CrossMark

ORIGINAL ARTICLE

Explanatory Flexibility and Explanatory Style in Treatment-Seeking Patients with Axis I Psychopathology

Ryan J. Lackner[1] · Michael T. Moore[2,3] · Julia R. Minerovic[1] · David M. Fresco[1]

Abstract Cognitive diathesis stress models of depression emphasize individual styles of attributing causal explanations to negative and positive events in life. The Attributional Style Questionnaire (ASQ) has traditionally been used to measure explanatory style, defined as an individual's habitual way of assigning causes to negative events. Explanatory flexibility, rather than focusing on the content of one's thoughts, emphasizes the extent to which individuals are able to make different attributions depending on the particular context of each event. The underlying notion is that individuals who are better able to adapt to the cues and demands of a stressful situation may be able to respond more effectively and are thereby less vulnerable to depression. Despite evidence attesting to its relevance to depression and anxiety disorders, explanatory flexibility has yet to be examined in a purely treatment-seeking sample of patients clinically diagnosed with Axis I psychopathology. The current study examined baseline levels of explanatory flexibility, along with explanatory style, in a sample of 171 treatment-seeking patients diagnosed with either major depressive disorder (MDD), generalized anxiety disorder (GAD), or at least one other Axis I disorder. Overall, the results replicate and extend past results indicating a distinction between explanatory flexibility and explanatory style. Furthermore, patients with MDD and GAD demonstrated lower levels of explanatory flexibility relative to patients with other Axis I disorders. Thus, explanatory flexibility may assist in our understanding of the etiology, maintenance, and treatment of emotional disorders, with particular relevance to MDD and GAD.

Keywords Explanatory flexibility · Explanatory style · Attributional Style Questionnaire

Introduction

The reformulated learned helplessness (Abramson et al. 1978) as well as hopelessness theories (Abramson et al. 1989), both derived from the original learned helplessness theory (Seligman 1974), are cognitive diathesis-stress models of depression that have been particularly influential in understanding the etiology, maintenance, and treatment of depression. Both models emphasize individual styles of attributing causal explanations to negative and positive events in life. Specifically, these theories maintain that vulnerability to depression arises with the development of a pessimistic explanatory style, defined as the tendency to attribute the arising of negative events to stable, global, and internal causes. Hopelessness theory (Abramson et al. 1989) emphasizes the former two dimensions and refers to this vulnerability factor as "generality," while subsequently placing less emphasis on the dimension of internality. Taken together, learned helplessness theory (Seligman 1974) along with its derivative theories, propose that an individual's propensity to become depressed is associated with how the individual interprets information or attributes causes to personal life experiences.

✉ David M. Fresco
fresco@kent.edu

[1] Department of Psychology, Kent State University, 226 Kent Hall Annex, Kent, OH 44242, USA

[2] Department of Psychology, Jackson State University, Jackson, MS, USA

[3] Present Address: Gordon F. Derner Institute of Advanced Psychological Studies, Adelphi University, Garden City, NY, USA

Pessimistic explanatory style (i.e., habitually viewing negative events as arising from stable, global, and internal causes) has traditionally been assessed using the Attributional Style Questionnaire (ASQ; Peterson et al. 1982), a self-report inventory in which respondents are presented with six hypothetical positive and six hypothetical negative situations and asked to rate the cause of the event along stable, global, and internal dimensions. In accordance with hopelessness theory (Abramson et al. 1989), explanatory style can be operationalized as a "generality" score, which is computed by averaging the values of the twelve stability and globality items across negative events to produce a score ranging from one to seven; higher scores on this measure indicate a more pessimistic style of responding. Previous research has demonstrated a link between explanatory style and various aspects of emotional disorders; for example, explanatory style has been shown to correlate with symptoms of both unipolar and bipolar depression (Seligman et al. 1988) and has also been implicated in the etiology, maintenance, and treatment of other emotional disorders (Abramson et al. 1999; Alloy et al. 1999, 2000; DeRubeis et al. 1990; Hollon et al. 1992).

Despite the potential clinical utility offered by explanatory style, targeting cognitive content as a specific mechanism of change may be a less fruitful approach than focusing instead on how information is processed. According to Hollon and colleagues, there are two distinguishable kinds of cognitions: *cognitive structures* and *cognitive products* (Hollon and Garber 1988; Hollon and Kriss 1984; Ingram and Hollon 1986; Kendall and Ingram 1989). Cognitive structures refer to the way in which information is processed, or represented in memory (Ingram and Hollon 1986). Examples of cognitive structures include the components of Beck's (1967, 1976) negative cognitive triad, or a negative schema about the self, world and future; these represent a framework, or structure through which an individual comes to organize and understand personal experiences. Cognitive products represent the end result of information processing and include, for example, negative statements about the self, directly accessible, conscious thoughts, and causal attributions. Importantly, researchers have cautioned against targeting the latter kind of cognition as a specific mechanism of change (Hollon and Kriss 1984; Safran et al. 1986), maintaining that targeting cognitive products is tantamount to targeting symptoms, an approach that is less likely to provide maximum clinical utility. In other words, focusing on *how* the individual processes information rather than the content of cognition itself may facilitate more central, "deep" change (Safran et al. 1986). Thus, researchers have recommended that treatment approaches should focus on core processes as critical change mechanisms in cognitive therapy for depression (Beck et al. 1979; Safran et al. 1986). More

recent evidence has shown that patients who simply become more optimistic by changing the contents of their thoughts evidence less durable treatment gains than patients who became more realistic in their thinking (DeRubeis et al. 2005).

One construct that emphasizes how information is being processed is explanatory flexibility, which refers to how flexible or rigid individuals are at assigning causes to events (Fresco et al. 2006a, b, 2007; Moore and Fresco 2007). Explanatory flexibility represents the degree to which individuals are able to make effective use of both historical and contextual factors when appraising the stressful situations that arise in their lives. As with explanatory style, the underlying vulnerability of the reformulated helplessness (Abramson et al. 1978) and hopelessness theories (Abramson et al. 1989), explanatory flexibility is also assessed using the ASQ; however, whereas explanatory style is an assessment of the cognitive content of the individual, as reflected by the average of values across the stability and globality items for negative events (i.e., generality score), explanatory flexibility is operationalized as the standard deviation of an individual's responses to negative life events along the stable and global dimensions on the ASQ (Fresco et al. 2007). A small standard deviation is conceptualized as the tendency to respond to negative situations in a more rigid fashion, whereas a large standard deviation indicates that the individual is able to view situations contextually and separately, and is therefore considered to be flexible in his or her casual attributions.

The role of rigidity in the pathogenesis of a variety of biomedical and psychological conditions has been a robust finding. For example, autonomic rigidity is associated with heart disease, hypertension, and various mood disorders (Thayer and Lane 2002), while affective flexibility is associated with enhanced adaptation to stress and negative life events (e.g., Rosenbaum and Ben-Ari 1985) and resilience to physical injury and illness (e.g., Miller et al. 1996). In particular, relatively recent research characterizes generalized anxiety disorder (GAD) as an inability to respond flexibly to environmental challenges (Thayer et al. 1996, 2000). Thayer et al. (1996, 2000) note that a global model of anxiety should encompass the role of attention in GAD. The same mode of rigid responding is also thought to increase risk of recurrence and relapse to depression (Teasdale 1999). Explanatory flexibility may reflect one such capacity or tendency to be flexible in one's cognitive processing. Thus, the present study adds to the ongoing investigation of individual variations of responsiveness in the face of negative events under the premise that individuals who are able to adapt contextually and flexibly to stressful situations are less vulnerable to depression and anxiety.

Considerable evidence offers validation for explanatory flexibility as a meaningful construct that adds to the current understanding of depression (Fresco et al. 2006a, b, 2007) and generalized anxiety disorders (Fresco et al. 2014) that is statistically distinct from explanatory style (Moore and Fresco 2007). For example, in a sample of 78 college students randomly selected from mass testing, explanatory flexibility was found to be modestly correlated with explanatory style ($r = -.27$; Fresco et al. 2007). Moreover, explanatory flexibility, but not explanatory style, moderated the relationship between negative life events to levels of self-reported depression measured 8 weeks later. Furthermore, Fresco et al. (2006b) found that explanatory flexibility and coping flexibility independently contributed to the prediction of latent negative affect, assessed via the *Beck Anxiety Inventory* (BDI; Beck et al. 1988) and cognitive and somatic indicators of the *Beck Depression Inventory-II* (BDI: Beck et al. 1996). This study also found that lower explanatory flexibility scores were not simply proxies from extreme responding in terms of explanatory style. Finally, two studies have shown that emotion provocation in the form of a negative mood priming challenge was associated with reductions in explanatory flexibility among individuals with either remitted major depression (Fresco et al. 2006a) or GAD (Fresco et al. 2014) as assessed by the Generalized Anxiety Disorder Questionnaire for DSM-IV (Newman et al. 2002), but not among healthy control participants. No other studies, to the authors' knowledge, have examined explanatory flexibility within the context of GAD.

Although previous studies have established the distinctiveness of explanatory flexibility from explanatory style and have demonstrated the useful contribution of explanatory style in understanding depression and anxiety, previous studies are limited in that the samples have been restricted to self-reported depressive and anxious psychopathology. Thus, the current study sought to extend and strengthen this work by examining explanatory flexibility in treatment-seeking patients who met diagnostic criteria as outlined in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-IV-TR*; American Psychiatric Association 2000) for a variety of disorders. Specifically, the current study examined levels of explanatory flexibility *and* explanatory style (generality) in a sample of patients seeking outpatient psychotherapy for one or more clinician-assessed *DSM-IV-TR* Axis I diagnoses. Given the relevance of depression and anxiety to the study of explanatory style and explanatory flexibility, patients were classified into one of three groups based upon current diagnoses: MDD, GAD, or psychiatric controls (No MDD or GAD). The hypotheses of the current study were:

1. MDD and GAD groups together would demonstrate lower levels of explanatory flexibility compared to the psychiatric control group.
2. As a clinical extension of Moore and Fresco (2007), explanatory style (i.e. generality) and explanatory flexibility would illustrate a low degree of correlation.

Method

Participants and Procedures

Data were obtained from a sample ($N = 171$) comprised primarily of college students ($n = 140$) with some community members ($n = 31$) seeking treatment within a university clinic at a large mid-western university. Of these participants, 120 were female (70.18 %) and the mean age was 22.66 years (SD = 5.01). The sample consisted of 141 Caucasian participants (82.46 %), 20 African-American participants (11.70 %), 2 Asian participants (1.17 %), 2 Hispanic participants (1.17 %), 6 participants who marked "other" (3.50 %), and one participant who failed to mark the item on race (.58 %). Participants completed the ASQ (Peterson et al. 1982) during intake, in addition to other baseline measures which are not included in the present analysis. All participants provided informed consent.

Clients were evaluated by clinic therapists with the Structured Clinical Interview for *DSM-IV-TR* Axis I Disorders (SCID-IV; First et al. 2002) prior to treatment. The SCID-IV was administered by one of six clinical psychology graduate students who were kept blind to participants' responses on the ASQ. Interviewers received extensive training in conducting the interview, including practicum training on administration techniques and 1 year of supervision rounds to establish accuracy of diagnoses. All diagnoses were confirmed by clinical supervisors, and diagnoses were given on a dichotomous, yes/no basis. Interrater reliability was established for the sample by having three interviewers rate each of 10 randomly selected SCID-IV interviews. The original interviewer recorded and rated the SCID-IV, while two other study interviewers, blind to the original ratings, independently rated the SCID-IV. Kappa (κ) was calculated by comparing each of the two additional ratings to the original rating, yielding two κ values for each interview. For this sample, interrater reliability across interviewers for the SCID-IV ranged from .79 to .87 (average κ = .83) for all diagnoses. Any discrepancies were addressed in case conference to establish consensus.

From the data obtained through SCID-IV interviews, individuals were subsequently placed into three groups

based on their diagnoses. The three groups were composed of clients with MDD, with no current or lifetime history of GAD ($n = 49$), GAD, irrespective of current or lifetime history of MDD ($n = 63$), and other Axis I disorders, with no current or lifetime history of MDD or GAD (psychiatric control group; $n = 59$). Specifically, the psychiatric control group was comprised of individuals diagnosed with eating disorder NOS ($n = 2$), panic disorder without agoraphobia ($n = 7$), anxiety disorder NOS ($n = 17$), post-traumatic stress disorder ($n = 8$), alcohol dependence ($n = 1$), anorexia nervosa ($n = 1$), alcohol abuse ($n = 2$), obsessive–compulsive disorder ($n = 2$), bulimia nervosa ($n = 2$), social phobia ($n = 4$), and adjustment disorder ($n = 13$). Within the patient subgroups, rates of comorbidity were 65 % (MDD), 77 % (GAD), and 44 % (psychiatric controls). For all individuals in either the MDD or GAD groups, MDD or GAD was identified as the primary diagnosis, allowing for other secondary and tertiary diagnoses.

The decision to allow MDD comorbidity in the GAD patient subgroup was made in accordance with previous research (Etkin and Schatzberg 2011), suggesting that depression-only individuals may represent an endophenotype dissociable from individuals with comorbid depression and anxiety. Specifically, Etkin and Schatzberg (2011) observed an increase in ventral cingulate activity and a dampening of amygdala activity during emotion regulation in both comorbid and depression-only groups; however, the depression-only group was able to compensate for this deficit by activating the anterior lateral prefrontal cortex, allowing for the regulation of emotional conflict. In addition to neural dissociability, other research has suggested that GAD is expected to create impairments over and above impairments due to MDD (Nisita et al. 1990; Roy-Byrne 1996). Thus, the current study grouped GAD and MDD comorbid individuals into the GAD subgroup.[1]

Measures

The Attributional Style Questionnaire is a self-report inventory that assesses causal attributions for six hypothetical positive and six hypothetical negative events along the dimensions of stability and globality that are each rated on a scale from 1 to 7. Higher ratings represent more pessimistic responses and more stable and global causes, while lower ratings represent more unstable and specific causes. A generality score is computed by averaging the

values of the 12 stability and globality items across negative events to produce a score that ranges from 1 to 7. In addition, explanatory flexibility can be computed by determining the standard deviation of these same 12 stable and global items for negative events. The ASQ has been shown to have adequate internal consistency ($\alpha = .70$ to .75; Peterson et al. 1982; Sweeney et al. 1986) in both psychiatric and undergraduate populations. Test–retest reliability for the negative event dimensions has ranged from $r = .52$ to .60 (Colin et al. 1981; Peterson et al. 1982; Sweeney et al. 1986). In the current study, the ASQ demonstrated adequate internal consistency ($\alpha = .77$).

The Structured Clinical Interview for *DSM-IV-TR*, Research Version (SCID-IV; First et al. 2002) is a widely used semi-structured interview that allows for current and lifetime diagnoses of Axis I disorders. The SCID-IV has demonstrated high inter-rater agreement (kappas) for current diagnosis with an overall weighted kappa of .82 and a range between .71 and .90 for specific diagnoses (Ventura et al. 1998).

Results

Explanatory Flexibility and Explanatory Style Between Diagnostic Groups

Groups did not significantly differ in age $F = (3,171) = .089$, $p = .966$. No significant differences were found between males ($M = 1.36$, $SD = .45$) and females ($M = 1.38$, $SD = .44$) on explanatory flexibility, $t(170) = -.259$, $p = .796$, nor were there differences between males ($M = 4.56$, $SD = .97$) and females ($M = 4.45$, $SD = .90$) on explanatory style, $t(170) = .737$, $p = .268$.

Patients diagnosed with MDD, GAD, as well as patients with all other *DSM-IV-TR* diagnoses were compared with regard to pre-treatment levels of generality and explanatory flexibility (see Table 1 for descriptive statistics). Omnibus tests were significant for both generality, $F(2, 168) = 6.97$, $p < .001$, Cohen's $f = .33$, and explanatory flexibility, $F(2, 168) = 3.59$, $p = .03$,

---

[1] Analyses were also conducted using non-comorbid MDD and GAD groups (i.e., MDD without GAD and GAD without MDD). Overall findings remain consistent; MDD and GAD groups together demonstrated lower levels of explanatory flexibility, $F(2, 127) = 4.61$, $p = .012$, and were more pessimistic, $F(2,127) = 4.66$, $p = .011$, compared to psychiatric controls.

Table 1 Pre-treatment generality and explanatory flexibility as a function of diagnostic group

Diagnosis	N	Generality M (SD)	Explanatory flexibility M (SD)
MDD	49	4.49 (.77)	1.31 (.50)
GAD	63	4.80 (.78)	1.31 (.42)
Controls	59	4.13 (1.06)	1.50 (.40)

MDD major depressive disorder, *GAD* generalized anxiety disorder, *Controls* treatment seeking patients with at least one DSM-IV-TR diagnosis other than MDD or GAD

$f = .21$. Follow-up planned contrasts, comparing participants with either MDD, GAD, or both, to participants diagnosed with another disorder, found significant differences for both generality, $t(168) = 3.64$, $p < .001$, $d = .56$, and flexibility, $t(168) = 2.68$, $p = .008$, $d = .41$, with both results approaching or exceeding Cohen's convention for a medium effect size (Cohen 1992).[2] Post hoc, exploratory analyses revealed that individuals with an adjustment disorder diagnosis ($n = 13$), included in the psychiatric control group, displayed relatively low levels of explanatory flexibility ($M = 1.35$, $SD = .39$) in the absence of a corresponding pessimistic style ($M = 3.74$, $SD = 1.12$).

Comorbidity Analyses

Rates of comorbidity were calculated for the sample as a whole and for each patient subgroup. Overall, 60 % of the sample received two or more diagnoses. On average, comorbid individuals met criteria for one additional diagnosis ($M = 1.11$, $SD = 1.05$). Number of diagnoses was similar for patients in the MDD group ($M = 1.00$, $SD = 1.03$), GAD group ($M = 1.42$, $SD = 1.10$), and psychiatric control group ($M = .78$, $SD = .90$). A oneway ANOVA determined that there was a significant difference in number of diagnoses between groups $F(2, 168) = 5.70$, $p = .004$. However, after controlling for instances of MDD–GAD comorbidity within the GAD group ($n = 26$), a subsequent oneway ANOVA revealed no significant differences in number of diagnoses between groups $F(2,168) = .929$, $p = .40$. Therefore, the patient subgroups were considered to be relatively similar with respect to diagnostic comorbidity. Nonetheless, a simple linear regression was calculated to evaluate symptom severity and comorbidity in relation to explanatory flexibility and style in each of the three groups. Results indicated that the addition of comorbidity as a predictor did not improve model fit in predicting explanatory flexibility $R^2 = .049$, $F(1, 127) = 1.075$, $p = .302$. However, the addition of comorbidity added significant variance accounted for in the criterion for explanatory style $R^2 = .106$, $F(1, 127) = 8.022$, $p < .01$.

Relationship Between Explanatory Flexibility and Explanatory Style

To replicate and extend the results of Moore and Fresco (2007), the correlations of generality and explanatory flexibility were compared between the three diagnostic groups used above (see Fig. 1). Averaged across all participants, the two variables were almost completely uncorrelated, $r(171) = -.05$, $p = .45$. Similar results were obtained in participants diagnosed with either GAD or MDD, or both (see Fig. 2), $r(112) = -.15$, $p = .13$, and participants diagnosed with another DSM-IV-TR disorder, $r(59) = .09$, $p = .50$. The above results are similar to findings obtained in two undergraduate, non-clinical samples (r's $= .02$ and $-.18$; Moore and Fresco 2007).

Discussion

The purpose of the current study was to examine baseline levels of explanatory flexibility and explanatory style, as measured by the ASQ, in a sample of treatment-seeking patients who met criteria for clinician assessed *DSM-IV-TR* disorders. A major limitation in previous work involving explanatory flexibility has been the use of samples with exclusively self-reported depressive and anxious psychopathology. It was hypothesized that MDD and GAD groups would demonstrate lower levels of explanatory flexibility compared to a psychiatric control group (Axis I diagnoses other than MDD and GAD). Additionally, it was hypothesized that explanatory flexibility and explanatory style would emerge as statistically distinct constructs, illustrated by a low degree of correlation. Findings from the current study largely support the hypotheses. First,
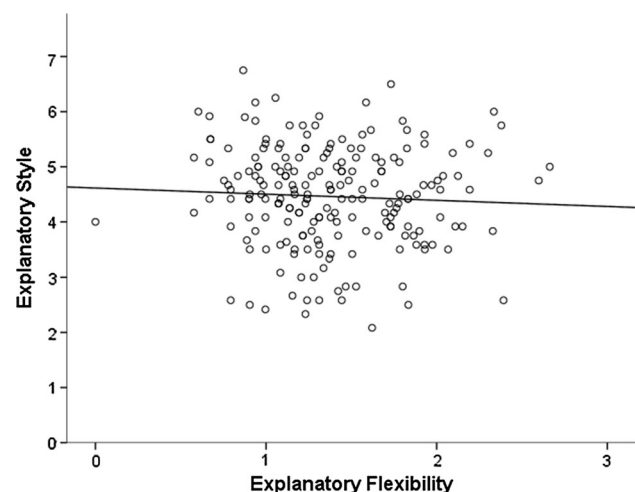
---

[2] In order to further explore the effect, we conducted the same analyses using the following 4 groups: pure MDD (n = 18), pure GAD (n = 16), comorbid GAD/MDD (n = 26), and controls (n = 46), with contrast weights of −1, −1, −2, and 4, respectively. Results support the contention that individuals with uncomplicated GAD or MDD as well as comorbid GAD and MDD evidence lower explanatory flexibility as compared to patients without GAD or MDD, $t(102) = 2.71$, $p = .008$, Cohen's $d = .26$.

Fig. 1 Correlation between explanatory style and explanatory flexibility across all patients. Pearson's $r = -.05$
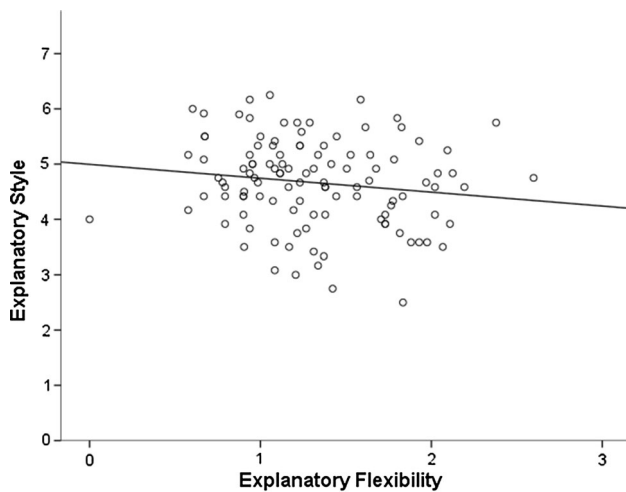
Fig. 2 Correlation between explanatory style and explanatory flexibility in MDD/GAD patients. Pearson's $r = -.15$

participants with MDD and GAD were both relatively pessimistic and rigid in their attributions of causal events, as compared to patients presenting with other Axis I diagnoses. In regards to the second hypothesis, there was no significant correlation between explanatory flexibility and explanatory style, indicating that although the two constructs may be somewhat related, they remain statistically distinguishable from one another.

The findings of the current study replicate and extend previous work (Fresco et al. 2007; Moore and Fresco 2007) demonstrating that explanatory flexibility and explanatory style are statistically distinguishable constructs measured by the ASQ. One important limitation of previous studies investigating explanatory flexibility thus far has been the use of strictly non-clinical samples. The current study illustrated that the relationship between explanatory flexibility and explanatory style (i.e., their psychometric nonequivalence) extends into clinical, treatment seeking populations.

As previously noted, the novelty of the current study was that it sought to examine explanatory flexibility in a treatment-seeking sample of patients with clinician assessed current *DSM-IV-TR* disorders. The findings indicated that patients presenting with MDD and GAD were both relatively pessimistic and rigid in their attributions of causal events, as compared to patients diagnosed with other Axis I disorders. These findings are consistent with previous theory in explanatory flexibility which posits that individuals with MDD (Fresco et al. 2006a, b, 2007) and GAD (Fresco et al. 2014; Thayer et al. 1996, 2000) respond rigidly to negative life events, as opposed to viewing situations contextually and separately, and therefore, enabling more flexibility in causal attributions.

Interestingly, one intriguing finding that emerged in exploratory, post hoc analyses was that individuals with an

adjustment disorder diagnosis ($n = 13$), who were included in the psychiatric control group, were found to display relatively low levels of explanatory flexibility ($M = 1.35$, $SD = .39$) in the absence of a corresponding pessimistic style ($M = 3.74$, $SD = 1.12$).[3] It is possible that these individuals were overwhelmed by an acute psychosocial stressor such that it became more difficult for them to incorporate content specific information, thus causing their attributions to be less varied and more rigid. Moreover, given that adjustment disorder is typically characterized as a disordered response to an external, psychosocial stressor, it may be that these individuals, although overwhelmed by an acute stressor, remain optimistic enough about other areas of life unrelated to the stressor, which may serve to counteract feelings of pessimism about their selves and the world. This finding might also be explained by the fact that many individuals with adjustment disorder display symptoms that are similar to that of depressive or anxiety disorders. However, more research is needed to fully address this question.

The current study, along with previous studies (Fresco et al. 2006a, b, 2007, 2014), strengthens the evidence that explanatory flexibility contributes to the understanding of the etiology, maintenance, and treatment of depression and anxiety. Explanatory flexibility has been shown to both predict long-term outcome of depressed mood (Fresco et al. 2006b, 2007) and serve as a mediator of improvement in CBT (DeRubeis et al. 1990; Hollon et al. 1992). Future research may want to focus on whether new or existing treatment approaches would benefit from including the flexibility with which one approaches negative life events as a target for treatment. Such an approach would ideally target an individual's cognitive processing instead of cognitive content, which may allow for more durable treatment gains (Hollon et al. 2005; Hollon and Kriss 1984; Safran et al. 1986). Treatments that invite individuals to engage in more non-judgmental and contextually based strategies (e.g., Mindfulness-Based Cognitive Therapy; Segal et al. 2002; Emotion Regulation Therapy (ERT; Mennin and Fresco 2014) may be particularly suited for the integration of such metacognitive processing treatment targets. Given the present study's contribution of demonstrating that individuals with clinically diagnosable MDD and GAD are relatively inflexible in their cognitive processing, explanatory flexibility stands as one clinically useful tool for capturing the flexibility, or rigidity of an individual's cognitive processing.

Findings of the current study should be viewed in conjunction with its limitations. First, the sample was restricted primarily to college-aged, Caucasian, females; thus, the

---

[3] The study findings remain consistent regardless of including patients with adjustment disorder in the psychiatric control group.

findings may not generalize to other populations. Second, the current study was unable to provide information regarding changes in explanatory flexibility in response to treatment. Future research will want to utilize longitudinal designs to monitor explanatory flexibility over time in response to various treatments. Third, SCID-IV inter-rater reliability was established using only 10 randomly selected cases; however, all diagnoses were made in consultation with licensed clinical psychologists, and discrepancies in diagnoses were discussed in length. Another limitation is that only SCID-IV and ASQ data were collected. Future studies may want to collect data on various mood indicators (e.g., rumination, worrying, trait anxiety, etc.) to analyze explanatory flexibility along such dimensions. Finally, responses on the ASQ are merely hypothetical responses and do not represent actual attributions made in response to real-life, negative events. A future line of research may want to examine explanatory flexibility in the context of patients responding to actual events in their lives.

In summary, findings from the current study contribute to the growing body of literature on explanatory flexibility and how it may assist in our understanding of the etiology, maintenance, and treatment of emotional disorders. The present study has established explanatory flexibility as a distinguishable construct among treatment-seeking patients presenting with a number of clinical diagnoses, with particular relevance to MDD and GAD. In future research, it will be important to corroborate existing self-report-based findings of explanatory flexibility by exploring the utility of behavioral or more objective measures. Such measures would strengthen the overall clinical utility of explanatory flexibility.

References

Abramson, L. Y., Alloy, L. B., Hogan, M. E., Whitehouse, W. G., Donovan, P., Rose, D. T., & Raniere, D. (1999). Cognitive vulnerability to depression: Theory and evidence. *Journal of Cognitive Psychotherapy, 13*, 5–20.

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review, 96*, 358–372.

Abramson, L. Y., Seligman, M. E. P., & Teasdale, J. D. (1978). Learned helplessness in humans: Critique and reformulation. *Journal of Abnormal Psychology, 87*, 49–74.

Alloy, L. B., Abramson, L. Y., Hogan, M. E., Whitehouse, W. G., Rose, D. T., Robinson, M. S., & Lapkin, J. B. (2000). The Temple-Wisconsin cognitive vulnerability to depression project: lifetime history of axis I psychopathology in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology, 109*, 403.

Alloy, L. B., Abramson, L. Y., Whitehouse, W. G., Hogan, M. E., Tashman, N. A., Steinberg, D. L., & Donovan, P. (1999). Depressogenic cognitive styles: Predictive validity, information processing and personality characteristics, and developmental origins. *Behaviour Research and Therapy, 37*, 503–531.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

Beck, A. T. (1967). *Depression: Clinical, experimental, and theoretical aspects* (Vol. 32). New York: University of Pennsylvania Press.

Beck, A. T. (1976). *Cognitive theory and the emotional disorders*. New York, NY: International Universities Press.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. A. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology, 56*, 893.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford Press.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the beck depression inventory-II*. San Antonio, TX: The Psychological Corporation.

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*, 155.

Colin, S., Sweeney, P. D., & Schaeffer, D. E. (1981). The causality of causal attributions in depression: A cross-lagged panel correlational analysis. *Journal of Abnormal Psychology, 90*, 14–22.

DeRubeis, R. J., Evans, M. D., Hollon, S. D., Garvey, M. J., Grove, W. M., & Tuason, V. B. (1990). How does cognitive therapy work? Cognitive change and symptom change in cognitive therapy and pharmacotherapy for depression. *Journal of Consulting and Clinical Psychology, 58*, 862.

DeRubeis, R. J., Hollon, S. D., Amsterdam, J. D., Shelton, R. C., Young, P. R., Salomon, R. M., & Gallop, R. (2005). Cognitive therapy vs medications in the treatment of moderate to severe depression. *Archives of General Psychiatry, 62*, 409.

Etkin, A., & Schatzberg, A. F. (2011). Common abnormalities and disorder-specific compensation during implicit regulation of emotional processing in generalized anxiety and major depressive disorders. *American Journal of Psychiatry, 168*, 968–978.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (2002). *Structured clinical interview for DSM-IV-TR Axis I disorders, research version, patient edition (SCID-I/P)*. New York: Biometrics Research, New York State Psychiatric Institute.

Fresco, D. M., Heimberg, R. G., Abramowitz, A., & Bertram, T. L. (2006a). The effect of a negative mood priming challenge on dysfunctional attitudes, explanatory style, and explanatory flexibility. *British Journal of Clinical Psychology, 45*, 167–183.

Fresco, D. M., Mennin, D. S., Moore, M. T., Heimberg, R. G., & Hambrick, J. (2014). Changes in explanatory flexibility among individuals with generalized anxiety disorder in an emotion evocation challenge. *Cognitive Therapy and Research, 38*, 416–427.

Fresco, D. M., Rytwinski, N. K., & Craighead, L. W. (2007). Explanatory flexibility and negative life events interact to predict depression symptoms. *Journal of Social and Clinical Psychology, 26*, 595–608.

Fresco, D. M., Williams, N. L., & Nugent, N. R. (2006b). Flexibility and negative affect: Examining the associations of explanatory flexibility and coping flexibility to each other and to depression and anxiety. *Cognitive Therapy and Research, 30*, 201–210.

Hollon, S. D., DeRubeis, R. J., Evans, M. D., Wiemer, M. J., Garvey, M. J., Grove, W. M., & Tuason, V. B. (1992). Cognitive therapy and pharmacotherapy for depression: Singly and in combination. *Archives of General Psychiatry, 49*, 774.

Hollon, S. D., DeRubeis, R. J., Shelton, R. C., Amsterdam, J. D., Salomon, R. M., O'Reardon, J. P., & Gallop, R. (2005). Prevention of relapse following cognitive therapy vs medications in moderate to severe depression. *Archives of General Psychiatry, 62*, 417.

Hollon, S. D., & Garber, J. (1988). Cognitive therapy. In L. Y. Abramson (Ed.), *Social cognition and clinical psychology: A synthesis* (pp. 204–253). New York, NY: Guildford Press.

Hollon, S. D., & Kriss, M. R. (1984). Cognitive factors in clinical research and practice. *Clinical Psychology Review, 4*, 35–76.

Ingram, R. E., & Hollon, S. D. (1986). Cognitive therapy for depression from an information processing perspective. In R. E. Ingram (Ed.), *Information processing approaches to clinical. Personality, psychopathology, and psychotherapy series* (pp. 259–281). San Diego, CA: Academic Press.

Kendall, P. C., & Ingram, R. E. (1989). Cognitive-behavioral perspectives: Theory and research on depression and anxiety. In P. C. Kendall & D. Watson (Eds.), *Anxiety and depression: Distinctive and overlapping features*. New York, NY: Academic Press.

Mennin, D. S. & Fresco, D. M. (2014). Emotion Regulation Therapy. In J. J. Gross (Ed.), *Handbook of Emotion Regulation* (2nd ed., pp. 469–490). New York: Guilford Press.

Miller, S. M., Shoda, Y., & Hurley, K. (1996). Applying cognitive-social theory to health-protective behavior: Breast self-examination in cancer screening. *Psychological Bulletin, 119*, 70–94.

Moore, M. T., & Fresco, D. M. (2007). The relationship of explanatory flexibility to explanatory style. *Behavior Therapy, 38*, 325–332.

Newman, M. G., Zuellig, A. R., Kachin, K. E., Constantino, M. J., Przeworski, A., Erickson, T., & Cashman-McGrath, L. (2002). Preliminary reliability and validity of the generalized anxiety disorder questionnaire-IV: A revised self-report diagnostic measure of generalized anxiety disorder. *Behavior Therapy, 33*, 215–233.

Nisita, C., Petracca, A., Akiskal, H. S., Galli, L., Gepponi, I., & Cassano, G. B. (1990). Delimitation of generalized anxiety disorder: Clinical comparisons with panic and major depressive disorders. *Comprehensive Psychiatry, 31*, 409–415.

Peterson, C., Semmel, A., von Baeyer, C., Abramson, L. Y., Metalsky, G. I., & Seligman, M. E. (1982). The attributional style questionnaire. *Cognitive Therapy and Research, 6*, 287–299.

Rosenbaum, M., & Ben-Ari, K. (1985). Learned helplessness and learned resourcefulness. Effects of noncontingent success and failure on individuals differing in self-control skills. *Journal of Personality and Social Psychology, 48*, 198–215.

Roy-Byrne, P. P. (1996). Generalized anxiety and mixed anxiety-depression: Association with disability and health care utilization. *Journal of Clinical Psychiatry,57*,86–91.

Safran, J. D., Vallis, T. M., Segal, Z. V., & Shaw, B. F. (1986). Assessment of core cognitive processes in cognitive therapy. *Cognitive Therapy and Research, 10*, 509–526.

Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2002). *Mindfulness-based cognitive therapy for depression: A new approach to preventing relapse*. New York: The Guilford Press.

Seligman, M. E. (1974). Depression and learned helplessness. In R. J. Friedman & M. M. Katz (Eds.), *The psychology of depression: Contemporary theory and research*. Oxford: Wiley.

Seligman, M. E., Castellon, C., Cacciola, J., Schulman, P., Luborsky, L., Ollove, M., & Downing, R. (1988). Explanatory style change during cognitive therapy for unipolar depression. *Journal of Abnormal Psychology, 97*, 13.

Sweeney, P. D., Anderson, K., & Bailey, S. (1986). Attributional style in depression: A meta-analytic review. *Journal of Personality and Social Psychology, 50*, 974.

Teasdale, J. D. (1999). Emotional processing, three modes of mind and the prevention of relapse in depression. *Behaviour Research and Therapy, 37*, S53–S77.

Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry, 39*, 255–266.

Thayer, J. F., Friedman, B. H., Borkovec, T. D., Johnsen, B. H., & Molina, S. (2000). Phasic heart period reactions to cued threat and nonthreat stimuli in generalized anxiety disorder. *Psychophysiology, 37*, 361–368.

Thayer, J. F., & Lane, R. D. (2002). Perseverative thinking and health: Neurovisceral concomitants. *Psychology and Health, 17*, 685–695.

Ventura, J., Liberman, R. P., Green, M. F., Shaner, A., & Mintz, J. (1998). Training and quality assurance with the structured clinical interview for DSM-IV (SCID-I/P). *Psychiatry Research, 79*, 163–173.