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Reducing hopelessness: the interaction of enhancing and depressogenic attributional styles for positive and negative life events among youth psychiatric inpatients

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Abstract

A recovery model of depression (Needles & Abramson, 1990 *Journal of Abnormal Psychology*, 99, 156–165) proposes that depressed individuals who exhibit an enhancing attributional style for positive events (i.e. make stable, global attributions) will be more likely to regain hopefulness and, thereby, recover from depression when positive events occur. While only a few studies have directly tested this model among clinical and nonclinical adult samples, none have tested a clinical sample of children and adolescents. Furthermore, prior studies testing this model have failed to examine the interactive role of an ‘enhancing attributional style’ for positive events with a ‘depressogenic attributional style’ for negative events, as prescribed by the hopelessness theory of depression (Abramson, Metalsky, & Alloy, 1989 *Psychological Review*, 96, 358–372). The current study presents data demonstrating that depressogenic and enhancing attributional styles interact to predict differential decreases in hopelessness. Implications of these findings and suggestions for future research also are presented. © 2003 Elsevier Science Ltd. All rights reserved.

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1. Introduction

Understanding how individuals develop, maintain, and recover from depression is central to managing the course of this often pervasive and unrelenting illness. Although prominent models

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of etiology and maintenance have been rigorously developed and tested in cognitive, behavioral, and social domains over the past few decades (e.g. Abramson, Metalsky, & Alloy, 1989; Beck, 1967, 1987; Coyne, 1976; Lewinsohn, Hoberman, Teri, & Hautzinger, 1985; Nolen-Hoeksema, 1991; Pyszczynski & Greenberg, 1987; Swann, 1990), much less research has been conducted on the recovery process itself (i.e. the mechanisms by which depression remits).

Studies that do examine the recovery process among depressed individuals are generally limited to treatment outcome studies focusing specifically on changes in cognitive, behavioral, and psychosocial variables as a product of empirically validated psychological, pharmacological or combined treatment techniques (for a review, see Pettit, Voelz, & Joiner, 2001). This scientific approach to recovery by mental health professionals is vital in the effective treatment of millions of depressed patients each year, and must be sustained. However, there also exists a large, untreated population of depressed individuals who appear to recover from depression through a natural, undirected process. What are the mechanisms underlying 'spontaneous' remission, and what role might they play in the recovery of professionally treated individuals?

Needles and Abramson (1990) first attempted to examine this process from a cognitive standpoint by developing a model of recovery based on the hopelessness theory of depression (Abramson et al., 1989). In brief, the hopelessness theory of depression is a vulnerability-stress model of depression causation wherein 'depressogenic attributional style' (serving as the vulnerability) interacts with negative life events (serving as the stressor) to produce hopelessness. More specifically, Abramson and her colleagues postulate that negative life events attributed to causes that are *stable* (not likely to change) and *global* (likely to affect many outcomes) may incite *generalized hopelessness* which, in turn, may lead to symptoms of hopelessness depression — a theoretical subtype of depression that overlaps considerably with DSM-IV (American Psychiatric Association, 1994) major depression. Hopelessness is defined by Needles and Abramson (1990) as '...the expectation that highly desirable outcomes will not occur, and that one is powerless to change the situation' (p. 156). As a necessary and sufficient cause of the symptoms of hopelessness depression, the presence of hopelessness is hypothesized to be responsible for the presence and duration of depressive symptoms. Further underscoring the critical role of hopelessness in the hopelessness theory, Needles and Abramson developed a corresponding model of recovery.

In their model of recovery from depression, Needles and Abramson (1990) proposed that increases in *hopefulness* (i.e. decreases in hopelessness) would lead to decreases in depressive symptomatology and, ultimately, the remission of depression. This being true, how does one develop hopefulness? Needles and Abramson proposed that the occurrence of positive life events would interact with 'enhancing attributional style' to incite the development of hopefulness. Enhancing attributional style is defined as the tendency to make *stable* (not likely to change) and *global* (likely to affect many outcomes) attributions for *positive* (rather than negative) life events. Thus, according to this model of recovery from depression, the occurrence of positive life events interacts with enhancing attributional style to promote the development (or restoration) of hopefulness, which may then lead to a reduction in depressive symptomatology.

Needles and Abramson (1990) provided the first empirical support for this model by examining college students who exhibited moderate to severe levels of depressive symptomatology, as well as hopelessness. Depressed participants were followed for 6 weeks, during which time levels of depressive symptoms, hopelessness, attributional style, and the occurrence of positive and negative life events were measured. Consistent with the recovery model, participants who exhibited enhanc-

ing attributional style and experienced an increase in positive life events demonstrated significantly restored hopefulness (i.e. reduction in hopelessness). Moreover, increased hopefulness resulted in the subsequent amelioration of depressive symptoms.

Beyond the preliminary empirical support provided by [Needles and Abramson \(1990\)](#), few studies have attempted to directly test this model of recovery among depressed individuals. Nonetheless, the few studies that have examined the Needles and Abramson model have produced generally supportive results. For example, [Edelman, Ahrens, and Haaga \(1994\)](#) examined levels of depressive symptomatology, hopelessness, attributional style, and the occurrence of life events over a three-week period among 94 college students experiencing depression. Consistent with the recovery model, enhancing attributional style tended to predict recovery from depressive symptoms in the presence of positive events. In contrast to the recovery model, however, hopefulness was not found to be a mediating factor between the interaction of enhancing attributional style and presence of positive events, and recovery from depressive symptoms.

A study conducted by [Johnson, Crofton, and Feinstein \(1996\)](#) examined depressive symptomatology, hopelessness, attributional style, and the occurrence of life events among a sample of 32 depressed adult psychiatric inpatients treated with antidepressant medication over an average period of 12 days. This was the first study to apply the recovery process posited by [Needles and Abramson \(1990\)](#) to a more controlled clinical sample of inpatients. Their findings indicated that, much like the Needles and Abramson results, depressed individuals who exhibited an enhancing attributional style and experienced positive life events were more likely than other patients to develop hopefulness, which then led to decreases in overall depressive symptoms. However, inconsistent with the Needles and Abramson model, [Johnson et al. \(1996\)](#) found that the *combined* main effects of an enhancing attributional style and the occurrence of positive life events — not their interaction — predicted the restoration of hopefulness.

To our knowledge, the most recent study to test the recovery model ([Johnson, Han, Douglas, Johannet, & Russell, 1998](#)) investigated levels of depressive symptomatology, hopelessness, attributional style, attributions for recent events, and the occurrence of life events among a sample of 52 depressed adult psychiatric inpatients treated with antidepressant medication over a 24 day period. Johnson and his colleagues found that internal, stable, global attributions for recent positive events mediated a significant association between internal, stable, global attributional style for positive life events and decreased hopelessness. [Johnson et al. \(1998\)](#) also found that attributions for positive events predicted decreases in depressive symptoms through the mediation of decreases in hopelessness, consistent with the [Needles and Abramson \(1990\)](#) model of recovery. Interestingly, the significant findings of this study involve, in all cases, an attributional style for positive events that includes a stable, global *and* internal component. This, of course, differs somewhat from Needles and Abramson who de-emphasize the importance of internality and hypothesize that only stable, global attributions are needed to create changes in hopelessness.

The research examining the [Needles and Abramson \(1990\)](#) model of recovery has been generally supportive. Further, the findings were similar across both clinical and nonclinical adult samples, suggesting a potentially similar recovery process in relatively natural (i.e. untreated) *and* controlled (treated) environments. However, we contend that the current conceptualization of the Needles and Abramson recovery model is incomplete. This recovery model has neglected to examine how depressogenic attributional style may impact recovery. According to hopelessness theory, a depressogenic attributional style leads to hopelessness and in turn, the symptoms of

depression. Then, after the onset of depression, the recovery model posits that the interaction of an enhancing attributional style and positive life events will lead to remission by decreasing hopelessness. However, the recovery model fails to account for the depressogenic attributional style that causally contributed to the onset of depression and is still operative in the depressed person. A depressogenic attributional style does not ‘disappear’ when a person develops hopelessness and the symptoms of depression. Rather, research suggests that the depressogenic style actually may become exacerbated as depressed mood increases (e.g. the mood state hypothesis, Persons & Miranda, 1992). Once depressed, a depressogenic attributional style (given the occurrence of negative life events) might lead to further increases in hopelessness, and consequently increase the severity and duration of depressed mood and symptoms. Thus, it is imperative that a recovery model of depression based on the hopelessness theory of depression examines the effect of depressogenic attributional style on the restoration of hopefulness. How does depressogenic attributional style impede or facilitate recovery from depression?

To understand how depressogenic attributional style may impact recovery, we revisited the impetus for the recovery model, i.e. the hopelessness theory of depression (Abramson et al., 1989). Fundamental to hopelessness theory, as described earlier, is the notion that certain individuals exhibit a depressogenic attributional style (i.e. stable, global attributions) that interacts with the occurrence of negative life events to produce generalized hopelessness. Hopelessness theory further describes this interaction through a titration model which states that more severe depressogenic attributional styles require negative events that are less severe to produce generalized hopelessness; conversely, less severe depressogenic attributional styles require negative events that are more severe to produce an effect. The importance of the titration model in light of our present investigation is that it indicates the existence of *varying* degrees of depressogenic attributional style for negative events across cognitively vulnerable individuals (i.e. one individual may have a less (or more) severe depressogenic attributional style than another individual).

Based on these assertions regarding depressogenic attributional style, we might also assume such characteristics to be true of enhancing attributional style for positive events. For example, a weaker enhancing attributional style (i.e. less global, less stable) may require a much more positive and significant life event to promote recovery from hopelessness, and ultimately, depression; conversely, a stronger enhancing attributional style (i.e. more global, more stable) may promote recovery with less positive and less significant life events. Although we propose that depressogenic and enhancing attributional styles are both continua, we do not intend to imply that they are on the *same* continuum. Rather, there is evidence that depressogenic and enhancing attributional styles are independent factors. In fact, prior studies have suggested that attributional styles for positive and negative events represent different constructs, with attributional style for positive events being more weakly associated with onset of depression than attributional style for negative events (see Sweeney, Anderson, & Bailey (1986), for a meta-analysis of 104 studies); also supportive of this idea is the consistently low degree of correlation between attributional style scales for positive and negative events (e.g. Zautra, Guenther, & Chartier, 1985). Needles and Abramson (1990) further state, ‘...even among those at risk for hopelessness depression, there may be a subset who have the hypothesized enhancing style for positive events and who thereby may be better able to recover.’ Theory and data thus support the notion that depressogenic and enhancing attributional styles are separate constructs and that both attributional styles may coexist within an individual.

Assuming the *coexistence* of depressogenic and enhancing attributional styles among depressed individuals, we hypothesize that existing levels of depressogenic attributional style for negative events may *interact* with enhancing attributional style for positive events in the restoration of hopefulness. Specifically, we propose that an enhancing attributional style should be most likely to reduce hopelessness when a depressogenic attributional style is not ‘working against it’. That is, the greater the level of enhancing attributional style compared to depressogenic attributional style, then the greater the recovery from hopelessness (assuming the equal occurrence of positive and negative life events). A high level of enhancing attributional style for positive events should lead to the recovery from depression (i.e. create higher levels of future hopefulness) when an individual has low levels of a depressogenic attributional style. Conversely, if a depressed individual exhibits a high degree of depressogenic attributional style and a low degree of enhancing attributional style, we would predict a slower recovery (i.e. lower levels of future hopefulness; higher levels of future hopelessness).

The above predictions are relatively straightforward because they are assuming equal rates of positive and negative life events and a large discrepancy between the levels of enhancing and depressogenic attributional style. The predictions become more complicated, however, if the levels of enhancing and depressogenic attributional styles are equivalent. Assuming equal levels of depressogenic and enhancing attributional style, then the occurrence rate of negative and positive life events should play a central role in predicting recovery from depression. For example, if a depressed individual exhibits a high degree of both depressogenic and enhancing attributional style then we would predict a more moderate recovery as the two styles work to offset one another. However, if the same individual experiences a positive life event, a strong enhancing attributional style for that event may predict a more rapid recovery. In this case, the absence of a negative life event leaves this individual’s strong depressogenic attributional style inactive, and only the enhancing attributional style will influence mood. Finally, there may exist the depressed individual who exhibits a low degree of both depressogenic and enhancing attributional style. Although less stable (*unstable*) and less global (*specific*) attributions toward negative life events might be predictive of a rapid recovery, this individual is also exhibiting more unstable and specific attributions for positive life events, which may temper rapid recovery. Without prior theory and research concerning these types of individuals, it becomes difficult to predict what kind of recovery might be observed. Examination of the recovery process among these types of individuals (i.e. low depressogenic and enhancing attributional style) could be a beneficial part of research examining the potentially interactive effects of depressogenic and enhancing attributional style.

The current study extends a test of the recovery model (Needles & Abramson, 1990) to a sample of child and adolescent psychiatric inpatients. While prior studies have examined clinical and nonclinical adult samples, the current study will be the first to apply the recovery model to a sample of youth. We refer to the current study as an ‘extended’ test of the recovery model because, while it is based on the Needles and Abramson (1990) model of recovery from depression, our hypotheses are not exclusively consistent with this model. Instead, we draw upon and combine depressogenic attributional style from the hopelessness theory (Abramson et al., 1989) and enhancing attributional style from the recovery model (Needles & Abramson, 1990) to formulate new hypotheses regarding the process of recovery.

We hypothesize that the *interaction* of depressogenic attributional style for negative events and

enhancing attributional style for positive events upon admission to the hospital will predict decreased hopelessness at discharge among a sample of psychiatric youth inpatients. We hypothesize that youth exhibiting a low degree of depressogenic attributional style for negative events and a high degree of enhancing attributional style for positive events upon admission will report the *lowest* levels of hopelessness at discharge; it is also hypothesized that youth exhibiting a high degree of depressogenic attributional style and a low degree of enhancing attributional style upon admission will report the *highest* levels of hopelessness at discharge. Finally, it is hypothesized that youth exhibiting a high degree of both depressogenic and enhancing attributional style will report levels of hopelessness similar to individuals exhibiting low depressogenic and high enhancing attributional styles (i.e. the lowest levels of hopelessness). Regarding this final prediction, it is noted that study participants are hospital inpatients in a stable and supportive environment. We argue that such an environment results in an absence of negative life events during the course of this study. In fact, [Needles and Abramson \(1990\)](#) have suggested that the reduction of negative life events as a function of being hospitalized is itself equivalent to experiencing a positive life event. It is thus assumed that, among individuals with high degrees of both depressogenic and enhancing attributional style, the occurrence of a ‘positive’ life event (i.e. being hospitalized) will result in an active enhancing attributional style leading to a stronger recovery (lower levels of future hopelessness). Regarding youth with a low degree of both depressogenic and enhancing attributional style, no specific prediction is made.

We also hypothesize that the *interaction* of depressogenic attributional style for negative events and enhancing attributional style for positive events upon admission to the hospital will predict changes in general depressive symptomatology (beyond hopelessness) at discharge. This finding would be consistent with aspects of the mediational component of the [Needles and Abramson \(1990\)](#) recovery model whereby effects of the hypothesized distal causal factors on recovery from depression are mediated by the hypothesized proximal cause — decreased hopelessness.

2. Method

2.1. Participants and procedures

Participants in the current study included 67 children and adolescents (32 boys, 35 girls), ages 7–17 years ($M=13.43$, $SD=2.57$). The majority of youth were Caucasian (44 of 67; 66%); 15 were African-American (22%); seven were Hispanic (10%); and one was Asian (2%). These participants represent a subset of a larger sample of inpatients (described below). Participants for the current study were selected from the larger sample if they produced a score greater than 11 on the Children’s Depression Inventory (CDI: [Kovacs, 1981, 1992](#)) upon admission to the hospital. This selection criterion was used so that we could appropriately examine potential recovery from depressive symptomatology among individuals actually experiencing depressive symptoms. In essence, those participants *not* selected for inclusion in the current study represented a nondepressed sample. There were no significant differences in age, gender, or ethnicity between the larger, original sample and the smaller sample selected for the current study.

Participants in the original, larger sample included 100 children and adolescents. All participants were psychiatric inpatients admitted to the child and adolescent units at the University of Texas

Medical Branch. The participants represent a consecutive period of admissions of those willing to participate. Informed consent was given from the parent or legal guardian as was assent from the minors for participation in the study.

Inclusion criteria for the study were that children were psychiatric inpatients and that they forthrightly completed all relevant measures. Children or adolescents who were unable (e.g. stated inability or difficulty reading; severe psychosis) or unwilling to participate were excluded.

Participant diagnoses were made using the fourth edition of the Diagnostic and Statistical Manual (DSM-IV; American Psychiatric Association, 1994) criteria based on direct interviews of the child and parent (or legal guardian) using the Lifetime Schedule for Affective Disorders and Schizophrenia (K-SADS-L; Puig-Antich, Chambers, & Klein, 1993) which assesses both current and lifetime psychiatric disorders in youth. Of those participants selected for the current study (i.e. individuals with an initial CDI score >11 [$N=67$]), 21 (31%) received a primary diagnosis of Major Depressive Disorder or Depressive Disorder Not Otherwise Specified; 17 (25%) received a primary diagnosis of Bipolar Disorder; 8 (12%) received a primary diagnosis of Attention-Deficit/Hyperactivity Disorder. The remaining primary diagnoses were Conduct Disorder (five cases), Psychosis Not Otherwise Specified (five cases), Intermittent Explosive Disorder (two cases), Organic Mood Disorder (two cases), Substance/Alcohol Abuse (two cases), Acute Adjustment Disorder (one case), Anxiety Disorder Not Otherwise Specified (one case), Post-Traumatic Stress Disorder (one case), Schizoaffective Disorder (one case), and Undifferentiated Somatoform Disorder (one case). Secondary diagnoses of study participants were unknown; thus comorbid conditions were not directly assessed.

After obtaining informed consent from the parent or legal guardian and participant assent, the Children's Depression Inventory (CDI), the Hopelessness Scale for Children (HSC: Kazdin, Rodgers, & Colbus, 1986), and the Children's Attributional Style Questionnaire (CASQ: Kaslow, Tanenbaum, & Seligman, 1978) were administered to the participants upon admission to the psychiatric unit. These questionnaires and rating scales were re-administered at the time of discharge.

The average length of hospitalization was 8.68 days for the original sample (range=2–24 days). For the smaller sample selected for the current study, average length of hospitalization was 9.10 days (range=2–24 days).

2.2. Measures

2.2.1. Children's Attributional Style Questionnaire (CASQ)

The CASQ (Kaslow et al., 1978) is a 48-item forced choice measure of causal explanations for 24 positive and 24 negative events. It has been widely used among clinical and nonclinical studies measuring attributional style in children and adolescents, ages 7 through 17 (for a review, see Joiner & Wagner, 1995). Hypothetical events are presented and participants are requested to select the response that explains why the event happened to them. Sixteen questions pertain to each of the three attributional dimensions of internality, stability, and globality. A score of 1 is assigned to each internal, stable, or global response and a score of 0 to each external, unstable, or specific response. For positive attributions, Gotlib, Lewinsohn, Seeley, Rohde, and Redner (1993) reported an alpha coefficient of 0.45, and a 1 year test–retest reliability of 0.48; for negative attributions, they reported an alpha coefficient of 0.58, and a 1 year test–retest reliability of 0.54.

Kaslow et al. (1978) and Seligman et al. (1984) also reported modest-to-adequate reliability and adequate validity for the measure.

For the purposes of the present study, consistent with the hopelessness theory of depression (Abramson et al., 1989) and the recovery model of depression (Needles & Abramson, 1990), and consistent with empirical work showing that stability and globality reflect the same factor but internality does not (Joiner & Rudd, 1996), analyses using the CASQ were limited to the negative attributional components of stability and globality (for negative events), as well as the positive attributional components of stability and globality (for positive events). The resulting combination of negative stability and negative globality is referred to as *depressogenic attributional style*; similarly, the combination of positive stability and positive globality is referred to as *enhancing attributional style*.

2.2.2. Children's Depression Inventory (CDI)

The CDI (Kovacs, 1981, 1992) is a reliable and well-validated 27-item self-report measure of depression with scores ranging from 0 (non-depressed) to 54 (extremely depressed). Each item has a set of three sentences which describes different depressive symptoms. Items are scored 0, 1 or 2. Kovacs (1981) reported adequate internal consistency and 1-month test-retest reliabilities (i.e. 0.86 and 0.72, respectively) for the scale as a whole. The scale has been adequately validated (e.g. correlates significantly ($r=0.55$) with clinician-rated depression; Kovacs, 1992).

2.2.3. Diagnoses

Diagnoses were based on information gathered from: (i) clinical interviews conducted by a psychiatry resident and faculty; and (ii) K-SADS-L interviews (Puig-Antich et al., 1993) conducted by a trained research assistant, with decision rules specified by the DSM-IV (American Psychiatric Association, 1994). Consensus diagnoses were reached using both of these sources of information. Although reliability was not formally assessed, the reliability of this assessment procedure has been repeatedly supported (for a review, see Ambrosini, 2000).

2.2.4. Hopelessness Scale for Children (HSC)

The HSC (Kazdin, French, Unis, Esveldt-Dawson, & Sherick, 1983) is a 17-item scale of true or false questions modified from the Hopelessness Scale for adults (Beck, Weismann, Lester, & Trexler, 1974). The HSC has been found to have adequate internal consistency ($\alpha=0.84$) and test-retest reliability ($r=0.49$ after 10 weeks) in clinical samples of adolescents (Spirito, Williams, Stark, & Hart, 1988).

3. Results

Means and standard deviations of, and inter-correlations between, all measures are summarized in Table 1. As seen, means, standard deviations, and inter-correlations were generally within expected limits and consistent with our hypotheses.

Consistent with prior research indicating that depressogenic attributional style (DepAtt) and enhancing attributional style (EnhAtt) represent separate constructs (e.g. Sweeney et al., 1986; Zautra et al., 1985), no significant correlation was found between depressogenic and enhancing

Table 1
Means and standard deviations of, and inter-correlations between, all measures

Variable	Age	Gender	T1 CDI	T2 CDI	T1 HSC	T2 HSC	T1 DepAtt	T2 DepAtt	T1 EnhAtt	T2 EnhAtt
Age	13.43 (2.57)									
Gender	0.10	1.52 (0.50)								
T1 CDI	0.32**	0.18	23.16 (8.41)							
T2 CDI	0.16	-0.14	0.36**	16.66 (8.86)						
T1 HSC	0.05	0.20	0.70**	0.16	6.22 (3.89)					
T2 HSC	-0.16	0.03	0.28*	0.51**	0.23	4.67 (3.34)				
T1 DepAtt	0.25*	-0.04	0.43**	0.32**	0.46**	0.34**	3.31 (1.30)			
T2 DepAtt	0.08	-0.28*	0.35**	0.59**	0.25*	0.49**	0.38**	3.02 (1.22)		
T1 EnhAtt	-0.13	-0.05	-0.25*	-0.13	-0.22	0.04	-0.13	-0.14	6.94 (2.71)	
T2 EnhAtt	-0.11	-0.03	-0.03	-0.44**	0.06	-0.35**	-0.04	-0.17	0.20	8.01 (3.11)

Note: N=67. Means (bold) and standard deviations (in parentheses) are presented on diagonal. Gender=1 for male, 2 for female; CDI=Children's Depression Inventory; HSC=Hopelessness Scale for Children; DepAtt=depressogenic attributional style (stable+global attributions for negative events); EnhAtt=enhancing attributional style (stable+global attributions for positive events); T1=Time 1; T2=Time 2.

* $p < 0.05$.

** $p < 0.01$.

attributional styles at either Time 1 or Time 2 (T1 DepAtt–T1 EnhAtt: $r=-0.13$; T2 DepAtt–T2 EnhAtt: $r=-0.17$).

Depressogenic attributional style and hopelessness (HSC scores) were significantly and positively correlated at both Time 1 and Time 2 (T1 DepAtt–T1 HSC: $r=0.46$; T2 DepAtt–T2 HSC: $r=0.49$). Enhancing attributional style and hopelessness scores were also correlated (negatively) at both Time 1 and Time 2, although their association at Time 1 only approached significance (T1 EnhAtt–T1 HSC: $r=-0.22$ [$p=0.07$]; T2 EnhAtt–T1 HSC: $r=-0.35$).

Depressogenic attributional style and depressive symptomatology (CDI scores) were significantly and positively correlated at both Time 1 and Time 2 (T1 DepAtt–T1 CDI: $r=0.43$; T2 DepAtt–T2 CDI: $r=0.59$). Also as expected, enhancing attributional style and depressive symptomatology were significantly and negatively correlated at both Time 1 and Time 2 (T1 EnhAtt–T1 CDI: $r=-0.25$; T2 EnhAtt–T2 CDI: $r=-0.44$).

Test–retest correlations for depressogenic and enhancing attributional styles, depressive symptomatology, and hopelessness, were also as anticipated, given that treatment-related change was expected [T1 DepAtt–T2 DepAtt: $r=0.38$; T1 EnhAtt–T2 EnhAtt: $r=0.20$ ($p=0.10$); T1 CDI–T2 CDI: $r=0.36$; T1 HSC–T2 HSC: $r=0.23$ ($p=0.06$)]. Age and T1 CDI were correlated such that older youth tended to report more depressive symptoms (AGE–T1 CDI: $r=0.32$); this relationship disappeared by Time 2. Similarly, age and T1 DepAtt were correlated such that older youth tended to report a stronger depressogenic attributional style (AGE–T1 DepAtt: $r=0.25$); once again, this relationship disappeared by Time 2.

3.1. Attributional interaction predicts changes in hopelessness

We hypothesized that a depressogenic attributional style for negative events would interact with an enhancing attributional style for positive events to predict decreases in hopelessness. Consistent with the recommendations of Cohen and Cohen (1983), a multiple regression procedure was used to test this prediction. The level of hopelessness at Time 2 (T2 HSC) served as the dependent variable. Time 1 level of hopelessness (T1 HSC) was first entered into the equation, thereby creating residual change scores in HSC from Time 1 to Time 2. Next, age, length of hospitalization, and gender values were entered, appropriately controlling for these variables. The depressogenic attributional style (T1 DepAtt) and enhancing attributional style (T1 EnhAtt) variables were entered next as main effects. Finally, the depressogenic attributional style and enhancing attributional style interaction (T1 DepAtt×T1 EnhAtt) was entered. Consistent with our hypothesis, the interaction of depressogenic and enhancing attributional styles upon admission to the hospital successfully predicted changes in hopelessness scores at discharge (*partial correlation* [pr]= -0.26 , $t[59]=-2.04$, $p<0.05$; see Table 2)¹.

To graphically depict our findings, we computed Time 2 hopelessness scores (T2 HSC) by inserting specific values for predictor variables (i.e. 1.5 standard deviations above and below the mean scores for DepAtt and EnhAtt) into the regression equation associated with the analysis reported above. Consistent with prediction, and as seen in resulting Fig. 1, youth exhibiting a

¹ If internal attributions for positive and negative events are also entered as covariates, the predictive effect of the attributional interaction becomes even more powerful.

Table 2

DepAtt \times EnhAtt at Time 1 predicting residual changes in HSC from time 1 to time 2

Order of entry of set	Predictors in set	<i>F</i> for set	<i>t</i> for within-set predictors	df	Partial correlation (<i>pr</i>)
1.	Control variable T1 HSC	3.54	1.84	1, 65 65	0.23
2.	Control variables Age Length Gender	1.08	–1.42 0.90 0.16	62 62 62	–0.18 0.14 0.02
3.	Main effects T1 DepAtt T1 EnhAtt	3.78*	2.71** 0.46	6, 60 60 60	0.33 0.06
4.	Interaction DepAtt \times EnhAtt	4.17*	–2.04	7, 59 59	–0.26

Note: $N=67$. HSC=Hopelessness Scale for Children; Length=length of hospitalization (in days); DepAtt=depressogenic attributional style (stable+global attributions for negative events); EnhAtt=enhancing attributional style (stable+global attributions for positive events).

* $p<0.05$.

** $p<0.01$.

high degree of depressogenic attributional style (High DepAtt) and a low degree of enhancing attributional style (Low EnhAtt) upon admission reported the highest levels of hopelessness at discharge (HSC=7.67). Youth exhibiting a low degree of depressogenic attributional style (Low DepAtt) and a high degree of enhancing attributional style (High EnhAtt) upon admission reported lower levels of hopelessness at discharge (HSC=5.16); similarly, as predicted, youth exhibiting a high degree of both depressogenic and enhancing attributional styles experienced lower levels of hopelessness at discharge (HSC=4.68), nearly equivalent to the low depressogenic/high enhancing individuals. Although no specific predictions were made for the final group, it is notable that youth exhibiting a low degree of both depressogenic and enhancing attributional style upon admission reported the lowest levels of hopelessness at discharge (HSC=1.17).

In a supplemental analysis, we wanted to address the question of whether *internal* attributions for positive and negative events are just as important as those which are stable and global in the restoration of hopefulness (as suggested by Johnson et al., 1998). To conduct this analysis, we used the total negative attributional composite (T1 Ntot; internal+stable+global) in place of depressogenic attributional style (T1 DepAtt), and the total positive attributional composite (T1 Ptot; internal+stable+global) in place of enhancing attributional style (T1 EnhAtt). Finally, we substituted a new attributional interaction product term (T1 Ntot \times T1 Ptot). Using a multiple regression procedure identical to the one described above, we found that the total negative composite and total positive composite interaction did not successfully predict changes in hopelessness ($pr=-0.13$, $t[60]=-0.98$, $p=ns$).

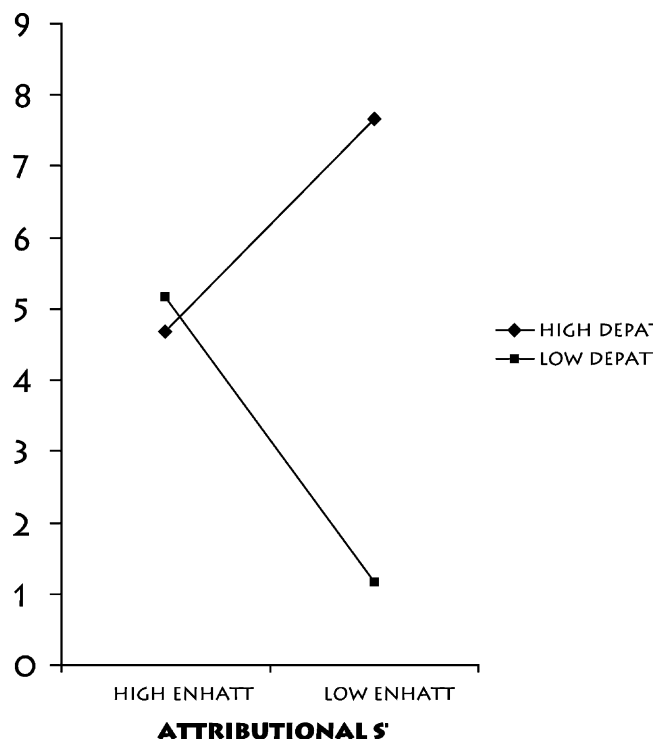


Fig. 1. Mean level of hopelessness as a function of depressogenic attributional style (DepAtt; high vs low) and enhancing attributional style (EnhAtt; high vs low). Hopelessness scores (HSC) were calculated by inserting specific values for predictor variables (i.e. 1.5 standard deviations above and below the mean scores for DepAtt and EnhAtt).

3.2. Attributional interaction does not predict changes in depressive symptoms

It was also hypothesized that the interaction of depressogenic attributional style for negative events and enhancing attributional style for positive events would predict changes in general depressive symptomatology. A multiple regression analysis was performed, using Time 2 level of depression (T2 CDI) as the dependent variable. Time 1 level of depression (T1 CDI) was entered first, thus creating residual change scores in CDI from Time 1 to Time 2. Next, we entered age, length of hospitalization, and gender values to control for both variables. We then entered the depressogenic attributional style (T1 DepAtt) and enhancing attributional style (T1 EnhAtt) variables as main effects. Lastly, we entered the depressogenic attributional style and enhancing attributional style interaction (DepAtt×EnhAtt). Contrary to our hypothesis, the interaction of depressogenic and enhancing attributional style upon admission did not successfully predict changes in depressive symptoms at the time of discharge ($pr=-0.11$, $t[59]=-0.83$, $p=ns$; See Table 3)².

² In line with the view that pre-selecting participants based on baseline T1 CDI scores affected our regression analysis, a CDI effect began to emerge once selection criteria were relaxed.

Table 3
 DepAtt×EnhAtt at Time 1 predicting residual changes in CDI from Time 1 to Time 2

Order of entry of set	Predictors in set	<i>F</i> for set	<i>t</i> for within-set predictors	df	Partial correlation (<i>pr</i>)
1	Control variable	9.72**		1, 65	
	T1 CDI		3.14**	65	0.37
2	Control variables	1.29	4, 62		
	Age		0.44	62	0.07
	Length		−0.57	62	−0.07
	Gender		−1.89	62	−0.23
3	Main effects	0.95		6, 60	
	T1 DepAtt		1.36	60	0.17
	T1 EnhAtt		−0.21	60	−0.03
4	Interaction	0.70		7, 59	
	DepAtt×EnhAtt		−0.84	59	−0.11

Note: *N*=67. CDI=Children's Depression Inventory; Length=length of hospitalization (days); DepAtt=depressogenic attributional style (stable+global attributions for negative events); EnhAtt=enhancing attributional style (stable+global attributions for positive events).

** $p < 0.01$.

4. Discussion

Results of this study demonstrate that a depressogenic attributional style (i.e. stable, global attributions) for negative events interacts with an enhancing attributional style (i.e. stable, global attributions) for positive events to predict changes in hopelessness among youth inpatients experiencing significant depressive symptomatology. Specifically, youth exhibiting a high degree of enhancing attributional style for positive events reported moderate levels of hopelessness upon discharge regardless of their degree of depressogenic attributional style for negative events. However, among youth exhibiting a low enhancing attributional style, those with a high depressogenic attributional style reported the highest level of hopelessness upon discharge (for the entire sample), and those with a low depressogenic attributional style reported the lowest level of hopelessness upon discharge (for the entire sample) (see Fig. 1).

These findings lend support for the recovery model presented by Needles and Abramson (1990), which states that an enhancing attributional style interacts with the occurrence of positive life events to predict decreases in hopelessness (or increases in hopefulness) among depressed individuals. Under the assumption that hospitalization can be regarded as a significant positive life event due to the resulting decrease in negative life events (as suggested by Needles & Abramson, 1990), participants in the current study (having experienced a 'positive' life event) demonstrated that having a high enhancing attributional style led to greater decreases in hopelessness among those exhibiting a high depressogenic attributional style — those who are particularly vulnerable to, and may have been experiencing, hopelessness depression.

It appears, however, that among youth exhibiting a low depressogenic attributional style (those not as vulnerable to hopelessness depression), there may be effects of having a strong enhancing attributional style that are detrimental to recovery. How can this initially counterintuitive finding

be explained? From the current study, it is difficult to determine with any certainty what process may be producing these results. It may be possible that depressed individuals who exhibit a low depressogenic attributional style represent victims of a more acute, reactive form of depression that is fundamentally different from individuals who experience hopelessness depression (Abramson et al., 1989). Perhaps, among these individuals, the process of attributing the occurrence of positive life events to reasons that are stable and global unexpectedly primes these individuals for future failure when their beliefs are falsified, leading to a slower recovery. Those who have low levels of both enhancing and attributional styles — participants who report the lowest levels of hopelessness at discharge — may be benefiting from the absence of stable or global attributions made toward positive *or* negative events. For this type of individual, maybe unstable (vs stable) and specific (vs global) attributions regarding any type of life event represent a more realist and productive approach to life, resulting in a faster recovery process. It should be clear that the explanation for our findings regarding the presence of an enhancing attributional style among individuals exhibiting a low depressogenic attributional style is purely speculative, and should be the focus of future studies which can explore this interesting pattern of results.

Our results did not support our hypothesis that the interaction of depressogenic and enhancing attributional styles would predict decreases in depressive symptoms, as measured by the CDI. Although our results for depressive symptoms do not support the recovery model of depression, there are a number of factors to consider when interpreting this null finding. First, our study utilized a self-report questionnaire to assess depression and did not examine the symptoms specifically associated with the hopelessness subtype of depression. The recovery model of depression is based on the hopelessness theory, which recognizes the heterogeneity of depression and explicitly proposes the existence of a cognitively mediated subtype of depression — ‘hopelessness depression’ (HD). Thus, we would expect the interaction of enhancing and depressogenic attributional style to predict decreases in hopelessness and specifically the symptoms of HD. By assessing depressive symptoms with the CDI we likely assessed a broad negative affect factor that characterizes depression, anxiety, and other emotional symptoms (Clark & Watson, 1991). Consistent with this explanation, a recent study by Hankin, Abramson, and Siler (2001), utilizing an adolescent sample, reported that depressogenic cognitive style predicted increases in hopelessness depression symptoms, but not non-hopelessness depression symptoms. Thus, it appears that attributional style may not predict levels of general negative affect, but rather is specific to the symptoms associated with theoretically derived hopelessness subtype of depression. It also is important to note that the temporal relationship between decreases in hopelessness and the subsequent decrease in depressive symptoms is not known. Thus, the relationship between the attributional interaction and future depressive symptomatology may have been affected by a somewhat restricted follow-up period (mean length of hospitalization=9.10 days).

There are limitations of the current study that should be noted when interpreting our results. First, our finding that depressogenic and enhancing attributional styles interact to predict changes in hopelessness appears to be a novel one, and is in need of replication. Second, although some aspects of our findings support the recovery model (Needles & Abramson, 1990) in a sample of youth inpatients (thus *adding* to the generalizability of their findings), it is possible that our interactive findings are not generalizable to adult populations. A final limitation of the current study is the absence of a life events measure, which may have provided empirical support for our presumption that hospitalization acted as a significant positive event among all participants.

Future studies examining the recovery process should fully test the mediational component of the recovery model while integrating the possible occurrence of attributional interactions across additional populations. Researchers are also encouraged to incorporate the effects of two additional types of inferences articulated explicitly in the hopelessness theory (i.e. consequence, and self worth implications) on recovery from depression. These are facets of hopelessness theory that are often overlooked in examinations of depressive onset, maintenance, and in this case, the recovery process.

Our findings have implications for the prevention and treatment of depressive disorders. Should future studies further demonstrate the attributional interactions found in the current study, the focus of treatment among depressed individuals may differ depending on the cognitive vulnerabilities that may or may not be present in a given individual. For example, individuals who exhibit a vulnerability to hopelessness depression may benefit from treatment focused on altering a depressogenic style of thinking; in contrast, depressed individuals who do not exhibit a depressogenic attributional style may benefit from treatment focused on altering an enhancing style of thinking that proves to be a potential precursor to failure. Similarly, among individuals who have recovered from depression, specific knowledge of individuals' attributional styles towards life events may guide preventive measures utilized to avoid recurrence.

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