

Examining Positive and Negative Affect as Outcomes and Moderators of Cognitive-Behavioral Therapy and Acceptance and Commitment Therapy for Social Anxiety Disorder

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Traditional cognitive-behavioral therapy (CBT) for anxiety disorders has been designed to target reductions in negative affect (NA) associated with defense-related processes. However, a subset of anxiety disorders, including social anxiety disorder (SAD), are also characterized by low positive affect (PA) resulting from separate deficits in appetitive-related processes. In contrast to CBT, “third-wave” approaches, such as acceptance and commitment therapy (ACT), align more consistently with motivational processes and, as a result, PA. However, the differential effect of CBT and ACT on PA and NA has yet to be investigated. Using secondary data from a randomized controlled trial, the present study sought to compare CBT’s ($n = 45$) and ACT’s ($n = 35$) effect on PA and NA in SAD. Findings were compared to a wait-list (WL) control

condition ($n = 31$), as well as normative data from a general adult sample. Baseline PA and NA were also examined as moderators and predictors of theory-relevant treatment outcomes. NA decreased significantly in both CBT and ACT from pre to posttreatment. Although ACT outperformed WL in reducing NA, this effect was not observed for CBT. PA increased significantly in both CBT and ACT from pre to posttreatment, with neither ACT nor CBT outperforming WL in increasing PA. Neither PA nor NA were found to moderate theoretically relevant treatment outcomes. Findings suggest that ACT and CBT share common treatment mechanisms, making them more similar than distinct. Further efforts should be focused on optimizing CBT’s and ACT’s influence on threat and reward learning, and elucidating common processes of change.

Keywords: social anxiety; cognitive-behavioral therapy; acceptance and commitment therapy; positive affect; negative affect

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avoidance of perceived threats and aversive outcomes (Gray, 1994). The defensive system is posited to generate certain forms of negative affect (NA) that are a central feature of anxiety disorders, such as fear and disgust (Davidson, 1994). In alignment with this defensive system, “first-” and “second-wave” cognitive-behavioral approaches for anxiety disorders almost exclusively focused on achieving reductions in NA—including fear and anxiety—and associated functional impairment (Barlow, Ellard, Sauer-Zavala, Bullis, & Carl, 2014; Carl, Soskin, Kerns, & Barlow, 2013; Craske, Meuret, Ritz, Treanor, & Dour, 2016). For example, exposure therapy works to lessen NA through a mechanism principal to the defensive system: fear extinction (Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014). Indeed, these strategies have consistently demonstrated effectiveness in reducing NA and related symptomatology (e.g., Craske et al., *in press*).

These traditional cognitive-behavioral approaches (referred to as “CBT”), however, have largely disregarded positive affect (PA)—one’s pleasurable engagement with the environment that results in positive emotional states (e.g., joy, interest, and enthusiasm)—an affective dimension that is generated by a separate motivational system related to appetitive and goal-directed behaviors (Davidson, 1994, 1998). Deficits in PA are predictive and characteristic of certain anxiety disorders (Khazanov & Ruscio, 2016; Kotov, Gamez, Schmidt, & Watson, 2010), as well as depression (e.g., Mineka, Watson, & Clark, 1998). CBTs have not been found to significantly increase PA for individuals with depression ($g = .37$, 95% CI [-0.004, 0.77], $p < .07$; Boumparis, Karyotaki, Kleiboer, Hofmann, & Cuijpers, 2016). Conceivably, CBT may also be insufficient for PA in anxious individuals. Thus far, the effect of CBT on PA for anxiety disorders has been examined in only one study through person-specific analyses on seven individuals with generalized anxiety disorder (Bosley, Fisher, & Taylor, 2018). PA was found to improve in only two of seven participants, and worsened in five of seven—even as NA improved over the course of treatment. Further investigation of CBT’s effect on PA for anxiety disorders with larger data sets is necessary.

Low PA appears to be particularly characteristic of social anxiety disorder (SAD; Watson & Naragon-Gainey, 2010). A meta-analysis examining cross-sectional and experience sampling studies found a moderate, inverse relationship between social anxiety and PA, $r = -.36$, 95% CI [-.31, -.40], that remained consistent after covarying for depressive symptoms ($r = -.21$; 95% CI [-.16, -.26]; Kashdan,

2007). These findings are paralleled with results harnessing longitudinal datasets, experimental designs, and structural equation modeling methods (Brown, Chorpita, & Barlow, 1998; Kashdan, Weeks, & Savostyanova, 2011; Khazanov & Ruscio, 2016; Sewart et al., 2019). Lack of explicit attention to the positive dimension of affect may in part explain less than optimal treatment response rates for CBT for SAD (see Loerinc et al., 2015).

The recent “third wave” in CBT has shifted away from directly decreasing NA and instead focused on acceptance and relinquishing control over maladaptive attempts to regulate internal mood states. The third-wave movement has ushered in modalities that emphasize previously unaddressed concepts, such as mindfulness of the present moment, acceptance of cognitions and feelings, and behaviors consistent with personal values (Hayes & Hofmann, 2017). One of the most widespread of these approaches is acceptance and commitment therapy (ACT), which focuses on making contact with the present moment and changing or persisting in behavior to reach value-consistent goals, or “psychological flexibility” (Hayes, Luoma, Bond, Masuda, & Lillis, 2006). When compared with CBT, ACT treatment philosophy aligns more consistently with appetitive and goal-directed behaviors. For example, the core ACT process of “committed action” requires engaging in activities that align with personal values, which may serve to recalibrate reward learning and increase positive affective states, such as pride, gratitude, and interest. Another core process of ACT, mindfulness, is defined by (a) “the self-regulation of attention so that it is maintained on immediate experience, thereby allowing for increased recognition of mental events in the present moment” and (b) “a particular orientation toward one’s experiences in the present moment, an orientation that is characterized by curiosity, openness, and acceptance” (Bishop et al., 2004; Fletcher & Hayes, 2005). While the goal of ACT is not to manipulate affective states, heightened awareness of the present moment may also serve to increase pleasurable engagement with the environment and PA.

Given that CBT and ACT appear to target the defensive and appetitive systems, respectively, it is likely these therapies also possess differential influence on PA and NA for SAD. Understanding how currently disseminated treatments impact affect and how baseline affect may predict treatment outcomes has high clinical utility, as such knowledge may be used to guide treatment planning. Furthermore, examining the effect of CBT and ACT on both PA and NA contributes to

dimensional understanding of mental disorders (Cuthbert, 2015).

We first hypothesized that CBT would more effectively decrease NA and symptoms when compared with ACT. Further, CBT is expected to be most effective for individuals with high NA, given that the defensive system is the primary target of CBT. Specifically, we hypothesized that reductions in NA would be greatest for individuals with high NA receiving CBT compared to ACT. With ACT strategies appearing to more directly target an appetitive reward system, we hypothesized that ACT would more effectively raise PA when compared with CBT and that increases in PA would be greatest for individuals with low PA compared to CBT.

To investigate these hypotheses, we utilized an existing data set to examine the main effects of CBT and ACT on PA and NA. We compared these main effects to a wait-list (WL) control condition to better contextualize pre- to posttreatment gains. Levels of PA and NA for each treatment condition were then compared to a normative sample. Next, we examined whether pretreatment levels of PA and NA acted as moderators of treatment-relevant outcomes for CBT and ACT, symptom reduction, and quality of life, and resulted in differential treatment response.

Method

PARTICIPANTS

Participants who met criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; American Psychiatric Association, 2000) for either principal or co-principal social phobia, generalized type, were randomly assigned and stratified by age and gender to ACT, CBT, or WL. Using a modified intent-to-treat approach, all participants who began psychotherapeutic treatment and had available data on variables of interest for at least one time point ($n = 111$) were included in the present analyses ($n = 35$ ACT, $n = 45$ CBT, $n = 31$ WL).

In addition to a diagnosis of generalized social phobia, individuals were eligible for participation if they were (a) between 18 and 60 years of age, (b) either medication-free or stabilized on psychotropic medication (i.e., 1 month for benzodiazepines and beta blockers, and 3 months for selective serotonin reuptake inhibitors [SSRIs]/serotonin-norepinephrine reuptake inhibitors [SNRIs] and heterocyclics), (c) psychotherapy-free or stabilized on alternative psychotherapies (not cognitive or behavioral therapies) that were *not* focused on their anxiety disorder for at least 6 months, and (d) English speaking. Exclusion criteria consisted of the following: (a) active suicidal ideation; (b) severe

depression (Clinical Severity Rating [CSR] ≥ 6 on a 0- to 8-point scale; Anxiety Disorders Interview Schedule for DSM-IV [ADIS-IV]; Brown, Barlow, & Di Nardo, 1994); (c) history of a psychotic disorder, bipolar disorder, mental retardation, or organic brain damage; (d) substance abuse or dependence within the previous 6 months using DSM-IV-TR criteria (American Psychiatric Association, 2000); and (e) serious medical conditions or pregnancy. Given there was an additional neuroimaging component to the study, neuroimaging-relevant exclusion criteria were (a) left-handedness, (b) metal implants, and (c) claustrophobia (see Craske, Niles, et al., 2014, for further details on inclusion/exclusion criteria). Participants were recruited via the Internet, local flyers and newspaper advertisements, and community referrals. Study procedures took place at the Anxiety and Depression Research Center located at the University of California, Los Angeles.

The sample was 50% female, with a mean age of 28.3 years ($SD = 6.7$). The sample was primarily White (46.8%), with the remainder of the sample identifying as Asian/Pacific Islander (20.7%), Hispanic/Latinx (18.0%), Black/African American (1.8%), and 12.7% as "other" race/ethnicity. At baseline, 20.0% of those receiving CBT ($n = 9$) and 22.9% of those receiving ACT ($n = 8$) reported being stabilized on psychotropic medication.

DESIGN

Random assignment, stratified by age and gender, was used to assign eligible participants to CBT, ACT, and WL. Participants were assessed (a) prior to treatment initiation (pretreatment [pre]), (b) immediately after the conclusion of treatment (posttreatment [post]), (c) 6 months after pretreatment assessment (6-month follow-up [6MFU]), and (d) 12 months after pretreatment assessment (12-month follow-up [12MFU]). Assessments comprised self-report questionnaires, laboratory paradigms to assess various moderators and mechanisms of treatment change (see Craske, Niles, et al., 2014), and a semistructured diagnostic interview.

TREATMENTS

Participants received 12 weekly, 1-hour individual CBT or ACT therapy sessions by advanced clinical psychology doctoral student therapists. Further information regarding therapist competence, integrity, and training can be found in Craske, Niles, et al. (2014). Treatment protocols used in each condition were based on orientation-consistent manuals (e.g., Eifert & Forsyth, 2005). Given that both ACT and CBT focus to some extent on avoidance reduction, treatments were matched on

the number of sessions dedicated to exposure but provided a different, orientation-consistent rationale. Following the conclusion of treatment, therapists provided follow-up booster phone calls to participants once per 6 months in an effort to reinforce progress consistent with therapy condition. Most participants completed therapy within 12 to 16 weeks (range: 11–18).

Cognitive-Behavioral Therapy

CBT treatment modules for generalized SAD were derived from standard CBT protocols (Hope, Heimberg, & Turk, 2010). Session 1 was focused on assessment, self-monitoring, and psychoeducation. Sessions 2–4 emphasized cognitive restructuring—specifically, errors of overestimation and catastrophizing regarding negative evaluation—these techniques were combined with strategies of hypothesis testing, self-monitoring, and breathing retraining. Rationale for exposure to socially relevant feared cues was presented in Session 5. Exposure—including in vivo, in vivo augmented with interoceptive exposure, and imaginal—was conducted in Sessions 6–11. Session 12 was focused on relapse prevention.

Acceptance and Commitment Therapy

ACT treatment modules for generalized social phobia were developed largely from Eifert and Forsyth (2005). Session 1 was focused on psychoeducation and orientation to ACT, which included experiential exercises (e.g., “finger trap”) and a discussion of acceptance and committed action. Sessions 2–3 centered on creative hopelessness and how attempts to control anxiety resulted in a reduction of valued life activities; accepting anxiety, rather than efforts to reduce, was encouraged. Sessions 4–5 directed participants in practices of mindfulness and cognitive defusion, while continuing to emphasize acceptance of anxiety. Sessions 6–11 largely focused on values identification and acceptance, with an additional component focusing on continued practice of acceptance, mindfulness, and defusion. When relevant, exposure—including in vivo, in vivo augmented with interoceptive exposure, and imaginal—was conducted in an effort to have participants practice mindfully observing and accepting anxiety while conducting values-consistent action. Session 12 was focused on what practices were helpful and how to continue moving forward after therapy has concluded.

OUTCOME AND MODERATOR/PREDICTOR VARIABLES

Given that CBT for SAD aims to reduce anxiety-related symptoms, social anxiety symptoms were used as the theory-relevant outcome measure for CBT when examining PA and NA as moderators/

predictors. In contrast to CBT, ACT aims to increase the capacity to experience the present moment (i.e., “psychological flexibility”) and foster a “life worth living.” Thus, quality of life (QOL) was used as the theory-relevant outcome measure for ACT when examining PA and NA as moderators/predictors, as QOL captures the importance of, as well as satisfaction with, different life domains.

PA and NA

In an effort to create a more valid and reliable index of self-reported PA and NA, scores from two well-validated self-report measures were combined following Z score transformation. PA was measured by the (a) positive affect subscale (PANAS-P; 10-item) of the Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988), a measure of dispositional affect in which participants indicate the extent to which they felt like a given affect-related adjective (e.g., “interested,” “distressed”) over the past week using a scale from 1 (*very slightly/not at all*) to 5 (*extremely*); and (b) high positive emotion items from the anhedonic depression subscale (MASQ-AD; 14-item; Kendall et al., 2016) of the Mood and Anxiety Symptom Questionnaire (MASQ; Watson & Clark, 1991) in which participants are asked to rate the strength of emotional experiences over the past week from 1 (*not at all*) to 5 (*extremely*). NA was measured by the (a) negative affect subscale (PANAS-N; 10-item) of the PANAS and (b) the general distress mixed subscale (MASQ-GDM; 15-item) of the MASQ. The PANAS and MASQ have been used frequently in socially anxious samples (see Kashdan, 2007, for a review) and across disorders to assess affective abnormalities (e.g., Brown et al., 1998; Sewart et al., 2019; Watson & Naragon-Gainey, 2010). These measures have also demonstrated both validity and good-to-excellent reliability in clinical and nonclinical populations (Crawford & Henry, 2004; Jolly, Dyck, Kramer, & Wherry, 1994; Keogh & Reidy, 2000; Mehrabian, 1997; Roesch, 1998; Watson, Clark, & Tellegen, 1988).

Social Anxiety Symptoms

Three well-validated self-report measures of social anxiety symptoms were also combined following Z score transformation to create a more reliable measurement of disorder-related symptomatology. These self-report measures consisted of the following: (a) the Liebowitz Social Anxiety Scale—Self-Report (LSAS-SR; Fresco et al., 2001), a 24-item measure designed to assess fear and avoidance in situations involving performance and social interaction of which each item rated on a 0 (*no fear/never avoid*) to 3 (*severe fear/usually avoid*) Likert scale; (b) the Social Interaction Anxiety Scale (SIAS;

Table 1
Baseline Sample Characteristics and Bivariate Correlations

	CBT M(SD)	ACT M(SD)	WL M(SD)	F value	p	Correlations			
						1	2	3	4
1. Negative affect									
PANAS	15.58 (7.60)	12.91 (8.18)	13.08 (6.91)	1.48	.23				
MASQ	41.03 (9.89)	39.59 (12.69)	37.11 (11.20)	1.07	.35				
Composite	0.18 (0.88)	-0.05 (1.01)	-0.17 (0.84)	1.39	.25				
2. Positive affect									
PANAS	16.43 (6.93)	15.85 (8.07)	15.10 (8.08)	.26	.77	-.117			
MASQ	34.39 (10.38)	35.21 (11.36)	32.15 (10.18)	.70	.50				
Composite	0.06 (0.82)	0.06 (0.99)	-0.13 (0.93)	.54	.59				
3. Social anxiety symptom composite	0.01 (0.70)	0.10 (0.86)	-0.14 (0.92)	.64	.53	.517**	-.217*		
4. Quality of life	0.15 (1.80)	0.08 (2.43)	-0.09 (1.48)	.13	.88	-.392**	.635**	-.435**	

Note. CBT = cognitive-behavioral therapy; ACT = acceptance and commitment therapy; WL = wait-list; PANAS = Positive and Negative Affect Schedule; MASQ = Mood Anxiety Symptom Questionnaire; Composite = composite of PANAS and MASQ; Social anxiety symptom composite = composite of Liebowitz Social Anxiety Scale—Self-Report, Social Interaction Anxiety Scale, and Social Phobia Scale; Quality of life = Quality of Life Inventory.

** $p > .01$, * $p > .05$.

Mattick & Clarke, 1998), a 20-item measure designed to assess cognitive, affective, or behavioral responses across various socially relevant situations of which each item is rated on a 0 (*not at all characteristic or true of me*) to 4 (*extremely characteristic or true of me*) Likert scale; (c) the Social Phobia Scale (SPS; Mattick & Clarke, 1998), a 20-item measure designed to assess anxiety related to observation by others of which each item is rated on a 0 (*not at all characteristic or true of me*) to 4 (*extremely characteristic or true of me*) Likert scale.

Quality of Life

The Quality of Life Inventory (QOLI; Frisch, 1994) was used to assess QOL, an outcome that corresponds with the overall goals of psychological flexibility and valued living within ACT. The QOLI assesses well-being and satisfaction with life across 16 domains (e.g., goals and values, friendships, health). This measure has demonstrated good internal validity and test-retest reliability (Frisch et al., 2005).

STATISTICAL ANALYSES

Analyses were performed in Stata 14.0 using the `xtmixed` command. We used multilevel modeling (MLM), a superior statistical approach for data with nested sources of variability, as it allows for examination of within- and between-subject change across time (pre, post, 6MFU, 12MFU) and by group (ACT, CBT, and WL). MLM accommodates unequal observations across individuals, allowing for the inclusion of participants missing one or more assessments.

Main Effects

For the main effect of treatment on PA and NA, time was modeled at Level 1 (pre, post, 6MFU, 12MFU) using a piecewise approach (e.g., Roy-Byrne et al., 2005) that examines two separate linear segments of change: Pre and post (S1) and post, 6MFU, and 12MFU (S2). This approach captures typical trends in treatment studies, wherein the greatest effect of treatment occurs by the end of treatment and is then subsequently maintained, or lessened, during follow-up assessments. Treatment group (CBT, ACT, and WL) was included at Level 2. All models included random effects of the intercept. Between-group differences were assessed via marginal means at each time point, and by comparing S1 in ACT, CBT, and WL, and S2 in ACT and CBT. The MLM equation for a model with random effects for the intercept can be referenced in Craske, Niles, et al. (2014).

Moderated Effects

To examine the moderating effects of baseline PA and NA on theory-relevant outcomes for ACT and CBT, time was modeled at Level 1 and condition and moderators at Level 2. Using a similar statistical approach as the main effects, we examined pretreatment to posttreatment and posttreatment to follow-up as two separate linear segments as described above. Separate moderator models were run for PA and NA. Omnibus three-way interaction effects were tested first (i.e., Time \times Condition \times Moderator). If moderation was not observed, we followed up with further testing to determine whether PA and NA functioned as a nonspecific predictor of treatment response to both CBT and ACT.

Table 2

Estimated Means and Confidence Intervals at Each Time Point for Cognitive-Behavioral Therapy (CBT), Acceptance and Commitment Therapy (ACT), and Wait-List (WL)

	CBT (95% CI)	ACT (95% CI)	WL (95% CI)
		Negative affect	
Baseline	0.17 [-0.08, 0.41]	-0.06 [-0.34, 0.22]	-0.15 [-0.45, 0.14]
Post	-0.34 [-0.61, -0.07]	-0.72 [-1.02, -0.42]	-0.34 [-0.64, 0.03]
6MFU	-0.44 [-0.69, -0.19]	-0.65 [-0.92, -0.39]	–
12MFU	-0.55 [-0.85, -0.24]	-0.59 [-0.90, -0.28]	–
		Positive affect	
Baseline	0.1 [-0.2, 0.3]	0.1 [-0.2, 0.4]	-0.1 [-0.5, 0.2]
Post	0.4 [0.1, 0.7]	0.3 [0.0, 0.7]	0.0 [-0.4, 0.3]
6MFU	0.4 [0.2, 0.7]	0.4 [0.1, 0.7]	–
12MFU	0.5 [0.1, 0.8]	0.4 [0.0, 0.7]	–

Note. CBT = cognitive-behavioral therapy; ACT = acceptance and commitment therapy; WL = wait-list; 6MFU = 6-month follow-up; 12MFU = 12-month follow-up.

Results

Means and standard deviations of PA and NA, along with theory-relevant outcomes examined in moderator analyses are presented in Table 1. No significant pretreatment group differences were found between CBT, ACT, and WL for variables of interest ($ps > .23$).

PA AND NA AS OUTCOME VARIABLES

Changes in PA and NA were examined by determining whether each slope differed significantly from zero from pre- to posttreatment (S1) for CBT, ACT, and WL and from 6MFU to 12MFU (S2) for CBT and ACT. Degree of slope change in these segments was then compared between groups. For Group \times Time interactions, we present beta values for pairwise slope comparisons (i.e., simple effects) and associated p values. Estimated means and 95% CI from the piecewise model for each group are displayed in

Table 2. Estimated slopes and effect sizes for pairwise group slope comparisons are further detailed in Table 3. Results are visually displayed in Figure 1.

Negative Affect

From pre- to posttreatment (S1), NA decreased significantly for both CBT ($b = -0.5$, 95% CI [-0.8, -0.3], $p < .001$) and ACT ($b = -0.7$, 95% CI [-0.9, -0.3], $p < .001$), but not WL ($b = -0.2$, 95% CI [-0.5, -0.1], $p = .18$). For S1 pairwise slope comparisons, ACT demonstrated larger reductions in NA than WL ($b = -0.5$, 95% CI [-0.9, -0.1], $p = .01$). CBT, however, did not outperform WL in reducing NA, although the observed effect approached significance ($b = 0.3$, 95% CI [-0.04, 0.7], $p = .08$). Inconsistent with our hypothesis, CBT and ACT S1 slopes did not significantly differ from each other ($b = -0.2$, 95% CI [-0.5, 0.2], $p = .39$). S2 change slopes were not significant in CBT ($p = .19$) or ACT ($p = .37$).

Table 3

Estimated Slopes (Change) From Pre- to Posttreatment (S1) and Post to 12MFU (S2), and Effect Sizes (Cohen's d) for Pairwise Group Slope Comparisons for CBT Versus ACT

	CBT beta (95% CI)	ACT beta (95% CI)	WL beta (95% CI)	CBT vs. ACT d	ACT vs. WL d	CBT vs. WL d
		Negative affect				
S1	-0.5 [-0.8, -0.3]***	-0.7 [-0.9, -0.3]***	-0.2 [-0.5, 0.1]	0.2	0.5	0.3
S2	-0.1 [-0.3, 0.1]	-0.1 [-0.1, 0.2]	–	0.0	–	–
		Positive affect				
S1	0.3 [0.1, 0.6]**	0.3 [0.01, 0.5]†	0.1 [-0.1, 0.4]	0.0	0.2	0.2
S2	0.04 [-0.1, 0.2]	0.01 [-0.1, 0.2]	–	0.03	–	–

Note. CBT = cognitive-behavioral therapy; ACT = acceptance and commitment therapy; WL = wait-list.

*** $p > .001$, ** $p > .01$, * $p > .05$, † $p = .053$.

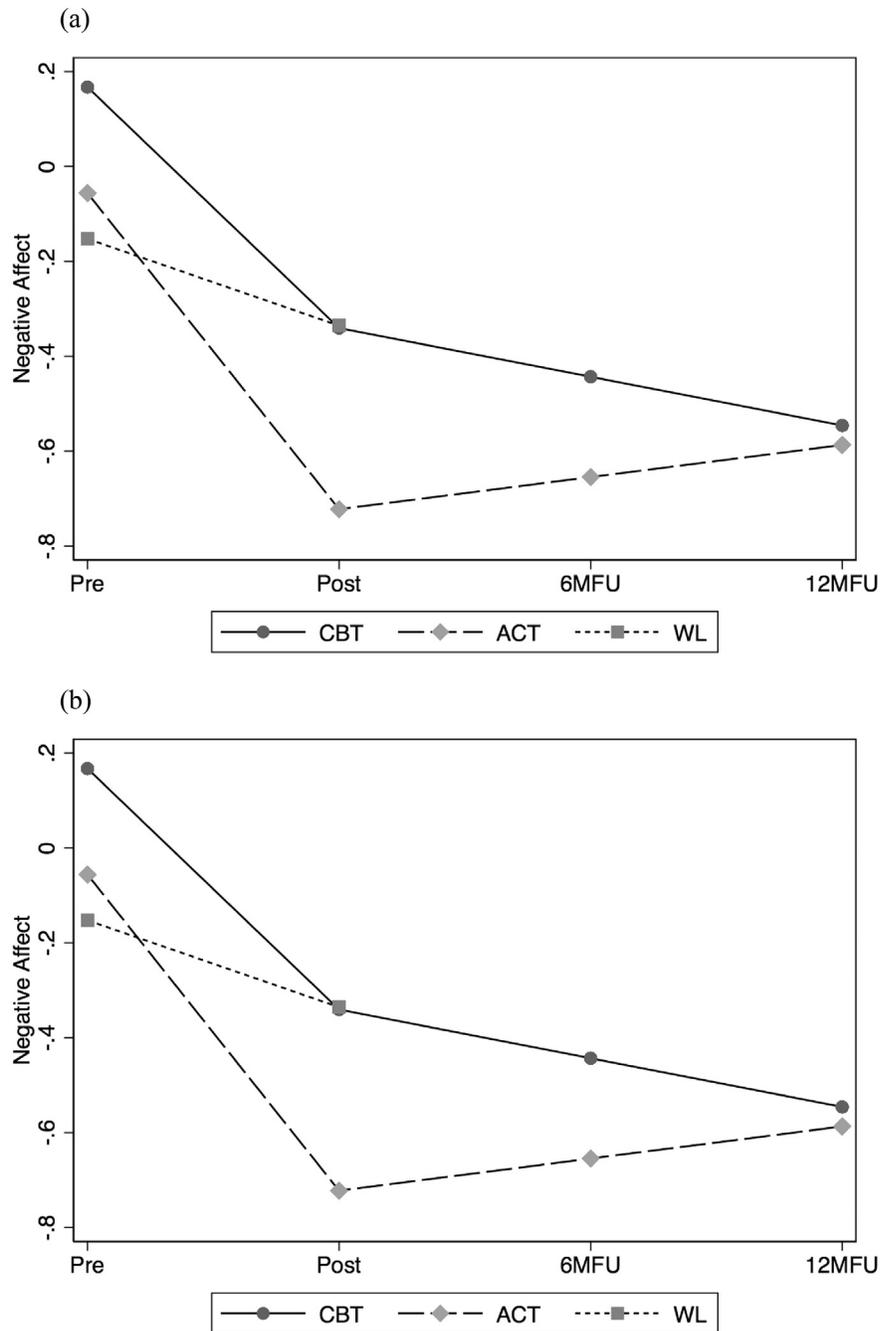


FIG. 1 Main effects of cognitive-behavioral therapy (CBT), acceptance and commitment therapy (ACT), and wait-list (WL) on (a) negative affect and (b) positive affect. Note. 6MFU = 6-month follow-up; 12MFU = 12-month follow-up.

Positive Affect

From pre- to posttreatment (S1), PA increased significantly for CBT ($b = 0.3$, 95% CI [0.1, 0.6], $p = .009$) and ACT ($b = 0.3$, 95% CI [0.01, 0.5], $p = .05$), but not WL ($b = 0.1$, 95% CI [-0.1, 0.4], $p = .37$). For S1 pairwise slope comparisons, neither ACT nor CBT demonstrated larger increases in PA than WL (ACT $b = 0.1$, 95% CI [-0.2, 0.5], $p = .48$; CBT $b = -0.2$, 95% CI [-0.6, 0.2], $p = .26$).

Inconsistent with our hypothesis, ACT and CBT S1 slopes did not significantly differ from each other ($b = -0.01$, 95% CI [-0.4, 0.3], $p = .70$). S2 change slopes were not significant in CBT ($p = .62$) or ACT ($p = .88$).

Nonclinical Sample Comparison

To further evaluate the degree to which PA and NA compared to normative levels across treatment, we referenced PANAS normative data from a general

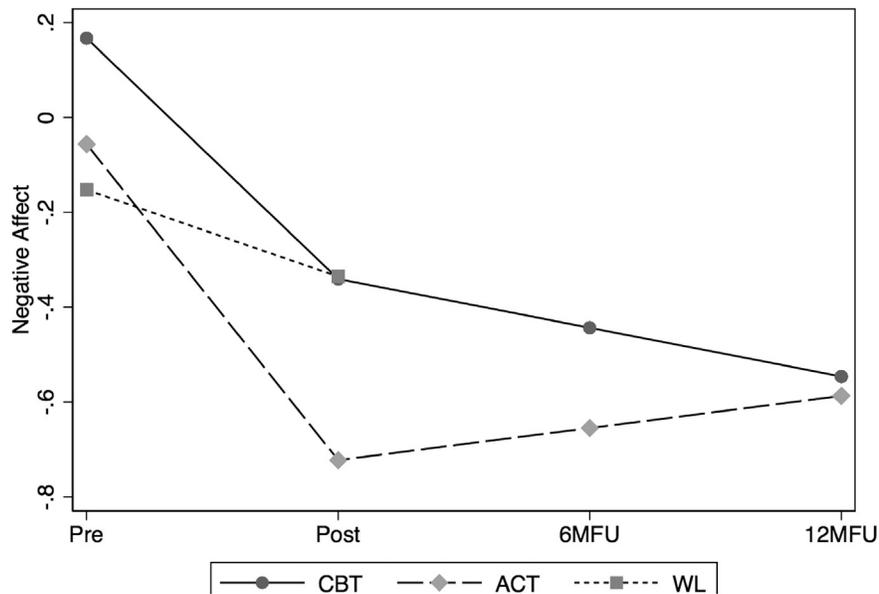


FIG. 2 Nonspecific treatment prediction of quality of life by positive affect. Note. 6MFU = 6-month follow-up; 12MFU = 12-month follow-up; CBT = cognitive-behavioral therapy; ACT = acceptance and commitment therapy; WL = wait-list.

adult sample recruited from various commercial and public service organizations (NA $M = 16.00$, $SD = 5.90$, PA $M = 31.31$, $SD = 7.65$; Crawford & Henry, 2004). We compared these nonclinical sample values to our sample's PANAS scores for CBT and ACT treatment completers.

At pretreatment, levels of NA were notably above normative values as indicated by average values in the 90th and 84th percentile for CBT and ACT, respectively. At posttreatment, NA had reduced to the 78th percentile for CBT and 63rd percentile for ACT. Twelve months following treatment, NA of those who had received CBT were averaged at the 74th percentile, while those receiving ACT were at the 69th percentile. Thus, NA scores approached the population average at posttreatment and reductions were generally maintained through 12MFU for both CBT and ACT.

PA was below normative values at pretreatment as indicated by average values in the 24th and 28th percentile for CBT and ACT, respectively. At posttreatment, PA had increased to the 41st percentile for both CBT and ACT. Twelve months following treatment, PA of those who had those received CBT were averaged at the 41st percentile, while those receiving ACT were at the 36th percentile. Thus, PA scores approached the population average at posttreatment and improvements were generally maintained through 12MFU for both CBT and ACT.

PA AND NA AS MODERATOR VARIABLES

PA and NA were evaluated as moderators of self-reported composite symptoms of social anxiety and QOL for CBT and ACT. Omnibus three-way interaction effects were tested first (Time \times Condition \times Moderator) for S1 and S2, and if not significant, we then tested PA and NA as nonspecific treatment predictors.

Social Anxiety Symptoms

NA was not found to differentially moderate social anxiety symptoms for CBT or ACT for S1 ($b = 0.4$, 95% CI $[-0.1, 0.8]$, $p = .09$) or S2 ($b = 0.1$, 95% CI $[-0.2, 0.2]$, $p = .85$). Nor did PA moderate social anxiety symptoms for CBT or ACT for S1 ($b = 0.04$, 95% CI $[-0.4, 0.5]$, $p = .88$) or S2 ($b = 0.2$, 95% CI $[-0.1, 0.4]$, $p = .30$). Further testing revealed that regardless of treatment condition neither NA nor PA significantly predicted outcome for S1 (NA $b = 0.1$, 95% CI $[-0.1, 0.3]$, $p = .50$; PA $b = -0.1$, 95% CI $[-0.4, 0.1]$, $p = .28$) or S2 (NA $b = 0.01$, 95% CI $[-0.1, 0.1]$, $p = .88$; PA $b = -0.01$, 95% CI $[-0.1, 0.4]$, $p = .27$).

Quality of Life

NA was not found to differentially moderate QOL for CBT or ACT for S1 ($b = 0.1$, 95% CI $[-0.7, 0.8]$, $p = .81$) or S2 ($b = -0.01$, 95% CI $[-0.5, 0.4]$, $p = .96$). Nor did PA moderate QOL for CBT or ACT for S1 ($b = -0.2$, 95% CI $[-1.0, 0.5]$, $p = .54$) or S2 ($b = 0.2$, 95% CI $[-0.2, 0.7]$, $p = .28$). Regardless of

treatment condition, NA did not significantly predict outcome for S1 ($b = -0.01$, 95% CI $[-0.4, 0.4]$, $p = .97$) or S2 ($b = -0.1$, 95% CI $[-0.3, 0.1]$, $p = .48$). In contrast, as shown in [Figure 2](#), PA predicted outcome for S1 ($b = -0.6$, 95% CI $[-1.0, -0.2]$, $p = .002$), but not for S2 ($b = 0.1$, 95% CI $[-0.2, 0.2]$, $p = .99$). Tests of simple effects revealed that among participants with low (1 *SD* below the mean) and average (mean) PA, the slope change in QOL was significant (low PA slope = 1.1, 95% CI $[0.6, 1.6]$, $p < .001$; average PA slope = 0.5, 95% CI $[0.3, 0.9]$, $p < .001$). Slope change was not found to be significant for individuals with high PA (1 *SD* above the mean; slope = 0.05, 95% CI $[-0.4, 0.5]$, $p = .82$). However, high PA individuals already demonstrated high QOL at pretreatment.

Discussion

Anxiety disorders have long been characterized by abnormal activation of a theorized defensive system that gives rise to elevated levels of NA (e.g., fear; [Gray, 1994](#)). As such, traditional CBT for anxiety disorders has been designed to target reductions in NA associated with defense-related mechanisms. However, a subset of anxiety disorders, including SAD, are also characterized by low PA resulting from separate deficits in appetitive learning processes (e.g., [Brown et al., 1998](#)). In contrast to CBT, “third-wave” approaches, such as ACT, have focused on new treatment targets that—although not a direct goal of these treatments—align more consistently with and facilitate appetitive and goal-directed behaviors and, as a result, PA. However, whether or not these treatment approaches actually exercise differential influence on appetitive and defensive systems has yet to be investigated. Thus, using secondary data from a previously conducted randomized controlled trial, the present study sought to compare CBT’s and ACT’s effect on PA and NA in SAD. The findings were compared to a WL control condition, as well as normative data from a general adult sample. In addition, we sought to investigate baseline PA and NA as moderators and predictors of theory-relevant treatment outcomes.

The present study has several main findings, some of which contrast with our initial hypotheses. First, NA decreased significantly in both CBT and ACT from pre- to posttreatment with these reductions maintained through follow-up assessments. However, contrary to our hypothesis, CBT was not found to outperform ACT in decreasing NA pre- to posttreatment. Moreover, whereas ACT outperformed WL in reducing NA, the difference in slopes between CBT and WL only approached significance ($p = .08$). Comparing posttreatment

NA to population average data, as reported in one reference study comprising an adult sample collected through various commercial and public service organizations, levels of NA approached normative values for CBT and ACT. Second, PA increased significantly in both ACT and CBT from pre- to posttreatment with these elevations maintained through follow-up assessments and approaching normative values for CBT and ACT. Again, contrary to our hypothesis, ACT was not found to significantly outperform CBT in increasing PA pre- to posttreatment. Neither ACT nor CBT were found to outperform WL in elevating PA pre- to posttreatment. Third, pretreatment levels of neither PA nor NA were found to moderate theoretically relevant treatment outcomes for either CBT or ACT.

In contrast with our hypothesized differential moderation, pretreatment levels of PA functioned as a nonspecific predictor of QOL in both ACT and CBT. While individuals with low to average levels of pretreatment PA experienced significant improvements in QOL, individuals with high pretreatment PA did not. As displayed in [Figure 2](#), this appears to be a ceiling effect due to individuals with high pretreatment PA reporting high levels of QOL prior to initiation of ACT and CBT, thus giving less potential for improvement. These results may be further explained by the “broaden-and-build” theory of positive emotion, wherein positive emotions enhance QOL through encouraging enduring personal resources and relationships and promoting exploratory and novel thoughts and actions—thus broadening one’s behavioral range ([Fredrickson, 1998, 2004](#)). Overall, our PA predictor findings corroborate a larger body of literature that implicates PA as a significant, positive predictor of various QOL dimensions (e.g., [Brennan, Singh, Spencer, & Roberts-Thomson, 2006](#); [Headey, Kelley, & Wearing, 1993](#); [Louro, Fernández-Castro, & Blasco, 2015](#); [Stauber et al., 2013](#)) and highlights the importance of further identifying psychotherapeutic strategies that more effectively increase PA.

One of the most notable findings is that although CBT significantly decreased NA pre- to posttreatment, this effect was not found to differ from WL. There may be a number of explanations. One possibility is that the treatment strategies (e.g., cognitive restructuring, exposure, and breathing retraining) targeted specific threat responses (e.g., fears of specific stimuli) without influencing general negative emotions.

Furthermore, while ACT and CBT were not observed to exercise a differential effect on NA, ACT did outperform WL with a moderate effect

size ($d = 0.5$). Although reducing NA is not a focus of ACT, it is likely that this reduction is a by-product of ACT-based strategies. For example, carrying out committed action for an individual with SAD may include spending more time socializing with friends, which may serve to lessen negative emotions. Why ACT outperformed WL and CBT did not, is unclear. Based on these contradictory findings, further research is needed to examine changes in threat-related processes as a result of CBT and ACT for anxiety disorders.

Another striking finding is that while ACT and CBT significantly increased PA, neither treatment outperformed WL pre- to posttreatment. This converges with previous findings that cognitive-behavioral and mindfulness-based psychotherapies are largely ineffective in heightening PA for depression (Boumparis et al., 2016; Dichter et al., 2009; Moore et al., 2013). Our results should be interpreted with the acknowledgment that CBT for SAD is not designed to target appetitive and goal-directed learning in the same manner as CBT for depression. This is not to say that strategies in CBT for SAD (e.g., exposure) should not also serve to indirectly increase PA through encouraging contact with feared situations that are likely to also be rewarding (e.g., social gatherings). ACT, however, seeks to improve QOL—a construct inherently related to reward attainment—and yet it does not outperform WL in increasing PA. These findings call for clinical scientists to identify psychotherapeutic strategies that more effectively target the appetitive approach system and—as a result—increase PA for anxiety disorders that demonstrate deficits, such as SAD and generalized anxiety disorder (Prenoveau et al., 2010). As with NA, further research is needed to examine changes in reward-related processes as a result of CBT and ACT for anxiety disorders.

These results corroborate and extend our initial outcome and moderator findings (Craske, Niles, et al., 2014). Comorbid depression previously failed to predict composite symptoms, which is consistent with our PA finding given that low PA is characteristic of depression (Watson, Clark, & Carey, 1988). Craske, Niles, et al. (2014) found extraversion to be a nonspecific significant linear predictor of composite symptoms ($z = 1.96, p = .05$), wherein participants higher in extraversion reported significantly fewer symptoms collapsed across follow-up assessments (Craske, Niles, et al., 2014). There is both self-report and psychophysiological evidence to suggest that PA forms the “conceptual core” of extraversion (Hermes, Hagemann, Naumann, & Walter, 2011; Lucas & Baird, 2004; Watson & Clark, 1997). This finding

diverges from our current analyses that found that pretreatment PA was neither a moderator nor a nonspecific predictor of symptom reduction. One potential explanation for these differing findings is that extraversion is a socially relevant trait, whereas PA encompasses more pleasurable engagement broadly. The ability to derive positive emotional states from social experiences may be particularly beneficial for enhancing treatment response to both CBT and ACT for individuals with SAD. Future research should investigate the dynamics between PA and social interaction in SAD and how their relationship may affect treatment outcome.

As the field moves more toward a dimensional understanding of psychopathology, it is essential that we begin to examine our standard psychotherapeutic treatments from this perspective (Brown & Barlow, 2009; Insel et al., 2010). This is the first study to our knowledge that examines changes in PA and NA as a result of CBT and ACT for anxiety disorders. Our findings raise important questions regarding the effectiveness of these interventions with respect to defense and reward systems. Furthermore, our findings suggest that individuals with low PA may benefit from augmenting CBT and ACT with strategies specifically designed to regulate positive emotion. For example, individuals with low PA may benefit from adjunctive training in how to “savor” past, current, or future positive experiences (Bryant, 2003) or “capitalize” on success (Carl et al., 2013; Langston, 1994). Indeed, protocols with such strategies have recently been shown to significantly improve PA, although results were not compared to WL (e.g., Craske et al., in press).

Our study possessed some limitations. Given that these two therapeutic approaches focus on avoidance reduction, exposure was employed in both treatment conditions for an equal number of sessions with an orientation-consistent rationale. As a result, it is possible that exposure may be driving effects in both ACT and CBT, making the differential impact of these approaches less detectable and more difficult to assess—or less present than suspected. Optimizing QOL is also a focus of CBT that has more recently been examined as a critical outcome of treatment success (e.g., Hofmann, Wu, & Boettcher, 2014). As a result, contending that QOL is not theoretically relevant to CBT as well is debatable. Given that PA and NA were not assessed throughout treatment, we were unable to examine these factors through mediational analyses that may have provided further insight into the mechanistic nature of threat and reward-related processes in CBT and ACT. Participants were deemed ineligible if they presented with

severe depression ($CSR \geq 6$ on a 0- to 8-point scale; Brown et al., 1994). While this is not an uncommon procedure for anxiety psychotherapy studies, this likely excluded individuals with anhedonia and as a result may have indirectly affected our PA findings. To further examine treatment change, we compared levels of PA and NA to a nonclinical sample collected in the United Kingdom with a mean age ($M = 42.9$) and demographic composition that was substantially different from our sample. Thus, these normative values do not generalize to our sample in an ideal manner. Last, we relied on self-report measures as the exclusive modality of treatment response, which may have inflated ratings (Loerinc et al., 2015). However, our treatment's main effects more often than not did not outperform WL. Future studies may address this limitation by employing experimental paradigms that more directly measure change in purported reward and threat-learning mechanisms during treatment.

Overall, these findings suggest that while ACT and CBT may approach psychotherapeutic treatment with different methods, they share common treatment mechanisms making them more similar than distinct. Still further research is needed to elucidate common processes of change in CBT and ACT. Future research should assess PA and NA during CBT and ACT for a more rigorous examination of these factors as potential mechanisms. Most importantly, efforts should be focused on enhancing CBT's and ACT's influence on threat and reward learning in an effort to better recalibrate PA and NA.

Conflict of Interest Statement

The authors declare that there are no conflicts of interest.

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