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Effects of a Transdiagnostic Group Treatment for Anxiety on Secondary Depression

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Abstract
Researchers have recently explored transdiagnostic anxiety treatments based on models of anxiety emphasizing a single common pathway across diagnostic categories. Results from a previous study (Norton and Hope, in press) indicated that a transdiagnostic approach was effective for both targeted and untargeted anxiety disorders. Consistent with the tripartite model, the transdiagnostic treatment should also influence symptoms of a broader pathology such as negative affectivity. This follow-up to Norton and Hope found significant decreases in depressed mood for clients undergoing transdiagnostic treatment for anxiety when compared to wait-list control participants. Although not statistically established, severity of depressive diagnoses seemed to generally decrease across treatment, whereas no change in severity occurred for those not receiving treatment.

Keywords: transdiagnostic treatment, anxiety disorder, comorbidity, depression

Introduction

Anxiety disorders rate among the most treatable psychological conditions. Meta-analyses of psychological and pharmacological treatments consistently show strong treatment effects of cognitive-behavioral treatments (CBT) and various pharmacological agents for anxiety disorders (Deacon and Abramowitz, 2004). Despite this, calls have been made for

the development of innovative but theoretically driven and evidence-based treatment approaches that will augment dissemination and accessibility (Office of the Surgeon General, 1999; Persons, 2003). In response to these calls, exploration has begun (Barlow et al., 2003; Erickson, 2003; Norton and Hope, in press; White-Lumpkin et al., 2002) on exploring transdiagnostic approaches to anxiety treatment. These transdiagnostic anxiety treatments are based on models of anxiety, such as the Clark and Watson (1991) tripartite model, that emphasize a single common pathology across the diagnostic categories as opposed to the DSM-IV implication of distinct disease states. These models hold that this core pathology ("negative affectivity") is not specific to anxiety but is also involved in depression (Clark and Watson, 1991; Eysenck, 1957).

In the only randomized, controlled trial of a transdiagnostic anxiety treatment conducted, to our knowledge, to date, Norton and Hope (in press) evaluated the efficacy of a 12-week group treatment among a sample of 23 individuals diagnosed with a range of anxiety disorders. Treatment included psychoeducation (1.5 sessions), cognitive restructuring (3.5 sessions), exposure and response prevention with feared stimuli whether via in vivo, role-played, imaginal, or interoceptive methods (6 sessions), and relapse prevention (1 session). Results of this trial provided preliminary support of the efficacy of the transdiagnostic treatment. Compared to waitlist controls, individuals completing treatment were less likely to meet diagnostic criteria for an anxiety disorder and had improved significantly on diagnostic severity measures and individualized fear-avoidance hierarchies. Currently, a multisite randomized controlled trial of a similar transdiagnostic anxiety treatment is being conducted in Canada. Although this project is not completed, preliminary data (Laposa et al., 2003) seem promising. In addition, others (Barlow et al., 2003; Erickson, 2003; Larkin et al., 2003; Schmidt, 2003; White Lumpkin et al., 2002) have conducted uncontrolled pre-post evaluations of similar transdiagnostic protocols and reported significant anxiety symptom reduction during treatment.

If the premise of a common core pathology is valid, and it is this pathology that is being targeted in cognitive-behavioral treatment (CBT), it should follow that CBT acting upon the core pathology should impact not only the targeted anxiety disorders but also untargeted secondary diagnoses related to negative affectivity. Studies have shown that despite targeting a primary anxiety diagnosis (PTSD [Blanchard et al., 2003], Generalized Anxiety Disorder [Borkovec et al., 1995], Panic Disorder [Brown et al. 1995; Tsao et al., 1998, 2002]), untargeted secondary anxiety diagnoses remitted or abated. Norton and Hope (in press) found that the aforementioned transdiagnostic anxiety treatment impacted not only primary anxiety diagnoses, but also significantly reduced the severity of secondary anxiety diagnoses. These results support the claim that CBT for anxiety disorders is effective for anxiety disorders in general, targeted or otherwise. Less evidence, however, is available regarding the effect of CBT for anxiety disorders on negative affect-related disorders in general. Blanchard et al. (2003) reported a decrease in comorbid depression after treatment for PTSD, although Brown et al. (1995) reported no such decline in comorbid depressive diagnoses after treatment for panic disorder. Tsao et al. (1998) reported a decline in comorbid depressive diagnoses after panic treatment, although this decline was smaller than that observed for comorbid anxiety diagnoses.

The purpose of this study was to examine the impact of a transdiagnostic anxiety treatment on negative affect-related disorders more generally by focusing on the treatment's influence on depressed mood and secondary depressive disorders.

Participants and Methods

Participants

Twenty-three individuals (60.9% women) meeting DSM-IV criteria for an anxiety disorder were recruited for participation in an anxiety treatment outcome study. Of these individuals, five had a clinically significant secondary diagnosis of major depression and three had a clinically significant secondary diagnosis of dysthymia. For inclusion in the study, participants were required to be (1) age 19 or older, (2) have primary DSM-IV diagnosis of an anxiety disorder, (3) demonstrate adequate proficiency in English, (4) be willing to accept random assignment, (5) be able to provide informed consent, and (6) show no evidence of serious suicidality, substance abuse, or other condition that would require immediate intervention. Participants who attended 75% or fewer of the sessions were considered to be dropouts. Four participants (three in immediate treatment), all women, dropped out from the study, yielding a final sample of 19. None of those who dropped out had comorbid depressive diagnoses. Primary anxiety diagnoses included panic disorder with or without agoraphobia ($n = 3$), social anxiety disorder ($n = 5$), obsessive-compulsive disorder ($n = 3$), generalized anxiety disorder ($n = 7$), and posttraumatic stress disorder ($n = 1$).

Ten of the completers were also receiving medication for their anxiety and were asked to provide a release to contact the prescribing physician. The participant and physician were informed of the general nature of the study and their agreement was sought to attempt to maintain the person on the same dosage and medications throughout the 12 weeks of active treatment. The participant was asked to inform his therapist of any medication adjustments the physician deems to be medically necessary during the course of the study. No medication changes were reported over the course of the treatment, save for one participant who successfully discontinued PRN benzodiazepine use. A full description of the sample is provided in detail elsewhere (Norton and Hope, in press).

Measures

All participants received a comprehensive assessment, consisting of a structured diagnostic interview and self-report questionnaires, at both pre- and post-treatment. One participant with a comorbid diagnosis of major depressive disorder completed questionnaires at post-treatment but did not return for a posttreatment structured diagnostic interview.

Anxiety Disorders Interview Schedule for DSM-IV

The Anxiety Disorders Interview Schedule for DSM-IV (ADIS-IV) (Brown et al., 1994) is a semistructured diagnostic interview designed to assess the presence, nature, and severity of DSM-IV anxiety, mood, and somatoform disorders. ADIS-IV interviewers, advanced doctoral students, and a licensed clinical psychologist were trained to rigorous standards for reliability with an expert ADIS interviewer. Because of practical constraints, ADIS in-

terviewers were not consistently blind to treatment condition or assessment point. Clinician Severity Ratings (CSR), subjective ratings to quantify the degree of severity for each diagnosis, were applied to each diagnosis. CSR range from 0 (not at all severe) to 8 (extremely severe/distressing), and a CSR of 4 (moderate impairment) is generally considered the cut-off for clinical significance.

Depression, Anxiety, And Stress Scales

The Depression, Anxiety, and Stress Scale (DASS) (Lovibond and Lovibond, 1993) is a 42-item self-report measure assessing the unique aspects of anxiety and depression as well as the common shared element of negative affectivity. The convergent and discriminant validity of the DASS subscales has also been consistently demonstrated. The Depression subscale correlates strongly with the Beck Depression Inventory, and the Anxiety subscale correlates strongly with Beck Anxiety Inventory, but the Depression and Anxiety subscales correlate only moderately with the nonrespective Beck Inventories (Antony et al., 1998; Brown et al., 1997; Lovibond and Lovibond, 1995). Furthermore, the Anxiety and Depression subscales significantly differentiate between patients with anxiety diagnoses or depressive diagnoses (Antony et al., 1998; Brown et al., 1997).

Mood and Anxiety Symptom Questionnaire

The Mood and Anxiety Symptom Questionnaire (MASQ) (Watson and Clark, 1991) is a reliable and valid (Keogh and Reidy, 2000) 90-item self-report measure also developed to assess the components of the tripartite model. The subscales of the MASQ were designed to be highly discriminative among anxiety, depression, and their common features. Consistent with predictions from the tripartite model, the nonspecific subscale tapping negative affectivity correlated moderately with both the somatic anxiety subscale (r = .58) and positive affect/anhedonia subscale (r = .58) whereas the correlation between the anxiety- and depression-specific factors was low (r = .23) (Keogh and Reidy, 2000). Estimates of convergent validity have shown that the anxiety-specific and depression-specific subscales relate strongly to other self-report measures of anxiety symptoms and depressive symptoms, respectively (Watson et al., 1995a,b).

Procedure

The study was conducted at the Anxiety Disorders Clinic of the University of Nebraska–Lincoln. Participants meeting inclusion criteria completed the questionnaires and were assessed by a trained diagnostician using the ADIS-IV. Participants were then assigned to either the immediate treatment condition or delayed treatment condition, using block randomization by primary diagnosis. After the treatment and delay periods, all participants were reassessed with the ADIS-IV and the same questionnaires. Details of the research design and treatment protocol are described in Norton and Hope (in press).

Results

As an initial test of the impact of the anxiety treatment on depressed mood, a 2 (Time: pre- vs. post-treatment) × 2 (Condition: immediate vs. delayed treatment) MANOVA, with repeated measures on the first factor, was computed using the Anhedonic Depression subscale of the MASQ and the Depression subscale of the DASS as the dependent variables. Significant main effects of Time, $F(2, 16) = 6.21$, $P = .01$, and Condition, $F(2, 16) = 4.07$, $P = .04$, were observed. The main effects, however, were modified by a significant Time × Condition interaction, $F(2, 16) = -3.64$, $P = .05$. Analysis of the interaction at a univariate level, using separate 2 (Time: pre- vs. post-treatment) × 2 (Condition: immediate vs. delayed treatment) ANOVA, indicated that participants in the immediate treatment condition improved on the MASQ-AD whereas no change was observed for waitlist control participants, $F(1, 17) = 7.50$, $P = .01$, $d = 1.33$. A nearly significant trend in the same pattern was observed for the DASS-D, $F(1, 17) = 4.06$, $P = .06$, $D = 0.98$ (Fig. 1 and Table 1).

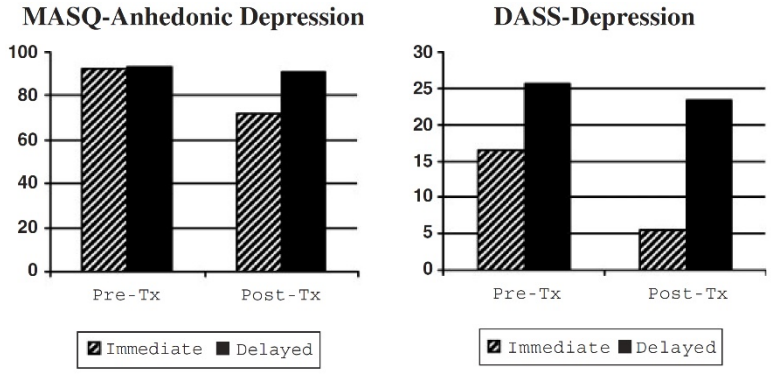

Figure 1. Change in self-reported depression by treatment condition.

Table 1. Change in self-reported depression by treatment condition

	Pre-Tx	Post-Tx	Pre-Tx	Post-Tx
Immediate	92.11 (5.17)	71.67 (5.60)	16.44 (4.24)	5.44 (3.49)
Delayed	93.00 (4.91)	91.00 (5.32)	25.70 (4.02)	23.30 (3.31)

Values are expressed as mean (*sd*).

ADIS CSR for Depressive Diagnoses were examined next. Only eight participants had an initial diagnosis of a depressive disorder. Therefore, due to the unacceptably low statistical power, the data were visually inspected but not statistically analyzed. Visual inspection (Fig. 2) of change in CSR shows that the depression CSR rating for three of four participants in the treatment condition improved to subclinical levels (CSR = < 4). In contrast, none of the participants in the control condition improved or worsened during the waitlist period. One participant receiving treatment showed a marked increase in depressive severity, such that depression became the primary concern, and significant marital difficulties became critical during this time.

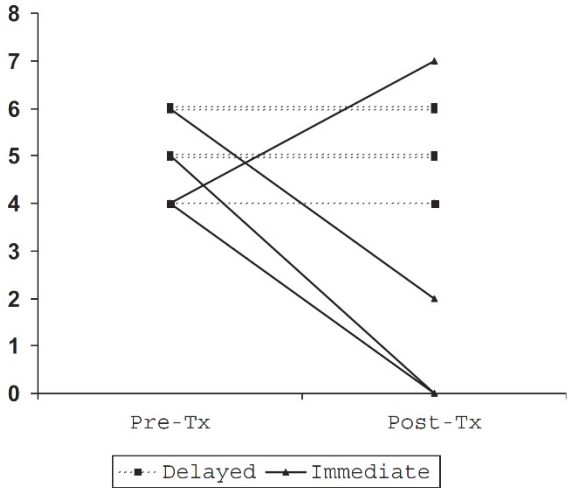

Figure 2. Change in individual ADIS clinician severity ratings of depressive diagnoses.

Discussion

The results of this study generally support the presumption that CBT for anxiety may actually impact a broader pathology, such as negative affectivity. Although no depression-specific techniques were introduced during treatment (such as behavioral activation, pleasant event scheduling, or identification and challenging of depressive automatic thoughts or schemata), indices of depression decreased significantly for those receiving treatment in comparison to waitlist control participants. Although not statistically established, it also seems that the severity of depressive diagnoses generally decreased across treatment, whereas no change in depressive disorder severity occurred for those not receiving treatment.

The results of these analyses may provide interesting support for the tripartite model (Clark and Watson, 1991) and other common pathology models of anxiety and depression (e.g., Eysenck, 1957). If clinical anxiety and depression were distinct disease entities, perhaps linked only at a predispositional level, then this effect of the anxiety treatment on depressive symptoms would not be expected. Of course, additional research employing attention-control conditions will be required to rule out the possibility of nonspecific treatment effects accounting for the changes in depressiveness. Furthermore, it is possible that the reduction in depressiveness may have resulted from other factors than changes in negative affectivity, such as decreased avoidance leading to greater behavioral activation. If the former, these data may suggest a preliminary affirmative response to Clark et al. (1994) question, "Can we treat the underlying personality variable rather than the specific manifest disorder?" (p. 113).

The principal limitation of this study is the limited sample size in general, and the very small number of individuals with comorbid depressive diagnoses in particular. The interaction effect sizes for indices of depression (d = 1.33 and 0.98) were similar to those we obtained on the indices of anxiety (Norton and Hope, in press) ($d \approx .75$–1.44). Given that the DASS and MASQ were specifically designed to maximally discriminate anxiety and

depressive symptoms, it is unlikely that the similarity in effect on anxiety and depression was a function of symptom overlap or imprecise measurement. A second limitation of this study was that all of the measures were either based on direct client report or clinician ratings based on client interviews. This opens the possibility that the data may have been influenced to some degree by factors such as over- or under-reporting symptom severity, biased or distorted recall, and so forth. As noted earlier, diagnostic interviewers were not consistently blind to condition or assessment period. The similar pattern of change on self-report outcome measures diminishes the credibility of experimenter bias effects as an explanation for the observed treatment effects.

One must be careful not to assume that CBT for anxiety represents a panacea for all negative affect spectrum disorders, nor should one infer that treating an anxiety disorder will automatically alleviate secondary depression. Clearly, much more research is necessary to better understand the pathological targets impacted by treatment, and extent of diffusion of treatment gains to non-targeted concerns. These data, however, in conjunction with those of Blanchard et al. (2003), Borkovoc et al. (1995), Brown et al. (1995), and Tsao et al. (1998, 2002) do provide preliminary evidence suggesting that CBT targeted to anxiety may have an impact beyond the level of specific diagnoses or even diagnostic genus. Trans-diagnostic treatments designed to target negative affect syndromes in general may prove valuable for more broadly mixed groups inclusive of all negative affect spectrum disorders, including depressive disorders. Evaluations of this possibility are reportedly underway (Barlow et al., 2003).

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