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# Response-Contingent Positive Reinforcement: Incremental Validity in Predicting Depression Severity

John Paul Carvalho

*University of Tennessee - Knoxville*

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To the Graduate Council:

I am submitting herewith a thesis written by John Paul Carvalho entitled "Response-Contingent Positive Reinforcement: Incremental Validity in Predicting Depression Severity." I have examined the final electronic copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Arts, with a major in Psychology.

Derek R. Hopko, Major Professor

We have read this thesis and recommend its acceptance:

Jenny Macfie, Todd Moore

Accepted for the Council:

Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

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Response-Contingent Positive Reinforcement: Incremental Validity  
in Predicting Depression Severity

A Thesis  
Presented for  
the Master of Arts  
Degree  
The University of Tennessee, Knoxville

John Paul Carvalho  
December 2008

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### **Abstract**

Insufficient response-contingent positive reinforcement (RCPR), or pleasure obtained through interaction with the environment that increases the likelihood of rewarding behavior, has been hypothesized to directly contribute to the onset and persistence of depression symptoms (Lewinsohn, 1974; Lewinsohn, Sullivan, & Grosscup, 1980). The present study examined the utility of RCPR in predicting the presence and severity of depression symptoms relative to other well-established risk factors that included gender, stressful life events, traumatic life events, childhood maltreatment, and cognitive vulnerability. Based on bivariate and hierarchical regression analyses, all variables except gender were significantly associated with the severity of depression symptoms, with RCPR most strongly related to depression symptom severity. The incremental validity of RCPR in predicting depression symptom severity also was established, with RCPR accounting for an additional 12% of the variance when added to a regression equation. Implications for the conceptualization and treatment of depression are discussed.

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## **Introduction**

A number of risk factors are linked to the development, persistence, and severity of clinical depression. These factors include female gender, marital separation or divorce, unemployment, exposure to trauma, physical and sexual abuse, other stressful and adverse life events, maladaptive cognitive styles, family history of depression, genetic predispositions, hormonal influences, physical pain and poor physical health, functional limitations, medical illness, socioeconomic status, financial limitations, poor social support, and limited coping resources (Alloy, Abramson, Walshaw, & Neeren, 2006; Kaelber, Moul, & Farmer, 1995; Kessler, 1997; Klein & Santiago, 2003; Lorant et al., 2007; Mazure & Keita, 2006; Neff et al., 2008; Person, Tracy, & Galea, 2006; Spasojevic & Alloy, 2001; Tennant, 2002; Vink, Aartsen, & Schoevers, 2008; Wareham, Fowler, & Pike, 2007). Often taking these factors into account, a number of psychological theories have been proposed that explicate the etiology, maintenance, and severity of clinical depression and depression symptomatology (Corveleyn, Luyten, & Blatt, 2005; Gotlib & Hammen, 2002).

Behavioral theories postulate depressive symptoms to be produced and maintained through reduced environmental reward, reinforcement of depressive behaviors, and punishment of healthy alternative behaviors (Ferster, 1973; Hopko, Lejuez, Ruggiero, & Eifert, 2003; Lewinsohn, 1974; Rose & Staats, 1988). Ferster (1973, 1974) described depression as the result of a reduction in positively reinforced behavior, in part attributable to patterns of escape and avoidance behavior. He stressed the role of the environment in prompting, shaping, and maintaining depressive behavior and the

need to analyze environment-behavior relationships to conceptualize and treat depression. Lewinsohn and colleagues subsequently expanded this pioneering work and highlighted a low rate of response-contingent positive reinforcement (RCPR) as the critical predictor of clinical depression (Lewinsohn, 1974; Lewinsohn & Graf, 1973; Lewinsohn & Libet, 1972). RCPR is defined as positive or pleasurable outcomes that follow an individual's behaviors within his or her environment and increase the likelihood of those behaviors. Minimal environmental (and social) reinforcement was proposed to result in the extinction of "healthy" adaptive behaviors and consequently the dysphoric mood and passivity that often characterize depression. A low rate of RCPR is a product of: 1) a decreased number of events that are potentially reinforcing for the individual, 2) decreased availability of these potential reinforcers in the environment, 3) inability to experience rewarding contingencies due to inadequate instrumental behaviors such as social skill, and 4) increased exposure to aversive stimuli (e.g., punishment) in the form of distressing, upsetting, or unpleasant events (Lewinsohn, 1974; Lewinsohn et al., 1980). The occurrence of such aversive events leads to avoidance behaviors that result in limited exposure to potentially rewarding activities and a lower rate of RCPR. Reduced RCPR is thought to be sufficient in producing the dysphoria and related symptoms observed in clinical depression (Lewinsohn, 1974; Lewinsohn, Sullivan, & Grosscup, 1980; MacPhillamy & Lewinsohn, 1974).

Supporting behavioral theory, a number of studies demonstrated relationships between pleasant events and mood state, with individuals reporting fewer positive events, decreased environmental reward, and more limited abilities to obtain reinforcement

endorsing increased depression severity (Bouman & Luteijn, 1986; Hopko, Armento, Cantu, Chambers, & Lejuez, 2003; Hopko & Mullane, 2008; Lewinsohn & Amenson, 1978; Lewinsohn & Graf, 1973; Lewinsohn & Libet, 1972; MacPhillamy & Lewinsohn, 1974). It also was shown that depressed individuals tend to engage in fewer rewarding interpersonal behaviors suggesting that insufficient social interaction and decreased social reinforcement may predict negative affect (Joiner, Lewinsohn, & Seeley, 2002; Lewinsohn & Shaffer, 1971; Libet & Lewinsohn, 1973). Important to note, however, a few studies have not supported the link between RCPR and mood variability (Biglan & Craker, 1982; Hammen & Glass, 1975; Sweeney, Shaeffer, & Golin, 1982).

In a related but more integrated paradigmatic behaviorism theory of depression, depression arises when positive emotional stimuli in the environment are limited or negative emotional stimuli are excessive (Staats & Heiby, 1985). Divergent from traditional behavioral theories, however, language-cognitive and emotional-motivational repertoires are hypothesized to impact whether such environmental circumstances ultimately result in depression. Similar to traditional behavioral theories, investigations of paradigmatic theories of depression have shown that the frequency and intensity of both positive and negative events are important predictors of depression symptom severity (Davis, 2001; Davis & Burns, 1999; Rose & Staats, 1988; Wilkinson, 1993).

Treatment outcome research has been another source of convincing evidence for the influence of environmental reinforcement on depression. Early behavioral treatments for depression demonstrated attenuation in symptoms through strategies designed to increase availability of positive reinforcement, including monitoring of pleasant events

and associated mood, activity scheduling, social skills development, and behavioral psychoeducation (Barrera, 1979; Brown & Lewinsohn, 1984; Lewinsohn & Atwood, 1969; Lewinsohn & Graf, 1973; Lewinsohn & Shaffer, 1971; Lewinsohn & Shaw, 1969; Lewinsohn et al., 1980; Sanchez, Lewinsohn, & Larson, 1980; Zeiss, Lewinsohn, & Munoz, 1979). More contemporary studies have shown that behavioral activation interventions (BA) may be just as effective as cognitive therapy in the treatment of major depression (Jacobson et al., 1996). Behavioral activation is based on the premise that increasing activities to promote environmental reinforcement leads to improvements in mood, thoughts, and quality of life (Hopko, Lejuez et al., 2003; Jacobson, Martell, & Dimidjian, 2001; Lejuez, Hopko, & Hopko, 2001; Martell, 2001). The efficacy of BA generally has been well supported (Cuijpers, van Straten, & Warmerdam, 2007; Ekers, Richards, & Gilbody, 2008; Hopko, Lejuez et al., 2003), with the most convincing support coming from a randomized trial comparing BA, cognitive therapy (CT), Paroxetine, and a medication placebo (Dimidjian et al., 2006). BA outperformed all other conditions with moderately to severely depressed participants when observed drop-out rate and relapse and recurrence problems for Paroxetine treatment were considered.

The revivification of behavioral interventions for depression has underscored the need for practical and reliable methods of measuring RCPR (Armento & Hopko, 2007; Kanter, Mulick, Busch, Berlin, & Martell, 2007). The Pleasant Events Schedule (PES; MacPhillamy & Lewinsohn, 1971) initially was developed to measure the frequency and intensity of positive events that an individual experienced. The frequency of positive events and corresponding pleasure were proposed to represent an approximate, yet valid,

method of quantifying RCPR (MacPhillamy & Lewinsohn, 1974). However, the scale is limited in that it is quite extensive (i.e., 320 items) and prompts for specific events that may be pleasant but not necessarily associated with increased RCPR. Accordingly, to better assess environmental reinforcement at a more functional level, the Environmental Reward Observation Scale was designed as an efficient, valid, and reliable measure of self-observed environmental reward (EROS; Armento & Hopko, 2007). There is a strong inverse relationship between the EROS and empirically-validated depression measures, as well as a moderate positive relationship between the EROS and the PES. Additionally, relative to the PES, the EROS was a stronger predictor of depression symptom severity, indicating that it may be a more valid measure of RCPR (Armento & Hopko, 2007). In a recent randomized controlled trial investigating the efficacy of behavioral activation in treating mildly depressed college students, depression symptoms significantly improved following treatment, while environmental reward as measured by the EROS significantly increased (Gawrysiak, Nicholas, & Hopko, 2008). These results support the behavioral hypothesis that depression symptoms are reduced as positive reinforcement becomes more available in the environment.

Given substantial evidence supporting a relationship between decreased RCPR and depression, it is important to determine the theoretical relevance of RCPR's association with the severity of depression symptoms relative to other well established risk factors. If decreased environmental reward accounts for significant variance beyond that associated with other well-established predictors of depression, in addition to further supporting behavioral models of depression, behavioral treatments for depression that

focus on increasing RCPR would be further validated as critical toward positive treatment outcome. As a move in this direction, the purpose of the present study was to establish the degree to which decreased RCPR contributed to depression symptom severity while controlling for gender, stressful and traumatic life events, childhood maltreatment, and cognitive vulnerability, factors empirically associated with depression onset and severity.

Gender has long been associated with depression, with females (21%) nearly twice as likely to develop the disorder as males (12%) (Kessler et al., 2003; Kessler et al., 1994). This sex difference emerges by middle adolescence and remains evident for sub-clinical depression symptoms as well as diagnosable clinical depression (Nolen-Hoeksema, 1994). Studies examining nonclinical samples have generally found females report more symptoms and greater severity of depression, although investigations of clinical samples are inconclusive (Angst & Dobler-Mikola, 1984; Ernst & Angst, 1992; Hammen & Padesky, 1977; Hankin, Mermelstein, & Roesch, 2007; Hildebrandt, Stage, & Kragh-Soerensen, 2003a; Hildebrandt, Stage, & Kragh-Soerensen, 2003b; Mitchell & Abbott, 1987; Petersen, Sarigiani, & Kennedy, 1991; Wareham et al., 2007; Williams et al., 1995; Young, Fogg, Scheftner, Keller, & Fawcett, 1990).

The relationship between stressful life events and depression has been consistently established through decades of extensive research (Kessler, 1997; Mazure, 1998; Paykel, 2003; Tennant, 2002). Abundant empirical data show the onset, recurrence, and severity of depression symptoms and episodes to be associated with the frequency, severity, and type of stressful events experienced (Brown & Harris, 1978; DeLongis, Coyne, Dakof, Folkman, & Lazarus, 1982; Dolan, Calloway, Fonagy, De Souza, &

Wakeling, 1985; Garnefski, Van Egmond, & Straatman, 1990; Grant, Sweetwood, Yager, & Gerst, 1981; Grosscup & Lewinsohn, 1980; Hammen, Davila, Brown, Ellicott, & Gitlin, 1992; Hammen, Mayol, DeMayo, & Marks, 1986; Kendler, Karkowski, & Prescott, 1999; Lewinsohn, Hoberman, & Rosenbaum, 1988; Monroe, Bromet, Connell, & Steiner, 1986; Monroe, Harkness, Simons, & Thase, 2001; Monroe, Kupfer, & Frank, 1992; Paykel et al., 1969; Reno & Halaris, 1990). Acute, chronic, major, minor, proximal and distal stressors have all been linked to the development of depression, with major and uncontrollable life events (e.g., loss of loved one) most associated with depression onset (Mazure, 1998). Further, the relationship between stressful life events and depression appears to be moderated by personality, genetic, environmental, cognitive, and social factors (Alloy, Abramson, Walshaw et al., 2006; Caspi et al., 2003; Coyne & Whiffen, 1995; Mazure, 1998; Paykel, 1994; Tennant, 2002).

Traumatic life events, or experiences that involve threat to oneself or a loved one, are a subset of stressful life events that, while most often associated with posttraumatic stress disorder, can also serve as precursors to major depression and predictors of depression severity (Galea et al., 2002; Green, 1994; Kessler, Davis, & Kendler, 1997; O'Donnell, Creamer, & Pattison, 2004; Schumm, Briggs-Phillips, & Hobfoll, 2006). Major traumatic experiences such as rape, assault, death of a loved one, injuries, and car accidents have all been linked to depression (Hinton, Tiet, Tran, & Chesney, 1997; Kessler et al., 1997; McQuaid, Pedrelli, McCahill, & Stein, 2001; Resick, 1983; Warshaw et al., 1993). There is evidence of a dose-effect relationship, where a higher number and greater severity of traumatic events increases likelihood and severity of

depression symptoms (Green et al., 2000; Kendler, Kuhn, & Prescott, 2004; Mollica, McInnes, Poole, & Tor, 1998; Schumm et al., 2006; Tanskanen et al., 2004). This cumulative effect has been found to occur in both clinical and nonclinical populations (Vrana & Lauterbach, 1994).

Moreover, adversities experienced during childhood, such as loss events (e.g., death of a parent), life-threatening accidents, and interpersonal traumas significantly increase the risk of depression and other psychiatric disorders during adulthood (Bemporad & Romano, 1992; Heim, Newport, Mletzko, Miller, & Nemeroff, 2008; Hill, 2003; Kessler et al., 1997). Extensive literature indicates that childhood maltreatment, in the form of sexual, physical, or emotional abuse or emotional or physical neglect, contributes to the onset and severity of depression (Bemporad & Romano, 1992; Bernet & Stein, 1999; Brown, Cohen, Johnson, & Smailes, 1999; Cole & Eamon, 2007; Courtney, Johnson, & Alloy, 2008; Gibb, Chelminski, & Zimmerman, 2007; Harkness & Monroe, 2002; Hill, 2003; Margo & McLees, 1991; Moskvina et al., 2007; Surrey, Swett, Michaels, & Levin, 1990; Zlotnick, Mattia, & Zimmerman, 2001; Zlotnick, Ryan, Miller, & Keitner, 1995). Such maltreatment may activate biological or cognitive vulnerability to depression, increasing the risk for developing depression, and may be moderated by environmental and genetic factors (Bradley et al., 2008; Cukor & McGinn, 2006; Gibb, 2002; Heim et al., 2008; Hill, 2003; Penza, Heim, & Nemeroff, 2003; Weiss, Longhurst, & Mazure, 1999).

Cognitive vulnerability-stress models of depression posit that individuals who exhibit maladaptive cognitive patterns are more vulnerable to depression when

confronted with negative life events. Beck's theory of dysfunctional attitudes (1967, 1987) and the hopelessness theory of depression (Abramson, Metalsky, & Alloy, 1989) are the most well-known of these models. Hopelessness theory postulates that inferential style (i.e., the causal attributions, inferred consequences, and inferred characteristics about the self that individuals make in response to life events) is a critical factor in determining risk and severity of depression. Depressive episodes are more common when individuals interpret negative life events as important, the result of stable (enduring) and global (widespread) causes, as likely to result in additional negative consequences or outcomes, and as indicating that he or she has limited worth, abilities, desirability, or is in any way deficient. According to hopelessness theory, this pattern of dysfunctional thinking represents a cognitive vulnerability to depression (CVD).

A number of retrospective (Alloy, Lipman, & Abramson, 1992; Sturman, Mongrain, & Kohn, 2006) and prospective investigations have provided strong support for the hopelessness theory of CVD as a predictor of depression onset and severity (Abela, 2002; Abela & Brozina, 2004; Abela & Seligman, 2000; Abramson et al., 1998; Bohon, Stice, Burton, Fudell, & Nolen-Hoeksema, 2008; Fresco, Alloy, & Reilly-Harrington, 2006; Hankin & Abramson, 2001; Hankin, Abramson, Miller, & Haeffel, 2004; Hankin, Abramson, & Siler, 2001; Metalsky & Joiner, 1992; Metalsky, Joiner, Hardin, & Abramson, 1993). The most compelling findings come from the Temple-Wisconsin Cognitive Vulnerability to Depression Project (Alloy et al., 2000; Alloy, Abramson, Whitehouse et al., 2006), which was longitudinal in design and addressed both retrospective reporting of depressive disorders and prospective occurrence. In the

retrospective analysis, participants defined as being at high-risk (HR) for CVD were three times more likely to have had MDD, twice as likely to have had minor depression, and reported greater severity of depressive episodes compared to low-risk (LR) participants (Alloy et al., 2000). In the same sample at 2.5 year follow-up, when controlling for history of depression and initial depression symptoms, HR participants were nearly seven times as likely to develop MDD and over three times as likely to develop minor depression compared to LR participants. Further, CVD was just as likely to predict initial onsets and recurrences of MDD, indicating CVD may be an enduring vulnerability (Alloy, Abramson, Whitehouse et al., 2006). While HR and LR groups were defined in terms of both Beck's dysfunctional attitudes theory and the hopelessness theory, subsequent analysis (Haefel et al., 2003) indicated the retrospective results were most attributable to the hopelessness theory of CVD.

RCPR, gender, stressful and traumatic life events, childhood maltreatment, and cognitive vulnerability have all been convincingly linked to depression. However, few studies have attempted to compare the relative validity of these factors as they might account for the severity of depression symptoms. In one longitudinal comparison of behavioral and cognitive constructs, attributional style and dysfunctional attitudes were more strongly related to depression symptom severity than a factor comprised of positive reinforcement and attributional style towards positive events (Reno & Halaris, 1989). Another comparison of depressive cognitions to mood-related activities found dysfunctional attitudes, automatic (depressive) thoughts, and engagement in pleasurable and unpleasant activities to be inter-related and significantly correlated with depression

severity, with mood-related activity associated with depression even when observed independent of cognitive measures (Wierzbicki & Rexford, 1989). Other studies have shown that frequency and intensity of pleasant and unpleasant events independently predict depression symptom severity, with mixed results as to which is the more powerful predictor (Davis, 2001; Davis & Burns, 1999; Lewinsohn & Amenson, 1978; Rehm, 1978).

Insufficient research on this topic as well as conflicting results necessitate further evaluation. The above studies are limited in that they either compared composite measures of cognitive and behavioral functioning without independently focusing on environmental reward (Reno & Halaris, 1989), did not directly investigate incremental validity and relative strength of RCPR (Wierzbicki & Rexford, 1989), or only compared positive environmental reinforcement to negative external stimuli, limiting the ability to assess the strength of these factors against those from other domains (Davis, 2001; Davis & Burns, 1999; Lewinsohn & Amenson, 1978; Rehm, 1978). The present investigation is unique in incorporating a potentially more valid measure of RCPR (the EROS) and directly comparing its relationship to the severity of depression symptoms relative to well-established risk factors. Specifically, this investigation assessed the incremental validity of RCPR (environmental reward) in predicting depression symptom severity when controlling well-established risk factors: gender, stressful life events, traumatic events, childhood maltreatment, and cognitive vulnerability, allowing for a stringent test of the role environmental reinforcement plays in further informing our understanding of depression severity, conceptualization, and treatment. It was hypothesized that each of

the measured variables (RCPR, gender, stressful life events, traumatic events, childhood maltreatment, and cognitive vulnerability) would be significantly correlated with the severity of depression symptoms, and that each would account for significant variance in predicting depression symptom severity, with the level of RCPR accounting for significant and unique variance while controlling for other variables.

## Method

### *Participants*

The sample included 127 male and female undergraduate students recruited from the University of Tennessee. Students volunteered to participate in the study based on an online description posted on the University's research participation website. Students aged 18 years and older were included in the sample. There were no additional exclusion criteria. The sample consisted of 50 men (39.4%) and 77 women (60.6%), with a mean age of 19.0 years ( $SD = 1.8$ ). Ethnic distribution was as follows: 105 Caucasians (82.7%), 13 African Americans (10.2%), 2 Latinos (1.6%), 5 Asian Americans (3.9%), and two participants identified as "other" (1.6%).

### *Measures*

The Environmental Reward Observation Scale (EROS; Armento & Hopko, 2007) is a 10-item measure that assesses environmental reward or response-contingent positive reinforcement on a 4-point Likert scale. The scale is intended to identify the magnitude of reinforcing events, the availability of reinforcement in the environment, and the ability of an individual to elicit that reinforcement. Sample items include "A lot of activities in my life are pleasurable," "It is easy for me to find enjoyment in my life," and "The activities I engage in usually have positive consequences." The EROS has strong internal consistency ( $\alpha = .85 - .90$ ) and excellent test-retest reliability ( $r = .84 - .85$ ), and correlates strongly with other commonly administered and psychometrically sound self-report measures of depression and anxiety, as well as the Pleasant Events Schedule

(Armento & Hopko, 2007). Internal consistency of the EROS in the study sample was strong ( $\alpha = .84$ ).

The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) is a measure of depression symptom severity which consists of 21 items, each of which is rated on a 4-point Likert scale (0-3 point anchors), with items summed to form a total score. Sample items include assessment of the frequency and intensity of “sadness,” “guilt,” and “concentration difficulty.” The instrument has excellent internal consistency ( $\alpha = .92$ ) as well as strong convergent validity with other measures of depression (Beck et al., 1996; Nezu, Ronan, Meadows, & McClure, 2000). Internal consistency of the BDI-II in the study sample was strong ( $\alpha = .85$ ).

The Life Experiences Survey (LES; Sarason, Johnson, & Siegel, 1978) is a 57-item measure that assesses potentially stressful life changes over the past year. It includes 10 events that are specific to students and three blank spaces for write-in events. Each experienced event is rated on a 7-point Likert scale ranging from “extremely negative impact” (-3) to “extremely positive impact” (+3). Sample items include “Death of a close family member,” “Change of residence,” and “Beginning a new school experience at a higher academic level.” The LES has demonstrated moderate reliability and is significantly related to a number of other stress-related measures (Sarason et al., 1978). In the present study, a negative event frequency score was calculated by summing the items that each participant labeled as having had a negative impact.

The Trauma History Questionnaire (THQ; Green, 1996) is a 24-item scale that assesses for the occurrence of various types of potentially traumatic events (crime-

related, general disaster and trauma, and physical and sexual experiences) with yes/no questions. The frequency of each event and the approximate age at occurrence are also evaluated. The THQ boasts moderate to high test-retest reliability as well as strong interrater reliability (Mueser et al., 2001). In the present study, an aggregate trauma exposure score was calculated by summing the frequencies of each type of traumatic event.

The Childhood Trauma Questionnaire (CTQ; Bernstein et al., 1994) retrospectively assesses experiences of abuse and neglect in the childhood and adolescent years (through age 18). Version 3 is a 28-item, self-report instrument that contains five subscales (emotional abuse, physical abuse, sexual abuse, emotional neglect, physical neglect). Items begin with the prompt “When I was growing up...” and include, “People hit me so hard that it left me with bruises or marks,” and “People in my family said hurtful or insulting things to me.” Each item is rated on a scale from 1 (“Never True”) to 5 (“Very Often True”). The CTQ has demonstrated acceptable reliability and validity, including moderate test-retest reliability, acceptable internal consistency, convergent validity with other measures of abuse, and a consistent five-factor structure (Bernstein, Ahluvalia, Pogge, & Handelsman, 1997; Bernstein & Fink, 1998; Scher, Stein, Asmundson, McCreary, & Forde, 2001). This study incorporated a total childhood maltreatment score by summing the average score of each subscale (omitting three Minimizing/Denial items).

The Cognitive Style Questionnaire (CSQ; Alloy et al., 2000; Haeffel et al., 2008) is a measure of cognitive vulnerability to depression based on the hopelessness theory of

depression (Abramson et al., 1989). The CSQ is a modified version of the Attributional Style Questionnaire (ASQ; Peterson et al., 1982) that uses a series of 7-point-response items to assess participants' attributions for 12 positive and 12 negative hypothetical events on dimensions of internality, stability, and globality, as well as probable consequences for each event and implications for the self. A negative event composite score was calculated by summing and averaging responses across the 12 negative hypothetical events as a measure of negative inferential style. The CSQ has been demonstrated as a reliable measure of cognitive vulnerability with strong construct validity (Haefel et al., 2008).

#### *Procedure*

All participants were assessed individually. Upon arrival to the research laboratory, participants initially completed informed consent procedures and a demographic form that included questions regarding age, gender, ethnic identity, and marital status. They then completed the battery of self-report instruments described above. Students were awarded course credit for participation in the study. The study was fully approved by the University of Tennessee Institutional Review Board.

## Results

### *Descriptive Statistics*

Descriptive statistics are provided in Table 1<sup>1</sup>. Based on independent-samples *t*-tests, there were no significant differences between males and females in depression symptom severity [ $t(125) = -1.71, p = .09$ ], RCPR [ $t(125) = 1.30, p = .20$ ], frequency of negative life events in the past year, [ $t(125) = -1.64, p = .10$ ], total number of traumatic events experienced, [ $t(85) = 1.27, p = .21$ ], childhood maltreatment history [ $t(125) = -0.42, p = .68$ ], or cognitive vulnerability to depression [ $t(125) = -0.24, p = .81$ ]. A Chi-square analysis also yielded no gender differences in terms of ethnic background ( $\chi^2(4) = 1.75, p = .78$ ), although males in the study were significantly older than females [ $t(125) = 2.52, p < .05$ ].

### *Bivariate Correlations*

Correlations among self-report measures and gender are presented in Table 2. As predicted, depression symptom severity as measured by the BDI-II was significantly and inversely correlated with RCPR ( $r = -.67, p < .001$ ). There also were significant positive correlations between depression symptom severity and negative life events ( $r = .50, p < .001$ ), traumatic events ( $r = .31, p < .001$ ), childhood maltreatment ( $r = .38, p < .001$ ), and cognitive vulnerability ( $r = .38, p < .001$ ). Interestingly, gender was not significantly correlated with depression ( $r = .15, p = .09$ ).

Pearson's test of dependent *r*'s indicated that the correlation between RCPR and depression symptom severity was significantly stronger than correlations between

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<sup>1</sup> All tables are located in the Appendix.

depression symptom severity and gender [ $t(124) = 6.88, p < .001$ ], negative life events [ $t(124) = 2.27, p < .05$ ], traumatic events [ $t(124) = 4.69, p < .001$ ], childhood maltreatment [ $t(124) = 3.81, p < .001$ ], and cognitive style [ $t(124) = 3.78, p < .001$ ].

### *Regression Analysis*

A hierarchical multiple regression analysis was conducted to determine the relative value of RCPR, gender, frequency of recent negative life events, cumulative history of traumatic life events, and cognitive vulnerability in accounting for depression symptom severity. Given study hypotheses, incremental predictive value of RCPR was assessed above that accounted for by the remaining variables. Accordingly, and in the context of strong bivariate relationships reported, the first step of the model involved regressing depression symptom severity on gender, negative life events, traumatic events, childhood maltreatment, and cognitive vulnerability. Results of the regression analysis are presented in Table 3. The first block of predictors explained 47.5% of the variance in depression symptom severity, with negative life events, traumatic life events, childhood maltreatment, and cognitive vulnerability significantly associated with depression [ $F(5, 120) = 21.94, p < .001$ ]. Gender was the only variable in step 1 not significantly related to depression symptom severity.

In step 2 of the regression model, the potential incremental value of RCPR (as measured by the EROS) in predicting depression symptom severity was assessed. The purpose for structuring the hierarchical regression analysis in this manner was to promote a stringent assessment of whether RCPR would account for significant incremental variance beyond other well-known depression risk factors in predicting depression

severity. In particular, reduced RCPR is proposed to have a powerful relation to depression severity such that it may contribute unique variance beyond other variables, based on behavioral theories that postulate decreased RCPR directly contributes to the experience and maintenance of dysphoria, anhedonia, and other associated symptoms of depression, in addition to increasing risk for the development of these symptoms (Ferster, 1973, Lewinsohn, 1974). When RCPR was added to the model in the second step, the amount of variance increased to 59.2% [ $F(6, 120) = 29.02, p < .001$ ], an increase of 11.7%. Change statistics indicated that the addition of RCPR to the regression model accounted for significant additive variance in predicting depression symptom severity [ $\Delta F(1, 120) = 34.28, p < .001$ ]. All variables with the exception of gender were significant predictors, with RCPR accounting for the most variance in predicting depression symptom severity ( $\beta = -.41$ ). Collinearity statistics for predictor variables were within the acceptable range (tolerance values *Range* = .69 to .98, variable inflation factor *Range* = 1.02 to 1.46; Hair, Anderson, Tatham, & Black, 1995).

## Discussion

Consistent with traditional behavioral theories of depression, the results support a convincing relationship between response-contingent positive reinforcement, or environmental reward, and the presence and severity of depression symptoms. The data may indicate RCPR is, as Lewinsohn proposed, a critical predictor of depression symptomatology (Lewinsohn, 1974). As predicted, RCPR was significantly and moderately correlated with depression symptom severity, and more strongly related to depression symptom severity than gender, stressful life events, traumatic life events, childhood maltreatment, and cognitive style. Interestingly, and possibly due to the sample's relatively low mean depression score and the finding that gender differences in depression severity have not consistently been observed when few symptoms are reported (Young et al., 1990), gender was not significantly related to depression severity. All other factors were significantly related to depression symptom severity in the predicted directions. RCPR accounted for significant and unique variance in predicting depression symptom severity when other established risk factors were controlled, accounting for an additional 11.7% of the observed variance in depression symptom severity.

These results are consequential for several reasons. Of primary significance, data suggest RCPR may be an equally if not more powerful predictor of the presence and severity of depression symptoms than a number of empirically validated demographic, environmental, and cognitive risk factors. This finding is consistent with behavioral theories of depression that posit depressive behaviors and emotions develop and persist when healthy alternative behaviors are not rewarded or reinforced in the natural

environment or when access to potential reinforcers is limited. This schedule of reinforcement effectively creates an extinction process whereby behaviors that might elicit rewarding outcomes are not emitted, not reinforced, and subsequently discontinue, being replaced by a more passive or ineffectual behavioral repertoire that is accompanied by depressive affect (Ferster, 1973; Lewinsohn, 1974). Whereas the other independent variables included in this design are often hypothesized to increase the risk for developing depression either indirectly or through mediating or moderating factors (Alloy, Abramson, Walshaw et al., 2006; Bemporad & Romano, 1992; Nolen-Hoeksema, 1994; Tennant, 2002), insufficient RCPR is theorized to directly produce and maintain the dysphoria, fatigue, anhedonia, and other somatic and cognitive symptoms that define depression (Lewinsohn, 1974; Lewinsohn et al., 1980). Therefore, it is logical that RCPR, by virtue of being a direct causal antecedent of depression, would be a stronger predictor of depression symptomatology and severity than other risk factors assessed in the study.

In addition to this theoretical support, the strong bivariate and multivariate relationships suggest the concepts of RCPR and environmental reward are important to consider in the conceptualization and treatment of depression. Indeed, behavioral activation interventions designed to increase RCPR by promoting increased overt behavior and corresponding access to environmental reinforcement have been successful in reducing depression symptoms and improving quality of life for many depressed patients (Cuijpers et al., 2007; Ekers et al., 2008). The present findings further support the rationale of such treatments and provide an argument for possibly integrating behavioral activation methods into depression treatments that do not traditionally focus

on behavioral principles. For instance, while traditional CBT incorporates guided activity into the first few sessions of treatment (Beck, Rush, Shaw, & Emery, 1979), the present findings and previous research (e.g., Jacobson et al., 1996) suggest CBT therapy may be strengthened by putting a stronger emphasis on the behavioral component of treatment, perhaps by including BA strategies throughout treatment and conceptualizing guided activity as a strategy to modify dysfunctional beliefs and simultaneously reduce behavioral avoidance and increase environmental reward. Such behavioral concepts may also be applied to non-behavioral treatments. Interpersonal psychotherapy for depression (IPT; Gillies, 2001), for example, addresses core problems with interpersonal deficits as well as role disputes and transitions. Understanding deficiencies in these areas as detrimental to eliciting RCPR and conceptualizing IPT interventions as methods to increase environmental reward may allow for a more comprehensive case conceptualization and provide more direction therapeutically. If RCPR is as influential to depression symptomatology and severity as the present findings suggest, current depression treatment interventions could be strengthened by the addition of or increased emphasis on techniques that increase patients' abilities to elicit positive reinforcement from their environments, such as behavior monitoring, activity scheduling, social skills training, relaxation training, and problem-solving strategies (Lewinsohn et al., 1980).

Further support for behavioral theory comes from the relationship observed between negative life events experienced in the previous year and depression symptom severity, especially when compared to the less powerful relationships between depression and traumatic life events and childhood maltreatment. Negative life events accounted for

the second highest proportion of variance in step 2 of the hierarchical regression analysis ( $\beta = .24$ ) and was more highly correlated ( $r = .50$ ) with depression symptom severity than any of the other measured variables other than RCPR. It is possible that the stronger relationship observed between depression and negative life events as measured by the LES is due to this measure's assessment of proximal stressors, compared to the more distal measures of trauma history and childhood maltreatment. This would be consistent with behavioral theories that state that aversive stimuli, in the form of negative events, contribute to a temporal reduction in environmental reinforcements, and subsequently, the development of depression symptoms (Lewinsohn et al., 1980). As only one of four main components of RCPR, it also fits with Lewinsohn's theory that the relationship between recent negative life events and depression would be weaker than that between RCPR and depression.

One major limitation of this study is its cross-sectional design. Because all measures were completed at one assessment session, it is illogical to assert causal relationships between depression severity and examined predictor variables. Therefore, it is plausible, albeit still consistent with behavioral theories of depression (Hopko & Lejuez, 2007; Lewinsohn, 1974; Martell, 2001), that the relationship observed between depression symptoms and RCPR is partially reflective of depressive emotions and behaviors reducing an individual's ability to elicit positive reinforcement from the environment rather than inadequate environmental reward preceding and accounting for all aspects of depression symptom severity. Probably the most parsimonious explanation is the existence of a bidirectional relationship, whereby reduced environmental reward

increases risk for developing depression as well as an impact of depression toward further limiting an individual's ability to obtain reinforcement from the environment that cyclically worsens depression (Coyne, 1976; Martell, 2001; Segrin, 2000). Also important to note, the observed relationship between decreased RCPR and depression symptom severity may partially be a function of response bias created by depression, with those who are more depressed being less likely to recognize and report positive reinforcement in their lives (DeMonbreun & Craighead, 1977). The cross-sectional design also limits the ability to interpret relationships between depression symptom severity and other risk factors. For example, the relationship between stressful life events and depression symptoms, while likely evidence that stressors act as antecedents to depression, may also indicate that depressed individuals are more prone to encountering stressful situations or are more likely to retrospectively report negative events due to response bias (Brown, 1989; Skodol, Dohrenwend, Link, & Shrout, 1990).

Future research that incorporates longitudinal designs and statistical strategies such as path analysis will be necessary to better determine causal relationships among all variables studied. Also important to address in future research is the degree to which each of the determining components of RCPR (i.e., potential reinforcers, availability of reinforcers, and individual ability to elicit reinforcers) contributes to depression onset, symptomatology, and severity (Lewinsohn, 1974). Determining which of these factors are most consequential toward our understanding of depression will help to elaborate on the current findings and further inform conceptualization and treatment of depression.

In summary, the findings of this study strongly support the notion that RCPR plays an important role in the development and maintenance of depression symptoms. Specifically, environmental reward appears to be more strongly related to the presence and severity of depression symptoms than several other empirically validated precursors to depression. These findings have broad implications for our understanding of the etiology and course of depression as well as how clinicians can most effectively treat the disorder.

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## Appendix

## Appendix A

Table 1.

### *Descriptive Statistics for Demographic Variables and Self-Report Measures*

	<u>Females (n = 77)</u>			<u>Males (n = 50)</u>			<u>Total Sample (N = 127)</u>		
	Mean	SD	%	Mean	SD	%	Mean	SD	%
Age (years)	18.68	0.92		19.48	2.57		18.99	1.80	
Caucasian			80.5			86.0			82.7
African American			13.0			6.0			10.2
Other Ethnicity			6.5			8.0			7.1
BDI	10.21	6.85		8.26	5.24		9.44	6.32	
EROS	29.71	4.71		30.82	4.61		30.15	4.68	
LES	6.03	3.76		4.94	3.45		5.60	3.66	
THQ	2.82	1.81		3.32	2.38		3.02	2.06	
CTQ	7.00	2.29		6.86	1.47		6.95	2.00	
CSQ	4.49	0.61		4.46	0.55		4.48	0.59	

BDI = Beck Depression Inventory-II total score; EROS = Environmental Reward Observation Scale total score; LES = Life Experiences Survey negative event frequency; THQ = Trauma History Questionnaire lifetime event frequency; CTQ = Childhood Trauma Questionnaire total score; CSQ = Cognitive Style Questionnaire negative event composite score

## Appendix B

Table 2.

*Bivariate Correlations Between Depression Symptom Severity and Predictive Factors*

	BDI	EROS	Gender	LES	THQ	CTQ	CSQ
BDI	1	-.67**	.15	.50**	.31**	.38**	.38**
EROS		1	-.12	-.41**	-.25**	-.34**	-.28**
Gender			1	.15	-.12	.03	.02
LES				1	.18*	.16	.15
THQ					1	.22*	.03
CTQ						1	.04
CSQ							1

\*p < .05      \*\*p < .01

For gender, male = 1 and female = 2; BDI = Beck Depression Inventory-II; EROS = Environmental Reward Observation Scale; LES = Life Experiences Survey; THQ = Trauma History Questionnaire; CTQ = Childhood Trauma Questionnaire; CSQ = Cognitive Style Questionnaire

### Appendix C

Table 3.

#### *Depression Symptom Severity as a Function of Established Risk Factors and RCPR*

Independent Variable	Standardized Coefficient ( $\beta$ )	SE	r	t	p
<b>Step 1</b>					
Gender	.11	.87	.14	1.56	.120
Negative life events	.36	.12	.43	5.20	<.001
Traumatic life events	.19	.21	.24	2.69	.008
Childhood maltreatment	.27	.22	.34	3.96	<.001
Cognitive vulnerability	.31	.72	.39	4.69	<.001
$R^2 = .48$					
<b>Step 2</b>					
Gender	.07	.77	.11	1.23	.222
Negative life events	.24	.11	.32	3.68	<.001
Traumatic life events	.13	.19	.18	2.03	.044
Childhood maltreatment	.17	.20	.23	2.62	.010
Cognitive vulnerability	.22	.66	.31	3.61	<.001
RCPR	-.41	.10	-.47	-5.86	<.001
$R^2 = .59$					
$\Delta R^2 = .12$					

Note: Negative life events were measured by the Life Experiences Survey, traumatic life events were measured by the Trauma History Questionnaire, childhood maltreatment was measured by the Childhood Trauma Questionnaire, cognitive vulnerability was measured by the Cognitive Style Questionnaire, and response-contingent positive reinforcement (RCPR) was measured by the Environmental Reward Observation Scale

### **Vita**

John Paul Carvalho graduated from Providence College in Providence, Rhode Island in 2004 with a B. A. in Psychology. From 2004 to 2006 he worked as a Senior Research Assistant and Study Coordinator for the Mood Disorders Research Program of Butler Hospital in Providence. In 2006 he entered the doctoral program in clinical psychology at the University of Tennessee, Knoxville. Since 2006 he has worked as a graduate research assistant under the supervision of Dr. Derek Hopko studying the efficacy of behavioral activation therapy in treating depressed cancer patients, as well as other topics dealing with depression and anxiety. In addition to his research pursuits, John has been working as a graduate student clinician at the University of Tennessee Psychological Clinic from 2007 to present. Since August 2008, he has also been working as therapist for Cherokee Health Systems in New Tazewell, Tennessee.