

Effects of perceived criticism on anxiety and depression during behavioral treatment of anxiety disorders

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Abstract

The present study explored the effect of perceived criticism (PC) on levels of anxiety and depression during behavioral treatment among patients diagnosed with obsessive-compulsive disorder (OCD) or panic disorder with agoraphobia (PDA). We posited that patients' perceptions of relatives' criticism and the degree to which they were upset by the criticism (UC) would be related to negative affect and discomfort during exposure. The sample included 75 patients with a primary diagnosis of OCD ($n = 43$) or PDA ($n = 32$) and their participating relatives. Measures of patients' weekly ratings of PC and upset about the criticism, anxious and depressed mood, and subjective discomfort during exposure treatment were analyzed using a mixed model regression approach (SAS Proc Mixed). Patients' anxious and depressed mood predicted greater discomfort during exposure. Patients who were more UC also had higher weekly ratings of anxiety and depression, and more discomfort during exposure sessions. Findings suggest that treatment outcome may be improved by attention to patients' reactions to their interpersonal environment.

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High expressed emotion (EE), typically operationalized as critical comments, hostility and emotional overinvolvement by relatives during a family interview, is a remarkably robust predictor of relapse upon psychiatric hospital discharge. Over the past several decades, high EE has predicted relapse in numerous studies of patients with schizophrenia, mood and other disorders (for review see [Butzlaff & Hooley, 1998](#)). Several recent studies have demonstrated that high EE or EE-related variables in relatives predict poor treatment outcome for children and adults with anxiety disorders (e.g., [Chambless & Steketee, 1999](#); [Crawford & Manassis, 2001](#); [Leonard et al., 1993](#); [TARRIER, Sommerfield, & Pilgrim, 1999](#)). A few studies have examined how EE may exert its effects. Studies of the physiological effects of EE suggest that patients respond to high

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EE relatives with increased heart rate and skin conductance responses (e.g., Hibbs, Zahn, Hamburger, Kruesi, & Rapoport, 1992; Sturgeon, Kuipers, Berkowitz, Turpin, & Leff, 1981; Tarrier, Vaughn, Lader, & Leff, 1979). These findings suggest that the presence of high EE relatives may act as a stressor that, in turn, exacerbates symptom severity.

Although the Camberwell Family Interview (CFI, Vaughn & Leff, 1976) is the standard measure of EE, it requires extensive time to administer and code and is not suitable for rapid or repeated assessment. Hooley and Teasdale (1989) developed the Perceived Criticism Measure (PCM) to facilitate rapid measurement of relatives' criticism from the patient's perspective. This measure assesses two conceptually related aspects of EE: how critical a patient perceives a relative to be perceived criticism (PC) and how upset the patient is by that criticism (UC). The simple 10-point rating scale of PC has proved a strong predictor of relapse upon hospital discharge for patients with various psychiatric conditions (see review by Butzlaff & Hooley, 1998). Similarly, in a sample of outpatients with panic-disorder with agoraphobia (PDA) or obsessive-compulsive disorder (OCD), we (Chambless & Steketee, 1999) found PC to be a stronger predictor of the main outcome measure than CFI measures of EE. Perceived upset added nothing to PC's prediction of outcome. Thus, for anxiety-disordered patients, it may be especially important to understand the role of patients' perceptions of criticism in determining response to treatment.

Hooley's measure also assesses how upset patients are by the criticism. In one of the few studies that examined the role of upset in predicting outcome, Miklowitz, Wisniewski, Miyahara, Otto, and Sachs (2005) found that how upset patients with bipolar disorder felt in response to criticisms from key relatives predicted treatment outcome (depressive and manic symptoms), although PC did not. They suggested that heightened sensitivity to criticism, rather than perception of criticism per se, merited further study. Thus, the extant literature is mixed as to which variable is more important, PC or sensitivity to that criticism.

Most patients with severe anxiety disorders respond positively to short-term outpatient behavioral treatment that includes prolonged exposure to feared situations (Barlow, 2002). Several studies suggest that processes and reactions that occur during treatment influence the effectiveness of exposure. Reduction (habituation) in discomfort between exposures to feared situations has been linked to positive outcomes for patients with OCD and PTSD (Foa et al., 1983; Jaycox, Foa, & Morral, 1998), and is considered an important mechanism through which exposure produces its effects (see Foa & Kozak, 1986). Both depressed and anxious mood prior to exposure can interfere with reduction in anxiety experienced during and following exposures (e.g., Foa et al., 1983; Mills & Salkovskis, 1988; Murphy, Michelson, Marchione, Marchione, & Testa, 1998). Some findings suggest that relationships with relatives can influence mood during exposure treatment. Low marital satisfaction has been linked to increased anxiety in vivo (Murphy et al., 1998). To our knowledge, no studies have examined the possible mechanisms by which mood state and criticism within the family environment influence the exposure treatment response for patients with anxiety disorders.

The present study used a prospective longitudinal design to elucidate the relationship between aspects of the family environment (perception of relatives' criticism and the degree of emotional upset this produces in the patient) and the progress of outpatients with OCD and PDA during behavioral treatment. We posited that weekly-PC and upset about (sensitivity to) this criticism would predict weekly levels of negative mood (anxiety and depression), and this in turn would influence discomfort during weekly or biweekly exposure sessions. Our exploratory hypothesis was that patients' perceptions of greater criticism from a key relative would be associated with more negative affect and higher anxiety during the exposure sessions. We also tested whether patients who were more upset by their relatives' criticism would report more anxious and depressed mood prior to exposure sessions and more discomfort during exposure.

Method

Participants

Participants were outpatients with primary diagnoses of OCD or PDA according to the Structured Clinical Interview for the DSM-III-R (SCID-P; Spitzer, Williams, Gibbon, & First, 1989) completed by trained masters level raters who achieved very high reliability ($\kappa = .94-1.0$) for these diagnoses. Participants were included if they were aged 18–65, had OCD or PDA symptoms for at least 1 year, displayed at least 1 h of

rituals per day for OCD participants or moderately severe avoidance behavior according to SCID criteria for PDA participants, and lived with a relative or partner for at least 3 months prior to beginning the study. Exclusion criteria were current symptoms or history of psychosis, evidence of organic etiology for primary anxiety disorder, or family members unwilling to participate in the study.

Of 102 patients who entered the trial, 75 completed the entire study protocol and had at least 18 of a possible of 30 (two for each of 15 treatment sessions) time points during treatment to permit analyses for the present study. Of the 75 participants, 73% were married or living with partners, 15% had never married, and 9% were divorced or separated but living with other relatives. Women constituted 73% of the sample; 83% were white, 13% African American, 3% Asian and 1% other. The average age was 35.8, ranging from 18 to 62 years old. Participants reported mean symptom duration of 12.5 years for their primary diagnosis¹ (range 1–54 years).

A primary live-in relative was selected for each patient. When the patient lived with more than one relative, the primary relative was chosen based on the importance of the relationship according to the following priority rules: (1) spouse/domestic partner, (2) most critical parent based on CFI ratings, (3) most critical other relative. Relatives included 54 spouses, 15 parents, and 6 other relatives (e.g., adult child, sibling).

Measures

PCM (Hooley & Teasdale, 1989): PC was assessed by having patients rate “how critical do you think your relative is of you?” on a scale from 1 (*not at all critical*) to 10 (*very critical*). Immediately following, patients responded to the question “how upset do you get when your relative criticizes you?” (UC) on a 10-point scale ranging from *not at all* to *very* upset. Test–retest reliability for the criticism portion of the PCM was moderately high ($r = .75$) in two studies over an interval of approximately 20 weeks (Hooley & Teasdale, 1989) and for a 2-week interval before treatment for patients in the present study. The PC rating has demonstrated adequate discriminant validity in relation to measures of depression, anxiety, and personality disorder traits (Chambless & Steketee, 1999; Hooley & Teasdale, 1989; Riso, Klein, Anderson, Ouimette, & Lizardi, 1996) and adequate predictive validity by its prediction of poor treatment outcome (Chambless & Steketee) and relapse upon hospital discharge (Hooley & Teasdale, 1989). Convergent validity in relation to CFI ratings of criticism and ratings of criticism from face-to-face interactions is somewhat mixed but within acceptable limits (Chambless, Bryan, Aiken, Steketee, & Hooley, 1999; Chambless et al., 2001).

Perceived upset has received less psychometric attention. Test–retest reliability for perceived upset over a 2-week interval was .68 for the parent study from which these data were drawn. Upset showed small and non-significant correlations with CFI measures (r 's < .11), whereas correlations with symptom severity and depression were significant, but small (r 's < .29). As expected, PC and upset proved to be moderately related ($r = .47$). Nonetheless they were distinct: corrected for attenuation due to lack of perfect reliability, their correlation was $r = .62$, far from 1.0. These data provide initial positive indication of the reliability and convergent and discriminant validity of the perceived upset measure.

Patients completed the PCM weekly for selected relatives during the course of behavioral treatment for their disorder; only the PCM for one primary relative per household was included in analyses. If more than one PCM was obtained within one treatment week (some sessions were held twice weekly), the averages of the scores for that week were used in the data analyses. In the present sample, average scores across time points for PC were 4.43 ($SD = 2.59$; range 1–10) and for perceived upset were 5.52 ($SD = 2.64$, range 1–10).

Weekly mood ratings: At the outset of each exposure session patients rated their mood over the past week separately for anxiety and for depression, on scales ranging from 0 = *none* to 8 = *severe*. In previous research, these self-reported anxiety and depression ratings showed good convergent validity (Fydrich, Dowdall, & Chambless, 1992). Daily ratings of anxiety averaged over 1 week correlated .58 with the STAI Trait Anxiety and .54 with the Beck Anxiety Inventory, and diaries of depression averaged over 1 week correlated .42 with the Beck Depression Inventory. Diary anxiety also showed evidence of discriminant validity.

¹Symptom duration was 14 years for participants with OCD and 10.6 years for those with PDA, differences that were not significant ($p > .10$).

Subjective units of discomfort (SUDS, [Wolpe, 1988](#)): Consistent with usual practice during exposure treatment, patients were asked to rate the intensity of their discomfort every 10 min during exposures. The scale ranged from 0 = *no discomfort* to 100 = *maximum discomfort ever felt*. [Thyer, Papsdorf, Davis, and Vallecorsa \(1984\)](#) demonstrated convergent validity in the significant correlation of SUDS ratings with physiological measures associated with anxiety. For the present study the highest SUDS rating occurring during the first 30 min of the exposure session was used as the criterion variable in analyses and represented variation in anxiety across exposure sessions.

Procedures

Details of the treatment and general procedures for the study are provided by [Chambless and Steketee \(1999\)](#). All patients and relatives provided written informed consent and completed a battery of measures before and after treatment. Patients and therapists also completed measures described above during or between therapy sessions. Behavioral treatment was conducted according to detailed manuals for OCD and PDA treatment. Therapists were masters or doctoral level clinicians, trained and supervised weekly by the local site investigators (GS and DC). Patients received a total of 22 sessions over 16 weeks. Treatment included two 90-min sessions for treatment planning, sixteen 90-min therapist-assisted exposure sessions (plus response prevention for OCD participants) of which 12 were held twice weekly and 4 once weekly. These sessions took place in relevant exposure locations. Treatment ended with 4 weekly 60-min office sessions focusing on self-directed exposure and relapse prevention. Relatives attended one session of treatment planning but did not participate in any exposure or maintenance sessions, and therapists were instructed not to discuss family problems during treatment. Therapist adherence to the protocol was assessed by having therapists from the other site rate a randomly selected sample of 25% of the tapes of the final four sessions conducted in the office using a treatment integrity checklist of prescribed and proscribed interventions devised for this study. All ratings were within acceptable guidelines.

Statistical analysis

Data were analyzed using a mixed model regression approach (SAS Proc MIXED, SAS Version 8.0, SAS Institute, Cary, NC). Mixed modeling accommodates the longitudinal design and the correlated nature of repeated measurements over time on the same individual and allows the incorporation of covariates. Further, it handles arbitrary numbers and spacing of measurements over time as well as data that are missing in ways predicted by either covariates in the model or the previously observed outcomes. Because we included only patients who completed treatment in the analysis, missing data from drop-outs was not a problem. To determine whether the pattern of missing data was related to outcome, we correlated the percent missing data on each outcome variable with the average value across all weeks. This is a simple form of a pattern-mixture model ([Little & Rubin, 1987](#)). A significant correlation would indicate that patients with substantial missing data had significantly different outcomes, requiring more sophisticated models to adjust for missing data. No significant associations were observed; we therefore employed the mixed model approach without further adjustments for missing data.

We hypothesized that the three types of variables measured (criticism, mood state, SUDS) constituted a hierarchy of time-series data from individuals, and that these within-subjects effects occur over time in a feedback loop. We constructed the conceptual model presented in [Fig. 1](#) in which distress in the anxiety-provoking situation, the dependent variable, was represented by SUDS. Two sets of independent variables, criticism/upset and weekly mood (anxiety, depression), were expected to predict SUDS. In addition, we hypothesized a hierarchy of these effects in which PC was expected to have an effect on UC, and these two variables, in turn, would influence weekly anxiety and depression. Based on the conceptual model, we predicted that (a) participants with higher PC and UC would have higher (more negative) mood state, (b) participants with higher mood state would have higher SUDS, (c) participants with higher PC/UC and mood state would have higher SUDS.

To test the overall model illustrated in [Fig. 1](#), we performed a series of Proc Mixed models analyses with random patient effects. Convergence criteria were met in all analyses, suggesting that no obvious numerical

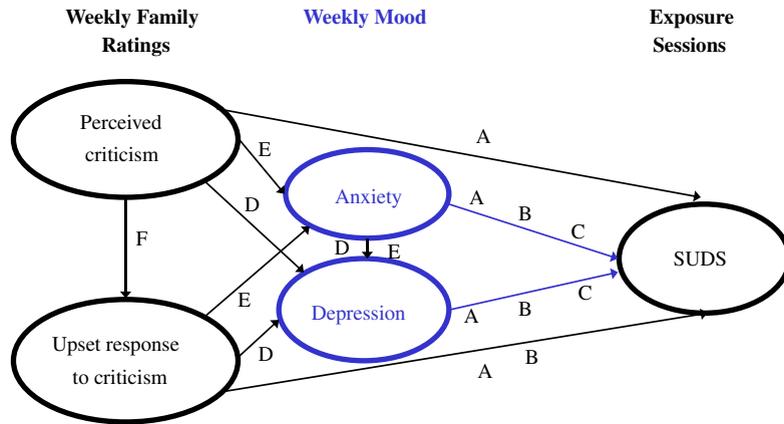


Fig. 1. Conceptual model of relationships among perceived criticism, upset response, mood state and subjective anxiety ratings (SUDS) during exposures. Letters represent variables included in the series of 6 analyses (see text).

Table 1

Significant mixed model solution paths or effects of perceived criticism, upset, mood state and anxiety during exposures for patients with OCD and PDA

Analysis model (see text)	Model fitting information (dependent variables)	Predictors	Unstandardized β	Standardized β	Standard error	t	$Pr > t $
<i>Direct effects</i>							
A, B, C	SUDS	Anxiety	2.9700	.2041	.7160	3.2	.0014
A, B, C	SUDS	Depression	1.3936	.1355	.5473	2.6	.0112
A, B	SUDS	UC	.8926	.1135	.3596	2.5	.0135
D	Depression	Anxiety	.5171	.3656	.0493	10.4	.0001
E	Anxiety	UC	.5557	1.0479	.0235	2.4	.0179
F	UC	PC	.5627	.5520	.2827	19.7	.0001
<i>Indirect effects</i>							
	SUDS	UC	.9753	.1264	.3526	2.6	.0061
	SUDS	Anxiety	3.1740	.2181	.6463	4.9	.0001
	SUDS	Depression	2.1320	.2072	.5015	4.3	.0001

SUDS = subjective units of discomfort during exposure, PC = perceived criticism, UC = upset regarding perceived criticism.

*Standardized β represent the magnitude of change, e.g., 1 unit increase in anxiety and SUDS go up by 0.2 *SD* or a small to medium change; 1 unit increase in UC and anxiety go up by over 1 *SD* or a large change.

problems were detected. Because the mixed model analysis used has no equivalent analogue to *R* and *R*² available for the ordinary least-squares regression analyses, these statistics are not reported here. Instead, standardized coefficients (*t* statistic, see Table 1) are given to facilitate interpretation of the figure.

Results

All significant statistical direct and indirect effects of the mixed model analyses of the predictor variables on SUDS are summarized in Table 1. The direct effects are represented by significant paths depicted in Fig. 2; the greater the magnitude of the statistic, the stronger the relationship of the two variables. The first three analyses (A, B, and C below) used SUDS during exposure as the criterion variable in relation to other predictors in the models. For the first analysis (Model A), SUDS was the dependent variable and all four predictors (PC, UC, weekly anxiety, weekly depression) were the independent variables. Findings from this analysis indicated that PC was not significantly related to SUDS, $p > .3438$. In the next analysis (Model B), the non-significant PC variable was omitted and UC, weekly anxiety and weekly depression were the independent variables with

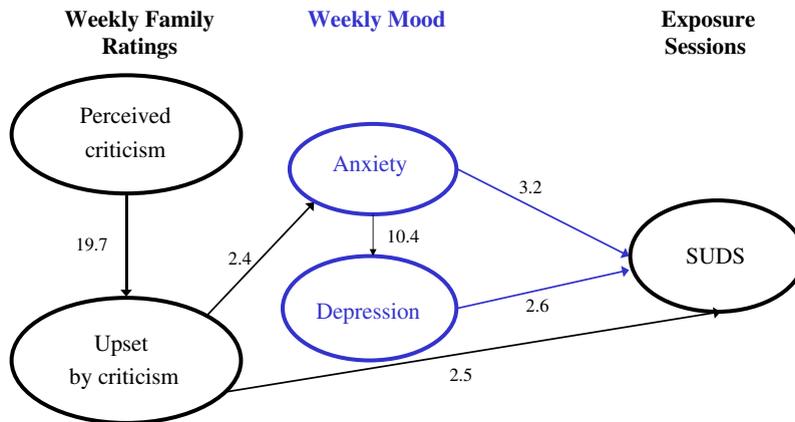


Fig. 2. Significant paths among perceived criticism, upset about criticism, mood state and subjective anxiety ratings (SUDS) during exposures (numbers represent *t*-coefficients).

SUDS as the dependent variable. This analysis revealed that weekly anxiety was not statistically significantly related to SUDS, $p > .068$, whereas UC and ratings of depression did predict anxiety during exposure. In the third analysis (Model C), SUDS was the dependent variable and weekly anxiety and depression were the independent variables; both mood variables were significantly related to SUDS.

The next 2 analyses (D and E) examined weekly depression and anxiety in relation to the other three predictors. For Model D, weekly depression was the dependent variable and PC, UC, and weekly anxiety were the independent variables; findings indicated that only weekly anxiety, but not PC and UC (p 's $> .231$ and $> .630$, respectively), was significantly related to weekly depression. In Model E, weekly anxiety was the dependent variable and PC, UC, and weekly depression were the independent variables; only PC was not significantly related to weekly anxiety ($p > .195$). For the final analysis (Model F) with UC as the dependent variable and PC as the independent variable, PC was significantly related to UC.

Direct and indirect effects were as follows. In the analysis with all four independent variables (Model A), the lack of statistical significance for PC indicates that PC did not have a direct effect on SUDS. However, PC indirectly affected SUDS scores via its direct effect on UC, which has direct effects on SUDS. That is, PC was associated with increased discomfort during exposure because of its relationship with upset, which directly predicted SUDS. In addition, an indirect effect of PC on SUDS was also evident through the pathway of significant adverse effect of UC on average weekly anxiety and of weekly anxiety on weekly depression, which predicted an increase in SUDS during exposure sessions. The second analysis (Model B) indicated that UC had a direct effect on SUDS, evident in its statistically significant relationship beyond the effects of anxiety and depression. The remaining analyses revealed that UC also had an indirect effect on SUDS through its relationship to anxiety. That is, the path from UC to anxiety and the path from anxiety to SUDS were both statistically significant. Anxiety also had direct effects on depression, and indirect effects on SUDS via depression. Finally, both anxiety and depression had direct effects on SUDS.

Discussion

In the present study, we sought to determine whether patients' reported anxiety during exposure treatment sessions was directly or indirectly associated with their perception of a key family member's criticism. We wanted to determine whether this effect was mediated by patients' emotional reaction (sensitivity) to PC and whether feelings of anxiety and depression played any role in this process. The findings from the mixed model regression analysis suggest that patient's perceptions of their relatives' criticism do influence their responses during exposure treatment on a week-to-week basis. However, the process by which this occurs appears to be somewhat complex. PC by itself has no direct impact, but PC about which patients are upset is associated with increased anxiety during exposure (SUDS) and also with increased general anxious mood assessed on a weekly basis. Not surprisingly, patients who are upset by PC also appear to suffer from anxious mood that is also

closely associated with depressed mood. These negative mood states appear to predict variation in anxiety during exposure sessions.

The present study is not a direct test of the influence of mood state or relative variables on habituation. We could not directly assess reduction of anxiety within or between exposure sessions (cf. Jaycox et al., 1998) because exposures were not constructed to permit repeated assessment of discomfort to the same exposure stimuli. Rather, in each subsequent session patients faced increasingly more difficult stimuli. Nonetheless, because our findings may have some bearing on habituation during exposure, we point to existing literature addressing this issue. That anxious and depressed mood predicted more anxiety during exposure is consistent with previous studies of OCD patients in which pretreatment depressed mood was associated with poorer outcome through its adverse effect on habituation during exposures (Foa et al., 1983). In that study, pretreatment anxious mood assessed at pretest, rather than weekly as we have done, had a direct negative effect on outcome rather than influencing outcome via habituation processes. Moreover, in analogue studies of the effects of induced depressed mood on treatment of spider phobia, negative mood negative mood assessed immediately before treatment sessions seemed to impair habituation and enhance return of fear (Mills & Salkovskis, 1988; Salkovskis & Mills, 1994).

The finding that sensitivity to PC, but not PC by itself, predicted greater anxious mood before exposure and higher distress during exposure is consistent with Miklowitz et al.'s (2005) report on bipolar patients. They observed that upset, but not PC, predicted worse outcome on negative mood measures after therapy. Their study and ours suggest the potential importance of reactivity to PC as an important variable in the treatment process.

Findings from the present study provide a possible mechanism of action for the adverse effect of PC on treatment outcome. As noted earlier, several studies have demonstrated a solid relationship between relapse and relatives' high EE assessed via a family interview. PC has also proved to be a strong predictor of relapse for patients diagnosed with major depression (Hooley & Teasdale, 1989) and with substance abuse (Fals-Stewart O'Farrell, & Hooley, 2001). Further, some studies indicate that patients with OCD react with greater physiological and subjective discomfort to higher levels of EE in parents (Hibbs et al., 1992). This greater reactivity may increase overall sensitivity to uncomfortable exposure experiences and account for poorer outcomes. For most patients with anxiety disorders, empirically effective treatment requires prolonged exposures to feared situations to reduce anxiety symptoms. Although causal interpretations cannot be clearly drawn from correlational data, our data suggest that living with relatives who are perceived as critical by patients who are highly sensitive to this criticism increases general anxious mood and also increases anxiety during exposures. As noted earlier, our study did not directly examine habituation of discomfort during exposure. Additional prospective research is needed to confirm that sensitivity to PC among anxious outpatients interferes with habituation of anxiety during and between exposure treatment sessions.

Confirmation of our preliminary findings may provide an important avenue to influencing treatment outcome, consistent with Keitner & Miller's (1990) model that calls for interventions that address both the patient's vulnerability and the family's competence. For example, perhaps patients could be trained to be less disturbed by their relatives' criticism, and relatives to moderate their negative attitudes and reduce their critical behaviors toward patients. For patients, such a treatment approach could include cognitive restructuring regarding perceptions of criticism or mindfulness and acceptance training to promote the ability to hear relatives' comments without having to reflexively treat the comments as upsetting. For relatives, likely treatment approaches include cognitive restructuring regarding the targets of criticism, as well as communication training to promote more straightforward and less aggressive communication patterns. Such family interventions have proved beneficial for patients with schizophrenia and bipolar disorder (Barrowclough & Tarrier, 1992; Miklowitz & Goldstein, 1997). Clearly, these suggestions are preliminary and require further research to confirm the present findings, followed by research regarding what specific techniques can actually reduce PC and upset regarding it. Our treatment protocol did not allow therapists to address family concerns, possibly limiting the generalizability of the findings to standard clinic settings.

Findings from the present study must be treated with caution because of the limitations of the dataset. Our sample included only 10 African American clients. Previous research on African Americans with schizophrenia has indicated different responses to criticism and relationship of criticism to outcomes than observed among white clients (e.g., Tompson et al., 1995). Our sample of black clients is too small to conduct

separate analyses for this group, but further research is needed to clarify whether culture, race or ethnicity influences findings. Although accommodated by the mixed model regression approach used, missing data would be much reduced by studies deliberately designed to train patients better on the measures and then encourage patients' cooperation in order to fully collect anxiety levels (SUDS) during exposures and self-report measures in between sessions. Another concern is that measures of PC and upset were collected at the same time, possibly making it more likely that their relationship is overestimated. Nonetheless, their different predictive capacities suggest they are measuring different constructs. Moreover, although our data collection procedure permits us to determine that mood and criticism variables preceded distress during exposure sessions, we cannot determine the temporal sequence of negative mood and PC and upset. Finally, although the time series analyses performed here afford a higher degree of confidence in the results than simple correlational analyses of relationships at one point in time, the data remain correlational in nature and clear causal conclusions cannot be drawn. However, we believe the present data draw us one step closer to understanding the mechanisms behind which interpersonal variables may affect treatment outcome in anxiety disorders.

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