10.29, such that a primary diagnosis of GAD in childhood was a significant predictor of adult Alcohol Dependence. All other logistic regressions predicting substance use disorders were non-significant. See Table 30.

Table 27

Logistic Regressions Examining Primary Diagnosis at Pretreatment as a Predictor of Any Anxiety, Depressive, and Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Any Anxiety Disorder					
Constant	.45 (.48)				
SP/AV					.37
SAD	-1.01 (.79)	.08	.36	1.72	.20
GAD/OAD	-.07 (.59)	.30	.93	2.96	.91
Any Depressive Disorder					
Constant	.69 (.50)				
SP/AV					.78
SAD	.29 (.84)	.26	1.33	6.94	.73
GAD/OAD	.44 (.63)	.45	1.56	5.35	.48
Any Substance Use Disorder					
Constant	.00 (.47)				
SP/AV					
SAD	.98 (.83)	.53	2.67	13.43	.23
GAD/OAD	.27 (.58)	.42	1.31	4.06	.64

Note. SP/AV=Social Phobia/Avoidant Disorder. SAD=Separation Anxiety Disorder. GAD/OAD=Generalized Anxiety Disorder/Overanxious Disorder of Childhood.

Table 28

Logistic Regressions Examining Primary Diagnosis at Pretreatment as a Predictor of DSM-IV Anxiety Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Generalized Anxiety Disorder					
Constant	2.83 (1.03)				
SP/AV					.38
SAD	-1.33 (1.29)	.02	.27	3.33	.30
GAD/OAD	-1.55 (1.10)	.03	.21	1.86	.16
Social Phobia					
Constant	.69 (.50)				
SP/AV					.64
SAD	.81 (.93)	.37	2.25	13.87	.38
GAD/OAD	.44 (.63)	.45	1.56	5.35	.48
Separation Anxiety Disorder					
Constant	21.20 (973.57)				
SP/AV					.17
SAD	-20.22 (9473.57)	.00	.00	-	1.00
GAD/OAD	-18.34 (9473.57)	.00	.00	-	1.00
Panic Disorder[1]					
Constant	2.83 (1.03)				
SP/AV					.52
SAD	-1.33 (1.29)	.02	.27	3.33	.30
GAD/OAD	-.41 (1.19)	.06	.67	6.90	.73
Agoraphobia					
Constant	21.20 (9473.58)				
SP/AV					.10
SAD	-20.22 (9473.58)	.00	.00	-	1.00
GAD/OAD	-17.62 (9473.58)	.00	.00	-	1.00
Obsessive-Compulsive Disorder					
Constant	21.20 (9473.57)				
SP/AV					.65
SAD	-19.70 (9472.57)	.00	.00	-	1.00
GAD/OAD	18.78 (9473.57)	.00	.00	-	1.00
Specific Phobia					
Constant	1.61 (.63)				
SP/AV					.25
SAD	-1.05 (.89)	.06	2.00	.35	.24
GAD/OAD	.25 (.80)	.27	6.07	1.28	.76

Note. SP/AV=Social Phobia/Avoidant Disorder. SAD=Separation Anxiety Disorder. GAD/OAD=Generalized Anxiety Disorder/Overanxious Disorder of Childhood.

Table 29

Logistic Regressions Examining Primary Diagnosis at Pretreatment as a Predictor of DSM-IV Depressive Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Major Depressive Disorder					
Constant	.69 (.50)				
SP/AV					.67
SAD	.29 (.84)	.26	1.33	6.94	.73
GAD/OAD	.60 (.64)	.52	1.81	6.35	.35
Major Depressive Episode					
Constant	.69 (.50)				
SP/AV					.78
SAD	.29 (.84)	.26	1.33	6.94	.73
GAD/OAD	.44 (.63)	.45	1.56	5.35	.48
Dysthymic Disorder					
Constant	2.08 (.75)				
SP/AV					1.00
SAD	19.12 (12118.64)	.00	2.02[8]	-	1.00
GAD/OAD	19.12 (6607.68)	.00	2.02[8]	-	1.00

Note. SP/AV=Social Phobia/Avoidant Disorder. SAD=Separation Anxiety Disorder. GAD/OAD=Generalized Anxiety Disorder/Overanxious Disorder of Childhood.

Table 30

Logistic Regressions Examining Primary Diagnosis at Pretreatment as a Predictor of DSM-IV Substance Use Disorders at 16-Year Follow-Up

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Alcohol Abuse					
Constant	.69 (.50)				
SP/AV					.37
SAD	1.61 (1.16)	.51	5.00	48.75	.17
GAD/OAD	.44 (.63)	.45	1.56	5.35	.48
Alcohol Dependence[1]					
Constant	1.25 (.57)				
SP/AV					.12
SAD	1.05 (1.19)	.28	2.86	29.56	.38
GAD/OAD	2.33 (1.16)	1,06	10.29	100.22	.05
Drug Abuse					
Constant	1.25 (.57)				
SP/AV					.47
SAD	1.05 (1.19)	.28	2.86	29.56	.38
GAD/OAD	.86 (.78)	.52	2.36	10.78	.27
Drug Dependence					
Constant	2.83 (1.03)				
SP/AV					.87
SAD	18.37 (12118.64)	.00	95027933.20	-	1.00
GAD/OAD	.75 (1.45)	.13	2.12	35.93	.60
Nicotine Dependence					
Constant	.69 (.50)				
SP/AV					.78
SAD	.29 (.84)	.26	1.33	6.94	.73
GAD/OAD	.44 (.63)	.45	1.56	5.35	.48

Note. SP/AV=Social Phobia/Avoidant Disorder. SAD=Separation Anxiety Disorder. GAD/OAD=Generalized Anxiety Disorder/Overanxious Disorder of Childhood.

[1] R^2=.08 (Cox & Snell), .17 (Nagelkerke). Model $\chi^2(2)=5.27$, $p = .07$

Exploratory analyses also assess for relationships between severity at posttreatment and outcome at long-term follow-up. Do variations in the clinical severity rating (CSRs) of anxiety or self-reported symptoms at posttreatment predict outcome at long-term follow-up? CSRs of anxiety at posttreatment for subjects who participated in RCT-2 were based on the scale of that time (0 to 4 scale). Subjects who participated in the third RCT were based on a 0 to 8 scale. The PI reviewed participant posttreatment diagnostic reports for subjects in RCT-3 and assigned a 0 to 4 rating for consistency.

Posttreatment CSR score did not significantly predict adult diagnostic status when diagnoses were collapsed into broad categories of anxiety, depressive, and substance use disorders. See Table 31. Additional logistic regression analyses examined posttreatment CSR scores as a predictor of individual DSM-IV diagnoses in adulthood. Posttreatment CSR score significantly predicted the presence of adult Obsessive-Compulsive Disorder, such that higher CSRs predicted increased risk for the disorder, demonstrating an odds ratio of .48. Logistic regressions examining CSRs predicting other anxiety disorders were all non-significant (see Table 32) as were logistic regressions predicting individual depressive diagnoses (see Table 33). All logistic regressions predicting substance use disorders were also non-significant. See Table 34.

Table 31

Logistic Regressions Examining Clinician Severity Rating at Posttreatment as a Predictor of Any Anxiety, Depressive, and Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Any Anxiety Disorder					
Constant	.46 (.34)				
Clinician Severity Rating	-.14 (.18)	.61	.87	1.23	.42
Any Depressive Disorder					
Constant	.85 (.36)				
Clinician Severity Rating	.16 (.21)	.78	1.18	1.78	.44
Any Substance Use Disorder					
Constant	.26 (.33)				
Clinician Severity Rating	.02 (.18)	.72	1.02	1.44	.94

Note. Clinician Severity Rating=Clinician rating of interference of most interfering disorder remaining in diagnostic profile at posttreatment on the Anxiety Disorders Interview Schedule-Parent version.

Table 32
Logistic Regressions Examining Clinician Severity Rating at Posttreatment as a Predictor of DSM-IV Anxiety Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Generalized Anxiety Disorder					
Constant	1.92 (.48)				
Clinician Severity Rating	-.24 (.23)	.51	.79	1.24	.30
Social Phobia					
Constant	1.41 (.41)				
Clinician Severity Rating	-.28 (.20)	.52	.76	1.12	.16
Separation Anxiety Disorder					
Constant	2.75 (.68)				
Clinician Severity Rating	-.19 (.32)	.44	.83	1.54	.55
Panic Disorder					
Constant	2.91 (.71)				
Clinician Severity Rating	-.40 (.30)	.38	.67	1.20	.18
Agoraphobia					
Constant	2.74 (.69)				
Clinician Severity Rating	-.01 (.36)	.49	.99	2.02	.98
Obsessive-Compulsive Disorder[1]					
Constant	3.87 (1.03)				
Clinician Severity Rating	-.74 (.37)	.23	.48	.98	.05
Specific Phobia					
Constant	1.36 (.41)				
Clinician Severity Rating	.21 (.25)	.75	1.23	2.03	.41

Note. Clinician Severity Rating=Clinician rating of interference of most interfering disorder remaining in diagnostic profile at posttreatment on the Anxiety Disorders Interview Schedule-Parent version.
[1] R^2=.07 (Cox & Snell), .17 (Nagelkerke). $\chi^2(1)=3.12$, $p = .37$ (Hosmer & Lameshow). Model $\chi^2(1)=4.87$, $p < .05$

Table 33
Logistic Regressions Examining Clinician Severity Rating at Posttreatment as a Predictor of DSM-IV Depressive Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Major Depressive Disorder					
Constant	.89 (.37)				
Clinician Severity Rating	.21 (.22)	.80	1.23	1.88	.34
Major Depressive Episode					
Constant	.85 (.36)				
Clinician Severity Rating	.16 (.21)	.78	1.18	1.78	.44
Dysthymic Disorder					
Constant	3.31 (.90)				
Clinician Severity Rating	.13 (.54)	.39	1.13	3.27	.82

Note. Clinician Severity Rating=Clinician rating of interference of most interfering disorder remaining in diagnostic profile at posttreatment on the Anxiety Disorders Interview Schedule-Parent version.

Table 34
Logistic Regressions Examining Clinician Severity Rating at Posttreatment as a Predictor of DSM-IV Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Alcohol Abuse					
Constant	1.19 (.39)				
Clinician Severity Rating	-.05 (.20)	.64	.95	1.41	.79
Alcohol Dependence					
Constant	2.76 (.67)				
Clinician Severity Rating	-.31 (.29)	.41	.73	1.30	.28
Drug Abuse					
Constant	2.17 (.53)				
Clinician Severity Rating	-.24 (.25)	.49	.79	1.27	.32
Drug Dependence					
Constant	3.63 (1.03)				
Clinician Severity Rating	-.13 (.49)	.34	.88	2.29	.79
Nicotine Dependence					
Constant	.81 (.36)				
Clinician Severity Rating	.13 (.20)	.76	1.13	1.69	.54

Note. Clinician Severity Rating=Clinician rating of interference of most interfering disorder remaining in diagnostic profile at posttreatment on the Anxiety Disorders Interview Schedule-Parent version.

Exploratory analyses also assessed for relationships between comorbidity at posttreatment and outcome at long-term follow-up. The number of posttreatment comorbid diagnoses did not significantly predict adult diagnostic status when diagnoses were collapsed into broad categories of anxiety, depressive, and substance use disorders. See Table 35. Additional logistic regression analyses examined comorbidity as a predictor of individual DSM-IV diagnoses in adulthood. Posttreatment comorbidity did not significantly predict the presence of any individual anxiety disorders (see Table 36), depressive diagnoses (see Table 37), or substance use disorders (see Table 38).

Table 35

Logistic Regressions Examining Comorbidity at Posttreatment as a Predictor of Any Anxiety, Depressive, and Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Any Anxiety Disorder					
Constant	.13 (.42)				
Comorbidity	.07 (.20)	.72	1.08	1.60	.72
Any Depressive Disorder					
Constant	.59 (.46)				
Comorbidity	.33 (.25)	.85	1.39	2.28	.19
Any Substance Use Disorder					
Constant	.06 (.42)				
Comorbidity	.12 (.20)	.75	1.12	1.67	.57

Note. Comorbidity=Number of diagnoses at posttreatment.

Table 36

Logistic Regressions Examining Comorbidity at Posttreatment as a Predictor of DSM-IV Anxiety Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Generalized Anxiety Disorder					
Constant	1.18 (.52)				
Comorbidity	.25 (.28)	.74	1.29	2.25	.37
Social Phobia					
Constant	1.07 (.48)				
Comorbidity	-.03 (.23)	.62	.97	1.51	.90
Separation Anxiety Disorder					
Constant	2.16 (.73)				
Comorbidity	.20 (.40)	.56	1.22	2.65	.61
Panic Disorder					
Constant	2.07 (.69)				
Comorbidity	.13 (.35)	.57	1.13	2.27	.72
Agoraphobia					
Constant	2.52 (.83)				
Comorbidity	.12 (.43)	.49	1.13	2.61	.78
Obsessive-Compulsive Disorder[1]					
Constant	3.42 (.96)				
Comorbidity	-.48 (.36)	.31	.62	1.26	.19
Specific Phobia					
Constant	1.18 (.52)				
Comorbidity	.25 (.28)	.74	1.29	2.25	.37

Note. Comorbidity=Number of diagnoses at posttreatment.

Table 37

Logistic Regressions Examining Comorbidity at Posttreatment as a Predictor of DSM-IV Depressive Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Major Depressive Disorder					
Constant	.62 (.47)				
Comorbidity	.37 (.26)	.87	1.45	2.42	.16
Major Depressive Episode					
Constant	.59 (.46)				
Comorbidity	.33 (.25)	.85	1.39	2.28	.19
Dysthymic Disorder					
Constant	3.46 (1.41)				
Comorbidity	.52 (.98)	.25	1.68	11.47	.60

Note. Comorbidity=Number of diagnoses at posttreatment.

Table 38

Logistic Regressions Examining Comorbidity at Posttreatment as a Predictor of DSM-IV Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Alcohol Abuse					
Constant	1.12 (.48)				
Comorbidity	-.01 (.23)	.63	.99	1.55	.95
Alcohol Dependence					
Constant	2.07 (.69)				
Comorbidity	.13 (.35)	.57	1.13	2.27	.72
Drug Abuse					
Constant	2.24 (.65)				
Comorbidity	-.24 (.28)	.46	.79	1.37	.40
Drug Dependence					
Constant	2.74 (1.01)				
Comorbidity	.53 (.70)	.43	1.70	6.68	.45
Nicotine Dependence					
Constant	.73 (.46)				
Comorbidity	.18 (.24)	.76	1.20	1.90	.44

Note. Comorbidity=Number of diagnoses at posttreatment.

Exploratory analyses also assess for (a) age and (b) time since treatment relationships. No significant relationships between age at time of initial treatment and outcome at long-term follow-up were observed (see Tables 39 to 42). Similarly, no significant relationships between time since initial study treatment and diagnostic outcomes at the present follow-up were observed (see Tables 43 to 46).

Table 39
Logistic Regressions Examining Age at Pretreatment as a Predictor of Any Anxiety, Depressive, and Substance Use Disorders at 16-Year Follow-Up

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Any Anxiety Disorder					
Constant	.18 (.42)				
Age	.01 (.05)	.92	1.01	1.10	.86
Any Depressive Disorder					
Constant	.73 (.45)				
Age	.04 (.05)	.94	1.04	1.15	.48
Any Substance Use Disorder					
Constant	.52 (.43)				
Age	-.03 (.05)	.89	.97	1.07	.55

Table 40

Logistic Regressions Examining Age at Pretreatment as a Predictor of DSM-IV Anxiety Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Generalized Anxiety Disorder					
Constant	2.25 (.71)				
Age	-.08 (.07)	.80	.92	1.06	.26
Social Phobia					
Constant	1.45 (.53)				
Age	-.05 (.06)	.85	.95	1.06	.36
Separation Anxiety Disorder					
Constant	2.22 (.71)				
Age	.04 (.08)	.88	1.04	1.23	.63
Panic Disorder					
Constant	2.63 (.83)				
Age	-.04 (.09)	.81	.96	1.14	.63
Agoraphobia					
Constant	2.23 (.71)				
Age	.08 (.09)	.90	1.09	1.30	.39
Obsessive-Compulsive Disorder					
Constant	2.78 (.89)				
Age	-.04 (.09)	.81	.97	1.16	.70
Specific Phobia					
Constant	.92 (.47)				
Age	.09 (.06)	.98	1.09	1.23	.13

Table 41
Logistic Regressions Examining Age at Pretreatment as a Predictor of DSM-IV Depressive Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Major Depressive Disorder					
Constant	.75 (.45)				
Age	.04 (.05)	.94	1.04	1.16	.40
Major Depressive Episode					
Constant	.73 (.45)				
Age	.04 (.05)	.94	1.04	1.15	.48
Dysthymic Disorder					
Constant	2.92 (.96)				
Age	.09 (.13)	.85	1.09	1.42	.50

Table 42
Logistic Regressions Examining Age at Pretreatment as a Predictor of DSM-IV Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Alcohol Abuse					
Constant	1.26 (.50)				
Age	-.02 (.06)	.89	.98	1.10	.77
Alcohol Dependence					
Constant	2.83 (.90)				
Age	-.07 (.09)	.78	.94	1.12	.47
Drug Abuse					
Constant	2.12 (.67)				
Age	-.04 (.07)	.84	.97	1.11	.62
Drug Dependence					
Constant	7.46 (5.05)				
Age	-.39 (.43)	.30	.68	1.60	.37
Nicotine Dependence					
Constant	1.18 (.49)				
Age	-.03 (.05)	.88	.97	1.08	.62

Table 43

Logistic Regressions Examining Time since Treatment as a Predictor of Any Anxiety, Depressive, and Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Any Anxiety Disorder					
Constant	.40 (1.17)				
Time since Treatment	-.01 (.07)	.86	.99	1.14	.89
Any Depressive Disorder					
Constant	2.46 (1.57)				
Time since Treatment	-.09 (.09)	.76	.91	1.10	.33
Any Substance Use Disorder					
Constant	.27 (1.17)				
Time since Treatment	.00 (.07)	.87	1.00	1.15	.98

Table 44

Logistic Regressions Examining Time since Treatment as a Predictor of DSM-IV Anxiety Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Generalized Anxiety Disorder					
Constant	1.21 (1.47)				
Time since Treatment	.03 (.09)	.86	1.03	1.22	.78
Social Phobia					
Constant	.71 (1.28)				
Time since Treatment	.02 (.08)	.88	1.02	1.19	.78
Separation Anxiety Disorder					
Constant	8.64 (7.17)				
Time since Treatment	-.35 (.40)	.32	.70	1.54	.38
Panic Disorder					
Constant	2.68 (2.16)				
Time since Treatment	-.02 (.13)	.76	.98	1.26	.86
Agoraphobia					
Constant	15.60 (13.77)				
Time since Treatment	-.73 (.76)	.11	.48	2.14	.48
Obsessive-Compulsive Disorder					
Constant	6.64 (5.02)				
Time since Treatment	-.24 (.28)	.45	.79	1.37	.39
Specific Phobia					
Constant	2.10 (1.64)				
Time since Treatment	-.04 (.10)	.80	.96	1.17	.71

Table 45
Logistic Regressions Examining Time since Treatment as a Predictor of DSM-IV Depressive Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Major Depressive Disorder					
Constant	2.47 (1.59)				
Time since Treatment	-.09 (.09)	.77	.92	1.10	.36
Major Depressive Episode					
Constant	2.46 (1.57)				
Time since Treatment	-.09 (.09)	.76	.91	1.10	.33
Dysthymic Disorder					
Constant	54.53 (34.24)				
Time since Treatment	-2.79 (1.83)	.00	.06	2.22	.13

Table 46
Logistic Regressions Examining Time since Treatment as a Predictor of DSM-IV Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Alcohol Abuse					
Constant	1.60 (1.43)				
Time since Treatment	-.03 (.09)	.82	.97	1.15	.74
Alcohol Dependence					
Constant	3.46 (2.50)				
Time since Treatment	-.07 (.15)	.70	.93	1.24	.63
Drug Abuse					
Constant	1.53 (1.61)				
Time since Treatment	.02 (.10)	.84	1.02	1.24	.84
Drug Dependence					
Constant	18.46 (20.15)				
Time since Treatment	-.84 (1.10)	.05	.43	3.75	.45
Nicotine Dependence					
Constant	.44 (1.24)				
Time since Treatment	.03 (.08)	.89	1.03	1.20	.66

As the majority of participants received additional therapeutic treatments subsequent to study participation, analyses examine the role of this interim variable on a continuous scale (i.e., PAT Ratings). No significant relationships between receipt of additional therapeutic services and outcome at long-term follow-up were observed (see Tables 47 to 50).

Table 47
Logistic Regressions Examining Additional Treatment (PAT Ratings) as a Predictor of Any Anxiety, Depressive, and Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Any Anxiety Disorder					
Constant	.75 (.56)				
Additional Treatment	-.24 (.23)	.51	.79	1.24	.30
Any Depressive Disorder					
Constant	1.44 (.64)				
Additional Treatment	-.21 (.25)	.49	.81	1.34	.42
Any Substance Use Disorder					
Constant	.96 (.57)				
Additional Treatment	-.30 (.23)	.47	.74	1.17	.20

Note. Additional Treatment=Additional treatment between posttreatment and 16-year follow-up. PAT Ratings= Participant Additional Treatment ratings.

Table 48

Logistic Regressions Examining Additional Treatment (PAT Ratings) as a Predictor of DSM-IV Anxiety Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Generalized Anxiety Disorder					
Constant	3.07 (.93)				
Additional Treatment	-.61 (.33)	.28	.54	1.04	.07
Social Phobia					
Constant	1.26 (.63)				
Additional Treatment	-.09 (.26)	.55	.91	1.51	.72
Separation Anxiety Disorder					
Constant	3.01 (1.12)				
Additional Treatment	-.22 (.43)	.35	.80	1.85	.60
Panic Disorder					
Constant	3.05 (1.08)				
Additional Treatment	-.32 (1.08)	.33	.73	1.59	.42
Agoraphobia					
Constant	4.09 (1.47)				
Additional Treatment	-.55 (.51)	.21	.57	1.56	.28
Obsessive-Compulsive Disorder					
Constant	4.02 (1.37)				
Additional Treatment	-.62 (.47)	.22	.54	1.35	.19
Specific Phobia					
Constant	1.54 (.70)				
Additional Treatment	-.02 (.29)	.56	.99	1.74	.96

Note. Additional Treatment=Additional treatment between posttreatment and 16-year follow-up. PAT Ratings= Participant Additional Treatment ratings.

Table 49

Logistic Regressions Examining Additional Treatment (PAT Ratings) as a Predictor of DSM-IV Depressive Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Major Depressive Disorder					
Constant	1.56 (.66)				
Additional Treatment	-.23 (.26)	.48	.80	1.34	.39
Major Depressive Episode					
Constant	1.44 (.64)				
Additional Treatment	-.21 (.25)	.49	.81	1.34	.42
Dysthymic Disorder					
Constant	4.16 (1.81)				
Additional Treatment	-.30 (.67)	.20	.74	2.74	.65

Note. Additional Treatment=Additional treatment between posttreatment and 16-year follow-up. PAT Ratings= Participant Additional Treatment ratings.

Table 50

Logistic Regressions Examining Additional Treatment (PAT Ratings) as a Predictor of DSM-IV Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Alcohol Abuse					
Constant	2.40 (.76)				
Additional Treatment	-.55 (.29)	.33	.58	1.01	.06
Alcohol Dependence					
Constant	3.05 (1.08)				
Additional Treatment	-.32 (.40)	.33	.73	1.59	.42
Drug Abuse					
Constant	2.23 (.84)				
Additional Treatment	-.17 (.33)	.44	.84	1.60	.60
Drug Dependence					
Constant	5.54 (2.38)				
Additional Treatment	-.80 (.76)	.10	.45	2.01	.29
Nicotine Dependence					
Constant	1.90 (.69)				
Additional Treatment	-.41 (.26)	.40	.67	1.12	.12

Note. Additional Treatment=Additional treatment between posttreatment and 16-year follow-up. PAT Ratings= Participant Additional Treatment ratings.

Given the finding that some participants characterized as treatment responders continued to meet criteria for an anxiety disorder at posttreatment, exploratory analyses included comparisons of the two types of treatment response (i.e., Primary No Longer Primary and Primary No Longer Present) and long term outcome. Type of treatment response did not significantly predict adult diagnostic status when diagnoses were collapsed into broad categories of anxiety, depressive, and substance use disorders. See Table 51. Additional logistic regression analyses examined type of treatment response as a predictor of individual DSM-IV diagnoses in adulthood. Primary No Longer Primary significantly predicted the presence of adult Obsessive-Compulsive Disorder, such that individuals whose primary diagnosis remained primary at posttreatment were significantly more likely than those whose primary disorder remained present to exhibit the disorder in adulthood, demonstrating an odds ratio of .06. Logistic regressions examining type of treatment response predicting other anxiety disorders were all non-significant (see Table 52) as were logistic regressions predicting individual depressive diagnoses (see Table 53). All logistic regressions predicting substance use disorders were also non-significant with the exception of Drug Abuse: Primary No Longer Primary significantly predicted the presence of adult Drug Abuse, such that individuals whose primary diagnosis remained primary at posttreatment were significantly more likely than those whose primary disorder remained present to exhibit Drug Abuse in adulthood, demonstrating an odds ratio of .07. See Table 54.

Table 51

Logistic Regressions Examining Responder Status Type as a Predictor of Any Anxiety, Depressive, and Substance Use Disorders at 16-Year Follow-Up

	B (SE)	95% CI for Odds Ratio			*p*
		Lower	Odds Ratio	Upper	
Any Anxiety Disorder					
Constant	.51 (.34)				
Primary No Longer Present					.42
Nonresponder	-.62 (.56)	.18	.54	1.63	.27
Primary No Longer Primary	-.80 (.83)	.09	.45	2.29	.34
Any Depressive Disorder					
Constant	1.10 (.37)				
Primary No Longer Present					.87
Nonresponder	-.36 (.61)	.22	.72	2.41	.60
Primary No Longer Primary	-.18 (.91)	.14	.83	4.99	.84
Any Substance Use Disorder					
Constant	.41 (.32)				
Primary No Longer Present					.87
Nonresponder	-.30 (.56)	.25	.74	2.23	.59
Primary No Longer Primary	-.12 (.83)	.18	.89	4.52	.89

Note. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Nonresponder: Primary diagnosis remained present in the diagnostic profile at posttreatment. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment.

Table 52
Logistic Regressions Examining Responder Status Type as a Predictor of DSM-IV Anxiety Disorders at 16-Year Follow-Up

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Generalized Anxiety Disorder					
Constant	1.74 (.44)				
Primary No Longer Present					.68
Nonresponder	-.06 (.77)	.21	.94	4.25	.94
Primary No Longer Primary	-.82 (.95)	.07	.44	2.81	.39
Social Phobia					
Constant	1.55 (.42)				
Primary No Longer Present					.17
Nonresponder	-1.01 (.63)	.11	.36	1.26	.11
Primary No Longer Primary	-1.26 (.87)	.05	.28	1.56	.15
Separation Anxiety Disorder					
Constant	2.94 (.73)				
Primary No Longer Present					.61
Nonresponder	-.80 (1.04)	.06	.45	3.45	.44
Primary No Longer Primary	-1.15 (1.30)	.03	.32	4.05	.38
Panic Disorder					
Constant	3.66 (1.01)				
Primary No Longer Present					.13
Nonresponder	-2.34 (1.16)	.01	.10	.93	.04
Primary No Longer Primary	-1.87 (1.48)	.01	.15	2.80	.21
Agoraphobia					
Constant	2.94 (.73)				
Primary No Longer Present					.65
Nonresponder	-.05 (1.26)	.08	.95	11.15	.97
Primary No Longer Primary	-1.15 (1.30)	.03	.32	4.05	.38
Obsessive-Compulsive Disorder[1]					
Constant	3.66 (1.01)				
Primary No Longer Present					.11
Nonresponder	-1.52 (1.26)	.02	.22	2.57	.23
Primary No Longer Primary	-2.75 (1.31)	.01	.06	.84	.04
Specific Phobia					
Constant	1.55 (.42)				
Primary No Longer Present					.91
Nonresponder	-.23 (.70)	.20	.80	3.14	.74
Primary No Longer Primary	.24 (1.16)	.13	1.27	12.30	.94

Note. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Nonresponder: Primary diagnosis remained present in the diagnostic profile at posttreatment. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment.
[1] R^2=.07 (Cox & Snell), .17 (Nagelkerke). Model $\chi^2(2)=4.90$, $p = .09$

Table 53

Logistic Regressions Examining Responder Status Type as a Predictor of DSM-IV Depressive Disorders at 16-Year Follow-Up

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Major Depressive Disorder					
Constant	1.10 (.37)				
Primary No Longer Present					.67
Nonresponder	-.33 (.61)	.22	.72	2.41	.60
Primary No Longer Primary	.69 (1.14)	.21	2.00	18.69	.54
Major Depressive Episode					
Constant	1.10 (.37)				
Primary No Longer Present					.87
Nonresponder	-.33 (.61)	.22	.72	2.41	.60
Primary No Longer Primary	-.18 (.91)	.14	.83	4.99	.84
Dysthymic Disorder					
Constant	3.66 (1.01)				
Primary No Longer Present					.87
Nonresponder	-.77 (1.44)	.03	.46	7.80	.59
Primary No Longer Primary	17.54 (15191.52)	.00	41422432.42	-	1.00

Note. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Nonresponder: Primary diagnosis remained present in the diagnostic profile at posttreatment. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment.

Table 54

Logistic Regressions Examining Responder Status Type as a Predictor of DSM-IV Substance Use Disorders at 16-Year Follow-Up

		95% CI for Odds Ratio			
	B (SE)	Lower	Odds Ratio	Upper	*p*
Alcohol Abuse					
Constant	1.39 (.40)				
Primary No Longer Present					.60
Nonresponder	-.61 (.63)	.16	.54	1.87	.33
Primary No Longer Primary	-.47 (.93)	.10	.63	3.83	.61
Alcohol Dependence					
Constant	3.66 (1.01)				
Primary No Longer Present					.07
Nonresponder	-2.63 (1.14)	.01	.07	.67	.02
Primary No Longer Primary	17.54 (15191.52)	.00	41422432.42	-	1.00
Drug Abuse[1]					
Constant	2.94 (.73)				
Primary No Longer Present					.04
Nonresponder	-1.62 (.92)	.03	.20	1.19	.08
Primary No Longer Primary	-2.66 (1.05)	.01	.07	.55	.01
Drug Dependence					
Constant	21.20 (6355.07)				
Primary No Longer Present					1.00
Nonresponder	-19.06 (6355.07)	.00	.00	-	1.00
Primary No Longer Primary	.00 (16467.21)	.00	1.00	-	1.00
Nicotine Dependence					
Constant	.85 (.35)				
Primary No Longer Present					.77
Nonresponder	.47 (.66)	.44	1.61	5.86	.47
Primary No Longer Primary	.07 (.91)	.18	1.07	6.31	.94

Note. Primary No Longer Present=Pretreatment primary diagnosis was no longer present anywhere in the diagnostic profile at posttreatment. Nonresponder: Primary diagnosis remained present in the diagnostic profile at posttreatment. Primary No Longer Primary=Pretreatment primary diagnosis was no longer the primary diagnosis at posttreatment.

[1] R^2=.11 (Cox & Snell), .20 (Nagelkerke). Model $\chi^2(2)=7.58$, $p < .05$

Correlations. Pearson product-moment correlations examined the relationship between (a) participant age, (b) time since treatment, (c) posttreatment CSR scores, and ratings on self-report forms at the present follow-up (i.e., BDI-II Total Score, BAI Total Score, SDS Overall Disability Score, QOLI Total Score, and DUSI-R Overall Problem Density Index). Older participant age significantly predicted increased time since treatment ($r = .92$, $p < .01$) and lower posttreatment CSR score ($r = -.27$, $p < .05$). Time since treatment also significantly predicted lower posttreatment CSR score ($r = -.25$, $p < .05$). Neither age, time since treatment, or posttreatment CSR score were significantly associated with any self-report form ratings at follow-up.

BDI-II Total Score was significantly and positively associated with BAI Total Score ($r = .65$, $p < .01$), SDS Overall Disability Score ($r = .71$, $p < .01$), and DUSI-R Overall Problem Density Score ($r = .79$, $p < .01$). BDI-II score was significantly negatively associated with QOLI Total Score ($r = -.69$, $p < .01$). BAI Total Score was significantly and positively associated with SDS Overall Disability Score ($r = .45$, $p < .01$). SDS Overall Disability Score was significantly and positively associated with DUSI-R Overall Problem Density Score ($r = .55$, $p < .01$). Finally, significant negative correlations were observed between both the SDS Overall Disability Score ($r = -.59$, $p < .01$) and DUSI-R Overall Problem Density Score ($r = -.63$, $p < .01$) with QOLI Total Score. See Table 55.

Table 55

Correlation Matrix: Age, Time since Treatment, Clinician Severity Rating at Posttreatment, and Self-Report Forms at 16-Year Follow-Up

	R							
Variable	1	2	3	4	5	6	7	8
Age[1]	-							
Time since Treatment[2]	.92**	-						
Clinician Severity Rating[3]	-.27*	-.25*	-					
Beck Depression Inventory-II (BDI-II)[4]	-.05	-.03	.01	-				
Beck Anxiety Inventory (BAI)[5]	-.09	-.06	.17	.65**	-			
Sheehan Disability Scale (SDS)[6]	.13	.08	-.06	.71**	.45**	-		
Quality of Life Inventory (QOLI)[7]	-.02	.01	.07	-.69**	-.49	-.59**	-	
Drug Use Screening Inventory-R (DUSI-R)[8]	-.14	-.10	.03	.79**	.63	.55**	-.63**	-

Note. Clinician Severity Rating=Clinician rating of interference of most interfering disorder remaining in diagnostic profile at posttreatment on the Anxiety Disorders Interview Schedule-Parent version. BDI-II= Total Score; BAI=Total Score; SDS=Overall Disability Score; QOLI=Total Score; DUSI-R=Overall Problem Density Index. *$p < .05$, **$p < .01$.

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APPENDIX A

COMMONLY USED ABBREVIATIONS

Commonly Used Abbreviations

Avoidant Disorder (AD)

Beck Anxiety Inventory (BAI)

Beck Depression Inventory-II (BDI-II)

Child and Adolescent Anxiety Disorders Clinic (CAADC)

Clinician Severity Rating (CSR)

Cognitive Behavioral Therapy (CBT)

Composite International Diagnostic Interview (CIDI)

Drug Use Screening Inventory-Revised (DUSI-R)

Generalized Anxiety Disorder (GAD)

National Comorbidity Survey-Replication (NCS-R)

Overanxious Disorder (OAD)

Quality of Life Inventory (QOLI)

Randomized clinical trial (RCT)

Separation Anxiety Disorder (SAD)

Sheehan Disability Scale (SDS)

Social Phobia (SP)

APPENDIX B

MEASURES

Beck Anxiety Inventory

Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by that symptom during the past month, including today, by circling the number in the corresponding space in the column next to each symptom.

	Not At All	Mildly but it didn't bother me much.	Moderately - it wasn't pleasant at times	Severely – it bothered me a lot
Numbness or tingling	0	1	2	3
Feeling hot	0	1	2	3
Wobbliness in legs	0	1	2	3
Unable to relax	0	1	2	3
Fear of worst happening	0	1	2	3
Dizzy or lightheaded	0	1	2	3
Heart pounding/racing	0	1	2	3
Unsteady	0	1	2	3
Terrified or afraid	0	1	2	3
Nervous	0	1	2	3
Feeling of choking	0	1	2	3
Hands trembling	0	1	2	3
Shaky / unsteady	0	1	2	3
Fear of losing control	0	1	2	3
Difficulty in breathing	0	1	2	3
Fear of dying	0	1	2	3
Scared	0	1	2	3
Indigestion	0	1	2	3
Faint / lightheaded	0	1	2	3
Face flushed	0	1	2	3
Hot/cold sweats	0	1	2	3
Column Sum				

Scoring **-** Sum each column. Then sum the column totals to achieve a grand score. Write that score here _____ .

Interpretation

A grand sum between **0 – 21** indicates very low anxiety. That is usually a good thing. However, it is possible that you might be unrealistic in either your assessment which would be denial or that you have learned to "mask" the symptoms commonly associated with anxiety. Too little "anxiety" could indicate that you are detached from yourself, others, or your environment.

A grand sum between ***22 – 35*** indicates moderate anxiety. Your body is trying to tell you something. Look for patterns as to when and why you experience the symptoms described above. For example, if it occurs prior to public speaking and your job requires a lot of presentations you may want to find ways to calm yourself before speaking or let others do some of the presentations. You may have some conflict issues that need to be resolved. Clearly, it is not "panic" time but you want to find ways to manage the stress you feel.

A grand sum that **exceeds 36** is a potential cause for concern. Again, look for patterns or times when you tend to feel the symptoms you have circled. Persistent and high anxiety is not a sign of personal weakness or failure. It is, however, something that needs to be proactively treated or there could be significant impacts to you mentally and physically. You may want to consult a physician or counselor if the feelings persist.

BDI-II

ID#_____ Date:_____

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the **one statement** in each group that best describes the way you have been feeling during the **past two weeks, including today**. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness

0 I do not feel sad.
1 I feel sad much of the time.
2 I am sad all the time.
3 I am so sad or unhappy that I can't stand it.

2. Pessimism

0 I am not discouraged about my future.
1 I feel more discouraged about my future than I used to be.
2 I do not expect things to work out for me.
3 I feel my future is hopeless and will only get worse.

3. Past Failure

0 I do not feel like a failure.
1 I have failed more than I should have.
2 As I look back, I see a lot of failures.
3 I feel I am a total failure as a person.

4. Loss of Pleasure

0 I get as much pleasure as I ever did from the things I enjoy.
1 I don't enjoy things as much as I used to.
2 I get very little pleasure from the things I used to enjoy.
3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings

0 I don't feel particularly guilty.
1 I feel guilt over many things I have done or should have done.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

6. Punishment Feelings

0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.

7. Self-Dislike

0 I feel the same about myself as ever.
1 I have lost confidence in myself.
2 I am disappointed in myself.
3 I dislike myself.

8. Self-Criticalness

0 I don't criticize or blame myself more than usual.
1 I am more critical of myself than I used to be.
2 I criticize myself for all of my faults.
3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes

0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10. Crying

0 I don't cry anymore than I used to.
1 I cry more than I used to.
2 I cry over every little thing.
3 I feel like crying, but I can't.

11. Agitation

0 I am no more restless or wound up than usual.
1 I feel more restless or wound up than usual.
2 I am so restless or agitated that it's hard to stay still.
3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest

0 I have not lost interested in other people or activities.
1 I am less interested in other people or other things than before.
2 I have lost most of my interest in other people or things.
3 It's hard to get interested in anything.

13. Indecisiveness

0 I make decisions about as well as ever.
1 I find it more difficult to make decisions than usual.
2 I have much greater difficulty in making decisions than I used to.
3 I have trouble making any decisions.

14. Worthlessness

0 I do not feel I am worthless.
1 I don't consider myself as worthwhile and useful as I used to.
2 I feel more worthless as compared to other people.
3 I feel utterly worthless.

15. Loss of Energy

0 I have as much energy as ever.
1 I have less energy than I used to have.
2 I don't have enough energy to do very much.
3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern

0 I have not experienced any change in my sleeping pattern.

1a I sleep somewhat more than usual.
1b I sleep somewhat less than usual.

2a I sleep a lot more than usual.
2b I sleep a lot less than usual.

3a I sleep most of the day.
3b I wake up 1-2 hours early and can't get back to sleep.

---

17. Irritability

0 I am no more irritable than usual.
1 I am more irritable than usual.
2 I am much more irritable than usual.
0 I am irritable all the time.

18. Changes in Appetite

0 I have not experienced any change in my appetite.

1a My appetite is somewhat less than usual.
1b My appetite is somewhat more than usual.

2a My appetite is much less than usual.
2b My appetite is such more than usual.

3a I have no appetite at all.
3b I crave food all the time.

19. Concentration Difficulty

0 I can concentrate as well as ever.
1 I can't concentrate as well as usual.
2 It's hard to keep my mind on anything for very long.
3 I find I can't concentrate on anything.

20. Tiredness or Fatigue

0 I am no more tired or fatigued than usual.
1 I get more tired or fatigued more easily than usual.
2 I am too tired or fatigued to do a lot of the things I used to do.
3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex

0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

S D S

INSTRUCTIONS: Fill in a numbered bubble that best describes your situation NOW.

1.

WORK

BECAUSE OF MY PROBLEMS, MY WORK IS IMPAIRED...

⓪ ① ② ③ ④ ⑤ ⑥ ⑦ ⑧ ⑨ ⑩

Not at All — Mildly — Moderately — Markedly — Very Severely (Cannot Work)

2.

SOCIAL LIFE/LEISURE ACTIVITIES

(with other people at parties, socializing, visiting, dating, outings, clubs, and entertaining)

BECAUSE OF MY PROBLEMS, MY SOCIAL LIFE/LEISURE IS IMPAIRED...

⓪ ① ② ③ ④ ⑤ ⑥ ⑦ ⑧ ⑨ ⑩

Not at All — Mildly — Moderately — Markedly — Very Severely (I never do these)

3.

FAMILY LIFE/HOME RESPONSIBILITIES

(For example, relating to family members, paying bills, managing home, shopping and cleaning.)

BECAUSE OF MY PROBLEMS, MY FAMILY LIFE/HOME RESPONSIBILITIES ARE IMPAIRED...

⓪ ① ② ③ ④ ⑤ ⑥ ⑦ ⑧ ⑨ ⑩

Not at All — Mildly — Moderately — Markedly — Very Severely (I never do these)

4.

WORK & SOCIAL DISABILITY SCALE

Mark the item that best describes your disability.
PLEASE FILL IN ONE NUMBERED BUBBLE BETWEEN 1 AND 5

Score	Definition
⑤	Symptoms radically change or prevent normal work or social activities.
④	Symptoms interfere with normal work or social activities markedly but they are not prevented or radically changed.
③	Symptoms interfere with normal work or social activities in minor ways.
②	Symptoms mild, but not interfering with normal work or social activities.
①	No complaints, normal activity.

QOLI (1994)

DIRECTIONS:
This survey asks how satisfied you are with parts of your life such as your work and your health. It also asks how important these things are to your happiness. Special definitions are used for words like “money”, “work,” and “play.” Keep these definitions in mind as you answer the questions. Answer every question, even if it does not seem to apply to you. It is your feelings and opinions that are important, so there are no right or wrong answers. Just give the answers that best describe you.

The survey asks you to describe how **important** and how **satisfied** you are with parts of your life such as work and health:

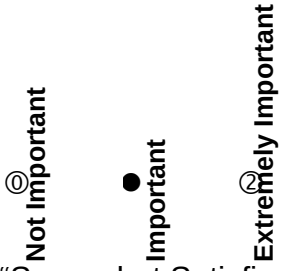
Important means how much this part of your life adds to your overall happiness. You can say how important something is by picking one of three choices: “Not Important” (0), “Important” (1), or “Extremely Important” (2).

Satisfied means how well your needs, goals, and wishes are being met in this area of life. You can say how satisfied you are by picking one of six choices from
“Very Dissatisfied” (-3) to “Very Satisfied” (+3).

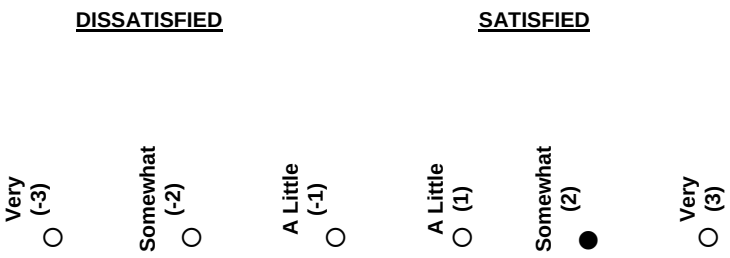
For each question, fill in the numbered bubble that best describes you.

EXAMPLE:

This is how you would answer if WORK was “Important” for your overall happiness:



You would answer this way if you were “Somewhat Satisfied” with your WORK:



				DISSATISFIED			SATISFIED		
	Not Important	Important	Extremely Important	Very (-3)	Somewhat (-2)	A Little (-1)	A Little (1)	Somewhat (2)	Very (3)
HEALTH is being physically fit, not sick, and without pain or disability.									
1. How **important** is HEALTH to your happiness?	⓪	①	②						
2. How **satisfied** are you with your HEALTH?				○	○	○	○	○	○
SELF-ESTEEM means liking and respecting yourself in light of your strengths and weaknesses, successes and failures, and ability to handle problems.									
3. How **important** is SELF-ESTEEM to your happiness?	⓪	①	②						
4. How **satisfied** are you with your SELF-ESTEEM?				○	○	○	○	○	○
GOALS-AND-VALUES are your beliefs about what matters most in life and how you should live, both now and in the future. This includes your goals in life, what you think is right or wrong, and the purpose or meaning of life as you see it.									
5. How **important** are GOALS-AND-VALUES to your happiness?	⓪	①	②						
6. How **satisfied** are you with your GOALS-AND-VALUES?				○	○	○	○	○	○
MONEY is made up of three things. It is the money you earn, the things you own (like a car or furniture), and believing that you will have the money and things that you need in the future.									
7. How **important** is MONEY to your happiness?	⓪	①	②						
8. How **satisfied** are you with the MONEY you have?				○	○	○	○	○	○
WORK means your career or how you spend most of your time. You may work at a job, at home taking care of your family, or at school as a student. WORK includes your duties on the job, the money you earn (if any), and the people you work with. (If you are unemployed, retired, or can't work, you can still answer these questions.)									
9. How **important** is WORK to your happiness?	⓪	①	②						
10. How **satisfied** are you with your WORK? (If you are not working, say how satisfied you are about not working.)				○	○	○	○	○	○
PLAY is what you do in your free time to relax, have fun, or improve yourself. This could include watching movies, visiting friends, or pursuing a hobby like sports or gardening.									
11. How important is PLAY to your happiness?	⓪	①	②						
12. How satisfied are you with the PLAY in your life?				○	○	○	○	○	○

				DISSATISFIED			SATISFIED		
	Not Important	Important	Extremely Important	Very (-3)	Somewhat (-2)	A Little (-1)	A Little (1)	Somewhat (2)	Very (3)
LEARNING means gaining new skills or information about things that interest you. LEARNING can come from reading books or taking classes on subjects like history, car repair, or using a computer									
13. How **important** is LEARNING to your happiness?	⓪	①	②						
14. How **satisfied** are you with your LEARNING?				○	○	○	○	○	○
CREATIVITY is using your imagination to come up with new and clever ways to solve everyday problems or to pursue a hobby like painting, photography, or needlework. This can include decorating your home, playing the guitar, or finding a new way to solve a problem at work.									
15. How **important** is CREATIVITY to your happiness?	⓪	①	②						
16. How **satisfied** are you with your CREATIVITY?				○	○	○	○	○	○
HELPING means helping others in need or helping to make your community a better place to live. HELPING can be done on your own or in a group like a church, a neighborhood association, or a political party. HELPING can include doing volunteer work at a school or giving money to a good cause. HELPING means helping people who are not your friends or relatives.									
17. How **important** is HELPING to your happiness?	⓪	①	②						
18. How **satisfied** are you with the HELPING you do?				○	○	○	○	○	○
LOVE is a very close romantic relationship with another person. LOVE usually includes sexual feelings and feeling loved, cared for, and understood. (If you do not have a LOVE relationship, you can still answer these questions.)									
19. How **important** is LOVE to your happiness?	⓪	①	②						
20. How **satisfied** are you with the LOVE in your life? (If you are not in a LOVE relationship, say how satisfied you feel about not having a LOVE relationship.)				○	○	○	○	○	○
FRIENDS are people (not relatives) you know well and care about who have interests and opinions like yours. FRIENDS have fun together, talk about personal problems, and help each other out. (If you have no FRIENDS, you can still answer these questions.)									
21. How **important** are FRIENDS to your happiness?	⓪	①	②						
22. How **satisfied** are you with your FRIENDS? (If you have no FRIENDS, say how satisfied you are about having no FRIENDS.)				○	○	○	○	○	○

				DISSATISFIED			SATISFIED		
	Not Important	Important	Extremely Important	Very (-3)	Somewhat (-2)	A Little (-1)	A Little (1)	Somewhat (2)	Very (3)
CHILDREN means how you get along with your child (or children). Think of how you get along as you care for, visit, or play with your child. (If you do not have CHILDREN, you can still answer these questions.)									
23. How **important** are CHILDREN to your happiness? (If you have no CHILDREN, say how important having a child is to your happiness.)	⓪	①	②						
24. How **satisfied** are you with your relationships with your CHILDREN? (If you have no CHILDREN, say how satisfied you feel about not having CHILDREN.)				○	○	○	○	○	○
RELATIVES means how you get along with your parents, grandparents, brothers, sisters, aunts, uncles, and in-laws. Think about how you get along when you are doing things together like visiting, talking on the telephone, or helping each other out. ***(If you have no living RELATIVES, circle the 0 ["not Important"] for question 25 and do not answer question 26).***									
25. How **important** are RELATIVES to your happiness?	⓪	①	②						
26. How **satisfied** are you with your relationships with RELATIVES?				○	○	○	○	○	○
HOME is where you live. It is your house or apartment and the yard around it. Think about how nice it looks, how big it is, and your rent or house payment.									
27. How **important** is your HOME to your happiness?	⓪	①	②						
28. How **satisfied** are you with your HOME?				○	○	○	○	○	○
NEIGHBORHOOD is the area around your home. Think about how nice it looks, the amount of crime in the area, and how well you like the people.									
29. How **important** is your NEIGHBORHOOD to your happiness?	⓪	①	②						
30. How **satisfied** are you with your NEIGHBORHOOD?				○	○	○	○	○	○
COMMUNITY is the whole city, town, or rural area where you live (It is not just your neighborhood). COMMUNITY includes how nice the area looks, the amount of crime, and how well you like the people. It also includes places to go for fun like parks, concerts, sporting events and restaurants. You may also consider the cost of things you need to buy, the availability of jobs, the government, schools, taxes, and pollution.									
31. How **important** is your COMMUNITY to your happiness?	⓪	①	②						
32. How **satisfied** are you with your COMMUNITY?				○	○	○	○	○	○

Subject Number:_____
Rater ID:_____
Date:_____

Participant Additional Treatment (PAT)

Instructions: Based on your review of the Services and Pharmacoepidemiology modules of the CIDI interview for this participant, rate the extent of additional treatment received by the participant since receiving services through the Kendall et al. (1997) study. Indicate your rating by circling the number that best reflects your impressions using the anchors provided. Note medication refers only to psychotropic medications. Therapy refers to any mental health intervention (i.e., not physical or occupational therapy).

0	1	2	3	4
No additional services received	The participant has received **some additional services** (e.g., has been on medication less than 1 year; has received 15 or less sessions of therapy)	The participant has received a **moderate amount of additional services** (e.g., has been on medication for 1-2 years; has received between 16-30 sessions of therapy)	The participant has received **a great deal of additional services** (e.g., on medication 2 years or more but not more often than not; more than 30 sessions of therapy but not in therapy more often than not)	The participant has **been receiving services more often than not** (e.g., on medication or in therapy more often than not during this time period)