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An investigation of outcome expectancies as a predictor of treatment response for combat veterans with PTSD: Comparison of clinician, self-report, and biological measures

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Abstract

Background—Outcome expectancy, or the degree to which a client believes that therapy will result in improvement, is related to improved treatment outcomes for multiple disorders. There is a paucity of research investigating this relation in regards to PTSD. Additionally, the bulk of the research on outcome expectancy and treatment outcomes has relied mostly on self-report outcome measures.

Methods—The relation between outcome expectancy on self-report measures, clinician-rated measures, and two biological indices (fear potentiated startle and cortisol reactivity) of PTSD symptoms was explored. The sample included combat veterans (N= 116) treated with virtual reality exposure therapy for PTSD.

Results—Results supported a negative association between outcome expectancy and both self-report and clinician-rated symptoms at the conclusion of treatment, but outcome expectancy was related to the magnitude of change during treatment for self-report measures only. Outcome expectancy was unrelated to biological measures of treatment response.

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Conclusions—These findings suggest that outcome expectancy may be related to patient and clinician perceptions of outcomes but not biological indices of outcome for PTSD.

Keywords

Outcome expectancy; post-traumatic stress disorder; multi-modal assessment; virtual reality; multilevel modeling

Outcome expectancy, the degree to which a client believes that therapy will result in improvement, is important to the therapeutic process¹ and empirically associated with improved outcomes. In a recent meta-analysis, early treatment outcome expectancies demonstrated a small but significant positive effect ($d = .24$) on treatment response². As a result, positive expectancies are considered critical to the effectiveness of psychological interventions³. The relation between expectancies and outcomes for posttraumatic stress disorder (PTSD), however have been relatively unexplored. Examining this association is necessary given that low expectations for treatment response is often cited as a barrier in providing evidence-based treatments for those with PTSD⁴.

Theoretical models of PTSD suggest that outcome expectancy is highly relevant to treatment response. A core process through which traumatic experiences can lead to psychopathology is the disruption of an individual's basic beliefs and assumptions⁵. PTSD is related to an increase in negative cognitions regarding self, others, and the world⁶. As such, individuals with PTSD with low expectations may have negative beliefs regarding their own ability to change, distrust in the therapeutic process, and avoid traumatic material. Indeed, a prior study suggested that feelings of an inability to change was associated with poorer treatment response for exposure therapy⁷. Elevated expectations may enhance mechanisms that are important to treatment, such as emotional engagement⁸. Positive expectations for treatment may allow for increased emotional engagement in exposure-based interventions, which would facilitate extinction and may increase adherence to the prescribed treatment regimen⁹.

In the treatment of PTSD, trauma-focused treatments have the consistent empirical support, many of which involve exposure to trauma memories and cues¹⁰. The only study to examine empirically the association between outcome expectancy and treatment response for those with PTSD, however, suggested a null relation for self-report and clinician-rated measures⁷. Although the association has not been examined extensively in PTSD specifically, the association between outcome expectancy and treatment response for exposure has been mixed. Four studies examined outcome expectancy as a predictor of response to exposure therapy for social phobia. Two supported a positive relation between outcome expectancy and treatment response in both clinician-rated and self-report measures^{11,12}. A third reported that expectancy was related to one of five self-reported outcomes but not clinician-rated measures¹³. The fourth found a positive relation between a single self-report inventory and expectancy¹⁴. Within obsessive-compulsive disorder (OCD), only one study demonstrated a significant relation between outcome expectancy and treatment response¹⁵. Others reported that it was unrelated to clinical outcomes for self-report and clinician-rated measures^{16–18}. There was no significant effect of outcome expectancy on treatment outcomes, including self-report and clinician-rated measures, in a sample of patients treated for generalized

anxiety disorder (GAD)¹⁹. In a study of individuals with specific phobia, higher outcome expectations were related to stronger reduction in symptoms across two self-report measures²⁰.

A critical limitation of the current literature on outcome expectations is an overreliance on self-reported outcomes²¹. This limits the ability to draw a firm conclusion that outcome expectancy significantly impacts clinical outcomes given the biases associated with self-report data. Indeed, recent discussions of exposure therapy have advocated for multi-modal assessment of outcomes that incorporate behavioral, physiological, and independent ratings as outcomes²². Research that combines self-report, clinician-rated, and biological measures of outcomes is needed. Alterations in acoustic startle reflex^{23–26} and increased cortisol reactivity²⁷ are considered biomarkers of PTSD symptoms. Both acoustic startle response²⁸ and cortisol reactivity^{29–31} have been used as objective biomarkers of PTSD treatment response²⁹; however they have not yet been studied in relation to outcome expectancy.

The present study examined the association between pretreatment outcome expectancy and treatment response in a sample of recently returning combat veterans diagnosed with PTSD. Participants underwent six sessions of virtual reality exposure (VRE) for PTSD as part of a larger clinical outcome trial²⁹. VRE use a computer-generated three-dimensional virtual environment to create stimuli in a virtual Iraq or Afghanistan. A meta-analysis investigating the use of VRE for the treatment of anxiety and specific phobias found that its was effective³², including for PTSD^{33,34}. Another meta-analysis specifically compared VRE to either in vivo exposure or control conditions and found a large mean effect size for virtual reality treatment compared to control conditions, and a small effect size favoring VRE relative to vivo based exposure treatment³⁵. Related work has suggested that outcome expectancy plays an important role in the treatment response for VRE^{11,20}, but this relation has never been examined for PTSD. The participants who endorsed greater outcome expectancy were hypothesized to demonstrate increased treatment response across self-report, clinician-rated, and biological measures compared to participants who endorsed lower outcome expectancy.

Methods

Participants

The total sample included 156 combat veterans. Inclusion criteria were meeting DSM-IV criteria for PTSD due to military trauma as verified by discharge paperwork. Exclusion criteria included a lifetime history of psychosis, bipolar disorder, current suicidal risk, current alcohol or drug dependence, pregnancy, and current use of medications that could confound outcomes (i.e., glucocorticoids, benzodiazepines, chronically used opioids). Participants were required to discontinue long-acting benzodiazepines for 1 month and short acting benzodiazepines for 2 weeks before screening. Participants taking other medications must have been taking stable doses for at least 2 weeks and were required to maintain their dose throughout the study.

For the present study, the 116 participants who completed session 1 and provided outcome expectancy data were included. There were no differences at baseline between those who

were enrolled in the study, completed treatment, or dropped out prior to the first session²⁹. Participants were between the ages of 23 and 55 ($M = 34.74$, $SD = 8.35$) and predominately male (94.8%). Self-reported ethnicities were as follows: White = 43.1%; Black = 47.4%; Hispanic = 6.0%; Asian = 0.9%; Other = 2.6%. Half (50.0%) of the sample was employed fulltime. A majority of the sample reported having an active or pending PTSD compensation claim with the VA (55.5%).

Measures

Clinician-Rated Measures

Clinician Administered PTSD Scale³⁶: The CAPS is a clinician-rated interview designed to diagnose current and lifetime PTSD. The CAPS targets the 17 specific PTSD symptoms from the *DSM-IV*³⁷ to assess the intensity and frequency of each symptom on a five-point Likert scale. Scores ranged from 0–120.

Clinician Global Impressions-Global Improvement and Severity of Illness (CGI-Global; CGI-Severity³⁸): The CGI-Global scale assesses clinical improvement of a patient on a 7-point scale that corresponds to discrete categories of wellness. A score of 1 indicates that the person is very much improved, 4 corresponds to no change, and 7 corresponds to very much worse. The CGI-Severity uses a similar methodology in which the clinician provides a rating as to the severity of the patient's illness.

Self-Report Measures

Outcome Expectancy³⁹: Outcome expectancy was assessed with a four-item measure that has been used extensively with clinical populations. Ratings were made on a 9-point Likert scale with scores ranging from 4 to 36. Higher scores indicated increased expectations for successful treatment. The measure has good internal consistency ($\alpha > .80$ ⁴⁰, repeated-measures reliability⁴¹, and is associated with treatment response for CBT¹⁴. Internal consistency for the current sample was good ($\alpha = .87$). This measure was administered at the end of the first treatment session, during which participants received the treatment rationale.

PTSD Symptom Scale⁴²: The PSS is a 17-item self-report instrument that corresponds to the DSM-IV PTSD symptoms. Participants provide a rating of symptoms on a 0–3 scale (0 = not at all, 3 = 5 or more times per week/very much) such that greater scores are indicative of more severe symptoms (range 0–51). Internal consistency was excellent across all assessment points with $\alpha = .89$ to $.96$.

Beck Depression Inventory-II⁴³: The BDI-II is a 21-item measure designed to assess the cognitive, affective, behavioral, motivational, and somatic symptoms of depression in adults and adolescents⁴⁴. Participants provide a rating of symptoms on a 0–3 scale with greater scores representing more severe symptoms (range 0–63). Internal consistency was excellent across all assessment points with $\alpha = .91$ to $.96$.

State Trait Anxiety Inventory⁴⁵: The STAI contains two subscales to assess state and trait anxiety. Only the state subscale was administered in the present study. The state subscale

includes 20 items rated on a 1–4 scale with higher scores indicating greater anxiety (range 20–80). Internal consistency was excellent across all assessment points with $\alpha = .88$ to $.93$.

Clinician Global Impressions-Self Report³⁸: The CGI scales measure global psychopathology similar to the other CGI scales. Participants rated their functioning at the start of each session.

Biological Measures

Trauma-Potentiated Startle²⁸: Trauma-potentiated startle is defined as increased magnitude of the startle reflex in the presence of trauma cues, and has recently been used as a biomarker of treatment response for exposure therapy. In the present study, participants were exposed to three 2-minute combat virtual reality scenes. Startle was elicited by four 40-ms, 106-dB white noise probes during each scene at a variable 15–45 second interstimulus interval. Responses were measured with electromyography (EMG) of the orbicularis oculi muscle contractions acquired using the EMG module of the Biopac MP150 system. Peak startle magnitude measured from 20–200 ms after acoustic probe onset was used as the startle response index. Responses were averaged across the four startle probes delivered during the three VR scenes. In order to correct for individual differences in baseline startle, post-treatment startle responses were calculated as percent change from baseline.

Cortisol Reactivity: Salivary cortisol samples were collected immediately before, after, and 15 minutes after the VR scenes presented in the startle paradigm. Samples were frozen and batch-processed using a chemiluminescent immunoassay. Inter-assay and intra-assay coefficients of variations were under 10%. A measure of cortisol change from the initial assay to the 15-minute assay was used in the present study as an indicator of treatment response as this difference was most affected by treatment²⁹.

Procedure

The present study is a secondary data analysis of a clinical trial examining the effect of d-Cycloserine on outcomes for PTSD relative to a pill placebo or benzodiazepine²⁹. An abbreviated methodology is provided below and readers are encouraged to consult²⁹ for a complete description of the methods.

Assessment schedule—Participants completed a total of 5 assessments during the study: baseline, posttreatment, 3-month follow-up, 6-month follow-up, and 12-month follow-up. All measures were completed at each assessment with the exception of the CGI scales and the outcome expectancy measure at baseline, which were completed at the first session.

Treatment—Participants completed a six-session treatment of virtual reality exposure therapy²⁹. Sessions lasted 90 minutes and were conducted by doctoral-level clinicians. The first session provided an overview of the treatment and the rationale for the approach. The remaining sessions involved exposure to virtual stimuli displayed through an eMagin Z800 head-mounted display. Virtual stimuli were modified to the specific patient such that the scene presented (e.g., Humvee caravan) and cues included (e.g., firefight, explosions) were

matched to the specific memory of a given patient. Therapists viewed the virtual environments on an independent computer and encouraged patients to complete 30–45 minutes of exposure. Exposures were followed by 15–20 minutes of processing. There were no homework assignments. Experimental medications (D-Cycloserine 50mg or alprazolam 0.25mg, compared to placebo) were administered 30 minutes prior to each exposure session, and participants and therapists were blind to the experimental medication condition.

Data Analytic Plan

An intent to treat (ITT) sample was used in the current study such that information from all 116 participants was used. Missing data were handled with FIML that is integrated into multilevel model. Of the 116 participants, 97 (83.6%) completed treatment.

Outcome expectancy was first correlated with all pretreatment measures. The relation between outcome expectancy, change during treatment, posttreatment scores, and change during the 1-year follow-up period for self-report and clinician-rated measures was assessed with multilevel models⁴⁶. A 2 level piecewise model was used in which level 1 assessed intraindividual change and level 2 assessed interindividual differences. The level 1 model included three fixed effects: an intercept that corresponded to posttreatment scores (β_0), a slope that corresponded to the change from pretreatment to posttreatment (β_1), and a slope that corresponded to change from posttreatment to 12-month follow-up (β_2). Random effects were included to model interparticipant variability at posttreatment, change during treatment, and change during follow-up. Outcome expectancy was examined as a person-level fixed effect to measure its association with posttreatment while controlling for other assessment points (β_{03}) and as an interaction term with the two slopes to determine if it was associated with change from pretreatment to posttreatment (β_{13}), and change from posttreatment to follow up (β_{23}). Dummy coded variables representing group membership were included as level 2 fixed effects for intercept, treatment slope, and follow-up slope. The d-Cycloserine group was treated as the reference group.

Biological measures were examined as predictors of treatment response at posttreatment only due to lack of available data collected at follow-up assessments. Startle data was available for 37 participants at posttreatment and cortisol data were available for 60 participants at posttreatment. Data loss on these measures was due to equipment error, loss to follow-up psychophysiological assessments, noisy data (EMG startle), or endocrine assay error (cortisol). The data were retained were believed to be of high quality. Regressions were used to examine the association between posttreatment startle and cortisol while controlling for group membership and pretreatment biomarker data.

Results

Descriptive statistics for outcome variables and outcome expectancy are presented in Table 1. Outcome expectations were positively correlated with pretreatment STAI ($r = -0.21$, $p = 0.039$), CGI-Global ($r = -0.28$, $p = 0.007$), CGI-SR ($r = -0.24$, $p = 0.019$). Outcome expectations were unrelated to other pretreatment measures.

Clinician-Rated Measures

Outcome expectancy was evaluated as a fixed effect of treatment slope, posttreatment scores (intercept), and follow-up slope for the clinician-rated measures (CAPS, CGI-Global, and CGI-Severity; Table 2). Outcome expectancy was associated with posttreatment scores on all measures (CAPS: $\beta_{03} = -1.18$, $p = 0.035$; CGI-Global: $\beta_{03} = -0.07$, $p < 0.001$; CGI-Severity: $\beta_{03} = -0.04$, $p = 0.038$). Outcome expectancy, however, was not associated with treatment slope (CAPS: $\beta_{13} = -0.43$, $p = 0.093$; CGI-Global: $\beta_{13} = -0.03$, $p = 0.097$; CGI-Severity: $\beta_{13} = -0.02$, $p = 0.480$) or follow-up slope (CAPS: $\beta_{23} < 0.01$, $p = 0.948$; CGI-Global: $\beta_{23} < 0.01$, $p = 0.481$; CGI-Severity: $\beta_{23} = -0.01$, $p = 0.472$). There was no support for differences in the effect of outcome expectancy across the treatment groups for all clinician-rated measures.

Self-Report Measures

Outcome expectancy was evaluated as a fixed effect for treatment slope, posttreatment scores (intercept), and follow-up slope for the self-report measures (PSS, STAI, BDI, CGI-SR; Table 3). Outcome expectancy was associated with posttreatment scores on all measures (PSS: $\beta_{03} = -0.85$, $p = 0.002$; STAI: $\beta_{03} = -1.04$, $p < 0.001$, BDI: $\beta_{03} = -0.78$, $p = 0.004$, CGI-SR: $\beta_{03} = -0.10$, $p < 0.001$). Furthermore, outcome expectancy was associated with treatment slope for the PSS ($\beta_{13} = -0.42$, $p = 0.003$), the BDI ($\beta_{13} = -0.26$, $p = 0.049$), and the STAI ($\beta_{13} = -0.36$, $p < 0.001$), but not the CGI-SR ($\beta_{13} = -0.02$, $p = 0.150$). Outcome expectancy was unrelated to follow-up slope for all measures (PSS: $\beta_{23} = 0.03$, $p = 0.207$; STAI: $\beta_{23} = 0.01$, $p = 0.709$, BDI: $\beta_{23} = -0.02$, $p = 0.488$, CGI-SR: $\beta_{23} < 0.01$, $p = 0.876$). There was no support for differences in the effect of outcome expectancy across the treatment groups for all self-report measures.

Biological Measures

Outcome expectancy was unrelated to the percent change in startle from pretreatment to posttreatment after controlling for experimental conditions ($b = 5.57$, $SE = 8.40$, $p = 0.512$). Similarly, outcome expectancy was unrelated to cortisol reactivity at posttreatment after controlling for pretreatment reactivity and experimental conditions ($b = 0.01$, $SE = 0.03$, $p = 0.719$). There was no support for differences in the effect of outcome expectancy across the treatment groups for the biological.

Discussion

Prior reviews have called for evaluations of the association between outcome expectations and outcomes across a range of modalities². The results of the present study extend this research to self-report, clinician-rated, and biological measures of treatment response for PTSD. Consistent with the current literature, there was clear support for the association between outcome expectations and self-report measures and mixed support for clinician-rated measures. The present study represents the first investigation of the relation between outcome expectancy and biologically based measures of treatment outcome, and results suggest that biological measures of outcome were unrelated to outcome expectations.

There are several explanations for the discrepancy in findings across outcome measures. First, outcome expectancy may be more closely tied to changes in patient perceptions of symptoms as opposed to more objective measures of outcome. Second, outcome expectations are cognitive phenomena²¹ and may be more closely related to cognitive symptoms or perceptions of symptoms. The biological measures in the current study assessed behavioral reactions to trauma-related stimuli²⁸. Third, patient report of symptoms, on self-report forms or during interviews, reflects perceptions of symptoms as opposed to performance in a given situation. Therefore, outcome expectancy may correlate with perceived gains in treatment as opposed to biological or performance changes from treatment. Additional work is needed to confirm this hypothesis through the use of behavioral tasks, which have been recommended as a valid indicator of response to exposure based interventions²². Such work should also explore patient perceptions of physiological changes as they relate to outcome expectancy. It may be the case that expectations have minimal effect on biological indices of arousal, but reduce the negative perception of such arousal. Alternatively, constructs assessed with the same method typically have stronger associations than those obtained with different methods⁴⁷. The stronger relation among the self-reported outcome expectations and self-reported PTSD symptoms relative to symptoms assessed via interviews or biomarkers may be attributed to shared method variance. Finally, the current sample size may have been insufficient to detect the effect of outcome expectancy with biological measures. Meta-analyses have found the effect of outcome expectancy to be small among self-report measures and the association may be smaller still among biological measures².

Additional work is needed to determine mechanisms by which outcome expectancy is associated with self-reported treatment response. For exposure-based interventions, it is possible that positive outcome expectancies may facilitate engagement in exposures and thus more extinction. Prior work has suggested outcome expectancy improves homework compliance, which in turn improves treatment response⁹. Outcome expectancies have also been associated with an improved therapeutic alliance⁴⁸. Expectations may exert a positive influence on patient report of symptoms through an enhanced therapeutic alliance.

The correlational results of the present study and prior work should be confirmed with experimental manipulations of outcome expectations at the start of treatment. The association between expectations and outcomes is well established and experimental work is required to establish a direct causal link. A prior study experimentally manipulated young women's expectations for menstrual-related mood symptoms⁴⁹. Moods were assessed prospectively across one complete menstrual cycle and the women's prospectively monitored moods matched the experimental manipulation. This supports the hypothesis that experimental manipulations can effectively alter expectancies and subsequent emotional experiences. It further suggests that outcome expectancy in psychological treatment could be experimentally modified in order to investigate a direct impact on treatment response. Such experimental work would rule out the possibility of third variables that may better explain this relation.

The study had several limitations of note. First, the majority of the sample was male and all experienced combat-related trauma. Therefore, these results should be replicated in samples

of women and those receiving treatment for PTSD resulting from a broader range of traumatic events. Second, the current study used an abbreviated treatment through VRE. The contrast in findings between self-report, clinician-rated, and biological outcomes may be specific to this treatment model. Third, the present study included pharmacological experimental conditions as part of the primary study, which may have influenced the relation between outcome expectancy and treatment response. Fourth, the current study did not evaluate constructs closely related to outcome expectancy such as outcome credibility⁵⁰. Future work is needed to tease apart the roles of these constructs in treatment response. Lastly, there was considerably greater missing data for on biological measures than on other modalities. These findings should be replicated with larger samples that contain greater quantities of complete data.

Conclusion

The findings of the present study provide continued support for the role of outcome expectancy in self-reported treatment response. Clinicians are encouraged to evaluate the outcome expectations of their patients at the start of treatment and should work to increase them in order to enhance outcomes. Indeed, many manualized protocols, including PE⁵¹, emphasize education, rapport, and patient motivation. These strategies likely enhance outcome expectancy, improve engagement in treatment, and facilitate overall outcomes according to patient report. Additional research is needed to determine if outcome expectancies are important for biological indices of change.

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Table 1

Descriptive statistics for outcome measures.

	Pretreatment		Posttreatment		3-Month Follow-Up		6-Month Follow-Up		12-Month Follow-Up	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
<i>Clinician Rated</i>										
CAPS	87.92	17.37	70.32	24.83	63.43	28.68	60.98	30.30	56.96	31.10
CGI-Global	3.62	0.52	2.69	0.98	2.51	0.93	2.35	1.05	2.20	1.03
CGI-Severity	4.05	0.47	3.59	0.86	3.22	1.04	3.22	1.12	3.11	1.131
<i>Self-Report</i>										
PSS	33.52	9.51	26.85	12.8	24.95	13.31	25.57	14.53	23.85	14.73
STAI	50.45	10.02	47.58	12.41	46.94	12.31	50.13	15.54	50.83	15.52
BDI	28.37	11.2	22.82	12.87	21.34	13.22	20.35	14.55	20.41	13.7
CGI-SR	3.49	0.86	2.79	0.94	2.84	1.17	2.63	1.13	2.68	1.49
Outcome Expectancy	25.52	4.47	-	-	-	-	-	-	-	-
<i>Biological</i>										
Startle	-	-	28.21	185.06						
Cortisol Reactivity	0.03	1.08	-0.03	1.07						

Note: CAPS = Clinician Administered PTSD Scale, PSS = PTSD Symptom Scale, STAI = State-Trait Anxiety Inventory, BDI = Beck Depression Inventory. Range of outcome expectancy scores: 4 – 36.

Table 2

Multilevel models of outcome expectancy predicting treatment response for clinician-rated measures.

	CAPS		CGI-Global		CGI-Severity	
	Coefficient	SE	Coefficient	SE	Coefficient	SE
Posttreatment β_{00}	67.83**	4.81	2.52**	0.19	2.76**	0.18
Alprazolam β_{01}	4.00	6.09	0.38	0.23	0.14	0.23
Placebo β_{02}	-2.44	6.25	-0.02	0.25	-0.08	0.24
Outcome Expectancy β_{03}	-1.18*	0.55	-0.07**	0.02	-0.04*	0.02
Treatment Slope β_{10}	-13.68**	2.28	-0.78**	0.12	-0.57**	0.14
Alprazolam β_{11}	2.14	2.97	0.28	0.16	0.2	0.19
Placebo β_{12}	-0.09	3.08	0.1	0.17	0.1	0.19
Outcome Expectancy β_{13}	-0.43	0.28	-0.03	0.02	-0.02	0.02
Follow-up Slope β_{20}	-0.99**	0.36	-0.02	0.02	-0.01	0.02
Alprazolam β_{21}	0.30	0.60	-0.02	0.03	0.01	0.03
Alprazolam β_{22}	-0.12	0.54	-0.02	0.02	0.02	0.03
Outcome Expectancy β_{23}	< 0.01	0.05	< 0.01	< 0.01	0.01	< 0.01

Note: CAPS = Clinician Administered PTSD Scale.

*
 $p < 0.05$.**
 $p < 0.01$.

Table 3
Multilevel models of outcome expectancy predicting treatment response for self-report measures.

	PSS		STAI		BDI		CGI-SR	
	Coefficient	SE	Coefficient	SE	Coefficient	SE	Coefficient	SE
Posttreatment β_{00}	27.82**	2.36	49.34**	1.99	23.35**	2.28	2.76**	0.18
Alprazolam β_{01}	-1.23	2.96	-0.46	2.72	-0.49	2.96	0.14	0.23
Placebo β_{02}	-2.73	2.89	-0.89	2.70	-3.11	2.91	-0.08	0.24
Outcome Expectancy β_{03}	-85**	0.26	-1.04**	0.26	-0.78**	0.24	-0.10**	0.02
Treatment Slope β_{10}	-4.28**	1.15	-1.11	1.00	-4.03**	0.88	-0.57**	0.14
Alprazolam β_{11}	0.19	1.46	0.16	1.30	-0.11	1.36	0.20	0.19
Placebo β_{12}	-1.34	1.56	0.07	1.38	-0.13	1.38	0.10	0.19
Outcome Expectancy β_{13}	-0.42**	0.13	-0.36**	0.12	-0.26*	0.13	-0.02	0.02
Follow-up Slope β_{20}	-0.17	0.12	0.26	0.18	-0.18	0.18	-0.01	0.02
Alprazolam β_{21}	0.23	0.23	-0.24	0.23	0.38	0.27	0.01	0.03
Alprazolam β_{22}	0.10	0.23	-0.03	0.25	-0.04	0.27	0.02	0.03
Outcome Expectancy β_{23}	0.03	0.02	0.01	0.02	-0.02	0.02	< 0.01	< 0.01

Note: PSS = PTSD Symptom Scale. STAI = State-Trait Anxiety Inventory. BDI = Beck Depression Inventory.

* $p < 0.05$.

** $p < 0.01$.