Cognitive Vulnerability as a Predictor Treatment Outcomes, Dropout, and Relapse During Cognitive Therapy for Depression

Dissertation Proposal

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Introduction

Major Depressive Disorder

Major depressive disorder (MDD) is a psychiatric disorder characterized by a depressed mood and/or anhedonia, as well a host of symptoms as outlined by the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), including changes in weight/appetite, sleep disturbances, difficulties concentrating, fatigue, feelings of guilt /worthlessness, psychomotor retardation or agitation, and suicidal ideation (American Psychiatric Association, 2013). Data from epidemiological studies suggest that MDD is one of the most common psychological disorders, with 12-month prevalence rates ranging from 5-10% and lifetime prevalence rates ranging from 13-16% (e.g., Hasin, Goodwin, Stinson, & Grant, 2005; Kessler et al., 1994; Kessler et al., 2003). It is also well established that people with MDD experience clinically significant functional impairment in their lives. For instance, a diagnosis of MDD is associated with significantly reduced quality of life (e.g., Kessler et al., 2003; Rapaport, Clary, Fayyad, & Endicott, 2005; Wittchen, Carter, Pfister, Montgomery, & Kessler, 2000). Moreover, MDD is a leading cause of disability worldwide, with Americans taking nearly 400 million disability days per year (Merikangas et al., 2007; World Health Organization, 2004). MDD is also rapidly emerging as one of the top causes for premature mortality worldwide (Murray & Lopez, 1996). MDD poses a significant economic burden to our

society through direct costs such as medical services (e.g., inpatient hospitalizations, emergency department visits) and pharmaceutical services, suicide-related costs, and workplace costs (Greenberg et al., 2003; Greenberg, Fournier, Sisitsky, Pike, & Kessler, 2015). Taken together, MDD is one of the most debilitating and costly mental health conditions that adversely affects millions of people worldwide.

Cognitive Theory of Depression

One of the frontline psychotherapeutic interventions for MDD is cognitive therapy (CT), which was pioneered by Aaron Beck in the 1960's. CT was developed from Beck's *cognitive theory*, which suggests that people with depression have a propensity for having negative automatic thoughts that exacerbate and maintain their depression symptoms (Beck, Rush, Shaw, & Emery, 1979). In particular, Beck posited that people with depression hold maladaptive schemas, including negative views about one's self, the future, and the external world (e.g., the cognitive triad; Beck et al., 1979). Based on this theoretical foundation, CT primarily teaches patients to identify their negative automatic thoughts, examine the evidence for and against them, and generate alternative responses that are often more objective and realistic (i.e., cognitive restructuring). Overall, it is this process of modifying negative cognitions that is proposed to facilitate the amelioration of depressive symptoms by helping people think more rationally, feel better emotionally, and behave more functionally.

One of the earliest assessments that was developed to measure cognitive dysfunction among people with depression was the Dysfunctional Attitude Scale (DAS; Weissman & Beck, 1978). The DAS specifically measures negative attitudes towards the

self, world, and future that are presumed to represent the underlying depressogenic assumptions made by people with depression (e.g., "If I fail at my work, then I am a failure as a person"). In the initial validation study of the DAS, it demonstrated strong psychometric properties, including strong internal consistency ($\alpha = .93$), test-retest reliability (.71), and correlations with measures of depression symptoms, including the Beck Depression Inventory (BDI, $r = .65$) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961; Weissman & Beck, 1978). Other early studies that sought to specifically examine the psychometric properties of the DAS found comparable results regarding it's internal consistency and reliability (e.g., Dobson & Breiter, 1983; Oliver & Baumgart, 1985; Weissman, 1980).

Since it's development, a multitude of studies have demonstrated that the DAS is significantly correlated with symptoms of depression in both clinical (inpatient and outpatient) (e.g., Beck, Steer, & Brown, 1993; Dobson & Shaw, 1986; Hamilton & Abramson, 1983; Ranieri et al., 1987; Silverman, Silverman, & Eardley, 1984) and non-clinical samples (Brown , Hammen, Craske, & Wickens, 1995; Olinger, Kuiper, & Shaw, 1987; Oliver & Baumgart, 1985). The DAS has even been able to discriminate between people diagnosed with depression who score higher on the DAS compared to those with other psychiatric disorders and non-depressed controls (e.g., Gotlib, 1984; Hedlund & Rude, 1995; Hill, Oei, & Hill, 1989; Hollon et al., 1986; Nelson et al., 1992). In other words, scores on the DAS tend to co-vary with either the presence or lack of depression symptoms.

Further supporting its construct validity, the DAS is also significantly associated with other measures of cognitive dysfunction akin to depression. Research in this area has found that the DAS is positive correlated (r = .53) with the Automatic Thought Questionnaire (ATQ; Hollon & Kendall, 1980), which is a self-report questionnaire that measures the frequency of negative cognitions about the self and is commonly used in CT process-outcome research (e.g., Hollon, Kendall, & Lumry, 1986; Nelson, Stern, & Cicchetti, 1992). Additionally, the DAS is positively correlated with the Cognitive Style Test (r = .44; CST; Blackburn, Jones, & Lewin, 1986) and the Cognitive Response Test (r = .37; CRT; Giles & Rush, 1983) – both of which measure distorted cognitions that are common in people with depression.

The DAS has even demonstrated predictive validity as evidenced by its capability to prospectively predict future depressive episodes. For example, one study found that dysfunctional attitudes measured at baseline significantly predicted the onset of depression symptoms three months later in undergraduate women reporting low levels of social support (Barnett & Gotlib, 1988). In a longitudinal study of women who remitted from their depression, the DAS was also found to be a significant predictor of future BDI and Hamilton Rating Scale for Depression (HRSD) scores six months later while controlling for initial symptom severity (Rush, Weissenburger, & Eaves, 1986). In fact, DAS scores accounted for 25% of the variance in scores on both the BDI and HRSD at the six-month follow-up time point. One interesting finding in this study was that the ATQ was not a significant predictor of subsequent depression, which lends support for the unique predictive validity of the DAS compared to other measures of cognitive

dysfunction (Rush et al., 1986). Another longitudinal study examined the temporal nature of dysfunctional attitudes in depressed patients as they achieved clinical remission. Although significant reductions in DAS scores was associated with clinical remission in depressed patients, they continued to endorse more dysfunctional attitudes than the non-depressed controls even when in remission (Eaves & Rush, 1984).

Taken together, these findings suggest that the DAS is a reliable and valid measure of dysfunctional attitudes that closely parallels Aaron Beck's *cognitive theory* whereby people with depression tend to exhibit negative cognitive styles that significantly contribute to their depressed mood. Accordingly, Beck's *cognitive theory* would posit that modifying such dysfunctional attitudes should be a key mechanism of change in reducing depression symptoms. Indeed, treatment studies have examined whether changes in DAS scores are associated with and predict treatment outcomes in CT for depression. For instance, one study found that DAS scores significantly decreased across 12 weeks of CT in a small sample of 27 patients with depression. Reductions in dysfunctional attitudes was also strongly correlated with reductions in BDI scores during CT (r = .67; Barber & DeRubeis, 2001). Another study of 30 patients with depression attending CT in a group format[1] found that DAS scores significantly decrease throughout treatment, and that these reductions were associated with post-treatment BDI scores (Furlong & Oei, 2002). However, one key limitation to these studies is that they only provide evidence for a concurrent association between changes in dysfunctional attitudes

[1] Although group CT was provided in this study, it should be noted that the intervention utilized the identical psychotherapeutic techniques emphasized by Beck in CT, such as cognitive restructuring for negative automatic thoughts. Additionally, this group CT intervention has been found to be efficacious in treating MDD.

and depression symptoms. That is, they do not speak to the direction and causality of this relationship.

More robust treatment studies have addressed this research question by examining whether early changes in dysfunctional attitudes prospectively predict later changes in depression symptoms during CT. In one randomized clinical trial comparing CT to Imipramine, reductions in DAS scores during the first six weeks of treatment prospectively predicted subsequent reductions in depression symptoms (DeRubeis et al., 1990). Importantly, this effect was only observed among the patients who received CT and not those treated with Imipramine. Another interesting finding in this study is that early changes in ATQ scores did not significantly predict subsequent changes in depression during the second half of treatment. This study is significant in that it showed that early changes in dysfunctional attitudes predicted subsequent reductions in depressive symptoms, but only among those who received CT, which indicates that this effect may be specific to CT and not other treatments for depression such as pharmacotherapy. Additionally, this finding was specific to the DAS and not other assessments of cognitive dysfunction (i.e., the ATQ), which may suggest that the DAS parallels Beck's *cognitive theory* better than other measures

Given the literature showing that dysfunctional attitudes remain elevated even after remitting from a major depressive episode (Eaves & Rush, 1984), some research has even investigated how dysfunctional attitudes predict risk of relapse following successful treatment with CT. One randomized clinical trial compared patients with depression who were randomly assigned to receive a 12-week course of CT, pharmacotherapy (a tricyclic

antidepressant), combined CT plus tricyclic antidepressant, or CT plus placebo. During the acute phase of treatment, reductions in dysfunctional attitudes were associated with clinical remission for all patients, regardless of treatment condition. Critically, patients with higher DAS scores at treatment termination were significantly more likely to relapse during the one-year follow-up period. Additionally, a discriminant function analysis revealed that post-treatment DAS and BDI scores correctly classified 75% patients who relapsed or maintained remission during the follow-up period (Simons, Murphy, Levine, & Wetzel, 1986). Of note, in these analyses, other key variables such as hopelessness, automatic thoughts, social adjustment, and self-control skills did not significantly discriminate between relapse or remission. It should be noted that this study did not examine whether the relationship between post-treatment dysfunctional attitudes and risk of relapse differed by treatment condition, which is a noteworthy oversight given that those patients who received CT (with or without a pill placebo) were significantly less likely to relapse compared to those who received pharmacotherapy alone.

Building off the literature demonstrating a link between dysfunctional attitudes and risk of relapse, some researchers have posited that dysfunctional attitudes may serve as a cognitive vulnerability that persists in remitted patients, but only become activated after an event triggers a sad mood. One important line of research by Zindel Segal and colleagues has specifically examined whether *cognitive reactivity* (i.e., mood-induced increases in dysfunctional attitudes) predicts relapse after successful treatment of depression (Segal, Gemar, & Williams, 1999). Segal and colleagues suggest that a sad mood is triggered by a prompting event that parallels the circumstances in which this

cognitive vulnerability was initially acquired (Segal & Ingram, 1994). In other words, this cognitive vulnerability makes it more likely that people with remitted depression will recall and utilize past associations between an event and a sad mood to use in further cognitive processing as they make interpretations about the events that are currently occurring in their lives (Teasdale, 1988). This philosophy is in harmony with Beck's *cognitive theory*, in which he states "Unpleasant – even extremely adverse – life situations do not necessarily produce a depression unless the person is particularly sensitive to the specific type of situation because of the nature of his cognitive organization" (Beck et al., 1979, p. 16). Early support for this cognitive reactivity hypothesis is provided by research suggesting that dysfunctional attitudes are indeed mood-dependent. That is, dysfunctional thinking tends to increase when one's mood worsens and decrease when one's mood improves. Moreover, this pattern was found only among those individuals who had experienced a major depressive episode, but not among those who have never been depressed (Miranda, Persons, & Byers, 1990).

In a randomized clinical trial testing this hypothesis, patients received either 20 weekly sessions of CT (with two tapered booster sessions thereafter) or six months of antidepressant medication (i.e., sertraline, paroxetine, or venlafaxine). At the end of treatment, 54 patients underwent a sad mood provocation where they were asked to think of a time in their lives where they felt sad and listen to piece of music that had been used in prior research to effectively induce a sad mood. In order to measure cognitive reactivity, the patients completed the DAS before and after the sad mood induction. The results revealed that patients who achieved remission through antidepressants (n = 29)

exhibited significantly greater cognitive reactivity (i.e., increases in dysfunctional attitudes) as they underwent the mood induction compared to patients who achieved remission through CT (n = 25). In order to determine whether increased cognitive reactivity after recovery was a significant predictor of risk for relapse, patients were re-contacted after 13 – 48 months (M = 31.7, SD = 11.3). Relapse was defined by meeting diagnostic criteria for a major depressive episode any time after treatment ended as assessed by the SCID-I. Irrespective of treatment condition, post-induction scores on the DAS were a significant predictor of relapse when controlling for pre-induction DAS scores (which did not significant predict relapse) (Segal et al., 1999).

These findings were replicated in a larger randomized clinical trial of 301 patients diagnosed with MDD who received six months of antidepressant medication or 20 weekly sessions of cognitive therapy. Although treatment response did not significantly differ between conditions, patients who achieved remission with antidepressant medication exhibited significantly greater cognitive reactivity following the sad mood provocation compared to those who achieved remission with CT (d = 0.42). Specifically, patients who remitted through antidepressants showed increased cognitive reactivity (DAS residual change score = 4.06) whereas patients who remitted through CT actually exhibited decreased cognitive reactivity (DAS residual change score = -2.76). Perhaps most importantly, regardless of treatment condition, the magnitude of cognitive reactivity following the sad mood provocation significantly predicted increased risk for relapse during the 18-month follow-up period (hazard ratio, HR = 1.42), even after controlling for prior depressive episodes and prior treatment (Segal et al., 2006).

In a more recent study, researchers examined the role of cognitive reactivity in predicting relapse and/or recurrence among patients who responded to treatment but were determined to be "high risk" (Jarrett et al., 2012). The study utilized a sample of 213 patients who received 16-20 sessions of CT and were classified as treatment responders based on two criteria: not having a diagnosis of MDD at the end of treatment and having a HRSD score ≤ 12. Despite their response to treatment, the researchers were specifically interested in studying "high risk" patients given their interest in examining predictors of relapse. Being "high risk" was operationalized as having at least one HRSD score ≥ seven during the last seven weeks of treatment. Following the acute phase of CT treatment, all high-risk responders were randomly assigned to an eight-month continuation phase of CT, fluoxetine, or a pill placebo. Interestingly, this study failed to replicate the aforementioned findings in that the identical sad mood induction did not elicit pre-post changes in dysfunctional attitudes. Ironically, follow-up analyses revealed that DAS scores significantly decreased during the sad mood induction for a subsample of patients (n = 41, 20%) whose mood was unchanged or even improved. Given that evidence for cognitive reactivity was unsubstantiated in this study, they investigated whether unprimed dysfunctional attitudes (i.e., pre-mood induction DAS scores) at the end of CT were predictive of relapse and/or recurrence during a 32-month follow-up period. The results revealed that unprimed dysfunctional attitudes did not significantly predict relapse and/or recurrence during the eight-month continuation phase. However, unprimed dysfunctional attitudes at post-treatment significantly predicted relapse and/or recurrence over 20 months (HR = 1.33) and over 32 months (HR = 1.32) after completion

of CT, even after controlling for depression symptom severity at the end of treatment. Furthermore, these effects did not differ among the three different treatment conditions during the continuation phase (Jarrett et al., 2012).

Taken together, these findings largely support Beck's *cognitive theory* and his conceptualization of the role that dysfunctional attitudes play in the nature and treatment of depression. First, evidence suggests that dysfunctional attitudes are associated with symptoms of depression (e.g., Beck et al., 1993; Dobson & Shaw, 1986; Hamilton & Abramson, 1983; Hollon et al., 1986; Weissman & Beck, 1978) as well as other measures of cognitive dysfunction (e.g., Blackburn et al., 1986; Giles & Rush, 1983; Hollon & Kendall, 1980). Second, dysfunctional attitudes have been shown to prospectively predict future depressive episodes (e.g., Barnett & Gotlib, 1988; Rush et al., 1986), indicating that these cognitive patterns may be relevant to the etiology of depression. Given that the primary therapeutic strategy of CT is to modify such maladaptive cognitions, dysfunctional attitudes should decrease over the course of CT treatment. This is indeed the case, as such changes in dysfunctional attitudes have even been shown to predict improved treatment outcomes in CT (e.g., Barber & DeRubeis, 2001; DeRubeis et al., 1990; Furlong & Oei, 2002). Additionally, some research failed to find this effect among those depressed patients receiving pharmacotherapy with an antidepressant, indicating that this effect may be specific to cognitive change mechanisms in CT (DeRubeis et al., 1990). Finally, some researchers even conceptualize dysfunctional attitudes as a cognitive vulnerability that confers greater risk for future depression, given that dysfunctional attitudes may become activated in the context of a stressful life event even in a patient

who has remitted from their depressive episode. This notion is consistent with the extant literature demonstrating that dysfunctional attitudes, either unprimed or primed with a sad mood induction as an index of cognitive reactivity, significantly predict relapse among patients successfully treated with CT for depression (Jarrett et al., 2012; Segal et al., 2006, 1999; Simons et al., 1986).

Despite the empirical support for the DAS as a measure of negative cognitive styles in depression, a handful of studies appear to have mixed results that are at odds with those described above. For example, Dobson and Shaw (1986) used three hospitalized samples to find that DAS scores were significantly higher compared to non-depressed psychiatric controls and non-psychiatric controls. However, in follow-up analyses of patients who had achieved remission from their depression, those patients who "remitted" still had an average BDI score of 12.80, which is indicative of mild depression. Given that the average BDI score for the non-psychiatric controls was only 3.8, it is not surprising that those in "remission" had higher DAS scores compared to the controls given that these two measures have been found to co-vary with each other. In order to appropriately test whether changes in the DAS are associated with reductions in depression in remitted samples, there must be equivalent levels of depression symptom severity when comparing those in remission to the control sample. A same critique can be made for a study conducted by Eaves and Rush (1984), where DAS scores for the non-depressed control group were lower than have generally been reported elsewhere, which suggests the possibility of an atypical sample (see review by Barber & DeRubeis, 1989). Additionally, Barber and DeRubeis (1989) computed a pooled mean and standard

deviation on the DAS from five prior studies and found that the patients who remitted from their depressive episode were less than one standard deviation higher on the DAS compared to the pooled control group. In other words, the difference between DAS scores in remitted patients and non-depressed controls may be smaller than originally reported (Barber & DeRubeis, 1989). Additionally, in the Hamilton and Abramson study (1983), found that inpatients with depression did not differ from non-depressed psychiatric inpatients or non-psychiatric controls in DAS scores following medication and/or non-cognitive therapy (i.e., milieu therapy, group therapy, occupational therapy). As such, the significant reductions that they observe in DAS scores for those with depression may not be specific to CT as previously hypothesized. In response to these methodological concerns, some researchers even argue that the DAS may not be an appropriate measure for assessing cognitive schemata (see Segal, 1988 for a review).

Overall, the extant literature that has empirically studied Beck's *cognitive theory* lends support for the role of dysfunctional attitudes in the phenomenology and treatment of MDD. As reviewed above, the majority of research suggests that dysfunctional attitudes play a key role in the onset, maintenance, and severity of MDD. Additionally, specifically targeting the modification of dysfunctional attitudes in CT appears to be crucial in helping people diagnosed with MDD achieve remission by reducing their depressive symptoms and improving their overall level of psychological functioning. However, those studies with discrepant findings or with key methodological issues point to the idea that dysfunctional attitudes capture only one facet of the role that negative cognitive styles play in depression and CT.

Learned Helplessness Theory of Depression

One important aspect of Beck's *cognitive theory* that may address some of these concerns is that a depressed person's negative cognitions and schema may be activated in response to particular life situations or circumstances (Beck, 1967, 1976; Beck et al., 1979, p. 16). This idea is fundamental to Martin Seligman's *learned helplessness theory of depression,* which provides a conceptual framework for how dysfunctional cognitive styles interact with stressful life circumstances in the development of depression. Seligman utilized translational research with animals to demonstrate that dogs who were repeatedly exposed to unavoidable and inescapable electric shocks later stopped attempting to escape the electric shocks at a later time when the possibility of escape was made available to them. Rather, the dogs would give up and lie passively in the new context, even when escape was indeed possible (Overmier & Seligman, 1967; Seligman & Maier, 1967). It was hypothesized that the repeated exposure to aversive stimuli led to the belief that the situation was uncontrollable – nothing that the dogs did had any impact on the administration of electrical shocks. Seligman asserted that this belief of uncontrollability fosters a sense of helplessness, which in turn interferes with learning adaptive behavioral responses (i.e., escaping the electric shocks).

Seligman coined this cognitive interpretation *learned helplessness,* and he noted that this phenomenon paralleled the experience of helplessness in people with depression (Maier & Seligman, 1976; Seligman, 1972). It was proposed that people with depression exhibit motivational, emotional, and cognitive deficits associated with learned helplessness (Seligman, 1975; Seligman, 1972). First, experiencing uncontrollable events

reduces a depressed person's motivation to respond behaviorally when they encounter an aversive situation at a later period in time. Second, through repeated exposure to uncontrollable events, the depressed person begins to believe that their responses will have little or no impact on their current situation. In behavioral terms, this exposure to an uncontrollable situation interferes with one's ability to perceive the contingency between their behavior and a specific outcome. Third, the combination of experiencing uncontrollable events and developing beliefs of learned helplessness cultivates in greater negative emotions, namely depression (Seligman, 1975; Seligman, 1972). Overall, Seligman emphasized that learning that one is helpless and responding behaviorally in accord with that belief was conceptualized to be a central psychological cause of depression.

Although the *learned helplessness theory of depression* was an innovative perspective that helped deepen our understanding of depression, it was limited in two ways that could not account for why certain people develop depression after encountering an uncontrollable stressor whereas other people do not. First, it did not account for differences in which a person believes that situations are viewed as uncontrollable for everyone (i.e., universal helplessness) versus situations that are viewed as uncontrollable for only some people (i.e., personal helplessness). Second, this theory does not adequately delineate the extent to which uncontrollability is chronic versus acute (i.e., the duration of the uncontrollable stressor), nor does it outline whether uncontrollability is general or specific (i.e., the extent to which beliefs of uncontrollability apply to many or few/no other situations).

Reformulated Learned Helplessness Theory of Depression

To address these limitations, Abramson and colleagues (Abramson, Seligman, & Teasdale, 1978) drew on attributional theory to develop their *reformulated learned helplessness theory,* which specifies how people develop depression based on the causal attributions that they make for negative life events along three critical dimensions. First, they proposed that people attribute the causes of negative life events to be due to either an *external versus internal* cause. Whereas people who feel universally helpless are more likely to make external attributions for negative life events, people who feel personally helpless are inclined to make internal attributions for these events. Importantly, it was hypothesized that such an internal attributions has the consequence of lowering one's self-esteem, which contributes to depression. Second, people make attributions about how *global versus specific* the causes of these negative events are. Global attributions are made when helplessness deficits are generalized and occur across a broad range of contexts and situations. On the other hand, specific attributions are made when helplessness is experienced in a situation-specific manner that does not generalize to other circumstances. Third, based on individual differences in the time course of helplessness beliefs, *stable versus unstable* attributions are made depending on whether the helplessness experienced is perceived as chronic or acute, respectively. In summary, this *reformulated learned helplessness theory* suggests that some people exhibit a *cognitive vulnerability* because they have a propensity to make global, stable, and internal attributions for the causes of negative life events, which puts them at a greater risk of developing depression (Abramson, et al., 1978).

In order to assess the *reformulated learned helplessness theory,* the Attributional Style Questionnaire (ASQ; Peterson et al., 1982) was developed specifically to measure the causal attributions that people make when they experience uncontrollable and aversive events in their lives. Specifically, the ASQ measures individual differences in the tendency to make global (versus specific), stable (versus unstable), and internal (versus external) attributions about the causes of negative life events. The ASQ presents 12 hypothetical events (six positive and six negative) and instructs respondents to vividly imagine themselves in that situation as if it was happening to them in that current moment. Additionally, half of the events are achievement related and the other half are affiliation (i.e., interpersonally) related. The purpose of having two different categories of events was to increase the generalization of this cognitive vulnerability across different situations. Given that these attributional tendencies reflect a cognitive "style," it would be expected that this cognitive vulnerability would be present across many different situations and domains. Respondents first consider the major cause of the event and provide one text response describing their attributed cause. Next, respondents answer three questions about the global, stable, and internal attributions made regarding the causes of the hypothetical events. These responses are provided using a 1-7 Likert scale, with higher scores indicating more negative causal attributions (i.e., greater cognitive vulnerability). Finally, one question asks about how important the situation would be to the respondent, which is also scored on a 1-7 Likert scale. A mean score is calculated for each type of causal attribution (i.e., global, stable, and internal), and a cognitive

vulnerability composite score is calculated by averaging each of these three subscale scores.

In the initial validation of the ASQ using 130 undergraduate students, respectable internal consistencies were found for the composite scales for positive and negative events (α = .75 and .72, respectively). However, reliability was rather low for each of the specific attributional dimensions (i.e., global, stable, internal) regardless of whether the events were positive or negative (αs ranging from .44 to .69). There was also no empirical evidence for the discriminability of achievement and affiliation events, which led researchers to largely drop the conceptual distinction between these two factors (Peterson et al., 1982). As such, scores are usually presented as a measure of cognitive vulnerability for negative events, positive events, and/or a composite measure of the two.

Despite its questionable reliability, the authors do provide evidence for the validity of the ASQ as a measure of cognitive vulnerability as outlined by the *reformulated learned helplessness theory.* One early study found that college students with elevated BDI scores made more global, stable, and internal attributions for the causes of negative outcomes compared to non-depressed college students. Moreover, the depressed college students were also more likely to attribute positive outcomes to unstable and external (but not global) causes (Seligman, Abramson, Semmel, & Von Baeyer, 1979). A separate study found that college students who made global, stable, and internal attributions for negative events related to school actually received lower grades during their freshman year (Peterson & Barrett, 1987).

Correlational research has also supported the relationship between cognitive vulnerability and depression symptoms. In two large independent samples, scores on the BDI were significantly associated with the global (r = .23 - .30) and stable (r = .14 - .15) subscales of the ASQ for negative events, as well as a composite measure of cognitive vulnerability (r = .22 - .26). A non-significant trend was observed for the internal subscale (r = .07 - .08, ps =.105 and .098). For positive events, scores on the BDI were significantly associated with the stable (r = -.14 - -.12) and internal (r = -.19 - -.16) subscales of the ASQ. Interestingly, the global subscale was not significantly associated with BDI scores for positive events (r = .02), and the composite measure of cognitive vulnerability was only significant in one sample (r = -.12) whereas a non-significant trend was observed in the other sample (r = -.09, p = .082). This study also demonstrated that the ASQ was able to adequately distinguish between people with high and low BDI scores (Blaney, Behar, & Head, 1980).

In one laboratory-based experiment, a learned helplessness behavioral paradigm was administered to undergraduates with either a global of specific attributional style (as indexed by scores on the ASQ). Participants were exposed to either uncontrollable bursts of noise, controllable bursts of noise, or no noise. Then, participants were put in a situation that was either similar or dissimilar to their prior experience and they were subsequently asked to complete an anagram task. Participants with a tendency to make global attributions who were exposed to the uncontrollable and aversive stimulus exhibited learned helplessness deficits that generalized to both similar and dissimilar

situations as measured by longer latencies to complete the anagram task (Lauren B. Alloy, Peterson, Abramson, & Seligman, 1984).

Longitudinal studies have also provided more robust empirical support for the *reformulated learned helplessness theory.* For instance, one prospective study of college students assessed cognitive vulnerability using the ASQ and depression symptoms using the BDI before and after a midterm examination. Results revealed that greater cognitive vulnerability before the midterm predicted later depression symptoms, but only if students experienced a negative life event by reporting that they were dissatisfied with their grade on the examination (Peterson & Seligman, 1981). Another study tested the causal role of attributions in the development of depression in 180 college students by administering the ASQ and BDI at two time points one month apart. The first finding was that, for negative events, the three ASQ subscales were all significantly correlated with BDI scores at both time points. However, for positive events, only the internal subscale (at both time points) and stable subscale (at time two) were negatively correlated with depression symptoms. Using cross-lagged panel correlational analysis (which allows researchers to test assumptions about causality from correlational data), there was evidence supporting the hypothesis that global and stable attributions made about the causes of negative outcomes were related to the development of later depression. However, evidence for such an effect was not found for the internal subscale (Golin, Sweeney, & Shaeffer, 1981).

Another longitudinal study used a prospective, naturalistic design to test whether people with greater cognitive vulnerability were more susceptible to experiencing

depression symptoms when encountering a negative life event at a later point in time. Using a sample of 227 undergraduates who were about to take a midterm examination, researchers examined the correlation between ASQ scores before the exam and depression symptoms immediately before and after receiving their midterm grade (approximately 11 and 16 days later, respectively). Among those students who received a poor grade on the midterm examination (i.e., the negative life event), global (r = .34) and internal (r = .34) causal attributions were significantly associated with residual changes in depression symptoms before and after receiving their grade. However, stable attributions were not significantly correlated with residual changes in depression symptoms (r = .04). In the absence of a negative life event (i.e., those who received a high grade on the exam), there were no significant associations between cognitive vulnerability and subsequent changes in depression symptoms (Metalsky, Abramson, Seligman, Semmel, & Peterson, 1982). Research has also demonstrated that attributional styles for positive and negative events moderate the relationship between positive/negative life events and depression symptoms one month later. Specifically, a tendency to make global/stable/internal causal attributions for negative events and specific/unstable/external attributions for positive events prospectively predicted higher levels of depression, but only when participants actually experienced a negative event or in the absence of positive events over the one-month time period (Fresco, Alloy, & Reilly–Harrington, 2006). A similar longitudinal study found a significant interaction between cognitive vulnerability and expectations about the presence or absence of positive and negative events in one's future. In a large sample of undergraduate college

students, cognitive vulnerability moderated the relationship between expectations at time 1 and depression symptoms at time 2. Specifically, more negative and pessimistic expectations at baseline were positively associated with depression symptoms six weeks later, but *only* for people who had high levels of cognitive vulnerability (one SD above the mean). However, there was no significant association between expectations and later depressive symptoms for those with average levels of cognitive vulnerability and there was evidence a negative relationship for those with low levels of cognitive vulnerability (Riskind, Rholes, Brannon, & Burdick, 1987). Overall, the authors concluded that expectations for negative life outcomes could precipitate depression symptoms in people with cognitive vulnerability, but not in those individuals without cognitive vulnerability.

Overall, the aforementioned studies provide empirical evidence for the *reformulated learned helplessness theory* as a diathesis-stress model of depression. That is, having a tendency to make global, stable, and internal attributions about the causes of life events is a cognitive vulnerability that serves as a "diathesis" for depression. However, this cognitive vulnerability primarily becomes activated in the context of a "stressor," which is conceptualized as the actual experience of a negative life event. It is this proposed diathesis-stress interaction between cognitive vulnerability and a negative life event which is proposed to lead to the development of depression (Lyn Y. Abramson, Seligman, et al., 1978).

Given the multitude of studies that have tested the *reformulated learned helplessness theory*, meta-analytic work has sought to summarize the relationship between cognitive vulnerability and depression symptoms. One meta-analysis of 104

studies consisting of nearly 15,000 participants found reliable associations between global, stable, and internal attributions and depression symptoms (Sweeney, Anderson, & Bailey, 1986). For negative outcomes, global (d = .37), stable (d = .34), and internal (d = .36), attributions were all significantly associated with depression symptoms. When creating a composite score of cognitive vulnerability, a stronger effect size was observed (d = .44). It should be noted that this meta-analysis pooled results from clinical, community, and university samples. In a comparison of these different samples, the relationship between cognitive vulnerability and depression was always stronger in clinical samples, although this comparison only reached statistical significance for global attributions. For positive outcomes, weaker effect sizes were observed for the association between global (d = - .12), stable (d = -.25), and internal (d = -.36) attributions and BDI scores. Moreover, the effect size was comparable when creating a composite measure of cognitive vulnerability for positive events (d = -.26) (Sweeney et al., 1986).[2] Although this meta-analysis provides strong evidence for the relationship between cognitive vulnerability and depression, it is also worth noting that the researchers included any study that incorporated a measure of global, stable, and internal attributions for positive and negative life events. However, this is not a meta-analysis of the specific relationship between the ASQ and depression. Nonetheless, it provides a useful and empirical summary of the link between cognitive vulnerability and depression. Further supporting this theory, these findings dovetail the extant literature in children and adolescents using the Children's Attributional Style Questionnaire (CASQ; Seligman et al., 1984). Another

[2] In the meta-analysis by Sweeney et al, (1986), the effect sizes reported above were weighted by sample size of individual studies and also corrected for attenuation due to measurement error.

meta-analysis of 28 studies involving over 7,500 youth demonstrated that global, stable, and internal attributions were positively associated with depression for negative outcomes (d = .41). A composite measures of cognitive vulnerability also yielded a stronger effect size (d = .50) (Gladstone & Kaslow, 1995).

Given that the *reformulated learned helplessness theory* posits that global, stable, and internal attributions contribute to the development of MDD, a variety of studies have examined this theory within the context of clinical samples. One study of males recruited through a Veterans Administration hospital compared the causal attributions made for negative and positive events in men with depression, men with schizophrenia, and men hospitalized for medical or surgical reasons. As expected, men with depression reported greater global, stable, and internal attributions for negative life events compared to the men with schizophrenia and non-depressed controls. For positive events, the depressed men also made less stable and internal (but not global) attributions relative to the other two comparison groups (Raps, Peterson, Reinhard, Abramson, & Seligman, 1982). Another study of female psychiatric patients observed significantly higher scores on the ASQ composite and each of the three subscales among those with endogenous, non-endogenous, and unremitted depression in comparison to non-depressed controls (Eaves & Rush, 1984). The ASQ has also been shown to discriminate between inpatients with depression and non-depressed inpatients and community participants (Hamilton & Abramson, 1983). Cognitive vulnerability has also been found to be positively associated with depression symptoms even after partialing out the contribution of general psychopathology in a clinical sample (*rs* ranging from .19 to .31; Johnson, Petzel, &

Munic, 1986). Research has also evaluated whether cognitive vulnerability is specific to depression or if it may be a transdiagnostic process that cuts across other psychiatric disorders. A multitude of studies have demonstrated that attributional styles for both negative and positive events can in fact discriminate between patients diagnosed with MDD and those diagnosed with anxiety disorders such as social anxiety disorder, panic disorder, agoraphobia, generalized anxiety disorder, and specific phobias (Fresco et al., 2006; Heimberg et al., 1989; Heimberg, Vermilyea, Dodge, Becker, & Barlow, 1987; Ingram, Kendall, Smith, Donnell, & Ronan, 1987). In all of these studies, patients with depression consistently tended to exhibit the greatest cognitive vulnerability for causal attributions about positive and negative events.

The body of research highlighting the strong link between cognitive vulnerability and depression begs the question of whether these negative attributional styles can be modified with CT, and if so, to what extent does this predict treatment outcomes. In Beck's highly influential *Cognitive Therapy of Depression* treatment manual, he outlines different aspects of cognitive dysfunction (e.g., cognitive triad, schemas, cognitive errors, faulty information processing) that may explain the cognitive substrates of depression. Given that Beck's proposed maladaptive cognitive styles in depression are rather broad, it is reasonable to hypothesize that cognitive vulnerability as outlined by the *reformulated learned helplessness theory* should be amenable to change through CT. Indeed, treatment studies have investigated whether cognitive vulnerability decreases during an acute course of CT and whether cognitive vulnerability predicts treatment outcomes.

One of the first studies to test this hypothesis administered the ASQ to 39 patients diagnosed with MDD at the beginning and end of CT, as well as at a one-year follow-up period. At all time points, greater cognitive vulnerability for negative events was significantly associated with depression symptoms (*r*s ranging from .56 to .64). Cognitive vulnerability significantly decreased from pre- to post-treatment during CT, and these changes were significantly associated with pre-post changes in depression symptoms ($r = .65$). Importantly, these improvements were stable over the one-year follow-up period, signifying that CT led to enduring changes in the global, stable, and internal attributions that depressed patients tend to make for negative life events (Seligman et al., 1988). One null finding from this study is that cognitive vulnerability assessed at intake did not significantly predict acute treatment outcomes during CT. However, there were a couple non-significant trends that are worth reporting. Where predicting BDI scores at the one-year follow-up, there were non-significant trends for pre-post changes in cognitive vulnerability ($r = -.31$, $p < .12$) and cognitive vulnerability at post-treatment ($p < .14$) as predictors of later depression when controlling for post-treatment BDI scores. Moreover, cognitive vulnerability at post-treatment was significantly correlated with BDI scores at the one-year follow-up ($r = .54$). Although these findings should be interpreted with caution, they do imply that cognitive vulnerability may be an important construct relevant to the risk of relapse following successful CT for depression (Seligman et al., 1988).

A randomized controlled trial sought to examine whether changes in cognitive vulnerability differed between patients receiving 16-20 sessions of CT or pharmacotherapy (i.e., Imipramine). Specifically, DeRubeis and colleagues (1990) were

interested in whether early reductions in cognitive vulnerability predicted subsequent symptom relief. The results of this study revealed that cognitive vulnerability did in fact decrease from pre-treatment to mid-treatment in both treatment conditions. Thus, reductions in global, stable, and internal attributions were not unique to CT *during the early stages of treatment* given that the same effects were observed among those receiving an antidepressant. However, early reductions in cognitive vulnerability did significantly predict subsequent reductions in depression symptoms for those patients receiving CT, but *not* for those patients treated with an antidepressant. Additionally, early changes in depression symptoms did not significantly predict later cognitive changes, which helps further clarify the nature of this causal relationship by ruling out the opposite sequence of change in cognition and depression (DeRubeis et al., 1990). In a review of this clinical trial, Hollon and colleagues (1990) report that the ASQ findings satisfied four of five criteria of statistical mediation established by Baron and Kenny (1986) and that cognitive vulnerability emerged as a potential mediator of CT's prophylactic effects on reducing depression symptoms and even risk of relapse/recurrence (Hollon, Evans, & DeRubeis, 1990, p. 126).

Another study examined changes in cognitive vulnerability among 27 patients diagnosed with MDD who received 12 weeks of CT. During treatment, patients experienced significant reductions in cognitive vulnerability for attributions about the causes of negative events as measured by the ASQ ($d = 3.45$). Patients also demonstrated improvements on a composite measure of cognitive vulnerability that was calculated by subtracting the ASQ negative events score from the ASQ positive events score ($d = 2.04$).

In other words, patient's attributions for negative life events became less global, stable, and internal as they progressed through CT. Additionally, this study found that changes in ASQ scores was significantly associated with changes in depression symptoms (i.e., BDI scores) while controlling for symptom severity as baseline (r = -.59; Barber & DeRubeis, 2001).

Extant literature suggests that cognitive vulnerability is indeed modifiable through psychotherapeutic interventions such as CT. However, the aforementioned studies only examine short-term outcomes by exploring how CT reduces cognitive vulnerability during an acute course of CT. To expand on this line of work, researchers have begun to examine the long-term effects of CT on cognitive vulnerability by predicting risk of relapse following successful treatment for depression.

To investigate this question, one randomized controlled trial studied relapse rates in 158 patients with recent MDD who remitted with antidepressant treatment (i.e., amitriptyline or fluoxetine) but were still exhibiting residual symptoms for a duration of two to 18 months after treatment ended (Paykel et al., 1999). Patients were randomized to receive medication/clinical management alone or in combination with 16 sessions of CT over 20 weeks plus two booster sessions (i.e., treatment phase). A follow-up assessment was then conducted at 48-weeks post-treatment to assess relapse (i.e., follow-up phase). Those patients who received CT in combination with medication/clinical management had a significantly lower risk of relapse over the entire 68 weeks during both the treatment and follow-up phases (29% in the CT condition versus 47% in the clinical management alone condition; HR = 0.54). Thus, in this difficult-to-treat sample of

patients who were experiencing residual symptoms of depression after responding to antidepressant treatment, CT proved to be beneficial in reducing the risk of relapse and ameliorating persistent severe residual symptoms (Paykel et al., 1999). In a follow-up examination of mediators of relapse prevention in this sample, an unexpected result was found in that greater cognitive vulnerability at the beginning of the treatment phase was found to be associated with *lower* risk of relapse even when controlling for depression symptom severity at baseline. However, exploratory analyses revealed that this effect was driven by individuals with a tendency to give extreme responses (i.e., a score of a 1 "totally disagree" or a 7 "totally agree") at the item level of the ASQ. This pattern of extreme responding on the ASQ was conceptualized as an absolutist, dichotomous, black-or-white attributional style. Furthermore, this cognitive vulnerability was also found to mediate the relationship between the observed effects of CT and the risk of relapse/recurrence (Teasdale et al., 2001). Overall, this study suggests that CT may reduce the risk of relapse, specifically by focusing on modifying the manner in which patients process and respond to depressogenic information (e.g., extreme responses), rather than trying to modify the specific content of their depressive thoughts (e.g., the specific causal attributions made).

A similar investigation tested whether CT would help maintain cognitive vulnerability reductions experienced by patients with MDD during acute phase fluoxetine treatment. Patients received eight weeks of fluoxetine treatment and remitters (N = 132) were randomized to a medication continuation with or without CT during a six-month treatment continuation phase. While all patients exhibited significant reductions in

cognitive vulnerability during the acute phase of treatment with fluoxetine, there were differential effects of treatment condition on cognitive vulnerability during the continuation phase. Specifically, the CT plus fluoxetine group maintained acute phase improvements in cognitive vulnerability, whereas the fluoxetine only group exhibited a worsening of cognitive vulnerability. However, despite these differences in attributional style changes, relapse rates did not differ among the continuation treatment conditions (Petersen et al., 2004).

All in all, the extant literature reviewed above demonstrates that the ASQ is significantly associated with concurrent symptoms of depression (e.g., Blaney et al., 1980; Seligman et al., 1979; Sweeney et al., 1986) and can adequately differentiate between those with depression and those with different psychiatric disorders or without depression (e.g., Eaves & Rush, 1984; Hamilton & Abramson, 1983; Heimberg et al., 1989, 1987; Ingram et al., 1987). Importantly, longitudinal research points to the interaction between cognitive vulnerability and the experience of a negative life event in the etiology of depression (e.g., Fresco et al., 2006; Metalsky et al., 1982; Peterson & Seligman, 1981; Riskind et al., 1987), which supports the diathesis-stress framework of the *reformulated learned helplessness theory.* Additionally, research suggests that cognitive vulnerability decreases during CT of depression (Barber & DeRubeis, 2001; DeRubeis et al., 1990; Seligman et al., 1988), and also serves as an important predictor of risk of relapse irrespective of treatment modality (i.e., CT or antidepressants) (e.g., Paykel et al., 1999; Petersen et al., 2004; Teasdale et al., 2001).

Despite the utility of the ASQ as a measure of cognitive vulnerability, a few limitations are worth discussing. First, a primary concern of the ASQ is the low reliability for the global, stable, and internal subscales for both positive and negative events (Peterson et al., 1982). According to commonly accepted standards for internal consistency (Cortina, 1993; Cronbach, 1951; Gliem & Gliem, 2003; Tavakol & Dennick, 2011), the original reliability of the individual subscales range from unacceptable to poor to questionable (mean $\alpha = .54$). Additionally, the composite measure of cognitive vulnerability for negative ($\alpha = .72$) events barely reaches the threshold for an "acceptable" level of internal consistency, with the composite for positive events not being much higher ($\alpha = .75$). What is perhaps most concerning is that many of the aforementioned studies do not even report estimates of internal consistency in their publications. Some studies even cite that the original ASQ has "acceptable" reliability based on the internal consistency of the composite scores, but then proceed to use individual ASQ subscales in their analyses without acknowledging the poor reliability of these subscales observed in prior studies.

In addition to concerns about the psychometric properties of the ASQ, there have been some inconsistent findings within the literature using the ASQ as a measure of cognitive vulnerability in the context of depression. For instance, one study found no significant group differences between scores on the ASQ among treatment-seeking patients diagnosed with depression and non-depressed controls from the general population. This null finding was observed when examining the ASQ composite scores or any of the three subscales (i.e., global, stable internal) for both negative and positive

events (Hargreaves, 1985). Similar null findings were observed in a comparison of depressed and non-depressed inpatients, who did not differ on ASQ scores despite the fact that the depressed group also endorsed significantly higher BDI scores. Interestingly, the depressed inpatients did report significantly more negative attributions for the causes of the single most stressful event that they experienced over the past six months (Miller, Klee, & Norman, 1982). These findings also remained unchanged in follow-up analyses where the researchers had blind evaluators provide ratings of global/stable/internal attributions based on the patient's reported stressful life event, indicating that the null findings were not due to poor reliability of the scales or patient's misunderstanding of the questions. One interpretation from these findings is that cognitive vulnerability may be more important stressful life events that are idiographic and personally relevant in comparison to hypothetical events as assessed by the ASQ.

Another study failed to replicate the findings of Metalsky et al. (1982) by showing that cognitive vulnerability among college students did not predict depression symptoms two weeks later following a disappointing performance on an examination. Additionally, follow-up analyses revealed that global, stable, and internal attributions for poor examination performance actually resulted in students making more plans and preparations to study for the next examination, which contradicts what would be expected from the *reformulated learned helplessness theory* (Follette & Jacobson, 1987). Some mixed findings have also been documented in treatment studies investigating the ASQ as a predictor of treatment outcomes. In a large randomized controlled trial comparing CT to antidepressants in a sample of patients with moderate-to-severe depression, a composite

measure of cognitive dysfunction that included the ASQ did not predict treatment outcomes as measured by the HRSD for either treatment condition (Fournier et al., 2009). However, it should be noted that this composite measure also consisted of the DAS, the Hopelessness Scale (Beck, Weissman, Lester, & Trexler, 1974), and the Rosenberg Self-Esteem Scale (Rosenberg, 1965). As such, this null finding should be interpreted carefully given that Fournier and colleagues assessed cognitive dysfunction more globally rather than a more nuanced measure of cognitive vulnerability that emphasizes attributional styles as outlined by the *reformulated learned helplessness theory.*

In order to address some of these limitations, particularly the questionable reliability of the ASQ, Peterson and Villanova (1988) developed the Expanded Attributional Style Questionnaire (EASQ) that was lengthened to include 24 hypothetical negative events in comparison to the ASQ which only included six negative events. Additionally, the EASQ does not assess causal attributions for positive events because the *reformulated learned helplessness theory* "is not explicitly concerned with positive events" in the etiology of depression (Peterson & Villanova, 1988, p. 87). The six negative events from the original ASQ were retained and the other 18 negative events were derived from the College Schedule of Recent Experience (CSRE) – a life events questionnaire designed for college students (Marx, Garrity, & Bowers, 1975). The psychometric properties of the EASQ were first assessed using a sample of 140 undergraduates who completed the EASQ and BDI at baseline, and then returned four months later to report on actual negative events that occurred to them during that time period. As expected, increasing the length of the EASQ increased the internal consistency

of the global (α = .88), stable (α = .85), and internal subscales (α = .66) – all of which were higher than those found in the original validation of the ASQ (Peterson & Villanova, 1988).

Supporting its construct and predictive validity, all three of the EASQ subscales were significantly associated with concurrent depression symptoms (global r = .40, stable r = .19, internal r = .18). Moreover, baseline scores on the EASQ global, stable, and internal subscales were significantly associated with their respective attributions for the causes of negative events that each participant actually experienced over the four month follow-up period (rs ranging from .18 to .36) (Peterson & Villanova, 1988). One surprising finding from this study is that the internal subscale was not significantly correlated with the other two subscales (i.e., global and stable), with the same finding observed among the three dimensions of causal attributions for negative life events that were actually experienced. The authors note the difficulty in assessing the internal/external dimension and suggest that it may be a multidimensional construct similar to an external versus internal locus of control (Peterson & Villanova, 1988). In the first study examining the factor structure of the EASQ, each of the three subscales emerged as distinguishable and replicable factors. However, the items on the internal subscale tended to have lower factor loadings and weaker internal consistency compared to the global and stable factors (T. E. Joiner & Metalsky, 1999). Given that each factor is assessed in the same manner, the authors conclude that the weaker psychometric properties of the internal subscale is likely a construct-related issue rather than a measurement-related issue.

Despite the improved reliability of the EASQ, a drawback of this measure was that respondents now had to provide responses for 72 total items (i.e., three ratings for 24 events), which created a lengthy and time-consuming measure of cognitive vulnerability. As such, two studies were conducted to see if a short form (i.e., EASQ-SF) could be created without compromising the psychometric properties of the EASQ (Whitley Jr, 1991). The validation study utilized data from 427 undergraduates to identify the most robust events from the EASQ to include in the short form. To do so, the researcher a priori established the number of events to be 12 "to shorten the scale while retaining enough items to provide an acceptable level of reliability" (Whitley Jr, 1991, p. 366). The 12 items that demonstrated the highest corrected item-total correlations for the internal subscale were selected for inclusion in the EASQ-SF given that the internal subscale has consistently demonstrated the lowest reliability among all of the subscales. The results indicated that, according to conventional standards, internal consistency was good for the global subscale ($\alpha = .87$), acceptable for the stable subscale ($\alpha = .79$), and questionable for the internal subscale ($\alpha = .65$). Moreover, these three subscales were significantly correlated with each other (*r*s ranging from .31 to 64), although it should be noted that the correlation was lowest for the internal subscale. Along these lines, the internal dimension was the only subscale that was not significantly associated with a measure of depression, as assessed by the short form of the Minnesota Multiphasic Personality Inventory (MMPI) depression scale (global $r = .26$, stable $r = .23$, internal $r = .09$). Finally, each of the EASQ-SF subscales was significantly correlated with their respective subscales measured by the long form (*r*s ranging from .89 to 94).

A second cross-validation study largely replicated these findings, although slightly lower internal consistencies were observed compared to study one (global α = .76, stable α = .79, internal α = .59). All subscales were significantly inter-correlated (*r*s ranging from .14 to 49), with the internal subscale again demonstrating the lowest correlation. In study two, two additional measures of depression were administered: the BDI and the depression scale of the Profile of Mood States (POMS; McNair, Droppleman, & Lorr, 1992). Contrary to study one, all of the EASQ-SF subscales were significantly correlated with both measures of depression (*r*s ranging from .14 to 26) (Whitley Jr, 1991). Overall, it was concluded that the EASQ could be shortened without significantly sacrificing reliability or loss of convergence with depression symptoms. A confirmatory factory analysis also provided further evidence for the validity of the EASQ-SF. The global (average loading = .49) and stable (average loading = .51) factors were strongly correlated with each other (r = .60), and weakly correlated (r = .28), with the internal factor (average loading = .31) (Whitley, 1991).

In a commentary on the development of the EASQ-SF, Christopher Peterson (who developed the original EASQ) provided data from eight different samples (N = 983) indicating that the EASQ-SF is uniformly less reliable than the long form of the EASQ. Indeed, internal consistencies from the original EASQ are higher for the global (mean α = .84 versus .75), stable (mean α = .86 versus .76), and internal (mean α = .59 versus .47) subscales. Additionally, analyses of the item-total correlations in these eight samples indicate that the "best" items (i.e., hypothetical events) from the EASQ differ from sample to sample and from dimension to dimension (Peterson, 1991). Nonetheless,

Peterson and Whitely Jr. conclude that the decision between using the EASQ and EASQ-SF is one that must be made by the researcher depending on the context of the research study (Whitley, 1991). The EASQ-SF appears to be an adequate measure of cognitive vulnerability, particularly for situations when the length and duration of the questionnaire is an important consideration

Overall, extant research has provided support for the validity of both the EASQ and EASQ-SF as a measure of cognitive vulnerability for depression. First, a multitude of studies demonstrate that scores on the EASQ are positively associated with measures of depression in both clinical and non-clinical samples, with correlations ranging from .31 to .51 (e.g., Ahrens & Haaga, 1993; Brown & Silberschatz, 1989; Chang & Sanna, 2001; Cheng & Furnham, 2001; Kwon, 1999; Luten, Ralph, & Mineka, 1997; Whitley, Michael, & Tremont, 1991). Cognitive vulnerability as assessed by the EASQ-SF tends to be higher in patients with chronic/recurrent MDD in comparison to those patients with non-chronic MDD and those with no lifetime history of psychopathology (Riso et al., 2003). Moreover, the EASQ has also demonstrated positive correlations with other symptoms critical to depression, such as hopelessness, suicidal ideation, and rumination (e.g., Ahrens & Haaga, 1993; Haaga et al., 1995; Hirsch & Conner, 2006). For instance, one study found that hopelessness was positively associated with suicidal ideation, but only among those with high levels of cognitive vulnerability (i.e., one SD above the mean on EASQ total scores; Hirsch & Conner, 2006). Low cognitive vulnerability (perhaps an index of cognitive resilience) mitigated the effects of traumatic life events on suicidal ideation (Hirsch, Wolford, LaLonde, Brunk, & Parker-Morris, 2009) and is associated

with a lower lifetime prevalence of suicide attempts (Hirsch & Rabon, 2015), even after controlling for levels of hopelessness and depression. Cognitive vulnerability has also been found to be associated with increased negative affect (i.e., sadness) after writing about negative events as a method for inducing a sad mood (Ahrens & Haaga, 1993). A similar laboratory-based experimental study found a stronger positive relationship between EASQ scores and depression symptoms among undergraduates randomly assigned to a rumination induction condition (Lo, Ho, & Hollon, 2010). Moreover, two studies have revealed that women tend to report higher EASQ scores compared to men, with this index of cognitive vulnerability co-varying with symptoms of depression (Boggiano & Barrett, 1991). The EASQ global and internal subscales have also been associated with more severe depression symptoms in an LGBT sample (Lindquist, Livingston, Heck, & Machek, 2017). Considered together, correlational research provides strong evidence for the validity of the EASQ as a measure of cognitive vulnerability akin to depression.

Extending this line of work, longitudinal studies provide more robust support for the role that cognitive vulnerability plays in the etiology of depression, which is posited by the *reformulated learned helplessness theory*. Two prospective studies found that EASQ scores significantly predicted increased depression symptoms over time, but only among students who experienced a negative life event: high school students getting rejected from a prestigious university and college students being rejected from joining a fraternity/sorority (Abela & Seligman, 2000). Another study found that undergraduates exhibited higher levels of depression and anxiety over the course of five weeks, but only

if they were high in cognitive vulnerability and also experienced a stressful life event. Additionally, this effect was qualified by a three-way interaction with religiousness, which acted as a buffer against these deleterious effects (Luten et al., 1997). Overall, such prospective studies are consistent with the diathesis-stress model of depression outlined by the *reformulated learned helplessness theory.*

In order to assess cognitive vulnerability using more sophisticated research methodologies, studies have implemented ecological momentary assessments (EMA) to examine whether cognitive vulnerability impacts the moment-to-moment attributions that people make for events they actually encounter in their daily lives, and whether these attributions relate to discrete fluctuations in mood (Swendsen, 1998). Using a sample of people from France, participants were signaled five times each day by electronic devices to provide reports of negative events, attributions, and depressed moods. Cognitive vulnerability as assessed with the EASQ-SF predicted causal attributions for negative events that participants actually encountered across diverse contexts in their environments. Moreover, these causal attributions predicted fluctuations in depressed mood throughout the day (Swendsen, 1998). These findings were partially replicated in a multi-site study using a nearly identical EMA methodology in two samples from the United States and France (Swendsen & Compagnone, 2000). After controlling for clinical and demographic variables, EASQ-SF scores emerged as the strongest predictor of the 4,000+ attributions that people actually made for the causes of negative events within the course of their daily lives. However, in contrast to the prior study, attributional styles did not predict depression levels following the experience of a negative life event. From a

cross-cultural perspective, one interesting finding from this study is that cognitive vulnerability was significantly higher among the American sample compared to the French sample, highlighting potential cultural differences in attributional styles that warrant consideration (Swendsen & Compagnone, 2000).

Given that evidence has accumulated for the utility of the EASQ as a measure of cognitive vulnerability, some studies have examined whether EASQ scores decrease during CT. In a highly-cited component analysis study of CT, 150 outpatients diagnosed with MDD were randomly assigned to one of three treatment conditions: 1) a treatment focused exclusively on the behavioral activation (BA) component of CT, 2) a treatment that included both BA and the cognitive restructuring skills to modify automatic thoughts (AT), but excluding the focus on core beliefs, or 3) the full CT treatment that used cognitive restructuring for automatic thoughts and core beliefs, as well as BA. Despite excellent therapist adherence, there was no evidence that the full CT package predicted superior acute or long-term outcomes (i.e., scores on the BDI-II and HRSD) compared to the BA and AT treatment conditions. Moreover, all treatments were equally effective in reducing cognitive vulnerability. The most surprising result of this study comes from secondary analyses examining mechanisms of change. Consistent with Beck's *cognitive theory,* the researchers predicted that early reductions in cognitive vulnerability would be associated with later decreases in depression, with this effect being the strongest for the CT condition. However, the opposite pattern emerged – early reductions in the EASQ global and stable (but not internal) subscales were associated with subsequent reductions in depression in the BA treatment, but not among those who received the full CT

treatment (Jacobson et al., 2000). In an attempt to explain these unexpected findings, they hypothesize that perhaps depressed patients who respond well to BA may be more likely to modify their negative causal attributions if they are regularly increasing their contact with naturally reinforcing contingencies in their environment. However, this hypothesis is merely speculative and is yet to be empirically tested.

In a follow-up investigation of this component analysis study, researchers tested whether extreme responding on the EASQ served as a predictor of acute outcomes and relapse (Ching & Dobson, 2010). Extreme responding was operationalized as a patient endorsing the lowest or highest response on any of the EASQ subscale items (i.e., a response of a one or a seven respectively). This was quantified in two ways – the total number and the mean number of extreme responses by each patient. Results of this study revealed that extreme responding on the EASQ did not predict or moderate acute treatment outcomes. Global attributions decreased for the CT group, while global and internal attributions decreased for the BA group. There were no significant changes in stable attributions across treatment. Contrary to what was expected, extreme responding did not significantly change across treatment for either the CT or BA condition, and changes in extreme responding did not co-vary with changes in scores on the BDI-II or HRSD. When examining long-term outcomes over a one-year follow-up, extreme responding did not significantly predict relapse in either group. In exploratory analyses, extremely low scores on the EASQ global subscale did predict relapse in the CT group when controlling for post-treatment BDI scores. However, this finding was no longer statistically significant after applying a Bonferroni correction to control for the number of

statistical tests conducted (Ching & Dobson, 2010). Although this study demonstrates that some facets of cognitive vulnerability decrease across treatment, these effects were not specific to CT. In fact, the patients who received BA surprisingly experienced reductions in more domains of the EASQ compared to those who received CT. Additionally, these findings were puzzling in that having a more dichotomous or extreme cognitive style was not predictive of acute treatment outcomes or relapse.

In a recent secondary analysis of this component analysis study, researchers examined the role of cognitive vulnerability and explanatory flexibility in the recovery from depression (Moore, Fresco, Schumm, & Dobson, 2017). Explanatory flexibility was conceptualized as the degree to which individuals balance their interpretation of events with historical and current contextual factors and make effective use of all available information. This construct was computed as the standard deviation of stable and global items of the EASQ. Results indicated that all patients demonstrated reductions in cognitive vulnerability, regardless of treatment condition or responder status. This effect was stronger among those patients classified as treatment responders (d = 1.69 versus .62). Pre-post changes in cognitive vulnerability was only associated with pre-post changes in HRSD scores for those in the AT condition. Patients did not exhibit increases in explanatory flexibility, although there was a non-significant trend for those who received BA. Pre-post changes in explanatory flexibility were also not correlated with pre-post change in HRSD scores, although there was a non-significant trend for the CT condition, with this correlation primarily being driven by those patients who responded to CT. When evaluating long-term outcomes over a two-year follow-up, there was a

significant two-way interaction between cognitive vulnerability and explanatory flexibility. Specifically, those patients with high cognitive vulnerability and high explanatory flexibility (i.e., "flexible pessimists") had the greatest risk of relapse, whereas those patients with low cognitive vulnerability and high explanatory flexibility (i.e., "flexible optimists") had the lowest risk (Moore et al., 2017).

In one other treatment study, researchers explored whether cognitive vulnerability as measured by the EASQ-SF differed among patients diagnosed with MDD and seasonal affective disorder. Additionally, researchers sought to test whether cognitive vulnerability predicted differential treatment responses in each of these groups. Although the two depressed groups did not differ on pre-treatment EASQ-SF scores, their cognitive vulnerability was greater than scores among non-depressed samples reported elsewhere. Furthermore, pre-treatment cognitive vulnerability was a significant predictor of a failure to respond to antidepressant pharmacotherapy in the MDD group. Cognitive vulnerability did not significantly predict outcomes of light therapy among those with seasonal affective disorder (Levitan et al., 1998). It should be noted that this study did not report whether cognitive vulnerability changed as a function of treatment and also did not examine individual EASQ-SF subscales as predictors of treatment response. There was also no CT condition, which would have permitted whether cognitive vulnerability predicts differential treatment response in CT versus antidepressant treatments.

To my knowledge, the aforementioned studies are the only treatment studies that have examined cognitive vulnerability as assessed by the EASQ or EASQ-SF within the context of CT and the *reformulated learned helplessness theory*. This is a noteworthy gap

in the literature given that both of these measures tend to have stronger reliability compared to the ASQ, which has been used much more frequent in CT treatment outcome studies. Additionally, both of these measures only assess negative hypothetical events whereas the ASQ also assesses positive events. This transition to focusing primarily on negative events appears to be more consistent with how the *reformulated learned helplessness theory* describes the etiology of depression. Although it would be posited that CT would be the treatment best suited for reducing cognitive vulnerability (i.e., EASQ/EASQ-SF scores) in patients with depression, findings remain mixed and inconclusive about whether this is indeed the case. First, although reductions in cognitive vulnerability have been observed in patients who participate in CT, other treatments such as BA have comparable effects, despite less emphasis being placed on modifying maladaptive cognitions such as negative causal attributions (Jacobson et al., 2000). Second, there is insufficient evidence to suggest that reductions in EASQ/EASQ-SF scores are concurrently or prospectively associated with short-term symptom reductions in CT or long-term outcomes (i.e., relapse, recurrence). Third, examining these measures in novel ways (i.e., extreme responding, explanatory flexibility) has largely been unable to predict acute treatment outcomes, and only one study identifying a significant predictor of relapse (Moore et al., 2017). All things considered, these mixed or null findings likely stem from either issues with measurement or theory. It could be the case that neither the EASQ nor the EASQ-SF sufficiently assess the construct of cognitive vulnerability and need to be modified and improved. It is also possible that the *reformulated learned helplessness theory* does not adequately specify the role that

cognitive vulnerability plays in the etiology of depression. In other words, it fails to provide a specific mechanism by which causal attributions (and their interaction with negative life events) actually lead to a person developing depression.

Hopelessness Theory of Depression

Although the *reformulated learned helplessness theory* provided an attributional framework for the foundations of learned helplessness, some researchers argued that it did not provide a fully articulated theory of depression that was supported by empirical evidence. Rather, it was contested that earlier conceptualizations (Abramson et al., 1978) emphasized an attributional model of what contributes to learned helplessness in humans and only briefly discussed the possible implications for depression. Specifically, Abramson and colleagues (1989) suggest that these global, stable, and internal causal attributions were over-emphasized in prior theories. Although empirical support for the link between causal attributions and depression has been well established, this facet of cognitive vulnerability serves as one of many proximal causes that lead to depression. Furthermore, a key distinction that Abramson and colleagues make is that they propose a causal mediation pathway for the etiology of depression, whereby the link between cognitive vulnerability and depression is mediated by hopelessness. Specifically, in their *hopelessness theory of depression,* they propose a chain of distal and proximal causes along a pathway that are hypothesized to culminate in the development of a specific subtype of depression that they call *hopeless depression* (Abramson, Metalsky, & Alloy, 1989).

In their *hopelessness theory,* the chain of events begins with the occurrence of a negative life event (a distal cause). In contrast to Seligman's theory, which posits that uncontrollability is a key contingency in promoting a sense of learned helplessness, Abramson et al. suggest that the event does not need to be viewed as uncontrollable. Rather, their theory proposes that a person merely needs to perceive that a negative life event has occurred in order to begin the series of event that are presumed to cultivate in hopelessness, and in turn, depression. Next in their etiological sequence are the attributions that people make for the causes of a negative life event that has a high level of importance to them. Consistent with the *reformulated learned helplessness theory*, they present a diathesis-stress model of depression whereby this cognitive vulnerability (i.e., negative attributions) interact with the experience of a negative life event to causally increase the likelihood of developing hopelessness and thus, depression (Abramson et al., 1989).

Additionally, a titration model is proposed for this interaction such that there is an inverse relationship between the level of a person's cognitive vulnerability and the severity of the negative life event needed to develop depression. For instance, an individual who has a high cognitive vulnerability may only need to experience a relatively mild negative life event in order to develop depression. Along the same lines, an individual with low cognitive vulnerability would need to experience a highly stressful life event in order to become depressed (Abramson et al., 1989). Overall, these aspects of the *hopelessness theory* were delineated to help address the question of why some people

develop hopelessness and depression after experiencing a negative life event whereas others do not.

Although cognitive vulnerability is viewed as an important diathesis in the etiology of depression in the *hopelessness theory,* there are a few key differences in contrast to the *reformulated learned helplessness theory*. First, the *hopelessness theory* deemphasizes the role that making internal attributions has in the etiology of depression. The *reformulated learned helplessness theory* postulated that making internal attributions for the causes of a negative life event would increase one's likelihood of developing depression through the mechanism of lowering self-esteem. However, Abramson and colleagues (1989) argue that attributing a life event to an internal cause may not be inherently maladaptive and consequently lower self-esteem. Rather, they propose that making an internal attribution may actually serve an adaptive function in certain contexts. For instance, if a woman is told by her boss that she may lose her job (negative life event) due to repeated failures to arrive on time and an incapability to complete her required duties (internal attribution), it may actually increase her motivation and effort to overcome these difficulties to keep her job (adaptive response). To support this notion, they cite a body of social psychological research which highlights that people's self-esteem is largely influenced by comparing themselves with others (e.g., Festinger, 1954; Morse & Gergen, 1970; Rosenberg, 1965; Schachter, 1959; Tesser & Campbell, 1983), not necessarily the internal or external attributions that people make for the causes of events. Moreover, they suggest that lowered self-esteem should occur when people have inferred negative characteristics about themselves (discussed below) that they view as

important to their general self-concept and are unlikely to be changed. As such, the *hopelessness theory* does not incorporate internal attributions for the causes of negative life events as a facet of cognitive vulnerability in the etiology of depression (Abramson et al., 1989). Although there are legitimate concerns regarding the internal subscale of the ASQ (e.g., low reliability, weak correlations with other subscales and measures of depression), it should be noted that this was decision was largely made on a theoretical basis rather than an empirical one.

A second modification of the *hopelessness theory* is the addition of two additional attributional styles that are viewed as cognitive vulnerabilities in the causal chain of events that may lead to the development of depression. First, Abramson and colleagues (1989) hypothesize that the inferred *negative consequences* of stressful life events moderate the relationship between the negative events experienced and the likelihood of developing hopelessness depression. This hypothesis emerged from research suggesting that the inferred negative consequences for stressful and negative life events impacts the risk of becoming depressed, independent of the causal attributions (i.e., global, stable, internal) made for these events. For instance, in a study of high school teachers reporting high levels of teaching-related stress, depression symptoms were significantly associated with negative inferences about the consequences of the stress associated with teaching in an urban school system (e.g., needing to miss future school days). However, contrary to the *reformulated learned helplessness theory*, depression symptoms were not related to the global, stable, or internal causal attributions regarding these stressful life events (Hammen & DeMayo, 1982). Another study examined the causal attributions and

consequences (e.g., degree of negative affect, uncertainty, predicted recurrence) of personally relevant negative events among three groups of college students: those who were moderately depressed, stressed but not depressed, and neither depressed nor stressed. Surprisingly, results revealed that the three groups of college students did not differ in their causal attributions. However, the moderately depressed college students were more likely to report negative consequences as a result of the stressful life event compared to the other two groups (Hammen & Cochran, 1981). As such, the *hopelessness theory* of depression establishes that, in addition to global and stable causal attributions, perceived negative consequences from a stressful life event are another aspect of cognitive vulnerability that serves as a proximal contributory cause in the etiology of depression.

A second aspect of cognitive vulnerability that was added to the *hopelessness theory* was the inferred *negative self-characteristics* in response to a stressful life event, including inferences about one's self-worth, desirability, adequacy, and personality. Similar to perceived negative consequences, these inferred negative self-characteristics are presumed to moderate the relationship between the negative life events and the risk of developing hopelessness depression. This addition to the *hopelessness theory* was primarily based on Aaron Beck's cognitive conceptualizations of patients with depression who tended to exhibit patterns of making exaggerated, over-general, and absolute conclusions about their self-worth in response to negative life events (Beck, 1976; Beck et al., 1979). These negative self-characteristics largely map onto the core beliefs (i.e., fundamental ideas about the self) of worthlessness, inadequacy, and unlovability

commonly reported by depressed people as outlined by Aaron and Judith Beck (Beck, 2011). Following the same logic as the aforementioned cognitive vulnerabilities, inferring negative self-characteristics (i.e., low self-worth) regarding a negative life event would causally lead the individual to feel hopeless and, eventually, depressed.

In summary, the *hopelessness theory* posits that cognitive vulnerability is comprised of the causal attributions (global and stable, but not internal), perceived consequences, and inferred negative self-characteristics that a person experiences in the context of a negative life event. Consistent with a diathesis-stress model, individual differences in these cognitive vulnerabilities interact with the experience of a negative life event in the development of depression. Critically, Abramson and colleagues (1989) underscore the importance of causal mediation in their theory on the etiology of hopelessness depression. That is, each causal factor contributes to the next causal factor in their model. When cognitive vulnerability interacts with a negative/stressful life event, it is proposed to engender a sense of hopelessness, which in turn leads to the onset of depression. They define hopelessness as being characterized by two key features: 1) pessimistic predictions about the occurrence of desirable and reinforcing outcomes (a negative outcome expectancy), and 2) expectations that one will be unable to change the likelihood a negative or aversive outcome (a helplessness expectancy). This hopelessness was hypothesized to be sufficient in and of itself to causally lead to developing depression.

Within the *hopelessness theory*, the key role of hopelessness in the etiology of depression was based off of longitudinal research providing evidence for the temporal

precedence of hopelessness predicting future depression symptoms. For instance, in a study of 91 mildly depressed undergraduate students, hopelessness predicted depression symptoms five weeks later while controlling for initial depression symptoms at baseline. However, other types of depressive cognitions were not significantly associated with later depression symptoms (Rholes, Riskind, & Neville, 1985). One longitudinal study of 103 undergraduates tested the interaction between expectations for future negative outcomes (i.e., hopelessness) and cognitive vulnerability (i.e., ASQ scores) when predicting depression symptoms (i.e., BDI-II scores) six weeks later. The results of this study demonstrated that, after controlling for initial severity of depression, there was a positive association between cognitive vulnerability and depression symptoms six weeks later, but *only* for those individuals with high levels of hopelessness (one SD above the mean). There was no significant association between attributional styles and later depression symptoms in people with average levels of hopelessness, and interestingly, there was a negative relationship between cognitive vulnerability and later depression symptoms in people who reported low levels of hopelessness (once SD below the mean) (Riskind et al., 1987). Another longitudinal study examined the interaction between a stressful life event (i.e., childbirth) and dispositional optimism-pessimism (a proxy for hopelessness) when predicting post-partum depression symptoms three weeks later. As expected, even after controlling for prenatal depression symptoms, high levels of optimism (the opposite of hopelessness) conferred reduced risk of developing later depression symptoms. Additionally, this buffer effect was most pronounced for women with low levels of depression during the prenatal period, suggesting that optimism serves as a protective

factor against developing later depression in the context of a stressful life event (Carver & Gaines, 1987). Taken together, these longitudinal studies provided support for hopelessness prospectively predicting the depression symptoms at a later time. Such empirical support provided the foundation for Abramson and colleagues' hypothesis that hopelessness is causally land temporally inked to the development of depression.

Given that the *hopelessness theory* emphasizes the causal relationship between hopelessness and depression, longitudinal research studies provide the most robust support for the mechanisms underlying the etiology of depression. However, it is important to also consider cross-sectional correlational research that supports the relationship between hopelessness and depression. In a study of hospitalized inpatients, patients with depression reported significantly greater hopelessness following success and failure in a behavioral task compared to patients with MDD and comorbid schizophrenia, patients with schizophrenia but not MDD, and non-psychiatric controls (Abramson, Garber, Edwards, & Seligman, 1978). A similar study found that patients hospitalized for a major depressive episode also endorsed significantly more hopelessness compared to non-depressed patients who were diagnosed with a range of anxiety disorders, personality disorders, and/or schizophrenia, as well as a non-depressed control group of community participants (Hamilton & Abramson, 1983). Another study of nearly 200 patients diagnosed with MDD demonstrated that they reported significantly higher scores of hopelessness compared to patients with generalized anxiety disorder or a group of psychiatric patients with no mood or anxiety disorder diagnosis (Beck, Riskind, Brown, & Steer, 1988).

Although these are merely a sampling of studies, the aforementioned research studies provide empirical support for the relationship between hopelessness and depression, both prospectively and concurrently. Given this body of literature, Abramson and colleagues (1989) specify in their theory that when a negative life event interacts with cognitive vulnerability to foster feelings of hopelessness, an individual may be at an increased risk to develop depression, specifically their hopelessness depression subtype.

The hopelessness depression subtype includes the following symptoms: sadness, retarded initiation of voluntary responses, apathy (i.e., anhedonia), suicidal ideation/behavior, fatigue, psychomotor retardation, sleep disturbances, difficulties concentrating, mood-exacerbated negative cognitions (e.g., self-blame, rumination), lowered self-esteem, and dependency. Overall, this hopelessness depression subtype largely maps onto the current nosology of MDD symptoms in the DSM-5, with the exception of a few key differences. First, one cardinal symptom reflects a motivational dysfunction in the initiation of voluntary responses, which stems from the helpless expectancy component of hopelessness. Additionally, they posit that the other principal symptom of sadness derives from the negative outcome expectancy facet of hopelessness. They also propose two additional symptoms that are not currently included in the DSM-5 nosology: lowered self-esteem and dependency. However, these two symptoms are conceptualized as ancillary and not experienced by all patients.

Overall, it should be noted that this taxonomy was largely driven by theoretical considerations, with the exception of the body of research linking hopelessness to depression and suicide (see Abramson et al., 1989 for a review). Additionally, the

hopelessness theory emphasizes that the hopelessness subtype of depression should be classified based on the amalgamation of causes, symptoms, course, therapy, and prevention of depression rather than symptomatology alone. For instance, the course duration of depression should be influenced by how long feelings of hopelessness last. From a therapeutic standpoint, any link in the etiological chain could serve as an opportunity for clinical intervention to treat patients with hopelessness depression. The experience of positive events that are experienced and associated changes in cognitive vulnerability (causes, consequences, self-characteristics) may also provide a path to recovery from hopelessness depression. Moreover, relapse and/or recurrence should be predicted by the reappearance of hopelessness after a person has remitted from their depression (Abramson et al., 1989).

In their highly influential publication, Abramson and colleagues presented preliminary evidence that contributed to the development of the *hopelessness theory of depression*. However, it should be emphasized that Abramson and colleagues did not provide a direct empirical test of their theory. Rather, they summarized and built off prior research spanning multiple subfields of psychology on attributional styles, hopelessness, and depression to propose a theory-based subtype of depression – hopelessness depression. Since it's original proposition, research has systematically examined the validity of the *hopelessness theory* and each of the components that make up the causal chain of events leading to hopelessness depression. As such, the empirical support for the *hopelessness theory* is discussed below.

One measure that was specifically developed to measure cognitive vulnerability akin to the *hopelessness theory* was the Cognitive Style Questionnaire (CSQ; Abramson & Metalsky, 1986). The CSQ is a modified and expanded version of the ASQ that specifically measures the three components that make up cognitive vulnerability according to the *hopelessness theory* of depression: causal attributions (global, stable, internal), negative consequences, and self-worth characteristics. In order to address the low reliability of the ASQ, the CSQ increased the number of hypothetical events from six to 12 positive and 12 negative events. An average score for each cognitive vulnerability facet is calculated, and a composite total score is calculated by averaging each of the subscales, with higher scores indicating greater cognitive vulnerability (i.e., more negative attributional styles). However, internal causal attributions are not included in this composite score given that the *hopelessness theory* does not view internal causal attributions as being central to the development of depression, but rather as a mechanism for decreasing self-esteem among people who are already depressed. Ratings for positive events are also not included in the composite measure of cognitive vulnerability given that the *hopelessness theory* primarily emphasizes negative life events as being crucial to their diathesis-stress model of depression. Moreover, the CSQ was primarily designed for use in college student populations, with the hypothetical events covering achievement and interpersonal related domains (e.g., academic performance, interpersonal relationships).

Haeffel and colleagues (2008) provided a review of the reliability and validity of the CSQ from over 30 studies. Overall, the CSQ demonstrated significantly improved

reliability compared to the ASQ, with internal consistencies ranging from .88 to .96 for the composite measure of cognitive vulnerability. Although the individual subscales (i.e., causes, consequences, self-worth) demonstrated lower reliability than the composite measure, internal consistencies were still strong as they ranged from .83 to .91 (see review by Haeffel et al., 2008). The test-retest reliability of the CSQ has also been established, with a correlation of $r = .80$ over a one-year period (Alloy et al., 2000). Research using factor analysis has also demonstrated that each of the three components of cognitive vulnerability load onto one factor, suggesting that these subscales assess one common factor of cognitive vulnerability (Hankin, Lakdawalla, Carter, Abela, & Adams, 2007). The major drawback of the CSQ is that it was designed specifically for college populations, which limits its generalizability and use in other contexts.

One of the earliest empirical tests of the *hopelessness theory* used a retrospective design by comparing the prevalence rates of depressive disorders among undergraduates who were determined to be either high-risk or low-risk based on their level of cognitive vulnerability (Alloy, Lipman, & Abramson, 1992). The researchers were particularly interested in the prevalence of MDD and their proposed hopelessness depression subtype over the past two years in a sample of undergraduates who were not currently depressed and were not exhibiting significant depression symptoms. Students who scored in the highest quartile of the ASQ composite for negative events were determined to be high-risk (n = 39), whereas those individuals in the lowest quartile were determined to be low-risk (n = 45). Using retrospective diagnostic interviews, the high-risk group had a greater prevalence of past MDD (51% vs. 18%) and hopelessness depression (42% vs. 11%), and

they also experienced significantly more episodes of these disorders than the low-risk group (one vs. zero episodes). In addition, the high-risk group had more severe major depressive episodes in the past. There was also a non-significant trend for the high-risk group to have longer major depressive episodes (10 weeks vs. five weeks) (Alloy et al., 1992). Overall, this study was influential in that it provided evidence for the notion that people who are cognitively vulnerable have more disadvantageous outcomes in key aspects of depression (e.g., prevalence rates, symptom severity, duration). Additionally, these results paralleled the hopelessness depression subtype that is proposed by the *hopelessness theory*.

These results were replicated in the Temple-Wisconsin Cognitive Vulnerability to Depression (CVD) Project – a two-site, prospective longitudinal study designed to examine the etiology of depression within the context of cognitive vulnerability (Alloy & Abramson, 1999). During phase I of the study, Alloy and colleagues used a retrospective design to recruit undergraduates from Temple University and the University of Wisconsin without a diagnosis of a current Axis I disorder, with the exception of past MDD that had remitted for a minimum of two months. A subsample of participants were identified as cognitively high-risk (n = 173) or low-risk (n = 176) based on scores on the CSQ and DAS, which were included to assess cognitive vulnerability as postulated by the *hopelessness theory* and Beck's *cognitive theory*, respectively. Those participants determined to be high-risk had to have scores in the highest quartile on both the CSQ and DAS, while those in the low-risk group scored in the lowest quartile. As expected, those individuals who were high-risk reported a significantly higher lifetime prevalence of

depressive disorders. This finding was consistent when assessing for DSM criteria for MDD (38.7% vs. 17%, odds ratio = 3.01), and even the project's specified criteria for hopelessness depression (39.9% vs. 11.9%, OR = 4.87). There was also a non-significant trend for the high-risk group having a higher lifetime prevalence of subclinical depression, coined "minor depression (22% vs. 11.9%, OR = 2.11, p = <0.08). No significant group differences were observed for other psychiatric disorders (e.g., anxiety disorders, substance/alcohol use disorders, personality pathology), indicating that cognitive vulnerability may be a specific risk factor that is unique to depression and not other mental health conditions. The high-risk group also endorsed more severe major depressive episodes than the low-risk group, although they did not differ with regards to earlier onset or episode duration (Alloy et al., 2000). Moreover, current depression symptoms were controlled for, which rules out the possibility that more severe current depressive symptoms may be accounting for reports of greater lifetime prevalence of depression.

Similar to the Temple-Wisconsin CVD Project, a similar retrospective high-risk behavioral paradigm was utilized to examine the relationship between cognitive vulnerability and the lifetime prevalence of psychological disorders among a large sample of undergraduates (N = 887) (Haeffel et al., 2003). This particular study used scores on the CSQ and the DAS to predict a lifetime history of a host of psychological disorders. After controlling for current depression symptoms, scores on the CSQ significantly predicted a lifetime history of MDD (OR = 1.37), but the DAS was not a significant predictor (OR = 0.94). To examine the unique contribution of each measure,

scores on the CSQ and DAS were simultaneously entered into the same logistic regression model. The same results held, with CSQ scores predicting a lifetime history of MDD (OR = 1.48), with scores on the DAS failing to reach significance (OR = 0.78). When examining the two-way interaction between the CSQ and DAS in secondary analyses, participants with high CSQ scores demonstrated the greatest lifetime prevalence rates of past RDC major depression, regardless of whether they had high or low DAS scores (Haeffel et al., 2003). These results suggest that cognitive vulnerability and dysfunctional attitudes may be distinct constructs, with the CSQ demonstrating a stronger ability to detect a past history of MDD.

Other studies have provided correlational evidence for the *hopelessness theory* using the CSQ as a measure of cognitive vulnerability. One study of 220 undergraduates found that the CSQ composite scores was positively correlated with symptoms of hopelessness depression as measured by the Hopelessness Depression Symptom Questionnaire (HDSQ; $r = .42$; Metalsky & Joiner, 1997) and also scores on the Hopelessness Scale ($r = .32$) (B. Gibb, Alloy, Abramson, & Marx, 2003). Another study using an undergraduate sample found that the CSQ was significantly correlated with scores on the BDI-II ($r = .36$) and was able to discriminate between those with and without a history of MDD. That is, those people who remitted from a major depressive episode endorsed significantly greater scores on the CSQ in comparison to controls who have never been depressed, although this effect was rather small (Cohen's $d = .29$) (Haeffel et al., 2005). In a cross-sectional study of undergraduates, CSQ scores significantly were significantly associated with concurrent depression symptoms, and

those individuals determined to be cognitively high-risk had significant greater depression compared to those who were classified as low-risk (Oliver, Murphy, Ferland, & Ross, 2007).

Overall, the aforementioned studies indicate that cognitive vulnerability as conceptualized by the *hopelessness theory* (i.e., attributions about the causes, consequences, low self-worth) is associated with a greater risk for clinically significant depressive disorders, including their proposed hopelessness depression subtype. However, one limitation to these studies is that they provide no test of the direction of this relationship given the correlational design using retrospective reports. That is, they do not delineate whether cognitive vulnerability contributes to the onset of depression, or on the contrary, if depression leads to the development of these cognitive styles.

To address this limitation, longitudinal studies have systematically investigated the temporal nature of the relationship between cognitive vulnerability and depression. The Temple-Wisconsin CVD Project followed a subsample of high- and low-risk undergraduates for 2.5 years during phase II of the study. Specifically, Alloy and colleagues were interested in whether cognitive vulnerability prospectively predicted the onset of future depressive episodes while controlling for important clinical covariates (e.g., sex, initial depression symptoms, prior MDD episodes). Indeed, they found that the high-risk group was at a significantly higher risk of developing future MDD (OR = 6.66), hopelessness depression (OR = 6.77), and minor depression (OR = 3.53) compared to the low-risk group. When examining other diagnoses, the two risk groups did not differ in incidence of anxiety disorders not comorbid with depression or other disorders.

Additionally, among those individuals with a past history of depression, those who were high-risk had a significantly greater likelihood of experiencing a recurrence of MDD, hopelessness depression, and minor depression (Alloy et al., 2006). Consistent with the *hopelessness theory*, these findings from the Temple-Wisconsin CVD Project provide support for the notion that cognitive vulnerability is a significant factor that is predictive of future onsets and recurrences of MDD and hopelessness depression.

Drawing on extant research demonstrating a powerful link between hopelessness and suicide, the *hopelessness theory* also speculates that suicidality may be a core symptom of hopelessness depression. Indeed, findings from the Temple-Wisconsin CVD Project support this notion by testing the link between cognitive vulnerability and suicidality. Cognitive vulnerability significantly predicted future suicidality such that those individuals classified as high-risk were significantly more likely to exhibit suicidality (28% vs. 12.6%) during the 2.5-year follow-up period, even after controlling for prior history of suicidality, which is known to be the most robust predictor of future suicidality (Joiner, 2007). Additionally, high cognitive vulnerability was associated with a greater lifetime prevalence of suicidality (Abramson et al., 1998).

Given that the *hopelessness theory* proposes a diathesis-stress model of depression, numerous studies have examined the interaction between cognitive vulnerability and life stress when predicting subsequent depression symptoms. A multitude of studies using both clinical and non-clinical samples have demonstrated that cognitive vulnerability as measured by the CSQ interacts with negative life events to consistently predict future depression symptoms over time periods ranging from days to

weeks to one year (e.g., Abela, Aydin, & Auerbach, 2006; Abela, Brozina, & Seligman, 2004; Abela & Seligman, 2000; Alloy, Reilly-Harrington, Fresco, Whitehouse, & Zechmeister, 1999; Alloy et al., 1999; Gibb, Beevers, Andover, & Holleran, 2006; Haeffel et al., 2007; Haeffel, Abramson, Brazy, & Shah, 2008; Hankin, Abramson, Miller, & Haeffel, 2004; Kwon & Laurenceau, 2002). Research has also replicated these prospective findings cross-culturally (e.g., Calvete, Villardón, & Estévez, 2008; Stewart et al., 2004) and also in children and adolescents (Abela & Seligman, 2000; Dixon & Ahrens, 1992; Hilsman & Garber, 1995; Kercher & Rapee, 2009; Prinstein & Aikins, 2004). Two other longitudinal studies found that while CSQ scores interacted with life stress to significantly predict the onset of future major depressive episodes or depression symptoms, this interaction did not significantly predict subsequent anxiety symptoms or the development of an anxiety disorder (Hankin et al., 2004; Metalsky & Joiner, 1992). Thus, similar to evidence described above, this cognitive vulnerability appears to be specific to depression and not anxiety disorders. Experience sampling studies also demonstrate that, among high-risk individuals, cognitive vulnerability predicts attributions for actual negative events experienced and subsequent increases in depressed mood (Swendsen, 1997). Another prospective study found that cognitive vulnerability as measured by the CSQ interacted with low self-esteem to predict depressive mood reactions four days later, but only for high school students who experienced a negative outcome academically (e.g., getting denied admissions from a competitive university) (Abela, 2002). Critically, this pattern of findings has been consistently replicated whether utilizing the CSQ as a composite measure of cognitive vulnerability or examining the

specific subscales of the CSQ. Overall, extant literature has largely supported the diathesis-stress nature of the *hopelessness theory* whereby cognitive vulnerability predicts future depression symptoms, particularly in the face of a negative life event. A strength of these studies is that they predict the development of future depression symptoms prospectively, which provides insight into the direction of the relationship between cognitive vulnerability and the etiology of depression.

Another key aspect of the *hopelessness theory* that has been empirically tested is the role of hopelessness as a potential mediator of the link between cognitive vulnerability and the onset of depression. The first study to test this mediation hypothesis utilized a sample of 152 undergraduates and assessed cognitive vulnerability using the EASQ and CSQ, symptoms of depression, anxiety, hopelessness, and negative life events at baseline and five weeks later. Consistent with the diathesis-stress component of this theory, each fact of cognitive vulnerability (i.e., causes, consequences, self-worth) interacted with negative life events to prospectively predict greater increases in depression symptoms over time. This effect was not present among those with low cognitive vulnerability, even in the face of life stress. Additionally, this pattern of findings was unique to depression, as there was no significant interaction when predicting state nor trait anxiety. In a test of mediation, two of the three interactions (causes and self-worth, but not consequences) were partially mediated by hopelessness (Metalsky & Joiner, 1992). Another study found a significant three-way interaction between global and stable causal attributions, low self-esteem, and a negative outcome on a college examination when predicting depression symptoms four days later. Moreover, this

relationship was mediated by hopelessness (Metalsky, Joiner, Hardin, & Abramson, 1993). Hopelessness has also been found to mediate the prospective relationship between cognitive vulnerability and suicidality, whereas this relationship was not mediated by other key risk factors for suicide such as past suicidality, past MDD, and symptoms of borderline personality disorder (Abramson et al., 1998). These mediation findings have also been replicated in children as well. In a sample of 60 youth psychiatric inpatients with ages ranging from nine to 17, cognitive vulnerability interacted with high levels of life stress to predict increases in depression (but not anxiety) symptoms two months after patients were discharged. Moreover, changes in hopelessness during this time period mediated the link between the cognitive vulnerability by negative events interaction and later depression symptoms (Joiner, 2000). One other study used a sample of 153 unemployed people and found that event-specific hopelessness (i.e., about future employment) mediated the link between the interaction between the stress of unemployment and cognitive vulnerability when predicting depression symptoms (Lynd-Stevenson, 1997).

Despite the aforementioned studies that have provided support for the role of hopelessness mediating the relationship between cognitive vulnerability and depression, other studies have failed to replicate these findings. Multiple studies have found that, although cognitive vulnerability interacts with the experience of negative life events to prospectively predict increases in depression symptoms, this relationship was not mediated by hopelessness. These null mediation findings have been found in adult and undergraduate samples (e.g., DeVellis & Blalock, 1992; Houston, 1995; Johnson, 1992;

Kapçi & Cramer, 2000; Morris & Tiggemann, 1999), and samples of children and adolescents (Abela, 2001; Hankin, Abramson, & Siler, 2001). Similarly, cognitive vulnerability has been found to interact with interpersonal stressors to predict increases in suicidality 10 weeks later in college students. However, hopelessness did not mediate this relationship (Joiner & Rudd, 1995). Considering this body of literature, evidence for the mediating role of hopelessness has been mixed and inconclusive. Although cognitive vulnerability is a strong predictor of future depression symptoms, this causal pathway outlined by the *hopelessness theory* is lacking sufficient empirical support and warrants attention in future research.

Finally, the remaining component of the *hopelessness theory* that has been investigated is the symptom profile of the hopelessness depression subtype that is proposed by Abramson and colleagues. In a recent systematic review, Liu and colleagues evaluate five studies that have empirically examined this subtype of depression (Liu, Kleiman, Nestor, & Cheek, 2015). First, a taxometric analysis was conducted in 531 outpatients with a primary diagnosis of MDD to observe the latent structure of symptoms in five proposed subtypes of depression, including the hopelessness depression subtype. This study failed to identify a discrete hopelessness depression subtype and also suggested that this symptom profile had poor internal consistency (Haslam & Beck, 1994). A second taxometric analysis study of 160 adolescent inpatients diagnosed with MDD demonstrated significant associations between hopelessness and nearly all of the hypothesized symptoms of the hopelessness depression subtype. However, when evaluating the latent structure of the relationship between hopelessness and these

symptoms, they did not find support for the existence of a hopelessness depression subtype (Whisman & Pinto, 1997). However, it should be noted that this study was limited by its small sample size, which may have precluded this study from successfully identifying a hopelessness depression subtype, particularly if it has a low base rate. The same research group conducted a study of 80 adult inpatients diagnosed with MDD and demonstrated differential hopelessness depression symptom profiles for those with high versus low levels of hopelessness. Specifically, patients who reported high levels of hopelessness were discriminated from those with low levels of hopelessness on two cardinal symptoms of hopelessness depression: retarded initiation of voluntary responses (a motivational symptom) and depressed affect (an emotional symptom). The high-hopelessness group also endorsed greater levels of suicidal ideation, social difficulties, and cognitive dysfunction (Whisman, Miller, Norman, & Keitner, 1995). Despite these promising findings on the discriminant validity of hopelessness depression, this study did find a lack of internal consistency for the hypothesized symptom profile of this depression subtype ($\alpha = .42$; Whisman et al., 1995). Interestingly, a study of 39 children with depression between the ages of seven and 13 found a good level of internal consistency ($\alpha = .81$) when creating a composite measure of hopelessness depression symptoms using items derived from a variety of empirically validated measures (see Abela, Gagnon, & Auerbach, 2007 for a list of measures). Additionally, this study found that children exhibiting cognitive vulnerability in the context of a negative life event were more likely to receive a diagnosis of hopelessness depression compared to children with no evidence of this diathesis-stress interaction (Abela et al., 2007). The final study

evaluating this hopelessness depression subtype used structural equation modeling in three large samples of psychiatric outpatient (N = 3128) and a sample of Air Force cadets (N = 1404) who completed the BDI-II. Data pooled from these four samples provided evidence for a unified cluster of hopelessness depression symptoms that was statistically distinguishable from other depression symptoms (Joiner et al., 2001). Collectively, these studies provide mixed evidence for Abramson and colleagues (1989) hopelessness depression subtype as a distinct syndrome that is at the core of their *hopelessness theory*.

Cognitive Vulnerability: Gaps in the Literature

Since it's original publication, substantial research has empirically tested the *hopelessness theory of depression.* Considering the extant literature as a whole, certain components of this theory have yielded mixed results, mainly the causal and mediating role of hopelessness in the etiology of depression and the symptom profile of the proposed hopelessness depression subtype. However, the one aspect of this theory that has consistently garnered the most robust empirical support is for the strong link between cognitive vulnerability (i.e., attributions about the causes, consequences, and low self-worth associated with experiencing a negative life event) and depression. Indeed, a recent meta-analysis summarized this relationship by examining 52 longitudinal studies comprised of 12,594 participants. Results revealed a positive correlation between cognitive vulnerability and subsequent depression symptoms (r = .27; Huang, 2015).

Despite this, less research has been devoted to examining whether cognitive vulnerability as outlined by the *hopelessness theory* decreases as a function of CT. Although research has shown that dysfunctional attitudes and global/stable/internal

causal attributions decrease in depressed patients participating in CT (e.g., Barber & DeRubeis, 2001; DeRubeis et al., 1990; Seligman et al., 1988), this research needs to be extended to the framework of the *hopelessness theory*, which also incorporates inferred negative consequences and perceived low self-worth as two key dimensions of cognitive vulnerability. Moreover, many prior studies revealing reductions in causal attributions during CT assessed cognitive vulnerability using the ASQ, which suffers from poor reliability and also includes attributions for positive events, which are de-emphasized in revised cognitive models of depression. This is problematic given that meta-analytic work suggests that attributions for negative life events demonstrate greater associations with future depression symptoms compared to attributions for positive life events (Huang, 2015). As such, testing whether CT reduces cognitive vulnerability in patients with MDD should be done with measures of cognitive vulnerability that have improved psychometric properties and emphasize negative events only (i.e., EASQ, EASQ-SF), which align more closely with the *hopelessness theory*. However, even studies that have done so have failed to demonstrate that reductions in cognitive vulnerability are specific to CT (Jacobson et al., 2000), which underscores the need for additional research on the effects of CT on changes in cognitive vulnerability.

Although research has evaluated how cognitive vulnerability changes during CT for depression, less research has utilized cognitive vulnerability as a predictor of acute treatment outcomes. That is, does cognitive vulnerability at baseline serve as a prognostic indicator of the extent to which a patient will respond to treatment? Research in this is critical because it may facilitate the identification of cognitively vulnerable individuals at

an intake assessment who are less likely to respond to a standard course of CT. Having such information could have direct clinical implications by informing treatment decisions made by clinicians at the outset of treatment, such as recommending that a patient with MDD may need different treatment (e.g., antidepressants) or additional treatment (e.g., combination of CT plus antidepressants). Additionally, if greater cognitive vulnerability does in fact predict a poorer prognosis in CT, perhaps clinicians can dedicate more time and efforts to modifying these negative attributional styles during treatment.

Although treatment response is a critical outcome to investigate, another key acute treatment outcome that warrants attention is that of dropout. Premature termination from treatment is an important issue because even highly efficacious treatments like CT are unlikely to benefit patients who dropout before receiving an ample "dose" of treatment. Meta-analytic work suggests that nearly 20% of patients will drop out if CT in controlled clinical trials (Gloaguen, Cottraux, Cucherat, & Blackburn, 1998), with dropout rates being dramatically higher (50%) in clinical settings that are more externally valid, such as private practice (Persons, Burns, & Perloff, 1988). To my knowledge, no studies have examined whether cognitive vulnerability predicts dropout in CT treatment. If cognitive vulnerability is indeed predictive of later dropout in CT, clinicians might be able to easily identify individuals at risk of dropping out early in treatment, and in turn, take differential efforts to reduce the incidence of dropout during CT.

Perhaps most importantly, a host of research studies have demonstrated that cognitive vulnerability as described by the *hopelessness theory* is associated with a greater lifetime prevalence of MDD, prospectively predicts the onset of future major

depressive episodes, and is associated with concurrent depression symptom severity (Alloy et al., 2000; Alloy et al., 2006). As such, it is reasonable to hypothesize that cognitive vulnerability may serve as an enduring and demonstrable risk factor for depression. Along these lines, some researchers conceptualize cognitive vulnerability as a "deep schematic cognitive structure" that may persist even after successful treatment (Teasdale et al., 2001, p. 348). In fact, some prominent CT researchers have even suggested that, "CT does not reduce the tendency for depressives to generate negative thoughts in distressing situations, but rather it inculcates a set of skills that helps them deal with these thoughts when they do occur" (Barber & DeRubeis, 1989, p. 450). Barber and DeRubeis (1989) refer to this as the activation-deactivation model, which postulates that CT does not actually modify the schema themselves, but rather the depressive schema becomes deactivated while making another schema more accessible and salient. In other words, the depressive schema becomes latent but remains intact, and may become reactivated when a significant prompting event occurs (e.g., experiencing a negative life event). As such, theoretical conceptualizations of cognitive vulnerability view this construct as a latent, trait-like cognitive style that is relatively stable and persists over time. This is supported by research suggesting that cognitive vulnerability persists over the lifespan (Burns & Seligman, 1989; Tiggemann, Winefield, Winefield, & Goldney, 1991) and predicts poor long-term functioning later in life (Peterson, Seligman, & Vaillant, 1988). Indeed, many theoretical models intellectualize cognitive vulnerability as a cognitive style that is "stable but not immutable" (Hankin & Abela, 2005, p. 35).

That is, although cognitive vulnerability tends to be relatively trait-like and stable over time, it is not resistant to change under certain circumstances (e.g., psychotherapy).

Within this framework, it is surprising that less research has been devoted to investigating whether cognitive vulnerability predicts risk of relapse after successful CT. This is a noteworthy oversight given that relapse remains a significant problem in MDD, which is notorious for it's chronic and recurrent course (American Psychiatric Association, 2013). In fact, one meta-analysis of 28 studies including 1,880 adults found that, even for those individuals successfully treated with CT, 30% will relapse within one year and 54% will relapse within two years after terminating treatment (Vittengl, Clark, Dunn, & Jarrett, 2007).

Despite these high rates of relapse, CT remains the frontline intervention for MDD given that meta-analyses suggest that, on average, patients treated with CT have lower rates of relapse compared to those treated with antidepressants (30% vs. 60%; Gloaguen et al., 1998). Furthermore, data from multiple large-scale randomized controlled trials indicate that patients withdrawn from CT have lower relapse rates, lower depression symptom severity, and higher rates of sustained remission compared to patients withdrawn from antidepressants (e.g., Dobson et al., 2008; Hollon et al., 2005; Kovacs, Rush, Beck, & Hollon, 1981). Although not statistically significant, it is also worth mentioning that there is a trend for patients with moderate-to-severe MDD who participate in and terminate from CT to have lower rates of relapse even compared to those who continue taking antidepressants during follow-up periods (31% vs. 47%; Hollon et al., 2005). Although these studies suggest that CT has enduring effects that

persist beyond treatment termination (see Hollon, Stewart, & Strunk, 2006 for a review), it remains imperative to identify what factors confer a greater risk of relapse, even after successful CT. Given it's strong association with past and future depressive episodes and it's enduring, trait-like nature, cognitive vulnerability may be a key factor in identifying a remitted patient's susceptibility for relapsing.

Some studies have found that dysfunctional attitudes (Simons et al., 1986) and mood-induced dysfunctional attitudes (i.e., cognitive reactivity; Segal et al., 2006, 1999) predict relapse after CT. However, other studies have failed to establish that cognitive vulnerability that emphasizes attributions for negative life events predicts greater risk for relapse after CT (e.g., Petersen et al., 2004; Teasdale et al., 2001). This has led researchers to examine novel methods for assessing cognitive vulnerability, such as "extreme responding" which may reflect a dichotomous and absolute cognitive style that may serve as a risk factor for relapse. However, results from these studies have been mixed and inconclusive, with some studies providing evidence for extreme responding predicting relapse (Teasdale et al., 2001), whereas others have failed to replicate this finding (Ching & Dobson, 2010).

Critically, very few studies have explicitly tested whether cognitive vulnerability according to the *hopelessness theory* predicts relapse after CT. This is a key limitation given that an advantage to the *hopelessness theory of depression* is that it provides a framework for not only the etiology of MDD, but also depressive relapse (i.e., the return of clinically significant depression symptoms following a brief period of remission). That is, people with greater cognitive vulnerability will be more likely to have relapses when

confronted with negative life events compared to people who do not exhibit these vulnerabilities (Abramson et al., 1989).

As reviewed above, various measures of cognitive vulnerability for depression have been used in cross-sectional, prospective, and treatment studies. In this study, cognitive vulnerability was assessed with the EASQ-SF *hopelessness theory* revision (EASQ-SF-HT). This measure of cognitive vulnerability was chosen for a few reasons. First, in contrast to the EASQ and EASQ-SF, this revised version also assesses perceived negative consequences and inferred low self-worth as attributions when experiencing a negative life event. This is a key revision given that it directly parallels the conceptualization of cognitive vulnerability in the *hopelessness theory*. Second, in contrast to the ASQ, versions of the EASQ have demonstrated stronger psychometric properties and should provide a more internally consistent assessment of cognitive vulnerability. Third, the EASQ-SF-HT also has a key advantage over the CSQ in that it is not specifically designed for college students, which may improve the generalizability of any research findings with this measure. Similar to the aforementioned measures, scores on the EASQ-SF-HT have been found to prospectively predict subsequent increases in depression symptoms, particularly for those with higher baseline levels of depression (Barnum, Woody, & Gibb, 2013). It has yet to be examined whether cognitive vulnerability as assessed by the EASQ-SF-HT – a more reliable and parsimonious assessment of cognitive vulnerability that has been revised to directly parallel the *hopelessness theory* – predicts risk for relapse after successful CT.

Purpose of This Study

This study primarily seeks investigate the extent to which cognitive vulnerability as outlined by the *hopelessness theory* decreases during an acute course of CT, and if greater cognitive vulnerability predicts poorer treatment response, dropout, and risk of relapse following CT. In this study, I utilize the EASQ-SF-HT as a measure of cognitive vulnerability to examine whether it serves as a significant predictor of acute treatment outcomes (i.e., depression symptom reductions) in CT. Moreover, no study to my knowledge has tested whether cognitive vulnerability predicts dropout during a course of CT. Given that little is known about the link between cognitive vulnerability and treatment engagement (i.e., dropout), it is essential to investigate if and how cognitive vulnerability predicts dropout from CT.

Additionally, relapse remains a common problem in MDD, even for those patients who respond to treatment. One possible explanation for the high risk of relapse in MDD is that some people may have greater cognitive vulnerability, even at the end of CT treatment. That is, those patients with a greater tendency to make negative attributions about the causes, consequences, and their self-worth in the context of negative life events may be at a greater risk of relapsing after responding to CT treatment. As reviewed above, a majority of studies investigating relapse in the context of MDD have mainly compared relapse rates among different treatment conditions (e.g., CT versus antidepressants). However, less research has been devoted to testing whether greater post-treatment cognitive vulnerability may increase one's risk of relapse after they have been successfully treated. As such, the primary aim of this study is to investigate whether

cognitive vulnerability as measured by the EASQ-SF-HT is a significant predictor of relapse following successful CT.

Overall, this study aims to build upon the prior literature by providing a more sophisticated and nuanced understanding of how cognitive vulnerability as outlined by the *hopelessness theory* may be impacting both acute treatment outcomes and long-term follow-up outcomes in patients diagnosed with MDD who participate in CT. There are four main objectives in this study: (1) test if cognitive vulnerability decreases during a 16-week course of CT, (2) evaluate how patients' cognitive vulnerabilities predict treatment response (i.e., changes in depression symptoms) during CT; (3) examine whether patients' cognitive vulnerabilities predict dropout from CT, and; (4) investigate whether cognitive vulnerability predicts risk of relapse among those patients who successfully responded to a course of CT.

Hypotheses

In evaluating these four main objectives, I propose the following primary hypotheses:

Hypothesis 1: The first aim of this study is to test whether cognitive vulnerability decreases during an acute course of CT. Researchers have largely conceptualized cognitive vulnerability as "stable but not immutable" (Hankin & Abela, 2005). That is, although cognitive vulnerability may represent a more enduring and trait-like cognitive style, it is still amenable to change under particular circumstances. I believe that CT, which seeks to modify maladaptive thinking patterns to reduce symptoms and improve functioning, is one such circumstance. Considering prior research evidencing reductions

in cognitive vulnerability across CT (e.g., Barber & DeRubeis, 1989; DeRubeis et al., 1990; Jacobson et al., 2000; Seligman et al., 1988), I hypothesize that cognitive vulnerability will significantly reduce across a 16-week course of CT. I expect this to be the case for the composite measure of cognitive vulnerability, as well as each individual subscale (i.e., global, stable, internal, consequences, self-worth).

Hypothesis 2: The second aim of this study is to evaluate how patients' cognitive vulnerabilities predict treatment response to CT (i.e., changes in depression symptoms). I hypothesize that greater cognitive vulnerability at intake (i.e., EASQ-SF-HT total scores) will be a robust predictor of subsequent slope of depression symptom change over the 16 weeks of CT. More specifically, I expect that greater cognitive vulnerability will predict shallower slopes of symptom change throughout CT. In other words, I believe that patients with greater overall cognitive vulnerability at intake will experience slower reductions in depression symptoms during CT.

A majority of CT treatment-outcome studies have not examined whether each of the five cognitive vulnerability subscales individually predict treatment outcomes during CT. As such, in order to provide more specificity on which dimensions of cognitive vulnerability may predict treatment response, I will also test whether scores on the EASQ-SF-HT global, stable, internal, consequences, and self-worth subscales at intake predict subsequent slopes of symptom change during CT for depression. Given prior research demonstrating that each of these subscales are associated with greater depression symptom severity, I predict that each of these five subscales will predict shallower slopes of symptom change throughout CT. In other words, I believe that patients with greater

cognitive vulnerability on each of these subscales at intake will experience slower reductions in depression symptoms during CT.

Hypothesis 3: The third aim of this study is to examine whether cognitive vulnerability at the intake assessment predicts dropout during a 16-week course of CT. Given that no studies to my knowledge have tested this systematically, I make no a prior hypothesis about the significance or direction of this relationship.

Hypothesis 4: The fourth and primary aim of this study is to investigate whether cognitive vulnerability predicts risk of relapse among those patients who successfully responded to a course of CT. Prior research has established that greater cognitive vulnerability predicts the onset of future major depressive episodes (Alloy et al., 2006) and predicts a greater risk of relapse after CT (e.g., Moore et al., 2017; Paykel et al., 1999; Teasdale et al., 2001). Based on this evidence, I predict that, among treatment responders, greater cognitive vulnerability at the end of treatment will confer greater risk for relapse in the one year following CT termination.

Given research suggesting that different dimensions of cognitive vulnerability as correlated with future depression symptoms (see meta-analysis by Huang, 2015), I will also test whether post-treatment scores on the EASQ-SF-HT global, stable, internal, consequences, and self-worth subscales predict greater risk of relapse. Doing so seeks to provide more specificity on which dimensions of cognitive vulnerability may predict risk of relapse among those patients who responded to CT. Specifically, I predict that each of the five EASQ subscales will predict greater risk of relapse in the year following treatment termination for those patients who were successfully treated with CT.

Methods

Data Collection and Cleaning

All research for this study was conducted in the Depression Treatment and Research Clinic (DTRC) located at the Ohio State University (OSU). The OSU institutional review board (IRB) reviewed and approved all research activities associated with this study prior to the beginning of data collection.

Participants

The sample is comprised of 126 adults with a primary diagnosis of MDD who participated in outpatient treatment as part of a research study examining predictors of treatment outcomes in CT for depression. Data collection for this research study began in October 2014 and ended in July 2016. All patients were provided treatment free-of-charge and were eligible to receive a total of 16 weeks of CT. Recruitment was conducted using advertisements and word-of-mouth in the central Ohio region. People who were interested in participating in the study were directed to contact the DTRC study staff to complete a pre-screening assessment by telephone. Upon first contact, participants completed a brief phone screen that assessed for the following criteria: likely to meet criteria for current MDD, stable on any antidepressant medication or not taking any antidepressant medication (with no plans to initiate pharmacotherapy during the course of

the study), and willing to commit to 16 weeks of treatment. Based on the pre-screening, those individuals deemed appropriate for the study were scheduled for an initial intake assessment. At the intake assessment, full inclusion and exclusion criteria were assessed to determine participant eligibility for enrolling in the study.

Inclusion criteria for participation in the treatment study were the following: (a) a diagnosis of MDD, according to criteria established by the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; American Psychiatric Association, 2000); (b) age of 18 years or older, and; (c) willing and able to provide informed consent. Exclusion criteria were the following: (a) current Axis I disorder other than MDD if it was determined to be the primary diagnosis and if it required treatment other than that being offered (e.g., a course of exposure therapy); (b) psychosis or Bipolar I disorder; (c) subnormal intellectual potential (IQ < 80); (d) clear indication of secondary gain (e.g. court ordered treatment), and; (e) current suicide risk sufficient to preclude treatment on an outpatient basis. In addition to meeting these criteria, patients on antidepressant medication were asked to maintain a stable dosage over the course of the study. All patients provided informed consent prior to participating in all research activities.

Out of 193 people invited to complete an initial intake assessment, 43 did not attend their scheduled appointment and one person only completed a portion of the assessment before opting out of the research study. Of the 149 people who completed the intake assessment, 23 were ineligible to participate in the research study. Potential patients were excluded for the following reasons: insufficient symptoms to be diagnosed with a MDD (n = 8), a history of Bipolar I disorder or manic episodes (n = 6), depression

did not constitute the primary diagnosis (n = 4), substance dependence within the last 6 months (n = 3), presence of a psychotic disorder (n = 1), and unwilling to end current psychotherapy (n = 1).

Sample demographics. The final sample consisted of 126 patients who met inclusion criteria to participate in the research study and receive CT. Enrollment in the study consisted of 74 females (59%) and 52 males (41%). The mean age of patients was 32 years (SD = 13.06, range 18-70). The ethnic composition of the sample was 82% white (n = 103), 7% African-American (n = 9), 8% Asian (n = 10), 2% Hispanic (n = 3), and 1% American Indian (n = 1). In terms of education level, the majority (72%) of the sample had completed part of college (n = 53), or earned a 4-year college degree (n = 30) or a 2-year college degree (n = 7). The majority of the sample (59%) reported having never been married, whereas 23% reported being currently married or cohabitating.

Diagnoses and symptom severity at intake. All 126 patients met diagnostic criteria for current MDD as assessed by the SCID-I (see measures section below) at intake. At intake, patients had a mean BDI-II score of 32.80 (SD = 8.81, range 11-56), indicating that, on average, patients were experiencing severe depressive symptoms. The majority of patients (71%) had at least one comorbid Axis I diagnosis. The number of comorbid diagnoses ranged from one to five. Anxiety disorders were the most common comorbidity, and among these, social anxiety disorder was reported most frequently (n = 54). Seventy-one patients reported recurrent depressive episodes (ranging from two to five previous episodes). Table 1 summarizes diagnostic specifiers and comorbid Axis I diagnoses across all patients.

Assessors and Therapists

Five advanced graduate students served as both study assessors and CT therapists. Patients were quasi-randomly assigned to assessors and therapists with only two constraints. First, the assessor who conducted a patient's intake assessment was never assigned to be their therapist. Second, the therapist's current caseload of patients was considered given that the therapist needed to have an opening to see a new patient. Therapists received approximately 60 hours of clinical training in CT, with a focus on experiential learning through role-play. Therapists received weekly individual and group supervision over the course of treatment from principal investigator, Daniel R. Strunk, Ph.D. Therapists followed the principles and procedures described in the *Cognitive Therapy of Depression* treatment manual (Beck et al., 1979).

Therapy Session Frequency

Therapy sessions were scheduled to take place twice per week for the first four weeks for all patients. After the fourth week of treatment, the majority of patients decreased their session frequency to one session per week, but patients were offered the option of continuing to schedule biweekly sessions up to week 12 of treatment. Following week 12 of treatment, all patients had sessions only once per week. The mean number of sessions that patients received in the first four weeks of treatment was 6.12 (SD = 1.51, range 1-8) and the mean number of sessions patients received in the first 12 weeks of treatment was 13.13 (SD = 3.88, range 1-24). Over the full course of treatment the mean number of sessions received per patient was 15.93 (SD = 5.14, range 1-28). These

represent conservative estimates of patients' session frequency as such estimates include those patients who were deemed treatment dropouts, as defined below.[3]

Follow-up Period

Patients were followed naturalistically for one year after treatment termination. Specifically, all patients were sent monthly surveys via Qualtrics. First, patients were asked to indicate the specific two-week time period where their depression symptoms were the worst since treatment ended. Second, they retrospectively completed the Patient Health Questionnaire-9 (PHQ-9; see measures section below) assessing their depression symptoms for this specific two-week time period that they indicated. Finally, patients were asked to describe any treatment changes since they terminated from CT, such as starting a new treatment (e.g., psychotherapy, medication) or modifying the dose of their medication. Patients were also asked to indicate the date of when this treatment change occurred.

Given that the primary outcome variable of interest for the follow-up period is risk of relapse, only patients who responded to CT during the 16 weeks of treatment could be included in analyses of relapse during the follow-up period. In other words, a patient could not be classified as having relapsed (i.e., developed a new major depressive episode) if they never responded to treatment and recovered from the depressive episode that was present during treatment. Treatment response was defined by the following two criteria: 1) not having a DSM-IV-TR diagnosis of MDD at the post-treatment assessment, and 2) a score less than 12 on the Hamilton Rating Scale for Depression (HRSD; see

[3] When those patients who were determined to be dropouts were excluded, the mean number of sessions per patient was: 6.44 over the first four weeks of treatment, 14.30 across the first 12 weeks of treatment, and 17.77 over the full course of treatment.

measures section below). Using these criteria, 72 of 126 patients were classified as treatment responders (57%) and thus, were eligible to be included in follow-up analyses. Of these 72 patients, 62 provided at least one follow-up response (M = 5.65, SD = 3.61).

Measures

Psychiatric Diagnoses. At the intake evaluation, psychiatric diagnoses were assessed with the Structured Clinical Interview for the DSM-IV (SCID-I; First, Spitzer, Gibbon, & Williams, 2002).

Predictor Variable: Cognitive Vulnerability.

Expanded Attribution Styles Questionnaire – Short Form – Hopelessness Theory Revision (EASQ-SF-HT). The EASQ-SF-HT is a self-report questionnaire that assesses patients' attributions to negative life events. Patients are presented with 12 hypothetical situations (sample items: "you are fired from your job," "you experience financial difficulties") and are asked to vividly imagine themselves in each of these situations as if it was happening to them right now. Specifically, patients are instructed to think about the primary causes of these negative life events if they were actually occurring in their life. First, patients provided a text description of the primary cause of this negative event. Then, patients answered five questions using a 1-7 Likert scale assessing their attributions for the causes of this event along the following subscales: global, stable, internal, consequences, and self-worth. Subscale scores for each of the 12 events were averaged, with higher scores indicating more negative attributions (i.e., greater cognitive vulnerability). In the present study, we used the same 12 events from the original EASQ-SF (Whitley Jr, 1991), and adapted this measure to include

attributional ratings on the consequences and self-worth dimensions as proposed by *hopelessness theory*. An EASQ total score was calculated by averaging each of the subscale averages, with the exception of the internal subscale which is consistent with the *hopelessness theory* of depression (Abramson et al., 1989). The EASQ-SF-HT has been used in prior research and has demonstrated strong psychometric properties (Barnum et al., 2013). Internal consistencies for the EASQ-SF-HT in the present study are as follows: EASQ total score α = .91 (pre-treatment) and α = .94 (post-treatment), global subscale α = .64 (pre-treatment) and α = .79 (post-treatment), stable subscale α = .80 (pre-treatment) and α = .87 (post-treatment), internal subscale α = .70 (pre-treatment) and α = .58 (post-treatment), consequences subscale α = .76 (pre-treatment) and α = .87 (post-treatment), and self-worth subscale α = .86 (intake) and α = .85 (post-treatment).

Acute Outcome Measures

Beck Depression Inventory II (BDI-II) **–** The BDI-II is one of the most commonly used self-report questionnaires to assess symptoms of depression (Beck, Steer, & Brown, 1996). The BDI-II consists of 21 items and asks patients to rate their depression symptoms on a 4-point Likert scale ranging from zero to three (sample item: 0 = "I do not feel sad"; 3 = "I am so sad or unhappy that I can't stand it"). The 21 items are summed to create a total score, with higher scores indicating more severe depressive symptoms. In the present study, the instructions for the BDI-II were modified to inquire about the past week (rather than the past two weeks) in order to assess the degree of symptom change on a session-to-session basis (for a similar modification, see Dimidjian et al., 2006). The BDI-II has demonstrated excellent internal consistency in both clinical

and non-clinical samples (α's > .92; Beck et al., 1996). Internal consistency in the present study was good at pre-treatment (α = .84) and excellent at post-treatment (α = .95).

Hamilton Rating Scale for Depression (HRSD) – The HRSD is a clinician-administered interview that assesses symptoms of depression (Hamilton, 1960). In the present study, the 17-item HRSD was modified to include the assessment of atypical symptoms (e.g., hypersomnia; for modification details see Reimherr et al., 1998). In order to assess inter-rater reliability of the HRSD, I calculated intraclass correlation coefficients (ICCs). To do so, an independent assessor observed a videotape of an assessment and completed the HRSD, which was compared to the HRSD ratings made by the assessor who conducted the original assessment. These ratings were completed for a subset of our sample (n = 30 patients) across all assessment time points (i.e., intake, week four, week 16). This amounted to a total of 60 HRSD ratings that were included in ICC analyses. ICCs in the present study were excellent (ICC = .95).

Dropout. Patients were classified as dropouts if they prematurely terminated treatment prior to receiving an adequate dose. Dropout was defined as terminating four weeks or more prior to their post-treatment assessment date which equates to a failure to complete 12 full weeks of treatment. Patients who missed sessions without declaring an intention to discontinue treatment were encouraged by their therapists to return to therapy. If a given patient's therapist had not been able to contact his/her patient after four weeks of repeated attempts, the patient was determined to be a dropout. If the patient returned to treatment at any time within this four-week period, they were not considered a dropout. Of the 126 patients, 17% (N = 21) were classified as dropouts. Of these 21

dropouts, 43% (N = 6) of patients dropped out early in treatment (i.e., prior to week four, but after completing at least two sessions) and 57% (N = 12) dropped out following the week four mark. One patient did not attend his/her first scheduled session and two patients only attended session one of treatment.

Follow-up Outcome Measures: Relapse

Patient Health Questionnaire – 9 (PHQ-9). The PHQ-9 is a brief, nine-item measure that assesses symptoms of depression that correspond directly to the nine DSM diagnostic criteria for MDD (Kroenke, Spitzer, & Williams, 2001). Given that the PHQ-9 consists of the actual nine criteria used to make a DSM diagnosis of MDD, this measure is frequently used in clinical and research settings as a diagnostic tool to make a diagnosis of MDD and to assess the severity of depression symptoms (Kroenke & Spitzer, 2002). Patients are instructed to consider how often they have bothered by their depression symptoms over the past two weeks. Each of the nine items is rated on a 4-point Likert scale ranging from zero ("not at all") to three ("nearly every day"). The nine items are summed to create a total score, with higher scores indicating more severe depression symptoms. A follow up, non-scored item assesses the extent to which the patient's depression symptoms have made it difficult for them to function in their daily lives such as at work, home, or socially (0 = "not difficult at all"; 3 = "extremely difficult"). Certain PHQ-9 score ranges have been established to correspond with minimal (0 – 4), mild (5 – 9), moderate (10 – 14), moderately severe (15 – 19), and severe (≥ 20) depression. The PHQ-9 has been empirically validated in 15 studies involving over 6,000 patients in primary care or outpatient settings and has demonstrated

comparable psychometric properties to other measures of depression symptoms. Moreover, a score of 10 or greater has been established as a clinical cutoff for the diagnosis of MDD, which has demonstrated a sensitivity of 88% and a specificity of 88% (Kroenke et al., 2001).

In the present study, relapse was defined as the first two-week period of the patient's worst depression symptoms where they had a score of 10 or greater on the PHQ-9. The date of relapse was coded as two weeks after the time when the patient indicated that his/her depression symptoms began in order to meet the two-week duration requirement in order be diagnosed with MDD according to the DSM. If a patient reported that they started a new treatment *and* had a PHQ-9 $\geq$ 10 either before or at the same follow-up response, they were classified as having relapsed under the assumption that they sought treatment due to a return of depression symptoms. If a patient did not indicate when their worst two-week period of depression symptoms was at the first follow-up response when they reported a PHQ-9 $\geq$ 10, their date of relapse was estimated as the middle point between the date of the follow-up response of when the PHQ-9 $\geq$ 10 and the date of the prior follow-up response where the PHQ-9 was less than 10 (i.e., when they were less symptomatic).

Patients were censored under two circumstances. First, a patient was censored if it was determined that the patient did not relapse by never reporting a PHQ-9 $\geq$ 10 during the follow-up period. In this case, the date of censoring was coded as the date of the final follow-up response that the patient provided. Second, a patient was censored if they

started a new treatment but did *not* report a PHQ-9 ≥ 10 either before or at the same time as when they started a new treatment.

A time to event variable (i.e., relapse or censor) was calculated as the amount of months after treatment termination when the event occurred. In all cases, the time to event variable rounded to the nearest half month (i.e., 0, 0.5, 1, 1.5, 2 months, etc.) for ease of interpretation.

Analytic Strategy

First, I will identify covariates to include in my primary analyses when predicting acute outcomes, dropout, and risk of relapse. In particular, I will examine whether the following demographic variables need to be statistically controlled for in my primary analyses: age, sex, medication status at intake, number of past major depressive episodes, and baseline depression severity scores (e.g., BDI-II, HRSD). To identify significant covariates in the models predicting acute outcomes, I will run one OLS regression model with all relevant demographic variables predicting outcomes (i.e., BDI-II, HRSD). To identify significant covariates in the models predicting dropout, I will run one logistic regression model with all relevant demographic variables predicting dropout. To identify significant covariates in the models predicting risk of relapse, I will run one Cox regression model with all relevant demographic variables predicting relapse. If any of the aforementioned demographic variables significantly predict outcomes in these models, then that variable will be included as a covariate in all models predicting that respective outcome.

Acute Outcomes. To predict acute outcomes (i.e., BDI-II, HRSD), I will use Hierarchical Linear Modeling (HLM) to estimate person-specific intercept and slope parameters in BDI-II and HRSD scores over time throughout 16 weeks of CT. I will utilize random coefficient models by modeling each patient's intercept and slope of symptom change as a random effect (Raudenbush, 2004). This approach allows for repeated measures of weekly symptom change on the BDI-II and HRSD during 16 weeks of CT to more precisely estimate symptom severity at the conclusion of treatment (for a similar appraoch see Strunk, Brotman, DeRubeis, & Hollon, 2010). The primary effects of interest in these HLM models are the interactions between cognitive vulnerability and time. The first cognitive vulnerability predictor of interest is the EASQ total score, which reflects a composite measure of cognitive vulnerability. Then, I will also model the following cognitive vulnerability predictors: EASQ global subscale, EASQ stable subscale, EASQ internal subscale, EASQ consequences subscale, EASQ self-worth subscale.

Dropout. To predict dropout, I will use logistic regression models, which are used when the dependent variable is categorical (i.e., dropout or non-dropout) (Hosmer Jr., Lemeshow, & Sturdivant, 2013). After identifying significant covariates to include in the logistic regression models, I will first enter the EASQ total score as a predictor of dropout. Then, in order to identify specific types of cognitive vulnerability that may predict treatment dropout, I will enter each of the EASQ subscales as individual predictors of dropout in separate logistic regression models.

Relapse. To predict relapse, I will use survival analyses, specifically using Cox regression models (Cox & Oakes, 1984). Cox regression (or proportional hazards regression) is a method for investigating the effect of an independent variable upon the time a specified event takes to occur (i.e., relapse or censor). Hazard ratios will be calculated as a measure of risk of relapse. After identifying significant covariates to include in the Cox regression models, I will first enter the EASQ total score as a predictor of relapse. Then, in order to identify specific types of cognitive vulnerability that may increase one's risk of relapse, I will enter each of the EASQ subscales as individual predictors of relapse in separate Cox regression models. Similar survival analyses have frequently been used in clinical trials and psychotherapy research to predict relapse after CT for depression (Hollon et al., 2005).

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Table 1. Major depressive disorder diagnostic specifiers, severity, and comorbidity.

Variable	% of sample and number of clients (N) meeting criteria
MDD specifiers	
One MDE (single episode)	44% (56)
≥ 2 MDE (recurrent)	56% (70)
With melancholic features	32% (40)
With atypical features	13% (16)
With seasonal pattern	2% (2)
MDD severity	
Mild	8% (10)
Moderate	65% (82)
Severe	22% (28)
Severity not assessed	5% (6)
Current Psychiatric Comorbidity	
Any Axis I comorbidity	71% (89)
Social anxiety disorder	43% (54)
Generalized anxiety disorder (GAD)	25% (32)
Specific phobia	20% (25)
Posttraumatic stress disorder (PTSD)	14% (17)
Dysthymia	6% (8)
Panic disorder	6% (7)
Agoraphobia	6% (7)
Obsessive-compulsive disorder (OCD)	4% (5)

Notes: MDD = major depressive disorder; MDE = major depressive episode. All psychiatric disorders were assessed using the Structured Clinical Interview for the DSM-IV (SCID-I; First, Spitzer, Gibbon, & Williams, 2002). The present study was designed and conducted before the DSM-5 was published.

Figure 1. Study flow chart.