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ORIGINAL ARTICLE

The Hopelessness Theory of Depression: Clinical Utility and Generalizability

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Abstract To date, "basic" research has dominated the empirical literature on hopelessness theory. The next logical step in this area of research is to determine if the theory can be used to help people. We conducted three studies to determine if the cognitive vulnerability factor featured in hopelessness theory could be reliably measured in diverse samples in a treatment context and if it could predict depressive therapeutic outcomes. Study 1 used a sample of male juvenile detainees ($n=296$; 70% from underrepresented groups) and found that cognitive vulnerability moderated the effectiveness of a social problem solving training intervention. Study 2 used a clinical sample of U.S. Veterans ($n=16$; 56% from underrepresented groups) enrolled in a cognitive behavioral therapy group for depression and found that cognitive vulnerability predicted post-therapy depressive outcomes. In both Study 1 and Study 2, higher levels of cognitive vulnerability resulted in poorer treatment outcomes (i.e., greater post-treatment levels of depressive symptoms). Study 3 used a clinical sample of U.S. Veterans ($n=76$; 67% from underrepresented groups) enrolled in a behavior activation group and found no effect of cognitive vulnerability on post-therapy depressive outcomes. The results of the three studies indicate that hopelessness theory's cognitive vulnerability construct can be reliably measured in diverse samples in real world clinical contexts and that it has the potential to be a useful predictor of clinical outcomes in the context of cognitively focused treatments.

Keywords Hopelessness theory · Cognitive vulnerability · Depression · Clinical context · Generalizability · Effectiveness

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According to the hopelessness theory of depression (Abramson et al. 1989), some individuals have a *cognitive vulnerability* that interacts with stressful life events to increase the likelihood of depression. Hopelessness theory defines cognitive vulnerability as the tendency of an individual to make particular kinds of inferences about the cause, consequences, and self-worth implications of stressful life events. Specifically, when faced with a stressful life event, an individual who has a cognitive vulnerability is likely to: (a) attribute the event to stable and global causes; (b) view the event as likely to lead to other negative consequences; and (c) construe the event as implying that he or she is unworthy or deficient. Individuals who generate these negative inferences are hypothesized to be at risk for hopelessness, which is viewed as a proximal and sufficient cause of depression.

Each component of the hopelessness theory has garnered at least some empirical support (Abramson et al. 1999; Haeffel et al. 2008; Liu et al. 2015; Russel et al. 2014). However, the most thoroughly tested and consistently

supported component of the theory is the cognitive vulnerability-stress hypothesis. A recent meta-analysis by Liu et al. (2015) found that 23 of the 24 studies conducted to date have supported the vulnerability-stress hypothesis featured in hopelessness theory [the one exception (Stange et al. 2013) also found support for the vulnerability-stress hypothesis, but only under conditions of low emotional clarity]. A review by Haeffel et al. (2008) found similarly strong support for the vulnerability-stress hypothesis. In brief, studies have found that cognitive vulnerability interacts with stressful events to predict the development of future depressive symptoms (see Haeffel et al. 2008 for review) and depressive disorders (Alloy et al. 2006; Hankin et al. 2004). These studies have shown that it is possible to take a group of never depressed individuals and predict which of them are most likely to develop depression based solely on individual differences in their cognitive style for interpreting life events (i.e., their level of cognitive vulnerability). For example, Hankin et al. (2004) found that cognitive vulnerability interacted with negative life events to predict clinically significant depression over a 2-year interval. This finding corroborates a much larger body of short-term prospective studies demonstrating that cognitive vulnerability interacts with stressors to predict depressive symptoms (Haeffel et al. 2008; Lakdawalla et al. 2007).

Taken together, prior studies support for the vulnerability-stress hypothesis featured in the hopelessness theory. The next logical step in this area of research is to determine if the theory has any clinical utility (Haeffel et al. 2008). Can measures of the vulnerability factor featured in hopelessness theory be used by therapists in real world clinical settings to help people? Specifically, can individual differences in cognitive vulnerability levels be used to determine *a priori* which patients are most likely to benefit from particular types of interventions?[1] The identification of moderators of intervention efficacy can shed light on for whom a particular intervention might work best (e.g., high vulnerable versus low vulnerable; Baker et al. 2008; Kazdin 2007). According to a recent review (Liu et al. 2015), hopelessness theory's cognitive vulnerability factor has been examined in one treatment study using a clinically depressed sample (Dobkin et al. 2007); this study examined the effect of CBT and adaptive inferential feedback on negative inferences and depressive symptoms. Four studies have targeted cognitive vulnerability in prevention designs using college samples (Dobkin et al. 2004; Haeffel 2010; Peters et al., 2011; Seligman 1999). To our knowledge, however, only one study has examined whether or not pretreatment levels of cognitive vulnerability could be used to predict post-therapeutic outcomes in a real world clinical setting. In this study, O'Conner et al. (2000) found that cognitive vulnerability, as conceptualized by the hopelessness theory, was a significant predictor of hopelessness outcomes in a hospitalized sample of parasuicide patients. Clearly, far more work is needed to establish the predictive power of cognitive vulnerability in real world clinical settings.

It is also important to underscore that almost all of the research on the cognitive vulnerability factor featured in hopelessness theory has used samples of relatively healthy youth and college students. (Liu et al. 2015). Using college students makes sense because they are ideal for testing a vulnerability-stress hypothesis in that they have varying levels of cognitive vulnerability, experience high levels of stress, and are at the peak age for developing depression (Hankin et al. 1998). However, focusing almost exclusively on this population leaves lingering questions about generalizability. It remains unclear if the reliability of the measurement of the cognitive vulnerability construct featured in the hopelessness theory generalizes to more diverse populations (e.g., underrepresented groups and older adults).

In summary, recent reviews by Liu et al. (2015) and Haeffel et al. (2008) indicate that the vulnerability-stress component of the hopelessness theory has proven its mettle (Popper 1959). However, it is unclear if the predictive power of cognitive vulnerability demonstrated in college samples generalizes to real world treatment settings and

[1] It is necessary to distinguish between the hopelessness theory of depression (Abramson et al. 1989) and its theoretical predecessor, the reformulated learned helplessness theory of depression (Abramson, Seligman, & Teasdale, 1978). Similarly to the hopelessness theory, the reformulated learned helplessness theory underscored the importance of causal attributions as vulnerability for depression. However, it defined cognitive vulnerability as the tendency to attribute negative life events to stable, global, and *internal* causes. The vulnerability factor featured in the hopelessness theory does not view attributions of internality as being central in the development of depression itself, but as an element that may decrease self-esteem among already-depressed individuals. In addition, the reformulated learned helplessness theory did not include the other two vulnerability components currently featured in the hopelessness theory—negative consequences and negative self-worth implications. Prior studies have examined the vulnerability factor featured in the reformulated learned helplessness theory in an intervention context. Prior randomized controlled trials have measured the vulnerability factor featured in the reformulated learned helplessness theory as a possible mediator of intervention efficacy. However, this theory is now over 40 years old and the measure that was used in these studies (the Attribution Style Questionnaire) is now outdated. Indeed, according to Liu et al., (2015) "… the still-prevalent focus on attributional styles (particularly as measured with the Attributional Style Questionnaire; Peterson et al., 1982) according to the reformulated learned helplessness theory (Abramson et al., 1978), rather than inferential styles according to the hopelessness theory, may be among the most significant obstacles to the advancement of the field" (p. 15). To our knowledge, cognitive vulnerability, as conceptualized by the hopelessness theory has not yet been focused on in a real world treatment context.

more diverse populations. It is possible that hopelessness theory is only important for identifying those who are at high-risk for developing depression, but not important once the disorder has developed. Indeed, the causes of mental and physical illness are not always important components of treatment planning and treatment. For example, smoking is thought to be a causal contributor to lung cancer, but quitting smoking alone would not be an adequate treatment for the illness. Determining the clinical usefulness of hopelessness theory remains an important empirical question. It is critical to determine if the cognitive vulnerability construct can be used to help patients and/or therapists in clinical settings.

We conducted three studies to determine the clinical utility and generalizability of hopelessness theory's cognitive vulnerability hypothesis. Study samples were chosen because they represented a diversity of ages, races, and problems of a clinical magnitude. A priority of this research was to maximize external validity in order to determine if cognitive vulnerability could be reliably measured and a useful predictor of clinical outcomes in real world applied settings. However, obtaining such external validity can be difficult because real world settings are not generally equipped to act as research centers. Fortunately, we found three clinical settings in which the therapists and administrators were willing to participate in our research. Study 1 used a sample of detained youth and studies 2 and 3 used samples of United States Veterans. The three studies covered a range of ages (from youth to adult), had a majority of participants from underrepresented racial groups, and had different treatment modalities (cognitively based and behavioral). Importantly, all three studies exhibited a high degree of external validity with staff members and therapists, rather than researchers, administering both the measures and the treatments. Also, all three studies used longitudinal rather than cross-sectional designs to ensure conclusions about temporal precedence could be made (Just et al. 2001). We evaluated the clinical utility of the hopelessness theory by assessing the extent to which baseline cognitive vulnerability levels could predict depressive outcomes in real world therapeutic contexts.

Like many theories of etiology, the hopelessness theory does not make predictions with regard to treatment implications. The theory is highly useful in predicting which individuals are at greatest risk for depression in the presence of stress, but the theory does not make claims about how cognitive vulnerability levels might influence the effectiveness of a given treatment. Thus, it is difficult to make specific predictions about how cognitive vulnerability will affect therapy outcomes. Needles and Abramson (1990) proposed a recovery model of depression based on the hopelessness theory of depression, however this theory focused on *enhancing* cognitve style (i.e., the interpretation of positive life events). The theory did not address the role of cognitive vulnerability in the prediction of therapy outcomes. We hypothesize, that cognitive vulnerability will be an indicator of a poorer prognosis because of its stability and rigidity. By early adolescence it is possible to detect meaningful and stable individual differences in how individuals cognitively interpret stressful life events (Cole et al. 2008; Nolen-Hoeksema et al. 1992). And, once cognitive vulnerability forms and stabilizes in early adolescence, it confers risk for depression throughout the life span (see Romens et al. 2009 for review). Research shows that cognitive vulnerability exhibits moderate to high stability during high school (Hankin and Abramson 2002), college (Alloy et al. 2000), and the rest of adulthood (Burns and Seligman 1989; Haeffel et al. 2005). Although highly stable, there may be unique environmental situations during which cognitive vulnerability can be altered. One situation is when it is directly targeted by a prevention or treatment intervention. Results of studies testing cognitive interventions for depression (e.g., cognitive therapy) demonstrate that cognitive vulnerability can be altered (e.g., Clark and Beck 2010; DeRubeis and Hollon 1995; Seligman et al. 1999). However, taken as a whole, research suggests that by early adulthood one is saddled (or blessed) with a level of cognitive risk that is relatively stable over time (Hankin 2008).

Thus, we predict that the presence of this cognitive risk factor would indicate a poorer prognosis and greater resistance to treatment. Higher levels of cognitive vulnerability likely reflect more extreme and entrenched cognitive styles, which are less amenable to change than more moderate and low levels of cognitive vulnerability. This means that individuals with higher levels of cognitive vulnerability should continue to have greater levels of cognitive vulnerability than those with moderate or low levels of cognitive vulnerability post-treatment, and thus, should report greater levels of depressive symptoms treatment (because it is associated with greater levels of depression concurrently and prospectively in prior work).

A second outcome of importance was with regard to the extent to which the vulnerability factor featured in the hopelessness theory could be reliably measured in non-undergraduate populations. To this end, we assessed the extent to which cognitive vulnerability could be reliably measured in these diverse samples of detained youth and Veterans, respectively. It is possible that these samples may differ from undergraduates in how they complete measures of cognitive vulnerability because of differences in age, intelligence, education level, reading ability, economic status, and cultural norms (Henrich and Norenzayan 2010). That said, we still hypothesized that measures of hopelessness theory's cognitive vulnerability factor would demonstrate reliability levels similar to those found in studies using college samples. Specifically, we expected

high levels of internal consistency as operationalized by Cronbach's α coefficient scores greater than 0.80. We also expected adequate levels of test–retest reliability over short prospective intervals (e.g., test–retest correlations greater than 0.6). At first blush, it might seem nonsensical to examine test–retest reliability in an intervention context because this is the situation in which one would expect a vulnerability factor to change. However, because cognitive vulnerability is a relatively stable factor that is resistant to change (see Romens et al. 2009 for review), and because the rank-order of cognitive vulnerability might be preserved even in the context of mean change due to an intervention (Haeffel and Hames 2014), we still expected to find test–retest reliabilities that were only slightly lower than those reported in studies with college students (test–retest reliabilities in these short-term longitudinal studies tend to range from 0.6 to 0.8; Haeffel et al. 2008). Those with higher levels of cognitive vulnerability at the start of therapy would still be expected to exhibit higher levels of vulnerability at the end of therapy relative to those who initially had lower levels of vulnerability.

Study 1: Method

Overview

We evaluated the clinical utility and reliability of cognitive vulnerability in a sample of juvenile detainees who were remanded for two weeks to state of Connecticut juvenile pre-trial detention institutions. Detained youth were randomly assigned to either a social problem solving training (SPST; Guerra and Slaby 1990) or treatment as usual (TAU). SPST is a cognitive-behavioral therapy that teaches children about negative emotions, perspective taking, cognitive flexibility, and how to more effectively cope with interpersonal stress and conflict. SPST may be particularly well suited for reducing an outcome like depression because of its focus on alleviating interpersonal stress, which is a potent predictor of depression in adolescents (Hammen 2009).

Preliminary research suggests that SPST might have possible effectiveness in reducing outcomes such as aggressive and violent behavior in randomized controlled trials (Guerra and Williams 2012). However, less work has examined its influence on comorbid symptoms affecting detained youth such as depression. Although SPST does not specifically focus on depressive vulnerabilities per se, a significant body of research suggests that cognitively based social problem solving interventions such as SPST can be effective in reducing depression (Gillham et al. 2007; Nezu et al. 2013; Possel et al. 2004; Shochet et al. 1997). These results support a broader body of research showing that a variety of interventions (interpersonal therapy, behavioral activation, problem-solving focused interventions) are capable of reducing depression. The commonalities among these empirically supported interventions appear to be that they are educational, focus on maladaptive behaviors and cognitions, are directive in nature, and are short term (Craighead and Dunlop 2014).

We used a 10-session SPST (Guerra and Slaby 1990; Guerra and Williams 2012) program guided by *Viewpoints* manual (Guerra et al. 1995). This manual was originally validated in a randomized controlled study, and based on the review of its empirical support, has been recommended for use with juvenile detainees in small group settings (Guerra and Slaby 1990; Guerra and Williams 2012). The main techniques used in *Viewpoints* include didactic education, in-session writing assignments, role-playing, and guided discussion. This SPST program is a particularly ideal intervention for detention facilities because of the brevity of the sessions and the ease with which the intervention can be administered by detention staff. The SPST program used was designed with high turnover rate of participants in mind. Each of the 10 one-hour sessions includes a 15-min recap session, which allows detainees to enter the program at any point during its 10-session run. Also, the program does not require the use of professional therapists or psychologists. Rather, the intervention can be delivered by trained correctional officers (COs) in the detention facilities directly.

The specific content of the TAU was established by the three detention centers. Specifically, during the study, the state detention facilities provided psychoeducational support groups to admitted children and adolescents in the areas of life skills, physical health and hygiene, orientation to detention services, anger management, and substance abuse prevention. It is important to highlight that TAU did not consist of any structured or controlled intervention programs, but rather the content of the daily groups was left to the discretion of the individual correctional officer in charge of TAU that day. Although there was some instruction in management of anger at the time of the study, there was no true social problem solving or cognitive behavioral intervention focused on mental health needs.

We had two primary hypotheses: (1) Cognitive vulnerability levels would predict intervention outcomes; specifically, we hypothesized that those exhibiting higher levels of cognitive vulnerability would be less likely to benefit from SPST than those with lower levels of cognitive vulnerability. In other words, those with higher levels of cognitive vulnerability pre-intervention would still have higher levels of depressive symptoms post-intervention, controlling for pre-intervention depressive symptoms. We did not expect pre-vulnerability cognitive vulnerability levels to predict TAU intervention outcomes as strongly as SPST

intervention outcomes because TAU did not have components focused on alleviating depressive symptoms; we did not expect any youth (those with either low or high vulnerability levels) to benefit from TAU and thus, there would be less pre-post variability in depressive symptoms for cognitive vulnerability to predict. Further, if there was variability in TAU it might be due to factors other than changes in cognitive vulnerability (e.g., time, social support, maturation, etc.). (2) Cognitive vulnerability, as measured by the Child Cognitive Style Questionnaire (CCSQ; Mezulis et al. 2006) would exhibit strong levels of reliability in detained youth over the two-week remand, which would be demonstrated by high levels of internal consistency (Cronbach's $\alpha > 0.6$) and good test–retest reliability across both treatment groups.

Participants

Participants were 296 adolescent males recruited through three pre-trial state-run detention centers in Connecticut, United States of America. The mean age of the participants was 14.97 years ($SD = 0.95$, range 11.36–16.94). The vast majority of participants (~70%) were from minority backgrounds (African–American: 48%, Hispanic: 30%, White: 18%, Other: 2%; Not Reported: 2%). The racial distribution of the sample was representative of the national population of male juvenile detainees in the United States (Juveniles in Residential Placement Report, 2010).

The following inclusion and exclusion criteria were used to recruit the sample: *Inclusion criteria*: (1) court order to stay in the facility for at least 14 days; (2) ability to understand and fluently reply in spoken English; (3) parental and youth consent to participate; and (4) minimum reading proficiency equivalent to grade four. *Exclusion criteria*: (1) Prior participation in the study (for children/adolescents who return to the facility after committing repetitive violations); (2) extreme "alarm" scores on the *Massachusetts Youth Screening Instrument–Version 2* (MAYSI-2; Grisso and Barnum 2003), which serves as a triage tool for decisions about the possible need for immediate intervention; juveniles who indicated high levels of psychological disturbances or suicidal ideation were excluded; (3) prior diagnosis of mental retardation or psychosis; and (4) under the guardianship of child protective services (Department of Children and Families). The study was approved by the proper authorities of all participating institutions.

Measures

Cognitive Vulnerability to Depression

The Child Cognitive Style Questionnaire (CCSQ; Mezulis et al. 2006) was used to measure the cognitive vulnerability factor featured in the hopelessness theory of depression. The CCSQ, using 5-point Likert scales, assesses participants' self-reported inferences for four hypothetical negative events on dimensions of stability, globality consequences, and self-worth. Children's responses to the 16 items (4 scenarios × 4 inference types) are averaged to create a cognitive vulnerability composite score. Thus, composite scores can range from 1 to 5 with higher values indicating greater levels of cognitive vulnerability. The CCSQ has demonstrated good internal consistency (Cronbach's α typically > 0.8; Mezulis et al. 2006), test–retest reliability (e.g., 2-week test–retest correlation of 0.81), and predictive validity (Mezulis et al. 2006) in non-clinical and non-incarcerated samples.

Depressive Symptoms

The Child Depression Inventory (CDI; Kovacs 1981) was used to measure level of depressive symptoms. The CDI is a 27-item self-report questionnaire that asks about depressive symptoms occurring during the past two weeks. Children are asked to choose one of three sentences that best describes them for each of the symptom clusters. They are scored from 0 to 2 with a higher score indicating greater symptom severity.

Procedure

All children and youth who were ordered by a Superior Court Judge to be detained for at least two weeks were assessed by detention staff for eligibility. Children and youth who met the inclusion/exclusion criteria were then introduced to a research member by detention staff. The researcher was allowed to speak privately with the child to explain the study and obtain consent. If the child agreed to participate, then the parents/legal guardians were contacted via telephone to obtain verbal consent (written consent was also obtained at a later time). If both parents/legal guardians and youth consented to participate, the youth were then administered the assessment battery. Youth were then randomly assigned to either SPST or TAU. COs delivered all groups, whether SPST ($n = 118$) or TAU ($n = 178$). All participating COs underwent training in SPST by the research team. Youth were assigned a study ID and all materials generated by this project were marked by the study ID only. The assessment materials used at baseline were administered again, approximately 14 days later, at the time of the juveniles' release. All assessment materials were removed from detention centers, and data was entered and processed off-site. The study was approved by both the detention center and Yale University human subject review boards.

Table 1 Means, standard deviations, and correlations for study 1

		1	2	3	4
1	CCSQ T1	–			
2	CCSQ T2	**0.54**	–		
3	CDI T1	**0.32**	0.16	–	
4	CDI T2	**0.48**	**0.26**	**0.73**	–
	M	1.63	1.61	8.69	6.93
	SD	0.50	0.60	5.96	6.53

Study 1. *CCSQ T1* child cognitive style questionnaire score at admission (n = 285), *CCSQ T2* child cognitive style questionnaire score at release (n = 220), *CDI T1* child depression inventory score at admission (n = 285), *CDI T2* child depression inventory score at release (n = 220). Higher scores on the CCSQ and CDI indicate greater levels of the construct being measured. Correlations in bold are significant at the $p < .05$ level

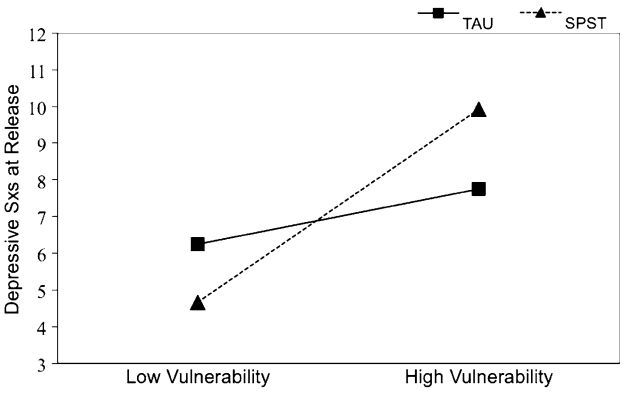

Fig. 1 Level of depressive symptoms at release as a function of cognitive vulnerability and condition (study 1)

Study 1: Results

Descriptive statistics and correlations for the primary study variables are in Table 1. The number of SPST sessions attended by the juveniles ranged from 1 to 14 with a mean of 5.4, and a mode of 10. Approximately 1/3 of participants completed the full program. Variation in sessions received was due to early release or transfer to another facility. All participants assigned to SPST and TAU, regardless of number of sessions completed, were included in the analyses. There was no correlation between number of sessions completed and either of the baseline variables (CDI, $p = .39$; CCSQ $p = .71$) or demographic variables (race, $p = .26$; age, $p = .95$). On average, youth's CCSQ scores did not significantly change from baseline to release ($t = 0.323$, $p = .75$).

Clinical Outcomes

Our first hypothesis was that those exhibiting high levels of cognitive vulnerability would be more likely to exhibit higher levels of depressive symptoms post-intervention than those with lower levels of cognitive vulnerability. To examine the effect of cognitive vulnerability on intervention effectiveness, we used hierarchical multiple regression. Continuous predictor variables were centered and entered into the regression equation in three steps. In step one, the baseline depression score (CDI) was entered to create a residual change score for the same depression measure post intervention (dependent variable). In step 2, the main effects of cognitive vulnerability (CCSQ score) and intervention condition (0 = TAU; 1 = SPST) were entered. And in step 3, the interaction of cognitive vulnerability and condition was entered. We examined the interaction to determine if the predictive power of cognitive vulnerability varied by intervention type. Individual variables within a given step were not interpreted unless the set as a whole was significant, thereby reducing Type I errors.

As expected, there were significant main effects of baseline depression ($b = 0.84$, standard error = .06, $t = 13.61$, $p < .001$, partial correlation = .76, change in $R^2 = 0.57$, Cohen's $f^2 = 1.33$) and cognitive vulnerability ($b = 3.31$, standard error = .74, $t = 4.48$, $p < .001$, partial correlation = .36, change in $R^2 = 0.06$, Cohen's $f^2 = 0.06$) on depressive outcomes. Youth with high baseline levels of cognitive vulnerability were most likely to have high levels of depression at release, even after controlling for baseline levels of depression. There was no main effect of condition ($p = .71$). Further, the cognitive vulnerability-by-condition interaction was significant ($b = 3.90$, standard error = 1.36, $t = 2.86$, $p = .005$, partial correlation = .24; change in $R^2 = 0.02$, Cohen's $f^2 = 0.02$). To graphically depict the cognitive vulnerability-by-condition interaction, we computed depressive symptom scores by inserting specific values for the moderating variable (i.e., 1 *SD* above and below the mean) into the regression equation. A simple slope analysis showed that the gradient of the simple slope for those with "low" and "high" levels of cognitive vulnerability was significantly different depending on intervention type ($t = -2.04$, $p = .04$). As can be seen in Fig. 1, SPST effectiveness depended on level of cognitive vulnerability. Specifically, youth receiving SPST reported higher levels of depressive symptoms at the end of treatment if they had higher, rather than lower, levels of cognitive vulnerability ($b = 5.43$, standard error = 1.19, $t = 4.56$, $p < .001$). The effect of cognitive vulnerability for those receiving TAU was not significant ($b = 1.69$, standard error = .88, $t = 1.91$, $p = .06$). Exploratory analyses showed no main effect ($p = .36$) or moderating effect of race ($p = .15$) on the association between the cognitive vulnerability × condition interaction and depressive outcomes.

Generalizability of Measurement

We examined the reliability of the CCSQ to measure cognitive vulnerability in detained youth by examining internal consistency and test–retest reliability. As hypothesized, cognitive vulnerability could be measured reliably in this diverse and high-risk sample. Cronbach's α for the baseline CCSQ was 0.83 and the test–retest correlation over the two-week interval was acceptable: 0.59 (95% confidence interval [CI]1: 0.45–0.70) in the TAU condition, and 0.50 (95% CI: 0.38–0.60) in the SPST condition.

Study 2: Method

Overview

We evaluated the reliability and predictive validity of cognitive vulnerability in a small sample of United States military Veterans. Veterans in this study were enrolled in a mental health outpatient clinic and referred by their individual providers (psychiatrists, psychologists, social workers, clinical nurse specialists) to a 16-week, cognitive-behavioral group therapy for depression (Group CBT-D). A total of three groups were run sequentially over a period of 12 months. Groups were co-led by a doctoral level psychologist and a pre-doctoral psychology intern. Data were collected for IRB approved Quality Assurance/Quality Improvement measurements, which means that this was not part of a research protocol but instead conducted as part of routine clinical care. As a consequence, this data is consistent with the principles of effectiveness studies (largely to maximize external validity), and there was no control group.

We had two primary hypotheses: (1) Cognitive vulnerability levels would predict depressive outcomes from pre- to post-intervention. We hypothesized that those with higher levels of cognitive vulnerability pre-intervention would have higher levels of depressive symptoms post-intervention, controlling for pre-intervention depressive symptoms. (2) Cognitive vulnerability, as measured by the Cognitive Style Questionnaire (Haeffel et al. 2008) would exhibit strong levels of reliability in Veterans; this would be demonstrated by high levels of internal consistency (Cronbach's $\alpha > 0.8$) and good test–retest reliability.

Participants

Participants were 16 males receiving therapeutic services in an outpatient mental health clinic at an urban VA medical center. Approximately two-thirds of participants were from minority backgrounds (African–American: 56%, White: 44%) and the mean age was 61 years old ($SD = 7.3$). All patients were diagnosed with Major Depressive Disorder, with a majority of them having a comorbid diagnosis of Post-Traumatic Stress Disorder (63%).

Measures

Depressive Symptoms

The Beck Depression Inventory (BDI; Beck et al. 1979) was used to measure depressive symptoms. The BDI is a 21-item self-report inventory that assesses depressive symptoms. The BDI has demonstrated strong psychometric properties in clinical samples (Beck et al. 1988).

Cognitive Vulnerability to Depression

The adult version of the Cognitive Style Questionnaire (CSQ; Haeffel et al. 2008) was used to measure the cognitive vulnerability factor featured in the hopelessness theory of depression (negative inferences for cause, consequence, and self-worth). The CSQ assesses participants' causal attributions for the 12 hypothetical negative events (6 achievement and 6 interpersonal) on dimensions of stability and globality; in addition, participants rate the probable consequences of each event and the self-worth implications of each event. Mean-item scores can range from 1 to 7, with higher scores reflecting more negative cognitive styles. The CSQ has good internal consistency, reliability, and validity (see Haeffel et al. 2008 for review) in college populations. The adult version of the CSQ used four negative scenarios (two work, two interpersonal). The CSQ was administered at session 1 and 16 weeks later, at the final session.

Procedure

The Group CBT-D followed a standard depression treatment protocol. Early sessions focused on education regarding the nature of depression and the interplay between behaviors, thoughts, and feelings. Group members were also educated about the role of behavioral inactivation and negative cognitive patterns in maintaining depression. Group members were then taught to identify, monitor, and schedule value-congruent activities (i.e., behavioral activation). Around session 6, group members began to identify negative automatic thoughts, assumptions, and core-beliefs and standard restructuring interventions were practiced for the remainder of the group (i.e., cognitive restructuring). Final sessions emphasized relapse prevention strategies. Group members completed measures of depression

Table 2 Means, standard deviations, and correlations for study 2

		1	2	3	4
1	CSQ T1	–			
2	CSQ T2	**0.66**	–		
3	BDI T1	**0.67**	**0.76**	–	
4	BDI T2	**0.84**	**0.77**	**0.90**	–
	M	4.51	4.78	30.79	27.95
	SD	1.31	0.97	8.63	11.24

Study 2. *CSQ T1* cognitive style questionnaire score at baseline (n = 16), *CSQ T2* cognitive style questionnaire score post-intervention (n = 12), *BDI T1* beck depression inventory score at baseline (n = 16), *BDI T2* beck depression inventory score post-intervention (n = 12). Higher scores on the CSQ and BDI indicate greater levels of the construct being measured. Correlations in bold are significant at the $p < .05$ level

and cognitive vulnerability at baseline (session 1) and end of treatment (session 16). Upon completions of Group CBT-D, participants were referred back to their individual providers.

Study 2: Results

Descriptive statistics and correlations for the primary study variables are in Table 2. There were no differences between those who completed the entire CBT-D group and those who did not (n = 4) on any of the baseline measures (BDI, $p = .43$; CSQ, $p = .28$). On average, Veteran's CSQ scores did not significantly change from baseline to post-intervention ($t = -1.62$, $p = .14$).

Clinical Outcomes

We hypothesized that Veterans exhibiting higher levels of cognitive vulnerability pre-intervention would be more likely to have higher levels of depressive symptoms post-intervention. To examine the predictive validity of cognitive vulnerability on intervention effectiveness, we used hierarchical multiple regression. The baseline depression measure (BDI) was entered in the first step of the regression equation to create a residual change score for the same measure post-intervention (dependent variable). In the second step, the main effect of cognitive vulnerability (CSQ) was entered. As predicted, CSQ score predicted changes in depressive symptoms, $b = 3.19$, standard error = 1.23, $t = 2.59$, $p = .03$, partial correlation = .68, change in $R^2 = 0.09$, Cohen's $f^2 = 1.00$. Individuals with higher levels of cognitive vulnerability reported higher levels of depressive symptoms post-intervention, even after considering baseline depression levels [which was also a significant predictor of post-intervention depression ($b = 1.01$, standard error = .16, $t = 6.35$, $p < .001$, partial correlation = .90, change in $R^2 = 0.82$, Cohen's $f^2 = 4.56$)]. Exploratory analyses showed no main effect ($p = .73$) or moderating effect of race ($p = .20$) on the association between the cognitive vulnerability and post-intervention depressive outcomes.

Generalizability of Measurement

We examined the reliability of the CSQ to measure cognitive vulnerability in Veterans by examining internal consistency and test–retest reliability. As hypothesized, cognitive vulnerability could be reliably measured in this diverse and high-risk sample. Cronbach's α for the baseline CSQ was 0.92 and the test–retest correlation over the 16-week interval was good at 0.66 (95% CI: 0.25–0.87).

Study 3: Method

Overview

The purpose of study 3 was to replicate the findings of Study 2 using another sample of United States military Veterans, but in the context of a non-cognitively based intervention. As in study 2, Veterans in this study were enrolled in a mental health outpatient clinic and referred by their individual providers to a group treatment. This group was a 12-week, behavioral activation (BA) group for depression that met once a week for 90 min sessions. A total of ten groups were run sequentially over a period of 16 months. Groups were led by a doctoral level psychologist or co-led by the doctoral level psychologist and a pre-doctoral psychology intern. Groups were comprised of 4 to 8 Veterans each. As in study 2, data were collected for IRB approved Quality Assurance/Quality Improvement measurements as part of routine evidence-based clinical intervention; thus, we did not follow an IRB-approved research protocol, and we did not have a control group.

We had two primary hypotheses: (1) We hypothesized that those exhibiting higher levels of cognitive vulnerability would be more likely to have higher levels of depressive symptoms post-intervention. (2) Cognitive vulnerability, as measured by the CSQ (Haeffel et al. 2008) would exhibit strong levels of reliability in Veterans; this would be demonstrated by high levels of internal consistency (Cronbach's $\alpha > 0.8$) and good test–retest reliability.

Participants

Participants were 76 Veterans (83% male) receiving therapeutic services in an outpatient mental health clinic at an urban VA medical center. All participants experienced

Table 3 Means, standard deviations, and correlations for study 3

		1	2	3	4
1	CSQ T1	–			
2	CSQ T2	**0.48**	–		
3	PHQ-9 T1	**0.66**	0.21	–	
4	PHQ-9 T2	0.23	0.15	**0.54**	–
	M	4.17	3.85	17.43	12.96
	SD	1.31	1.16	5.48	5.01

Study 2. *CSQ T1* cognitive style questionnaire score at baseline (n = 68), *CSQ T2* cognitive style questionnaire score post-intervention (n = 61), *PHQ-9 T1* patient health questionnaire at baseline (n = 72), *PHQ-9 T2* patient health questionnaire post-intervention (n = 60). Higher scores on the CSQ, and PHQ-9 indicate greater levels of the construct being measured. Correlations in bold are significant at the $p < .05$ level

clinically significant symptoms of depression and/or anxiety and were referred to 12-week group Behavioral Activation by their individual providers (psychiatrists, psychologists, social workers, clinical nurse specialists). Approximately two-thirds of participants were from minority backgrounds (African–American: 67%, White: 30%) and the mean age was 59 (range 41–78, $SD = 9.31$). The majority of participants (77%) carried diagnoses of a depressive disorder (e.g., Major Depressive Disorder, Depressive Disorder Not Otherwise Specified) and/or PTSD (61%).

Measures

Depressive Symptoms

Depressive symptoms pre and post treatment were assessed with the Patient Health Questionnaire-9 (PHQ-9, Kroenke et al. 2001). The PHQ-9 is a 9-item, self-report measure designed to make criteria-based diagnoses of depressive disorders. Respondents rated the frequency with which they experienced each of the symptoms of depression on a four-point scale (0 = *not at all*, 1 = *several days*, 2 = *more than half the days*, 3 = *nearly every day*) over the past two weeks. Items were summed for a possible total of 27. Diagnostic validity and psychometric properties have been well-established (Spitzer et al. 1999, 2000).

Cognitive Vulnerability to Depression

The adult version of the Cognitive Style Questionnaire (CSQ; Haeffel et al. 2008) was used to measure the cognitive vulnerability factor featured in the hopelessness theory of depression (see Study 2 for description). The adult CSQ was administered prior to beginning session one and one week following completion of the 12-week group.

Procedure

Group BA follows a standard BA protocol (Martell et al. 2013) adapted for Veterans with concomitant anxiety and difficulties in emotion regulation. The overall goal of BA is to help Veterans increase their access to natural rewards in their environment. Early sessions focus on education regarding the bidirectional relationship between symptoms of depression and anxiety and behavioral avoidance and in helping Veterans to monitor and develop more sophisticated coping skills to use in response to urges for behavioral avoidance or other problematic behaviors that interfere with engaging in valued activities. In subsequent sessions, Veterans are asked to monitor their daily activities and to begin to plan activities that, based on their own individualized assessments, are inherently mood elevating. Problem solving and addressing skill deficits that interfere with following through on goal activities are addressed throughout. Final sessions emphasize relapse prevention strategies. Group members completed measures of depression (PHQ-9) and cognitive vulnerability at baseline (session 1) and 13 weeks later (i.e., 1-week post-treatment). Upon completion of Group BA, participants were referred back to their individual providers.

Study 3: Results

Descriptive statistics and correlations for the primary study variables are in Table 3. There were no differences between those who completed the entire BA group and those who did not (n = 15) on any of the baseline measures (BDI, $p = .94$; CSQ, $p = .39$). On average, Veteran's CSQ scores did not significantly change from baseline to post-intervention ($t = 1.60$, $p = .12$).

Clinical Outcomes

We hypothesized that those exhibiting higher levels of cognitive vulnerability would be more likely to have higher levels of depressive symptoms post-intervention. To examine the predictive validity of cognitive vulnerability on intervention effectiveness, we used hierarchical multiple regression. The baseline depression measure (PHQ-9) was entered in the first step of the regression equation to create a residual change score for the same measure post-intervention (dependent variable). In the second step, the main effect of cognitive vulnerability (CSQ) was entered. As expected, the main effect of baseline levels of depressive symptoms was a significant predictor of post-intervention depressive symptoms, $b = 0.58$, standard error = .09, $t = 5.86$, $p < .001$,

partial correlation = .63, change in $R^2 = 0.40$, Cohen's $f^2 = 0.67$. In contrast to predictions, CSQ score did not predict changes in depressive symptoms, $b = 0.08$, standard error = .59, $t = 0.14$, $p = .89$, partial correlation = .02, change in $R^2 = 0.0$. Exploratory analyses showed no main effect (p = .37) or moderating effect of race (p = .88) on the association between the cognitive vulnerability and post-intervention depressive outcomes.

Generalizability of Measurement

We examined the reliability of the CSQ to measure cognitive vulnerability in Veterans by examining internal consistency and test–retest reliability. As hypothesized, cognitive vulnerability could be reliably measured in this diverse and high-risk sample. Cronbach's α for the baseline CSQ was = 0.91 and the test–retest correlation over the 13-week interval was fair at 0.52 (95% CI: 0.33–0.67).

Discussion

We conducted three studies to determine the generalizability and clinical utility of hopelessness theory's cognitive vulnerability construct. These three effectiveness studies are among the first to examine hopelessness theory in a real-world clinical context using real world therapists. The results of Studies 1 and 2 showed that cognitive vulnerability could have predictive validity in real world clinical settings with effect sizes in the small to medium range. Cognitive vulnerability influenced the effectiveness of a SPST intervention in youth and could predict levels of future depressive symptoms in U.S. Veterans administered a group CBT intervention. Study 3, however, did not support our hypothesis. Cognitive vulnerability did not predict future depressive symptoms in U.S. Veterans administered a group behavioral activation therapy.

Results also showed that cognitive vulnerability could be reliably measured in a highly diverse sample of male juvenile detainees (>70% minority) as well as two clinical samples of U.S. military Veterans (>60% minority). Both measures of cognitive vulnerability, the CCSQ and CSQ, demonstrated high levels of internal consistency (Cronbach's α >0.80) and test–retest reliabilities only slightly lower than that found in undergraduates, which tend to range from 0.6–0.8 (Haeffel et al. 2008). We suspect that the lower reliability results for the cognitive vulnerability measures reflect actual changes in cognitive vulnerability due to the interventions rather than unreliability of the measures.

Although cognitive vulnerability demonstrated predictive validity in Studies 1 and 2, the results of Study 3 did not conform to hypotheses. In Studies 1 and 2, individuals with high levels of cognitive vulnerability at pre-treatment appeared more treatment resistant than those with low levels of cognitive vulnerability. In Study 3, however, level of cognitive vulnerability at pre-treatment had no effect on intervention effectiveness. Taken together, these results indicate that the association between cognitive vulnerability and treatment outcomes may not be straightforward. Rather, the influence of cognitive vulnerability may depend on treatment modality and or other moderating factors. One explanation for the different findings is that individuals in Studies 1 and 2 were administered cognitively based interventions, whereas those in Study 3 were administered behavioral activation, which does not focus on altering cognitive processes (Dimidjian et al. 2006). Cognitive vulnerability may only predict outcomes for interventions for which it is part of the therapeutic focus. In contrast, cognitive vulnerability levels may matter very little for interventions that achieve their outcomes by other means. If the intervention's mechanism of action is not cognitive vulnerability (e.g., behavioral, motivational, neurochemical, etc.), then would might not expect it to predict treatment outcomes. Although speculation at this point, the reasoning is consistent with our findings that cognitive vulnerability did not predict post-intervention depressive outcomes for TAU in Study 1 or for behavioral activation therapy in Study 3. Another plausible explanation for the results is that behavioral activation is effective for reducing a larger range of depression levels than is cognitive therapy. At least one study (Dimidjian et al. 2006) suggests that behavioral activation therapy may be superior to cognitive interventions for treating severe depression, but similarly effective for mild to moderate depression. Given the strong positive correlation between depression levels and cognitive vulnerability, this would then mean that behavioral activation is effective across the cognitive vulnerability spectrum (low, moderate, and high levels). In contrast, cognitive therapy may only be effective for those with low and moderate levels of depression (and thus, low and moderate levels of cognitive vulnerability on average, given their positive correlation). Thus, pre-treatment levels of cognitive vulnerability would only be predictive of outcomes for cognitive interventions because there is differentiation in who is getting better. However, pre-treatment levels of cognitive vulnerability would not be predictive of behavioral activation outcomes because this intervention is effective regardless of cognitive vulnerability level. Clearly, future research is necessary to replicate the current findings and understand the treatments for which pretreatment levels of cognitive vulnerability can predict post intervention outcomes.

The results underscore the potential importance of identifying individual difference variables that can be used to predict prognosis and guide treatment choices. By examining the interaction of cognitive vulnerability and treatment type in Study 1, our study was able to identify a subgroup of male juvenile detainees for whom SPST training was not as effective as it needed to be. According to Lilienfeld (2007), identifying moderating factors that lead to poor outcomes is largely neglected in psychology research despite its real-world importance. Thus, this study provides clues about a potentially important moderator of intervention effectiveness—cognitive vulnerability. If we had only examined the main effect of intervention, we would have missed identifying two subgroups of male juvenile detainees for whom SPST led to different outcomes and, given further research may potentially even be contraindicated. Similarly, the results of Studies 2 suggest that assessing cognitive vulnerability could be an additional strategy, outside of depression severity, to identify individuals who might benefit from group CBT. Taken together, these preliminary findings indicate that assessing cognitive vulnerability could be a cost-effective strategy for gathering information that could potentially inform treatment providers making a referral and/or the course of treatment. It is notable that the CSQ and CCSQ are easy to administer (both are paper and pencil questionnaires), easy to score (there is no reverse scoring, responses are simply averaged), and can be reliably completed by youth, college students, and older adults from a variety of backgrounds and races.

There were a number of strengths of the current research. For example, we conducted three independent studies with diverse samples to test the predictive validity and reliability of the cognitive vulnerability construct featured in the hopelessness theory. The use of three independent samples allowed for replication of results and provided a rigorous examination of the generalizability of hopelessness theory's cognitive vulnerability hypothesis. Another strength is the use of clinical samples in real world therapeutic situations. It is often difficult to obtain samples involved in state government organizations such as detention center and VA facilities. Further, by using these samples, we increase the external validity of our research designs as the participants in our studies were sampled from "real world" clinical contexts. Members of the participating organization rather than a research team administered the interventions in our studies; this allows us to draw conclusions about the usefulness of hopelessness theory in real world contexts. Finally, in all analyses, pretreatment levels of depressive symptoms were controlled statistically. This conservative data analytic approach establishes the incremental validity of cognitive vulnerability and ensures that the results are not due to factors sharing variance with initial levels of depressive symptoms.

It is also important to note limitations. The sample size for Study 2 (Veteran participants) was small. This sample was recruited from an outpatient clinic that was only running one small CBT-D group at a given time. The result found in this study was significant and in the small to medium effect size range, even when considering baselines depression levels. The study also provides additional support for the feasibility of administering the CSQ in a clinical context. However, it will be important to replicate the findings in larger samples particularly given the null results found in Study 3 for the same population. It is possible that cognitive vulnerability is a better predictor in younger samples (youth and college students) than in older samples. A second limitation is the relatively low level of internal validity. We were willing to sacrifice some level of internal validity because our central research question was whether or not a real world therapist could administer the CSQ and CCSQ to a diverse set of patients and use it to predict therapeutic outcomes. However, this means we could not control for measures of therapist adherence or concomitant treatment (e.g., medications), and we only included participant's self-reported changes rather than including clinician-rated changes. Along these same lines, the participants in Study 1 likely missed more treatment sessions than if the study were implemented in a research setting (a majority of participants did not complete all 10 SPST sessions). Thus, it is possible that SPST could have been more effective in reducing cognitive vulnerability if administered in a more idealized fashion and setting where attrition could be limited. Thus, these findings should be considered preliminary. Finally, it is unclear if the results are specific to the vulnerability factor featured in the hopelessness theory. Future research should examine competing cognitive vulnerability factors (e.g., Beck's theory) to determine which of them has the most predictive power.

In conclusion, recent research has provided compelling support for hopelessness theory's cognitive vulnerability hypothesis. However, little work has examined whether or not hopelessness theory can be used to help people in clinical contexts. The results of three independent studies using diverse samples in a clinical context indicate that cognitive vulnerability can be measured in diverse samples of children and adults and, given further research, has the potential to be a useful tool when making treatment decisions (at least for cognitively based interventions). These results corroborate past research and suggest that hopelessness theory has implications for not only the etiology of depression, but also its treatment.

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Compliance with Ethical Standards

Conflict of interest Gerald J. Haeffel, Rachel Hershenberg, Jason T. Goodson, Sascha Hein, Amanda Square, Elena L. Grigorenko, John Chapman declare that they have no conflicts of interest.

Ethical Approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed Consent Informed consent was obtained from all individual participants in Study 1. Data for Studies 2 and 3 were collected for IRB approved Quality Assurance/Quality Improvement measurements, which means that this was not part of a research protocol but instead conducted as part of routine clinical care. As a consequence, this data is consistent with the principles of effectiveness studies (largely to maximize external validity). This paper was supported by the VISN 4 Mental Illness Research, Education, and Clinical Center, Philadelphia Veterans Affairs Medical Center, Philadelphia, PA.

Animal Rights No animal studies were carried out by the authors for this article.

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