Changes in Affect During Treatment for Depression and Anxiety

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Abstract

We tested the hypothesis that the tripartite model (Clark & Watson, 1991) can be extended to account for change during treatment for anxiety and depression. Forty-one patients treated naturalistically in private practice with cognitive behavior therapy completed weekly measures of depression, anxiety, negative affect (NA), positive affect (PA), and anxious arousal (AA). Consistent with the model, NA was associated with anxiety and depression during treatment, PA was more strongly related to depression than to anxiety, and AA was more strongly related to anxiety than to depression. As predicted, symptoms of depression and anxiety and NA all decreased during treatment. As predicted, AA also decreased, particularly for patients with panic disorder. PA increased during treatment, but only for patients who showed a significant decline in depression and only over an extended period of treatment. Nearly two-thirds of the variance in anxiety change was accounted for by changes in depression and NA, and just over three-fourths of the variance in depression change was accounted for by changes in anxiety and NA, indicating that much of the change in anxiety and depression across the course of treatment is shared in common.

Introduction

Depression and anxiety are among the most common psychological disorders, with lifetime prevalence rates for depression at just over 16 percent (Kessler et al. 2003) and for the anxiety disorders at nearly 29 percent (Kessler et al., 2005). Not only are these conditions common, they are also highly comorbid. Nearly two thirds of individuals with depression meet diagnostic criteria for an anxiety disorder (e.g., Mineka, Watson, & Clark, 1998), and as many as 50 percent of individuals who meet criteria for an anxiety disorder are depressed (e.g., Brown et al., 2001).

Given the substantial overlap of anxiety and depression, researchers and clinicians alike have long been interested in understanding the relationship between them. One of the most influential theories about the relationship between anxiety and depression is the tripartite model proposed by Clark and Watson (1991; Watson, Clark et al., 1995; Watson, Weber, et al., 1995; Watson, Weise, Vaidya, & Tellegen, 1999). In this model, a general distress factor characterized by high levels of negative affect (NA) is common to both anxiety and depression. A positive affect (PA)/ anhedonia factor[1] that is characterized by low levels of positive affect (PA) or pleasurable engagement with the environment is specific to depression, and a third factor, variously referred to as anxious arousal (AA) or somatic arousal, is specific to anxiety. A revision to the model was later proposed to better account for the heterogeneity among the anxiety disorders (Mineka, Clark, & Watson, 1998). The revised model, termed the integrative hierarchical model, followed from additional data suggesting that high levels of AA were more

characteristic of panic disorder rather than all of the anxiety disorders (Brown, Chorpita, &

Barlow, 1998; Zinbarg & Barlow, 1996).

The tripartite model has achieved broad empirical support across children, younger and

older adults, college students and psychiatric patients (e.g., R. Beck et al., 2001; Brown,

Chorpita, & Barlow, 1998; Cook et al. 2004; Joiner, Catanzaro, & Laurent, 1996; Lonigan,

Phillips, & Hooe, 2003; Marshall et al. 2003; Watson, Clark, & Carey, 1988; Watson, Clark et

al., 1995; Watson, Weber, et al., 1995). However, some studies have found more limited support

for the model, perhaps reflecting differences in data analytic techniques (e.g., Burns & Eidelson,

1998; Wetherell, Gatz, & Pedersen, 2001), less clear applicability in elderly samples (e.g., J. G.

Beck et al. 2003; Shapiro, Roberts, & Beck, 1999; Wetherell, et al., 2001; but see Cook et al.,

2004), or differences in measures of the three factors (e.g., Burns & Eidelson, 1998; Mineka et

al., 1998; Wetherell, et al., 2001). Taken together, there is a good deal of support for the central

tenets of the tripartite and integrative hierarchical models, namely that NA characterizes both

anxiety and depression, low PA is more characteristic of depression than anxiety, and AA is

more characteristic of anxiety, especially panic disorder, than depression. Most support for the

tripartite model comes from cross-sectional studies, and few studies have been carried out in

patients receiving treatment.

In the study reported here, we tested the hypothesis that the tripartite model can be

extended to the pattern of changes in depression and anxiety over time—and, in fact, during

treatment. We predicted that over the course of treatment, patients would show reductions in

symptoms of anxiety, symptoms of depression, and NA, and increases in PA. We predicted that

reductions in panic would be associated with reductions in AA, especially for patients with panic disorder. Tests of these hypotheses have both theoretical and clinical importance. They have theoretical importance because they extends the boundaries of the explanatory power of the tripartite model, and they have clinical importance because increased understanding of the relationship between anxiety and depression over time and during treatment has the potential to inform clinicians' work with anxious depressed patients and even to lead to new and improved treatments for these patients.

To our knowledge, only three studies have assessed change in NA, PA, or AA over time and during treatment, and all these studies were of carefully selected patients with depression. Mohr et al. (2005) showed that over the course of a 16-week telephone-administered psychotherapy, depressed multiple sclerosis patients who were randomly assigned to either cognitive behavioral (T-CBT) or supportive emotion focused therapy (T-SEFT) showed decreases in depression symptoms and increases in PA (NA and AA were not assessed). The patients who received T-CBT showed a greater decrease in most measures of depression and a greater increase in PA than patients who received T-SEFT.

Tomarken and colleagues (Tomarken, Dichter, Freid, Addington, & Shelton, 2004) measured change in depression, anxiety, NA, PA, and AA in outpatients with depression across 12 weeks of medication treatment (bupropion [wellbutrin] SR). During the first six weeks of treatment (phase one), patients were randomly assigned to receive medication or placebo. During the last six weeks of treatment (phase two), all patients knowingly received medication. NA, PA, and AA were assessed before and during treatment using the Mood and Anxiety Symptoms

Questionnaire (MASQ), a measure developed by Watson and colleagues to test the tripartite model (Watson, Clark, et al., 1995; Watson, Weber et al., 1995). During phase one, depressed patients who received medication showed a significantly larger decrease in depression and NA and a significantly larger increase in PA than patients who received placebo. No change in AA or anxiety occurred in either group during phase one. In phase two (medication for all), all patients showed a decrease in depression, anxiety, NA and AA. Only the group who received medication in both phases showed a significant increase in PA. There was no significant group difference in the rate of decline in depression symptoms during phase two, although the patients who received medication in both phases had a very low level of symptoms at the end of treatment.

Finally, a third study reported preliminary findings on changes in PA, NA, and AA across 16 weeks of treatment for depression with cognitive behavior therapy or paroxetine (Paxil) (Schmid, Freid, Hollon, & DeRubeis, 2002). PA, NA, and AA were assessed at pre-treatment, mid-treatment (week 8), and post-treatment. Regardless of treatment type, all patients experienced a significant decrease in NA and AA, and a significant increase in PA over the course of treatment. Changes in NA and PA were more rapid for patients receiving medication, but by the end of treatment there were no differences between the treatment groups. Data on comorbid anxiety disorders were presented for half the sample, and there were no differences in PA, NA, or AA at pre-treatment for depressed patients with and without a comorbid anxiety disorder. Unfortunately, data regarding change among individuals with comorbid anxiety were not presented.

These studies support the notion that the tripartite model's predictions about depression can be extended to account for changes over the course of treatment. As predicted by the (extended) model, the studies showed that treatment was associated with decreased depression, decreased NA, and increased PA. None of these studies, however, tested whether the tripartite model's predictions about anxiety can be extended to account for changes over the course of treatment. In the present study, we collected weekly measures of symptoms and affect in order to address whether the tripartite model's predictions about depression and anxiety can be extended to account for changes during treatment in patients who have symptoms of depression *and* anxiety.

The study reported here also sought to extend the tripartite model by assessing the degree to which change during treatment reflects change in common or distinct features of anxiety and depression. According to the tripartite model, general emotional distress, or NA, is common to both anxiety and depression. An intriguing and as yet unanswered question regarding the relationship between measures of anxiety, depression, and NA is how much of the variance in change during treatment is accounted for by common aspects of anxiety and depression, including NA, versus how much is accounted for by distinct aspects of anxiety and depression. The answer to this question is theoretically important in that it can further extend the tripartite model by illuminating the nature of the change that occurs during treatment. In addition, understanding the amount of variance in change that is common and specific can guide treatment development by informing us about what actually changes during treatment.

Thus, the present study examined whether the tripartite model can characterize change in anxiety and depression across the course of treatment in a naturalistic and highly comorbid sample, and whether change during treatment is best captured by common or specific aspects of anxiety and depression. We tested several hypotheses based on the tripartite model. First, we hypothesized that during treatment, NA would be related to both anxiety and depression, PA would be more strongly related to depression than anxiety, and AA would be more strongly related to anxiety than depression. Second, we tested the hypothesis that the tripartite model can be extended to account for change during treatment for anxiety and depression. We tested the hypotheses that as depression decreased, NA would be significantly reduced and PA would be significantly increased. Consistent with the integrative hierarchical model, we further hypothesized that as anxiety decreased, NA would be significantly reduced, and AA would also be significantly reduced, but primarily for those patients with panic disorder. Finally, we assessed the proportion of the variance in change during treatment that is attributable to common versus specific aspects of the anxiety and depression measures.

Method

Participants

Participants were recruited via fliers announcing the study that were part of the new patient intake packets at the San Francisco Bay Area Center for Cognitive Therapy (SFBACCT). Forty-four individuals (30 women; 14 men) consented to be in the study. The sample was primarily white (n=40), with a mean age of 35.75 (SD = 13.31). Diagnoses were assigned by the treating clinician at the beginning of treatment using the fourth edition of the Diagnostic and

Statistical Manual (DSM-IV-TR; APA, 2002). More than two-thirds of the participants (n = 28; 68.3%) were diagnosed with at least one mood disorder and at least one anxiety disorder. One participant with an adjustment disorder with mixed anxiety and depression was included in this group. Six participants (14.6%) were diagnosed with mood disorder only, and seven participants (17.1%) were diagnosed with anxiety disorder only. Mood disorder diagnoses included major depressive disorder (n = 24) and dysthymia (n = 10). Anxiety disorder diagnoses included panic disorder (n =6), generalized anxiety disorder (n = 16), social phobia (n =17), specific phobia (n = 1), and obsessive compulsive disorder (n =3). Eleven participants had more than one anxiety disorder. Nearly a third (n = 13) of the sample also carried an Axis II diagnosis. Because the study addressed the relationship between depression and anxiety symptoms, three participants without diagnoses of major depressive disorder or an anxiety disorder were excluded, leaving a final sample size of 41.

Measures

Beck Depression Inventory (BDI). The BDI is a widely-used, reliable, and valid self-report measure that assesses the presence and severity of depression symptoms (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961; Beck & Steer, 1987). The 21 items are each scored from 0 to 3. Participants were asked to choose the statements that best described the way they had been feeling for the past week. Cronbach's alpha (Cronbach, 1951) at intake for the BDI was .87.

Burns Anxiety Inventory (Burns AI). The Burns AI is a 33-item self-report measure that assesses the presence and severity of various anxiety symptoms, including thoughts, feelings, and physical symptoms experienced in the last week (Burns & Eidelson, 1998). Participants

responded to each item using a 0 (not at all) to 3 (a lot) Likert scale. The Burns AI was chosen

because it is a part of standard clinical practice at the SFBCCT; it assesses a wide range of

anxiety symptoms; it is sensitive to change during treatment (e.g., Persons, Roberts, & Zalecki,

2003), and it is internally consistent and related to other measures of anxiety symptoms (e.g.,

Burns & Eidelson, 1998; Persons et al., 2003). Cronbach's alpha at intake for the BAI was .92.

A subscale of eight items from the Burns AI was used to assess *Anxious Arousal (AA)*.

Persons et al. (2003) developed this measure of AA by selecting items from the Burns AI that

approximated the AA symptom list presented by Watson, Weber, et al. (1995). The eight items

included fear of physical illness/dying; pain/tightness in chest; tingling in toes/fingers; sweating;

trembling/shaking; dizziness/lightheadedness; choking sensations; and hot flashes/cold chills. In

addition to its face validity, Persons et al (2003) found that this measure was sensitive to change

during treatment and related to a measure of depression that did not contain overlapping anxiety

items. In the present study, coefficient alpha for the AA scale at intake was .82, and the average

alpha across the first 12 weeks of treatment was .79.

Positive and Negative Affect Schedule (PANAS). Experienced emotion was assessed

using the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988),

which contains 20 emotion adjectives. The PANAS was designed to measure Positive affect

(PA) and Negative affect (NA), two factors that have been reliably produced in several studies of

emotion (e.g., Watson & Tellegen, 1985; Watson, et al., 1999; Zevon & Tellegen, 1982). To fill

out the PANAS, participants were instructed to indicate using a 5 point Likert scale (1 = very

slightly or not at all; 5 = extremely) the extent to which they felt each emotion during the past

week. These instructions were chosen to be consistent with the week time frame covered by the

BDI and Burns AI. Cronbach's alphas at intake for the PA and NA subscales were .80 and .84,

respectively.

Treatment

Participants completed the three measures weekly in the waiting area prior to their

therapy session. Two of these measures, the BDI and Burns AI, are part of standard clinical

practice to track progress, and they are often discussed during a weekly session. The PANAS

was added to this packet of questionnaires but was not discussed during the therapy sessions.

Participants were treated with individual, case formulation-driven cognitive behavior therapy

(CBT) (Persons, 1989, 2005). The case formulation was used to guide an individualized

treatment plan for each patient. Idiographic treatment plans were based on nomothetic evidence-

based cognitive behavioral (Nathan & Gorman, 2002), especially Beck's cognitive therapy for

depression (Beck, Rush, Shaw, & Emery, 1979) and anxiety (Beck, Emery, & Greenberg, 1985).

Typical interventions included self-monitoring, activity scheduling, cognitive restructuring,

contingency management, social skills training, and exposure. Treatment goals included the

reduction of depression and/or anxiety, as well as other individualized goals such as improving

interpersonal relationships, among others.

Sixteen participants were treated by the second author, a PhD level clinical psychologist

with 20 years of experience. The remaining participants were treated by PhD level clinicians

with between 1 and 7 years experience. Participants received an average of 18 sessions of

therapy (SD = 12.67; median = 16; mode = 8). All but one participant (who received only two sessions of therapy), received at least six sessions of therapy.

Seventy percent (n = 29) of the participants were also taking medication. Fifteen participants were taking what could be classified as an antidepressant (Celexa, Effexor, Lexapro, Paxil, Prozac, Wellbutrin, Zoloft); four were taking both anti-anxiety (Ativan, Buspar, Klonopin Xanax) and antidepressants; one was taking only anti-anxiety medications; five were taking anti-anxiety or antidepressants in combination with an insomnia medication (Trazodone, Remeron, Provigil, Prosom); one was taking an antidepressant in combination with a stimulant (Strattera); one was taking a stimulant only, and one was taking an insomnia medication only. Thirteen participants were taking one medication; nine were taking two medications; three were taking three medications, and four were taking four medications.

Results

Relationships Between Depression, Anxiety, NA, PA, and AA

The tripartite model makes specific predictions about the relationships between anxiety, depression, and emotional states. In order to evaluate whether these predictions held in our sample over the course of treatment, we examined within-subject correlations between the weekly measures. That is, for each participant at each week, we computed a correlation between the measures of symptoms and emotional states in order to assess whether symptoms and emotion were related over the course of treatment. We examined within-subject correlations because they can be less susceptible than between-subject correlations to systematic response biases (e.g., Watson et al., 1999) such as acquiescence. Within-subject correlations were

calculated as simple Pearson correlation coefficients within each participant at each of the

multiple administrations of the measures for that participant. Only participants with at least 5

administrations of the measures (n = 36) were used. Table 1 presents the means of these within

subject correlation coefficients across all of the participants. As predicted, the BDI and Burns AI

were significantly positively related to NA and significantly negatively related to PA.

Importantly, the magnitude of the correlation between PA and the BDI was greater than the

correlation between PA and the Burns AI, t (34) = 2.14, $p < .05$, supporting our prediction that

PA is more strongly related to symptoms of depression than anxiety (e.g., Watson, Weber, et al.,

1995). The correlation between AA and the Burns AI is not readily interpretable due to item

overlap between the AA and Burns AI scales, as well as common method variance, because both

AA and Burns AI were measured with items on a single scale (the Burns AI). When we excluded

the AA items from the Burns AI, the correlation between AA and the "purged" Burns AI scale

was .48. A paired t-test showed that, as predicted, this correlation was higher than the correlation

between the BDI and AA, t (43) = 2.33, $p < .05$. Thus, consistent with expectations based on the

tripartite model, AA was more strongly related to symptoms of anxiety than depression.

Analysis of Change

We used mixed effects growth curve models to evaluate change in measures of

depression, anxiety, positive and negative affect, and anxious arousal during treatment (Bryk &

Raudenbush, 2002). At Level 1 of the analysis, these models evaluate change within each

individual by regressing the dependent variable on time (using the multiple administrations of a

measure across time for that individual) and estimating an intercept and slope parameter for each

individual. At Level 2 of the analysis, the models estimate intercept and slope parameters from variables that vary across participants, in order to examine systematic differences between participants. Mixed effects growth curve models have a number of advantages over traditional models of repeated measures data. These models treat change as a continuous trajectory across multiple time points rather than as a comparison between distinct time points. This fact makes it possible to take advantage of all available data, even when length of time between measurement occasions varies. In addition, earlier models assume that change can be adequately characterized by a fixed parameter for all participants. Mixed effects models estimate intercept and slope parameters for each participant, thus accounting for heterogeneity across participants both in starting levels and in change over time. These models are described in detail by Raudenbush and Bryk (2002) and Singer and Willett (2003)[2].

We employed mixed effects linear growth curve models to assess change across time in treatment (measured in weeks). We chose to model the first 12 weeks of therapy because (a) missing data beyond 12 weeks were not random, as 50% of patients had 16 or fewer sessions of psychotherapy (cf. Barkham et al., 2006); and (b) there is evidence that cognitive behavior therapy typically causes significant change in 12 weeks (e.g., Ilardi & Craighead, 1994; Tang & DeRubeis, 1999)[3]. Except where noted below, the estimation procedure for all models was full maximum likelihood.

For most models in which significant change did occur, change was curvilinear across time, such that change was more rapid nearer the beginning than later in treatment. A change trajectory of this sort has been observed in cognitive behavior therapy (e.g., Ilardi & Craighead,

1994; Tang & DeRubeis, 1999) and in psychotherapy more generally (Howard, Kopta, Krause, & Orlinsky, 1986; Lambert, Hansen, & Finch, 2001). The curvilinear effect was evidenced by a significant, positive quadratic fixed effect for the BDI, Burns AI, and NA analyses (all ps < .0001). A series of transformations were considered to account for this effect, following the ladder of transformations suggested by Mosteller and Tukey (1977; see also Singer & Willett, 2003). A logarithmic transformation satisfactorily accounted for curvilinearity in all of the models, with quadratic effects no longer being significant (all ps > .35). We thus used log-transformed time, ln(time[in weeks]+1), in all analyses presented below.

We tested models of change in BDI, Burns AI, NA, AA, and PA scores across the course of treatment[4]. Each measure was regressed on time since intake. The intercept is thus the estimated score at intake and the slope is the rate at which the score is expected to change across the course of treatment. Results are presented in Table 2 and shown graphically in Figure 1. For ease of interpretation, the values in Figure 1 have been rescaled with a z-transformation based on means and standard deviations at intake. The scores on the measures at the beginning of treatment were in the mildly to moderately elevated range, with the possible exception of the AA subscale (standardization data are not available).

BDI, Burns AI, and NA scores decreased significantly across the course of treatment[5]. These results are consistent with the expectation of improvement in symptoms, and with the prediction from the tripartite model that NA represents a feature shared with depression and anxiety. As predicted, AA also decreased during treatment. Based on the integrative hierarchical model (Mineka et al., 1998), we predicted that initial AA would be highest in individuals with

panic disorder and that reductions in AA would be most evident in this group. To test this

hypothesis, we classified participants as having a panic disorder diagnosis (N=6) or having no

panic diagnosis (N=35). We entered the diagnostic variable in the model as a level 2 fixed effect,

predicting starting levels and rate of change in AA symptoms. As expected and shown in Figure

2, individuals with panic disorder had higher initial levels of AA, $t(39) = 3.18$, $p < .01$, and

showed a greater reduction in AA symptoms with treatment, $t(286) = -2.38$, $p < .05$, than those

without panic disorder. Individuals without panic disorder did not show any significant change in

AA symptoms across treatment when they were considered separately from those with panic

disorder, $t(286) = -1.07$, n.s. Given the small sample size, these results must be interpreted with

caution.

Contrary to predictions, PA did not increase during treatment, even as depression

decreased. The finding is particularly surprising given that BDI and PA scores were significantly

negatively correlated in this sample ($r = -.34$, $p < .05$). We conducted post-hoc analyses to

determine if there were circumstances under which an increase in PA did occur. Like all post-hoc

analyses, they should be interpreted with caution.

We reasoned that increases in PA may have been more likely to occur in patients who

showed a full and prolonged recovery from a hefty level of initial depression symptoms. To test

this idea, we included eight participants who had at least moderate levels of depression

symptoms at intake (BDI >= 18) and were no longer depressed after 20 weeks (BDI <=10)

(Kendall, Hollon, Beck, Hammen, & Ingram, 1987). Within this group, PA increased

significantly over the course of 20 weeks, $t(78) = 2.06$, $p < .05$. Initial PA in this group was

14.87± 4.82 s.e. The rate of change across log transformed time was 4.86±2.35 s.e., indicating a typical increase of about 14 points across 20 weeks. It is notable that even within this select group, there was no significant evidence of change in PA if only the first 12 weeks were considered, $t(53) = .35$, n.s. even though this group showed a reduction in BDI score from 29 to 10 points by week 12, $t(53) = -3.85$, $p < .0005$. Given the small sample size in this group, we consider these results to be suggestive and not conclusive.

Common and Specific Change

Across the course of treatment, the anxiety, depression, and NA measures were strongly intercorrelated (see Table 1). In order to examine the extent to which the change participants showed during treatment was captured by the measure of general distress (NA) or by the distinct measures of depression and anxiety symptoms, we modeled change in each measure (BDI, Burns AI, and NA) while controlling for scores on the other two measures. For instance in examining the specific change in the BDI, the prediction equation for BDI scores included the concurrent BAI and NA scores, as well as slope, intercept, and residual terms. Table 3 presents parameter estimates for rates of change in these models. Results indicated that the BDI measured change over the course of treatment that was not captured by the other two measures (Burns AI and NA). Similarly, the Burns AI measured change over the course of treatment that not captured by the BDI and NA. However, the NA measure showed no change over the course of treatment when BDI and Burns AI scores were controlled.

To examine the *degree* to which the changes in BDI and Burns AI scores were specific to these measures, we partitioned the variance associated with change over time into specific and

non-specific portions. The non-specific portion was that portion of the change in the measure that was shared in common with the other measures, whereas the specific portion was captured uniquely by that measure. For this partitioning of variance, models were rerun using restricted maximum likelihood, which isolates variance components during the estimation procedure. We used a modified version of the pseudo-R^2 associated with mixed models (Raudenbush & Bryk, 2002), a statistic that compares variance components between models in order to determine the proportional reduction in variability when predictor variables are added. We were interested in what portion of the variance in an outcome measure (level 1 variance) was unique to the outcome measure of interest when scores on the other outcome measures were controlled[6]. This analysis showed that 22% of the change during treatment in BDI scores was specific to the BDI, and 78% was shared with the other measures. The analysis also showed that 36% of the change during treatment in Burns AI scores was specific to the Burns AI, and 64% of the change was shared with the other measures.

Discussion

We tested the hypothesis that the tripartite model could be extended to account for relationships between anxiety and depression over the course of treatment in this naturalistic and highly comorbid sample. We predicted and found that NA was related to both anxiety and depression, PA was more strongly related to depression than anxiety, and AA was more strongly related to anxiety than depression. The linkage between AA and anxiety must be interpreted with caution, however, as the items to assess each construct were drawn from the same measure

(Burns AI). Moreover, despite its high reliability, our measure of AA has less validity data than the other measures used in the study, thus further tempering our conclusions regarding AA.

In addition, we predicted and found that decreases in depression and anxiety symptoms corresponded to decreases in NA across 12 weeks of treatment. AA also decreased significantly during treatment; consistent with our predictions and with the integrated hierarchical model (Mineka et al., 1998), this effect was largely specific to individuals with panic disorder. In fact, when individuals without panic disorder were considered separately from those with the disorder, there was no evidence of change in AA levels across treatment for this group. It is important to note, however, that our sample contained only 6 individuals with panic disorder, and thus our findings must be interpreted somewhat cautiously. Taken together, these findings have both theoretical and practical implications. First, by examining the linkages among depression, anxiety, and NA across the course of treatment, our findings suggest that the tripartite model can indeed be extended to account for changes in anxiety and depression during treatment. Second, the findings extend the tripartite model to a naturalistic and highly comorbid sample, thus bolstering the model's external validity.

Contrary to our predictions, PA did not significantly increase across treatment in the entire sample. This finding stands in contrast to three previous studies of treatment for depression (Mohr et al., 2005; Schmid et al., 2002; Tomarken et al., 2004). Why did we not find an increase in PA? First, changes in PA may happen later in the course of treatment, after symptoms of anxiety and depression remit. That is, patients' energies may first be directed towards recovering from depression. After depression symptoms have remitted, patients may

then be able to focus on increasing pleasurable engagement with the environment. Consider the example of an athlete whose knee injury requires surgery. The early focus of recovery is to heal tissue damage and reduce pain. It is only later that moving around, even running, becomes enjoyable once again. In our post-hoc analyses, we found tentative support for this idea. Participants who showed a full recovery from at least a moderate level of depression symptoms evidenced a significant up tick in PA, but only when a period of 20 rather than 12 weeks in treatment was considered. This finding suggests that changes in PA and depression across time may not occur simultaneously, though the post-hoc analysis and our small sample size points to the importance of replicating this finding in future studies.

Second, PA may not have exhibited much change because it was not a central focus of treatment. The cognitive behavior therapy provided in the study was heavily influenced by the theorizing of Beck. Thus, the clinical focus was more often on reducing symptoms than on increasing pleasure or engagement with the environment. Participants' attention to their anxiety and depression symptoms was also heightened by asking them to complete weekly depression and anxiety measures. Other cognitive behavior therapy models place more emphasis on pleasure and positive affect, such as Lewinsohn's behavioral model (e.g., Lewinsohn, Hoberman, & Hautzinger, 1985), behavioral activation (e.g., Martell, Addis, & Jacobson, 2001), and self-system therapy (e.g., Vieth et al. 2003). For example, behavioral activation encourages patients to get moving again in order to get them back in contact with the reinforcers that activate their behavior and increase pleasure and satisfaction. An important goal of self-system therapy is to help patients increase behavior in the service of meaningful and motivating personal goals.

Given that PA is associated with levels of engagement with the environment and positive social relationships, both of which may serve as protective factors against depression (e.g., Keltner & Kring, 1998), specific efforts to elevate PA with psychotherapeutic interventions could be useful both in improving patients' quality of life and in preventing relapse. Studies designed to unpack the ways in which PA may or may not change over the course of treatment are an exciting avenue for future research.

Third, the high level of comorbidity in the sample may have contributed to the relative lack of change in PA. The tripartite model was developed to help distinguish anxiety from depression, and the data generated in support of the model have for the most part been collected from fairly "pure samples" of depressed patients that were characterized by low levels of PA (e.g., Mohr et al, 2005; Tomarken et al., 2004). More than two thirds of the participants in the present study had both anxiety and depression, and this level of comorbidity is likely higher than in the three other studies that assessed change in affect during treatment.

We also examined the extent to which changes in anxiety and depression during treatment reflected aspects common across symptoms and general distress or aspects specific to anxiety and depression symptoms. Our results provide support for both common and specific change. That is, across the course of treatment, some of the variance in change was common to the measures of anxiety, depression, and NA, and some of the variance in change was specific to the measures of anxiety and depression symptoms. With respect to specific change, just over a third of the variance in change in anxiety during treatment was specific to anxiety and not accounted for by changes in depression or NA. Similarly, just under a fourth of the change

variance in depression was specific to depression. Thus, while unique features of anxiety and depression are present that may require therapeutic attention and assessment, our findings suggest that much of the change in anxiety and depression across the course of psychotherapy is shared in common. A single symptom measure of the elements common to depression and anxiety could capture the majority of symptom change that occurs in psychotherapy. The use of a single measure could provide significant advantages in time and efficiency that outweigh the relatively smaller loss of information about change that results from not measuring depression and anxiety separately. The proportions of shared variance in change across the three measures are consistent with previous theories (e.g., Barlow, 2000) and findings (e.g., Persons et al., 2003) proposing that change in anxiety and depression shares common elements. Moreover, these findings support the notion that treating general anxiety and depression with a single treatment may be useful (Barlow et al., 2004; Hayes et al., 1999).

It is important to acknowledge some limitations to this study. First, although we found that change in symptoms and reported affect occurred during treatment, because these patients were treated in an uncontrolled naturalistic setting and received cognitive behavior therapy and/or pharmacotherapy as their clinical needs dictated, we cannot make any claims about what caused those changes. Second, because treatment was idiographic, the order and choice of interventions was different for each case, and this fact may have affected the results of our analyses. Third, diagnoses were assigned clinically rather than with research-quality interviews. Nevertheless, most of these weaknesses are inextricably tied to one of the strengths of this study,

namely its investigation of an unselected sample of highly comorbid patients treated naturalistically in a real world clinical setting.

In summary, our findings indicate that most of the predictions of the tripartite and integrated models (Clark & Watson, 1991; Mineka et al., 1998) can be extended to account for change during treatment of anxiety and depression in a comorbid sample treated in the community. NA was associated with anxiety and depression during treatment, PA was more strongly related to depression, and AA was more strongly related to anxiety. Symptoms and NA changed over the course of treatment, and much of the change in anxiety and depression appears to be shared in common. AA also changed over the course of treatment, particularly for patients with panic disorder, a finding that is consistent with an extended model, but tempered by our small number of individuals with panic disorder. PA increased during treatment, but our post-hoc analysis found this only for patients who showed a significant decline in depression and only over an extended period of treatment.

Acknowledgements

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Footnotes

[1]Because because the items assessing the high pole of PA are more strongly linked to the underlying PA factor than anhedonia items (Watson, Weber et al., 1995), we chose to focus solely on PA in this study.

[2]One assumption of this modeling approach is that change over time can be expressed as a continuous curve. Some traditional alternatives do not make this assumption. Instead, they use only first and last scores, so that no trajectory of change is necessary. For instance, hierarchical regressions can be used, with residual change scores (final scores residualized relative to initial scores) as the index of change across the time period. This approach is limited by its restricted use of the available data, as well as by the fact that it assumes a fixed process of change across all participants (Curran, 2000). Because they have different assumptions, regression and growth curve approaches can produce different results (Schnall, Schwartz, Landsbergis, Warren, & Pickering, 1998; Stoolmiller, Duncan, Bank, & Patterson, 1993). While the assumptions of growth curve models are in general less restrictive, we also ran multiple regression analyses in order to ensure that the assumption of continuous change was not distorting our conclusions. Regression analyses used residual change scores where appropriate, with mean-centered initial scores as covariates. Because the results were similar in all cases, only the mixed model growth curves are presented here.

[3]Follow-up analyses that extended the time frame to 16 and to 20 weeks led to virtually identical conclusions in all cases.

[4]The BDI measure showed significant evidence of positive skew, which was substantially reduced with a square-root transformation. We ran all analyses on both the raw and transformed BDI scores and found no substantive differences in results. For ease of interpretation, we present here the models using the raw BDI scores.

[5]We conducted the analyses for the Burns AI without the 8 items comprising the AA scale, and the pattern of results was identical. That is, Burns AI significantly decreased over the course of treatment.

[6]This proportion was calculated as follows:

$$\frac{\sigma^2(control) - \sigma^2(control + time)}{\sigma^2(random) - \sigma^2(time)}$$

In this equation, $\sigma^2(random)$ is the variability in the outcome measure when no predictor variables are included in the model, $\sigma^2(time)$ is the variability in the measure after controlling for changes over time, $\sigma^2(control)$ is the variability in the measure when controlling for concurrent scores on the other measures (e.g., BDI scores controlling for Burns AI and NA), and $\sigma^2(control + time)$ is the variability controlling both for concurrent scores on the other measures and time. The statistic isolates the variability in the outcome measure that is related to change over time and establishes the proportion of that variability that is specific to the measure of interest.

Figure Legends

Figure 1. Change During Treatment on Measures of Symptoms and Affect. Scores have been

rescaled using a z-transformation.

Figure 2. Changes During Treatment on Measures of AA.

Table 1

Within subjects correlations between the PANAS, BDI, Burns AI

	PA	NA	BDI	Burns AI	AA
PA	—				
NA	-.21*	—			
BDI	-.34*	.59*	—		
Burns AI	-.22*	.60*	.62*	—	
AA	-.14	.29*	.36*	.58*	—

Note: An asterisk indicates significance at $p <.05$ (mean correlation exceeding twice the standard error of the mean).

Table 2: Change in BDI, Burns AI, NA, AA and PA During Treatment

Measure	Starting Value	Rate of change
BDI	$17.84 \pm 1.44, p < .0001$	$-.3.06 \pm .52, p < .0001$
Burns AI	$26.78 \pm 2.34, p < .0001$	$-4.62 \pm .92, p < .0001$
NA	$25.89 \pm 1.34, p < .0001$	$-2.58 \pm .69, p < .001$
AA	$3.42 \pm .61, p < .0001$	$-.54 \pm .26, p < .05$
PA	$22.69 \pm 1.01, p < .0001$	$.56 \pm .58$, n.s.

Note. Parameter estimates presented with standard errors of estimation. P-values assess the probability the parameter is equal to 0. Change in log-transformed weeks.

Table 3: Specific Change in BDI, Burns AI, and NA

Measure	Specific change
BDI	$-1.44 \pm .35, p < .0001$
Burns AI	$-2.06 \pm .75, p < .01$
NA	$.06 \pm .51$, n.s.

Note. Parameters represent rates of change across time when scores on the other two measures have been controlled. Parameter estimates presented with standard errors of estimation. Change across log-transformed time (in weeks). Intercepts are not presented because the inclusion of control variables makes intercepts difficult to interpret.
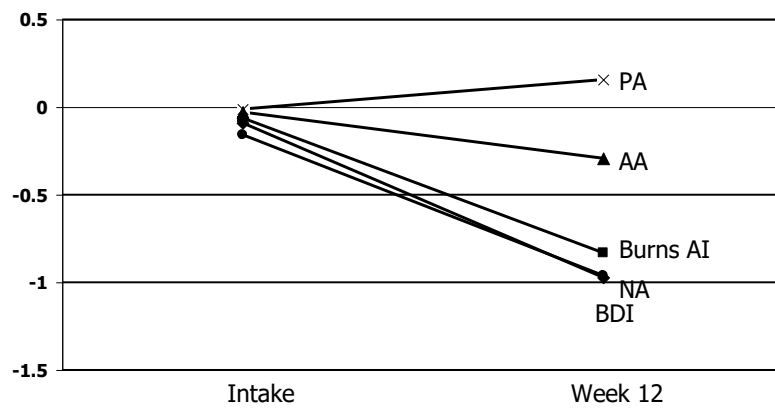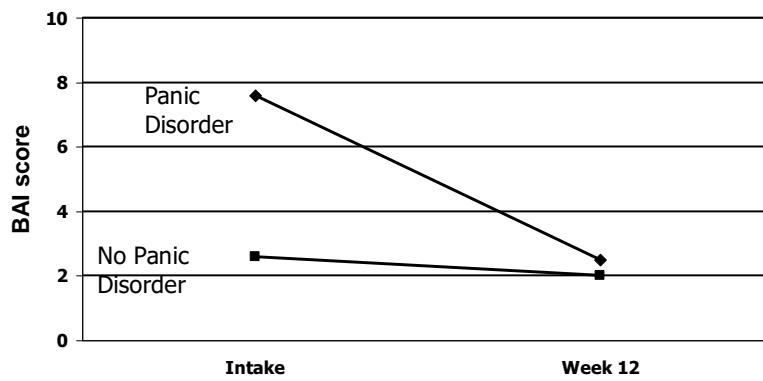
Figure 1

Figure 2