



HHS Public Access

Author manuscript

J Trauma Stress. Author manuscript; available in PMC 2021 August 01.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Published in final edited form as:

J Trauma Stress. 2020 August ; 33(4): 488–499. doi:10.1002/jts.22570.

Prolonged Exposure and Sertraline Treatments for Posttraumatic Stress Disorder Also Improve Multiple Indicators of Social Functioning

Belinda Graham¹, Natalia M. Garcia¹, Hannah E. Bergman², Norah C. Feeny², Lori A. Zoellner¹

¹Department of Psychology, University of Washington, Seattle, Washington, USA

²Department of Psychological Sciences, Case Western Reserve University, Cleveland, Ohio, USA

Abstract

Trauma survivors with posttraumatic stress disorder (PTSD) frequently also suffer from difficulties in social functioning that range across emotional, cognitive, and environmental domains. A detailed evaluation of the differential impacts of effective PTSD treatments on social functioning is needed. Men and women ($N= 200$) with chronic PTSD received 10 weeks of prolonged exposure (PE) or sertraline in a randomized clinical trial and were followed for 24 months. A secondary data analysis examined changes in social functioning with regard to fear of intimacy; receipt of social support; and distress, avoidance, and negative cognitions in social situations. Effects were examined between treatments over time, controlling for baseline functioning. There were large, durable improvements across all indices. Compared to sertraline, PE was more efficient at reducing fear of intimacy and distress from negative social cognitions by posttreatment, $ds = 0.94-1.14$. Patients who received sertraline continued to improve over the course of follow-up, $ds = 0.54-1.17$. The differential speed of therapeutic effects may argue for more direct mechanisms in cognitive behavioral interventions versus cascade effects in serotonin reuptake inhibitors. Notably, both treatments produced substantial social benefits for trauma survivors with social functioning difficulties, and effect sizes were comparable to typical reductions in PTSD, depression, and anxiety.

Many individuals with posttraumatic stress disorder (PTSD) report difficulties with social interactions and relationships; as such, a more comprehensive understanding of the impact of PTSD treatments on social functioning is needed (Schnurr & Lunney, 2016). This is particularly important given that impaired social functioning not only impacts individuals with PTSD but also impacts partners, families, places of work, and communities (Olatunji et al., 2007). Despite strong evidence that trauma-focused psychotherapy and pharmacotherapy reduce PTSD, anxiety, and depressive symptoms (e.g., Cusack et al., 2016; Jonas et al.,

Correspondence concerning this article should be addressed to Belinda Graham, Oxford Centre for Anxiety Disorders and Trauma, University of Oxford, The Old Rectory, Paradise Square, Oxford, UK, OX1 1TW. belinda.graham@psy.ox.ac.uk. Belinda Graham is now at Oxford Centre for Anxiety Disorders and Trauma, Department of Experimental Psychology, University of Oxford, United Kingdom.

Natalia Garcia is now at VA Puget Sound Health Care System, Seattle, Washington, USA. Hannah Bergman is now at Montana VA Health Care System, Bozeman, Montana.

2013; Watts et al., 2013), social functioning outcomes that are central to well-being are less thoroughly studied, including factors such as an individual's openness to close relationships, general social support, and anxiety in social situations. Furthermore, treatment comparisons that specifically investigate social functioning outcomes are lacking. Thus, the current study examined this emerging area of focus in the PTSD literature and addressed the question of whether two established treatments for PTSD, prolonged exposure (PE) therapy and sertraline, produce clinically meaningful gains in social functioning.

Meta-analytic findings indicate that poorer social support is one of the most consistent predictors of PTSD (Brewin et al., 2000; Dinenberg et al., 2014; Guay et al., 2006; Ozer et al., 2008; Xue et al., 2015). As such, a lack of positive social relationships is not only a risk factor for PTSD but may increase vulnerability to PTSD symptom relapse if it is not improved in treatment. In a sample of combat veterans, higher PTSD symptom severity was associated with more severe deficits in social support prior to the start of treatment (Price et al., 2013). Indeed, PTSD has been associated with poorer quality of life across several domains, such as physical health, mental health, work, social and family relationships, and functioning at home (Olatunji et al., 2007). These impairments are often corroborated by friends and family, with some studies indicating that military veterans with PTSD and their romantic partners report significant relationship distress, intimacy difficulties, and more general relationship problems compared to veterans without PTSD and their partners (Monson et al., 2009; Riggs et al., 1998). Furthermore, PTSD symptoms are likely to further exacerbate these existing deficits in social functioning. Avoidance of trauma reminders may lead to loss of interest in activities, avoidance of social situations and activities, social isolation, and emotional numbing. In turn, these symptoms can reduce opportunities for interpersonal interactions, creating barriers to intimacy and compromising social functioning. Moreover, hyperarousal symptoms, including irritability, anger, and poor concentration, may contribute to social discord and interpersonal difficulties. Taken together, there is likely a vicious cycle wherein poor social functioning and symptoms of PTSD mutually reinforce each other.

Although social functioning has been examined separately in some psychotherapy and pharmacotherapy PTSD treatment studies, no study to our knowledge has included both types of treatment. In the current study, we addressed this gap in the literature by performing an in-depth evaluation of the impact of PE and sertraline on various facets of social functioning, both of which are empirically supported treatments for PTSD that produce, on average, moderate-to-large effects on PTSD symptoms (e.g., Jonas et al., 2013; Watts et al., 2013). However, the treatment modalities are quite different and may target associated deficits in social functioning through distinct mechanisms. Sertraline, a selective serotonin reuptake inhibitor (SSRI), involves taking a medication daily, whereas PE, a trauma-focused psychotherapy, involves working with a therapist to approach the trauma memory and trauma-related reminders via imaginal exposure and in vivo exposure. In PE, patients may be encouraged to approach social interactions and situations as part of their in vivo exposures, to the extent that they represent previously avoided trauma-related stimuli, and to examine negative social cognitions that emerge through imaginal exposure and processing of the trauma memory. According to emotional processing theory (Foa & Kozak, 1986), this would lead to a reduction in PTSD symptoms and, potentially, improvement in social

functioning in part due to disconfirmation of maladaptive beliefs about social situations. In comparison, individuals who receive sertraline may begin to feel more inclined to engage in pleasurable activities, decrease isolation, and increase social interactions as their PTSD symptoms are reduced. According to behavioral activation theory (e.g., Jacobson et al., 2001), this would lead to improvements in mood and symptoms more gradually. Thus, a side-by-side evaluation of these treatments may indicate distinct mechanisms of change and even provide theoretical support for drivers of improvements in social functioning.

Randomized controlled trials (RCTs) for PTSD psychotherapies, specifically PE, that have examined social functioning have reported significant improvements after active treatment, which were maintained at follow-up evaluations. For example, individuals who received PE, cognitive restructuring (CR), or a combination of the two reported significantly better social functioning at 3-month follow-up, with large effect sizes ($ds = 1.60\text{--}2.50$) compared to individuals in a relaxation-only condition (Marks et al., 1998). Similar results were found among a sample of female assault survivors, who reported significantly higher levels of social functioning after receiving PE alone or in combination with stress inoculation training (Foa et al., 1999) and PE alone or combined with CR (Foa et al., 2005; Rauch et al., 2009) when compared to individuals in a waitlist condition. In two studies that reported long-term outcomes among female sexual assault survivors (e.g., 5–10 years posttreatment), individuals who received PE or cognitive processing therapy (CPT) reported they maintained gains in social and/or work-related functioning (Larsen et al., 2019; Wachen et al., 2014). Thus, there is strong evidence that psychotherapies for PTSD, such as PE, improve social functioning overall, but detailed evaluation of specific components is lacking.

Few RCTs have examined the effect of PTSD pharmacotherapy treatments on social functioning, either as a primary or secondary outcome, after a 10- or 12-week trial of the medication. In a meta-analysis of 35 short-term RCTs for PTSD pharmacotherapies (medication vs. placebo), only four included studies reported outcomes related to social functioning (Stein et al., 2006). Of these four studies, three found that social functioning was significantly improved by an SSRI (fluoxetine or paroxetine) compared to the placebo (Connor et al., 1999; Marshall et al., 2001; Tucker et al., 2001), whereas the other study found no significant difference between pharmacotherapy and placebo (Butterfield et al., 2001). In other PTSD pharmacotherapy RCTs, adult outpatients with PTSD who received fluoxetine (Malik et al., 1999) or paroxetine (Marshall et al., 2006) reported trend-level or significantly improved social functioning at posttreatment compared to individuals in the respective placebo groups. No effect sizes were reported in any of the PTSD pharmacotherapy studies. Overall, the results from the emerging PTSD psychotherapy and pharmacotherapy literature on social functioning indicate global improvements in social functioning compared to waitlist conditions or placebo.

There is a need to examine social functioning as a multifaceted construct. However, in all of the previously mentioned literature, social functioning is defined quite broadly as social adjustment (Foa et al., 1999; Larsen et al., 2019; Marks et al., 1998; Wachen et al., 2014), social disability (Connor et al., 1999), social life (Tucker et al., 2001), and impairment in social functioning (Butterfield et al., 2001; Marshall et al., 2001, 2006). Given that general social functioning can be viewed as a broader measure of overall functioning (Rauch et al.,

2009), a more nuanced approach needs to be taken when considering how to define social functioning within the context of PTSD. As PTSD is associated with social withdrawal and overly negative beliefs about the self and others, it is important to specifically examine avoidance, cognitions, and distress across a variety of social situations that may interfere with day-to-day functioning (Olatunji et al., 2007). For many individuals, trauma-related social functioning impairments also extend to difficulties with trust and closeness in intimate relationships (Monson et al., 2009). In addition, given that perceived social support greatly influences posttrauma recovery (Brewin et al., 2000; Dinenberg et al., 2014; Guay et al., 2006; Ozer et al., 2003; Xue et al., 2015), it is important to assess individuals' perceptions of positive social support in their recovery environments. Although prior studies have examined social functioning as an outcome, the scope of existing literature does not match the clear clinical complexity of these issues.

We examined social functioning within and across PE and sertraline treatments for individuals with chronic PTSD. Extending to 24-months posttreatment, we investigated the differential impact of these treatments across multiple measures of interpersonal functioning, including specific situations and broader interactional aspects. Based on evidence of improved social functioning in general with PTSD treatment (e.g., Foa et al., 2005; Stein et al., 2006), we hypothesized that there would be improvements across all specific measures of social functioning, with gains achieved in treatment and maintained to 24-month follow-up. In particular, we expected improvements among individuals with poorer social functioning prior to treatment, as we did not expect improvements among patients who were not initially experiencing difficulties. We hypothesized that these effects would be observed among participants receiving both PE and sertraline.

Method

Participants

Men and women ($N= 200$) participated in a multi-site, randomized treatment trial and received PE or sertraline for chronic PTSD. Eligible participants were between 18 and 65 years of age and had a current primary diagnosis of chronic PTSD per criteria in the fourth edition, text revision of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)*; American Psychiatric Association [APA], 2000). Participant characteristics are shown in Table 1. Additional trial details are reported in the main outcome paper (Zoellner et al., 2019).

Procedure

The present study received appropriate ethical approvals from the institutional review boards at the University of Washington (04-0541-D) and Case Western Reserve University/University Hospitals of Cleveland (08-03-47). Participants were screened on the phone via a semistructured interview to determine initial eligibility. Potentially eligible participants were invited for an intake interview, which was conducted by trained independent evaluators and included informed consent and an assessment of demographic characteristics and diagnostic status. If an individual was interested and eligible, they were invited for a randomization visit, during which they completed self-report assessments, including all measures of social

functioning. Participants then received up to 10 weeks of PE or sertraline. Interview and self-report measures were repeated at posttreatment, 3-month, 6-month, 12-month, and 24-month follow-ups. Independent evaluators remained blinded to treatment allocation throughout active treatment and follow-up.

Treatments

Prolonged Exposure.: The PE treatment condition (Foa et al., 2007) consisted of 10 weekly sessions (90–120 min) delivered by masters- or PhD-level clinicians who had received training in PE. The sessions included psychoeducation, a breathing technique for managing anxiety, a gradual approach to the trauma memory through imaginal exposure, gradual in vivo exposure to previously avoided trauma reminders, processing of trauma-related thoughts and feelings, and between-session homework. Sessions were recorded and clinicians received ongoing supervision. An external rater assessed fidelity for 10% of the sample and indicated that PE providers completed 90% of essential components with no protocol violations. Therapist competence was rated on a scale from 1 (*inadequate*) to 3 (*adequate or better*), and average competence was high across the therapists ($M = 2.73$, $SD = 0.32$).

Sertraline.: The sertraline condition consisted of 10 weekly sessions (up to 30 min, with the first session up to 45 min) with board-certified psychiatrists who followed a standard titration algorithm and manual (Brady et al., 2000; Marshall et al., 2001). The sessions included ongoing assessments of symptoms and side effects, adjustment of medication dose, and general support. Sertraline dose was titrated upward as tolerated and clinically indicated from 12.5 mg/day to 300 mg/day. The average final dose was 115 mg/day ($SD = 78.0$), and continuation was provided free of cost over the follow-up period. An external rater rated fidelity for 10% of the sample, indicating that providers completed 96% of essential components with no protocol violations.

Measures

PTSD Diagnosis and Symptom Severity—The PTSD Symptom Scale—Interview (PSS-I; Foa et al., 1993) is a 17-item, clinician-rated measure used to assess *DSM-IV-TR* criteria for PTSD (APA, 2000). For the present study, the PSS-I was used to determine current PTSD diagnosis (i.e., *DSM-IV* PTSD Criteria B–D) and symptom severity. Items are rated on a 4-point Likert scale ranging from 0 (*not at all or only one time*) to 3 (*5 or more times per week/almost always*), based on symptom frequency and/or severity over the past 2 weeks; total scores range from 0 to 51, with higher scores indicating a higher level PTSD severity. The PSS-I has demonstrated good convergent validity ($r = .93$) and interrater reliability ($r = .95$; Foa et al., 1997; Foa & Tolin, 2000). In the current sample, the total score internal consistency was acceptable, Cronbach's $\alpha = .65$. Ten percent of cases were rerated for diagnostic reliability, which was excellent, intraclass coefficient (ICC) = .99.

Psychiatric Disorders—The Structured Clinical Interview for *DSM-IV* (SCID-IV; First et al., 2002) is a semistructured, clinician-rated interview used to assess whether *DSM-IV* diagnostic criteria are present, exist at subthreshold levels, or are absent for Axis I disorders (APA, 2000). The SCID-IV was used to assess inclusion and exclusion criteria. This

measure has demonstrated good interrater reliability and validity (e.g., Lobbstaal et al., 2011). In the current study, 10% of cases were rerated, and interrater reliability was acceptable, $\kappa = 0.80$.

Fear of Intimacy—The Fear of Intimacy Scale (FIS; Descutner & Thelen, 1991) was included as an indicator of in-depth interpersonal functioning within close relationships. It is a 35-item, self-report measure used to assess an individual's capacity to exchange thoughts and emotions of personal significance with another individual who is highly valued (e.g., "I would feel comfortable expressing my true feelings to them"). Items are rated on a 5-point Likert scale that ranges from 0 (*not at all characteristic of me*) to 4 (*extremely characteristic of me*); total scores range from 0 to 140, with higher scores indicating more fear of intimacy. The measure has demonstrated high test-retest reliability ($r = .89$; Descutner & Thelen, 1991) as well as high construct validity, evidenced by strong associations with related constructs (e.g., loneliness, $rs = .30-.54$), and good discriminant validity with trait anxiety in an adolescent sample (Sherman & Thelen, 1996). In the current sample, internal consistency was good, Cronbach's $\alpha = .89$.

Social Interactions—The Inventory of Social Interactions (ISI; Amir et al., 2003, 2005) was included as a broad indicator of anxiety across 13 social situations that may interfere with day-to-day functioning in individuals with PTSD. The ISI is a self-report measure with three subscales: Negative Cognitions (20 items), Avoidance (13 items), and Distress (13 items). The Negative Cognitions subscale is used to assess distress from negative cognitions related to social situations (e.g., "I'm making a fool out of myself"), the Avoidance subscale assesses avoidance of 13 different social situations (e.g., parties, public speaking, participation in meetings/classes), and the Distress subscale assesses distress in the same 13 social situations. Items are rated with respect to how they are generally experienced, using a 5-point Likert scale that ranges from 0 (*not distressed/never avoid*) to 4 (*extremely distressed/always avoid*). The ISI has demonstrated good divergent validity such that it has been shown to discriminate between socially anxious and non-socially anxious individuals (Amir et al., 2003). Total subscale scores range from 0 to 80, for Negative Cognitions; 0 to 52, for Avoidance; and 0 to 52, for Distress, with higher scores reflecting higher levels of severity. In the current sample, internal consistency was good across subscales, Cronbach's $\alpha = .89-.96$.

Social Support—The Inventory of Socially Supportive Behaviors (ISSB; Barrera et al., 1981) was included to measure the extent to which individuals receive social support, which is believed to buffer the risk for PTSD. The ISSB is a 40-item, self-report measure that is used to assess the perceived frequency of receiving supportive actions (e.g., "Talked with you about some interests of yours"). Items are rated on a 5-point Likert scale ranging from 1 (*not at all*) to 5 (*about every day*) with respect to frequency during the past 2 weeks; total scores range from 40 to 200, with higher scores indicating higher levels of perceived social support. The measure has demonstrated good test-retest reliability ($r = .88$) and adequate construct validity ($rs = .32-.40$; Barrera et al., 1981). In the current study, internal consistency was good, Cronbach's $\alpha = .96$.

Data Analysis

The present study is a secondary data analysis from a larger PTSD treatment trial (Zoellner et al., 2019). Using multilevel modeling, predictor variables were baseline scores on measures of social functioning (FIS, ISSB, and ISI), treatment condition (PE, sertraline) and time (posttreatment, 3-month, 6-month, 12-month, or 24-month follow-up), as well as two interaction terms: (a) Baseline Social Functioning x Time and (b) Baseline Social Functioning x Treatment x Time. We accounted for baseline impairment in the models to evaluate the effect among participants for whom a potential change in social functioning was most relevant (i.e., those with deficits at baseline). Dependent variables were scores on measures of social functioning (FIS, ISSB, and ISI). Site was included as a covariate, and missing data were handled using restricted maximum likelihood estimation. The random intercept model that covaried for site was the best fit for the data across measures. Significant interactions were further probed in the acute phase (i.e., pretreatment to 3-month follow-up) and maintenance phase (i.e., 3-month follow-up to 24-month follow-up) separately, reflecting the best fit for the data. Effect sizes were calculated following Feingold's (2009) guidelines. Analyses were conducted using SPSS (Version 22).

Results

Fear of Intimacy

When examining FIS scores, on which higher scores reflect more fear of intimacy, there were main effects of baseline fear of intimacy, $F(1, 211.01) = 339.42, p < .001$; and time, $F(5, 629.35) = 6.45, p < .001$, which were modified by both a baseline FIS x Time interaction, $F(5, 631.15) = 15.30, p < .001$; and a baseline FIS x Treatment x Time interaction, $F(6, 482.30) = 2.16, p = .046$ (Figure 1, Panel A).

To examine the three-way interaction, we examined the acute (i.e., pretreatment to 3-month follow-up) and maintenance phases (i.e., 3-month follow-up to 24-month follow-up) separately. During the acute phase, the baseline Fear of Intimacy x Treatment x Time interaction was present, $F(3, 298.01) = 4.54, p = .004$. Specifically, for participants who received PE, baseline fear of intimacy interacted with time, $F(2, 182.49) = 21.43, p < .001$, such that those with higher FIS scores showed better improvement over time, $M + 1 SD: d = 1.14; M - 1 SD: d = 0.14$. Similarly, for participants who received sertraline, there was an interaction between baseline fear of intimacy and time, $F(2, 129.29) = 5.14, p = .007$, that showed the same pattern of improvement for individuals with higher FIS scores, $M + 1 SD: d = 0.50; M - 1 SD: d = 0.17$.

For the maintenance phase (i.e., from 3-month follow-up to 24-month follow-up), the three-way interaction persisted, $F(4, 226.58) = 2.66, p = .034$. For participants in the sertraline condition in particular, there was a baseline Fear of Intimacy x Time interaction, $F(3, 115.35) = 3.51, p = .018$, such that those with higher baseline FIS scores continued to show improvement over time, $M + 1 SD: d = 0.54; M - 1 SD: d = 0.10$; however, this was not the case for participants in the PE condition, $p = .453$. In summary, higher baseline ratings of fear of intimacy were related to more improvement in fear of intimacy over time. For

individuals with higher baseline levels of fear of intimacy, PE produced large acute gains, but sertraline showed more consistent improvement over the follow-up period.

Negative Cognitions in Social Interactions

When examining negative cognitions during social interactions as measured using the Negative Cognitions subscale of the ISI, where higher scores reflect higher levels of negative cognitions in social situations, there was a main effect of baseline severity, $F(1, 230.87) = 206.69, p < .001$, which was modified by a baseline Negative Cognitions x Time interaction, $F(5, 392.21) = 8.32, p < .001$, and a baseline Negative Cognitions x Treatment x Time Interaction, $F(6, 380.69) = 2.66, p = .015$.

To investigate the three-way interaction, we examined the acute (i.e., pretreatment to 3-month follow-up) and maintenance phase (i.e., from 3-month follow-up to 24-month follow-up) separately. During the acute phase, the baseline Negative Cognitions x Treatment x Time interaction remained, $F(3, 298.02) = 3.27, p = .022$. For individuals who received PE, baseline levels of negative cognitions interacted with time, $F(2, 155.89) = 16.69, p < .001$, such that those with higher ratings of negative cognitions showed more improvement over time, $M + 1 SD: d = 0.94; M - 1 SD: d = 0.18$; however, this was not the case for those in the sertraline condition, $p = .167$.

For the maintenance phase, the three-way interaction also persisted, $F(4, 131.89) = 3.35, p = .012$. For individuals who received sertraline, there was a baseline Fear of Intimacy x Time interaction, $F(3, 51.40) = 3.40, p = .025$, such that those with higher levels of baseline negative cognitions continued to show improvement over time, $M + 1 SD: d = 1.17; M - 1 SD: d = 0.46$; but this was not the case for participants who received PE, $p = .745$. In summary, higher baseline ratings of negative cognitions were related to more improvement in negative cognitions over time. For individuals with higher levels of negative cognitions in social interactions, PE produced large acute gains, but sertraline showed consistent improvement over the follow-up period (Figure 1, Panel B).

Avoidance of Social Interactions

When examining avoidance of specific social interactions as measured using the Avoidance subscale of the ISI, where higher scores reflect higher levels of avoidance, there was a main effect of baseline Avoidance, $F(1, 239.33) = 288.92, p < .001$, which was modified by a baseline Avoidance x Time interaction, $F(5, 449.71) = 5.44, p < .001$. The baseline Avoidance x Treatment x Time interaction did not reach significance, $p = .063$.

To examine the two-way interaction, we examined the acute (i.e., pretreatment to 3-month follow-up) and maintenance phase (i.e., from 3-month follow-up to 24-month follow-up) separately. During the acute phase, the baseline Avoidance x Time interaction was present, $F(2, 275.25) = 8.05, p < .001$. For individuals with higher baseline ratings of avoidance, there was a large decrease in avoidance during the acute phase, $M + 1 SD: d = 0.72; M - 1 SD: d = 0.13$. In the maintenance phase, the two-way interaction was lost, $p = .276$, and only a main effect of baseline avoidance remained $F(1, 95.74) = 69.42, p < .001$. In summary, higher baseline avoidance was related to larger improvements in avoidance in the acute phase, which were maintained through follow-up (Figure 2, Panel A).

Distress During Social Interactions

When we examined distress reported during specific social interactions, as measured using the ISI Distress subscale, where higher scores reflect higher levels of distress in social interactions, there were main effects of baseline Distress, $F(1, 216.21) = 284.79, p < .001$, and time, $F(5, 632.53) = 2.59, p = .025$, that were modified by a baseline Distress x Time interaction, $F(5, 636.03) = 30.81, p < .001$.

To examine the two-way interaction, we assessed the acute (i.e., pretreatment to 3-month follow-up) and maintenance phase (i.e., 3-month follow-up to 24-month follow-up) separately. During the acute phase, the baseline Distress x Time interaction was present, $F(2, 326.57) = 61.16, p < .001$. For participants with higher baseline ratings of distress, there was a large decrease in symptoms during the acute phase, $M + 1 SD: d = 1.03; M - 1 SD: d = 0.06$. During the maintenance phase, the two-way interaction was lost, $p = .085$, and only a main effect of baseline distress remained $F(1, 129.05) = 89.07, p < .001$. In summary, higher initial baseline levels of distress in social interactions were related to more improvement in distress in social interactions in the acute phase (Figure 2, Panel B).

Socially Supportive Behaviors

When examining the receipt of socially supportive behaviors, as measured using the ISSB, where higher scores reflect higher levels of social support involvement from others, there were main effects of baseline severity, $F(1, 202.94) = 223.09, p < .001$, and time, $F(5, 645.21) = 12.11, p < .001$, that were modified by a baseline Social Support x Time interaction, $F(5, 641.46) = 14.73, p < .001$, as well as a baseline Social Support x Treatment x Time interaction, $F(6, 482.04) = 2.47, p = .023$.

To examine the three-way interaction, we examined the acute (i.e., pretreatment to 3-month follow-up) and maintenance phase (i.e., 3-month follow-up to 24-month follow-up) separately. During the acute phase, the three-way interaction was not significant, but the baseline Support x Time interaction remained, $F(2, 323.50) = 29.91, p < .001$, such that individuals with higher ratings of baseline support showed a decrease in support, $M + 1 SD: d = 0.49$, whereas those with lower baseline ratings of support showed an increase in support, $M - 1 SD: d = 0.39$. When we examined the maintenance phase, the two-way interaction was lost, $p = .538$, and only a main effect of baseline support remained $F(1, 130.98) = 61.16, p < .001$. In summary, individuals with both higher and lower baseline ratings of social support involvement from others moved toward a more balanced level of social support during the acute phase (Figure 2, Panel C).

Discussion

Although it is well documented that poor social functioning constitutes a critical posttrauma risk factor, is often exacerbated by PTSD symptoms, and contributes to significant public and personal costs (Olatunji et al., 2007; van Minnen et al., 2015), such deficits are rarely targeted directly in PTSD treatments. As such, reduction in PTSD symptoms may be a necessary but not sufficient way of conceptualizing posttrauma recovery. The results of the current study provide evidence that clinicians providing PE or sertraline for PTSD can

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

expect substantial improvements across various indices of social functioning, stretching beyond symptom reduction to concrete and highly protective social benefits. In the current study, patients who initially presented with significant social functioning deficits made large improvements during treatment that were maintained over the 24-month follow-up period. Large effects were observed in both treatment modalities for individuals with higher baseline social functioning deficits, although PE appeared to work faster in decreasing fear of intimacy and distressing negative thoughts about social situations, and sertraline continued to improve social functioning deficits over time.

The comparison of psychotherapy and pharmacotherapy treatment effects may suggest distinct processes of change, though both treatments appear to be effective in addressing social functioning difficulties that occur alongside PTSD. Consistent with research suggesting that social support is a pathway through which exposure therapy reduces PTSD symptoms (Bourassa et al., 2020), it is possible that teaching patients to approach feared but objectively safe situations leads to increased participation in social life, which in turn contributes to PTSD symptom reduction. In a sample of combat veterans who received PE, a higher level of social support over the course of treatment was related to larger reductions in PTSD symptom severity (Price et al., 2018). In the current study, PE was more efficient at reducing fear of intimacy and negative cognitions in social situations. It is possible that teaching patients that they can experience, express, and tolerate a full range of trauma-related emotions with their therapist (Foa et al., 2007) creates a model for emotional engagement and interpersonal connection that increases capacity for emotional intimacy in other relationships, especially among individuals who begin treatment with difficulties in this area. It is also possible that tackling negative trauma-related cognitions during the imaginal exposure and processing components of PE provides opportunities for patients to engage in cognitive shifts that are critical for driving PTSD symptom reduction (e.g., Cooper et al., 2017; Zalta et al., 2014). The more efficient reduction of negative social cognitions (e.g., "I'm making a fool out of myself" or "people are criticizing me") during PE compared with sertraline suggests that developing a more realistic perspective on trauma-related cognitions may also generalize to developing alternatives to distressing negative social cognitions.

Among individuals who received sertraline, improvements in social functioning during the acute phase of treatment were slower but continued during the subsequent 2-year follow-up period, underscoring the importance of long-term assessments in research and suggesting mechanisms of change that are distinct from psychotherapy. This is consistent with the ongoing benefits of continuing SSRIs for anxiety disorders beyond an initial acute treatment phase (see Ballenger, 2004; Stein et al., 2006). This slower improvement may reflect a cascade of effects initiated by sertraline, such that PTSD symptoms reduce first and create the conditions necessary for subsequent improvements in social functioning. This may include increased inclination toward pleasurable activities, reduced isolation, and increased opportunities for positive social interactions. According to Harmer and colleagues (2009), the ongoing effects from antidepressants on depression symptoms are not due to a delay in neuropharmacological actions but rather due to a delay between the effects on the processing of emotions and subsequent impact on mood. Similarly, when treated with sertraline, a reduction in PTSD symptoms may lead to increased behavioral activation, which in turn

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

may include approaching previously avoided stimuli, including social triggers, and ultimately help to gradually challenge previously held negative beliefs surrounding particular social situations.

Impairments in social functioning have long been known to be associated with PTSD, and recent changes to diagnostic criteria in the 11th revision of the *International Classification of Diseases* (World Health Organization, 2018) now formally spotlight disturbances in relationships within the classification of PTSD. This diagnostic emphasis on social factors means it is critical to understand the ability of existing PTSD treatments to resolve interpersonal deficits and evaluate whether a more explicit focus on social functioning is needed. Although the present study had several strengths, some limitations should be kept in mind. Treatment responders continued sertraline over the follow-up period, making it difficult to know whether the trajectory of social functioning gains would be maintained if they went off medication or to disentangle the effects of ongoing, but infrequent, interactions with their provider. There were no placebo or waitlist control conditions, as unblinded pharmacological treatment reflects standard clinical practice. The lack of a waitlist control condition also means we cannot rule out the possibility that the observed social functioning gains were a function of regression to the mean or improvement over time; however, the average time since trauma for participants in this study was over 12 years, challenging the likelihood that social functioning deficits would have improved in the absence of intervention. Moreover, prior studies have reported substantial improvements in social functioning in active treatment compared with waitlist conditions (Foa et al., 2005; Monson et al., 2006, 2012; Rauch et al., 2009). The measures used in the present study are well-validated and pragmatic indicators of severity. However, psychometric properties of social functioning measures lack precise clinical cutoffs, challenging our ability to interpret changes compared with population norms, and the study design did not include third-party or behavioral observations. However, this range of measurement tools provided a broad assessment of social functioning that has not been included in PTSD treatment studies to date (e.g., Monson et al., 2012). Finally, diagnostic instruments used to evaluate criteria from the fifth edition of the *DSM* (*DSM-5*) were not yet available when the study was carried out, but it is likely that a high proportion of participants also met *DSM-5* criteria.

Clinicians can expect that PE and sertraline will improve multiple aspects of social functioning, especially among individuals who begin treatment with poorer social functioning, with gains achieved during treatment and maintained up to at least 2 years. This speaks to the broader potential for the cost-effectiveness of these treatments beyond reducing PTSD symptoms (e.g., Le et al., 2014; see van Minnen et al., 2015). Although it is possible that additional interventions could offer further enhancements among individuals with lingering problems, social impairments that occur alongside chronic PTSD were addressed in the present study. Future research could utilize a time-lag design to address questions about the directionality of effects between symptom reduction and improvements in social functioning and explore specific hypothesized mechanisms of change. Additional studies could also explore these effects with regard to other evidence-based PTSD treatments, among different cultural groups, and across the lifespan. Researchers and clinicians who develop and deliver PTSD treatments have a shared goal to help trauma survivors with PTSD get back to their lives, and the field increasingly acknowledges that true recovery

extends beyond reducing PTSD symptoms to increasing life satisfaction more broadly. The current research provides strong support that PTSD treatments have positive impacts well beyond symptom reduction and suggests that patients who begin treatment with broad and possibly complex difficulties in multiple life domains can reasonably hope for better outcomes.

Acknowledgments

This study was funded by grants to Dr. Zoellner and Dr. Feeny from the National Institute of Mental Health (R01 MH066347, R01 MH066348) and the William T. Dahms, M.D., Clinical Research Unit, funded under the Cleveland Clinical and Translational Science Award (UL1 RR024989). This manuscript is also the result of work supported by resources from the Wellcome Trust [205156], the U.S. Department of Veterans Affairs (VA) Office of Academic Affiliations, Advanced Fellowship Program in Mental Illness Research and Treatment, and the VA Puget Sound Health Care System (Seattle, Washington).

References

American Psychiatric Association. (2000). Diagnostic and statistical manual of mental disorders (4th ed., text revision). American Psychiatric Association.

Amir N, Beard C, & Bower E (2005). Interpretation bias and social anxiety. *Cognitive Therapy and Research*, 29(4), 433–443. 10.1007/s10608-005-2834-5

Amir N, Bower E, Briks J, & Freshman M (2003). Implicit memory for negative and positive social information in individuals with and without social anxiety. *Cognition & Emotion*, 17(4), 567–583. 10.1080/0269993030230 [PubMed: 29715733]

Ballenger JC (2004). Remission rates in patients with anxiety disorders treated with paroxetine. *The Journal of Clinical Psychiatry*, 65(12), 1696–1707. 10.4088/jcp.v65n1216 [PubMed: 15641876]

Barrera M, Sandler IN, & Ramsay TB (1981). Preliminary development of a scale of social support: Studies on college students. *American Journal of Community Psychology*, 9(4), 435–447. 10.1007/BF00918174

Bourassa KJ, Smolenski DJ, Edwards-Stewart A, Campbell SB, Reger GM, & Norr AM (2020). The impact of prolonged exposure therapy on social support and PTSD symptoms. *Journal of Affective Disorders*, 260, 410–417. 10.1016/j.jad.2019.09.036 [PubMed: 31539674]

Brady K, Pearlstein T, Asnis GM, Baker D, Rothbaum B, Sikes CR, & Farfel GM (2000). Efficacy and safety of sertraline treatment of posttraumatic stress disorder: A randomized controlled trial. *JAMA*, 283(14), 1837–1844. 10.1001/jama.283.14.1837 [PubMed: 10770145]

Brewin CR, Andrews B, & Valentine JD (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology*, 68(5), 748–766. 10.1037/0022-006X.68.5.748 [PubMed: 11068961]

Butterfield MI, Becker ME, Connor KM, Sutherland S, Churchill LE, & Davidson JRT (2001). Olanzapine in the treatment of post-traumatic stress disorder: A pilot study. *International Clinical Psychopharmacology*, 16(4), 197–203. https://journals.lww.com/intclinpsychopharm/Abstract/2001/07000/Olanzapine_in_the_treatment_of_post_traumatic.3.aspx [PubMed: 11459333]

Cooper AA, Zoellner LA, Roy-Byrne P, Mavissakalian MR, & Feeny NC (2017). Do changes in trauma-related beliefs predict PTSD symptom improvement in prolonged exposure and sertraline? *Journal of Consulting and Clinical Psychology*, 85, 873–882. 10.1037/ccp0000220 [PubMed: 28504542]

Connor KM, Sutherland SM, Tupler LA, Malik ML, & Davidson JR (1999). Fluoxetine in post-traumatic stress disorder. Randomised, double-blind study. *British Journal of Psychiatry*, 175(9), 17–22. 10.1192/bj.p.175.1.17

Cusack K, Jonas DE, Forneris CA, Wines C, Sonis J, Middleton JC, Feltner C, Brownley KA, Olmsted KR, Greenblatt A, Weil A, & Gaynes BN (2016). Psychological treatments for adults with posttraumatic stress disorder: A systematic review and meta-analysis. *Clinical Psychology Review*, 43, 128–141. 10.1016/j.cpr.2015.10.003 [PubMed: 26574151]

Descutner CJ, & Thelen MH (1991). Development and validation of a Fear of Intimacy Scale. *Psychological Assessment: A Journal of Consulting and Clinical Psychology*, 3(2), 218–225. 10.1037/1040-3590.3.2.218

Dinenberg RE, McCaslin SE, Bates MN, & Cohen BE (2014). Social support may protect against development of posttraumatic stress disorder: Findings from the Heart and Soul Study. *American Journal of Health Promotion*, 28, 294–297. 10.4278/ajhp.121023-QUAN-511 [PubMed: 23941102]

Feingold A (2009). Effect sizes for growth-modeling analysis for controlled clinical trials in the same metric as for classical analysis. *Psychological Methods*, 14(5), 43–53. 10.1037/a0014699 [PubMed: 19271847]

First MB, Spitzer RL, Gibbon M, & Williams JBW (2002). Structured clinical interview for DSM-IV-TR Axis I disorders, research version, patient edition. (SCID-I/P) Biometrics Research, New York State Psychiatric Institute.

Foa EB, Cashman L, Jaycox L, & Perry K (1997). The validation of a self-report measure of posttraumatic stress disorder: The Posttraumatic Diagnostic Scale. *Psychological Assessment*, 9(4), 445–451. 10.1037/1040-3590.9.4.445

Foa EB, Dancu CV, Hembree EA, Jaycox LH, Meadows EA, & Street GP (1999). A comparison of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault victims. *Journal of Consulting and Clinical Psychology*, 67(2), 194–200. 10.1037/0022-006X.67.2.194 [PubMed: 10224729]

Foa EB, Hembree EA, Cahill SP, Rauch SA, Riggs DS, Feeny NC, & Yadin E (2005). Randomized trial of prolonged exposure for posttraumatic stress disorder with and without cognitive restructuring: Outcome at academic and community clinics. *Journal of Consulting and Clinical Psychology*, 73(5), 953–964. 10.1037/0022-006X.73.5.953 [PubMed: 16287395]

Foa EB, Hembree EA, & Rothbaum BO (2007). Prolonged Exposure (PE) therapy for PTSD: Emotional processing of traumatic experiences, therapist guide. Oxford University Press.

Foa EB, & Kozak MJ (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, 99(1), 20–35. 10.1037/0033-2909.99.1.20 [PubMed: 2871574]

Foa EB, Riggs DS, Dancu CV, & Rothbaum BO (1993). Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. *Journal of Traumatic Stress*, 6(4), 459–473. 10.1002/jts.2490060405

Foa EB, & Tolin DF (2000). Comparison of the PTSD Symptom Scale–Interview version and the Clinician-Administered PTSD Scale. *Journal of Traumatic Stress*, 13(2), 181–191. 10.1023/A:1007781909213 [PubMed: 10838669]

Guay S, Billette V, & Marchand A (2006). Exploring the links between posttraumatic stress disorder and social support: Processes and potential research avenues. *Journal of Traumatic Stress*, 19(3), 327–338. 10.1002/jts.20124 [PubMed: 16788995]

Harmer CJ, Goodwin GM, & Cowen PJ (2009). Why do antidepressants take so long to work? A cognitive neuropsychological model of antidepressant drug action. *British Journal of Psychiatry*, 195(2), 102–108. 10.1192/bjp.bp.108.051193

Jacobson NS, Martell CR, & Dimidjian S (2001). Behavioral activation treatment for depression: Returning to contextual roots. *Clinical Psychology: Science and Practice*, 8(3), 255–270. 10.1093/clipsy.8.3.255

Jonas DE, Cusack K, Forneris CA, Wilkins TM, Sonis J, Middleton JC, Feltner C, Cavanaugh MD, Brownley KA, Olmsted KR, Greenblatt A, Weil A, & Gaynes BN (2013). Psychological and pharmacological treatments for adults with posttraumatic stress disorder (PTSD). Agency for Healthcare Research and Quality. <https://europepmc.org/article/med/23658937>

Larsen SE, Fleming CJ, & Resick PA (2019). Residual symptoms following empirically supported treatment for PTSD. *Psychological Trauma: Theory, Research, Practice, and Policy*, 11(2), 207–215. 10.1037/tra0000384

Le QA, Doctor JN, Zoellner LA, & Feeny NC (2014). Cost-effectiveness of prolonged exposure therapy versus pharmacotherapy and treatment choice in posttraumatic stress disorder (the Optimizing PTSD Treatment Trial): A doubly randomized preference trial. *Journal of Clinical Psychiatry*, 75(3), 222–230. 10.4088/JCP.13m08719

Lobbestael J, Leurgans M, & Arntz A (2011). Interrater reliability of the structured clinical interview for DSM-IV Axis I disorders (SCID I) and Axis II disorders (SCID II). *Clinical Psychology & Psychotherapy*, 18(1), 75–79. 10.1002/cpp.693 [PubMed: 20309842]

Malik ML, Connor KM, Sutherland SM, Smith RD, Davison RM, & Davidson JR (1999). Quality of life and posttraumatic stress disorder: A pilot study assessing changes in SF-36 scores before and after treatment in a placebo-controlled trial of fluoxetine. *Journal of Traumatic Stress*, 12(2), 387–393. 10.1023/a:1024745030140 [PubMed: 10378176]

Marks I, Lovell K, Noshirvani H, Livanou M, & Thrasher S (1998). Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring: A controlled study. *Archives of General Psychiatry*, 55(4), 317–325. 10.1001/archpsyc.55.4.317 [PubMed: 9554427]

Marshall RD, Beebe KL, Oldham M, Zaninelli R (2001). Efficacy and safety of paroxetine treatment for chronic PTSD: A fixed-dose, placebo-controlled study. *American Journal of Psychiatry*, 158(12), 1982–1988. 10.1176/appi.ajp.158.12.1982 [PubMed: 11729013]

Marshall RD, Lewis-Fernandez R, Blanco C, Simpson HB, Lin SH, Vermes D, Garcia W, Schneier F, Neria Y, Sanchez-Lacay A, & Liebowitz MR (2006). A controlled trial of paroxetine for chronic PTSD, dissociation, and interpersonal problems in mostly minority adults. *Depression and Anxiety*, 24(2), 77–84. 10.1002/da.20176

Monson CM, MacDonald A, Vorstenbosch V, Shnaider P, Goldstein ES, Ferrier-Auerbach AG, & Moccia KE (2012). Changes in social adjustment with cognitive processing therapy: Effects of treatment and association with PTSD symptom change. *Journal of Traumatic Stress*, 25(5), 519–526. 10.1002/jts.21735 [PubMed: 23073971]

Monson CM, Schnurr PP, Resick PA, Friedman MJ, Young-Xu Y, & Stevens SP (2006). Cognitive processing therapy for veterans with military-related posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, 74(5), 898–907. 10.1037/0022-006X.74.5.898 [PubMed: 17032094]

Monson CM, Taft CT, & Friedman SJ (2009). Military-related PTSD and intimate relationships: From description to theory-driven research and intervention development. *Clinical Psychology Review*, 29, 707–714. 10.1016/j.cpr.2009.09.002 [PubMed: 19781836]

Olatunji BO, Cisler JM, & Tolin DF (2007). Quality of life in anxiety disorders: A meta-analytic review. *Clinical Psychology Review*, 27(8), 572–581. 10.1016/j.cpr.2007.01.015 [PubMed: 17343963]

Ozer EJ, Best SR, Lipsey TL, & Weiss DS (2003). Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. *Psychological Bulletin*, 129(1), 52–73. 10.1037/0033-2909.129.1.52 [PubMed: 12555794]

Price M, Gros DF, Strachan M, Ruggiero KJ, & Acierno R (2013). The role of social support in exposure therapy for Operation Iraqi Freedom/Operation Enduring Freedom veterans: A preliminary investigation. *Psychological Trauma: Theory, Research, Practice, and Policy*, 5(1), 93–100. 10.1037/a0026244

Price M, Lancaster CL, Gros DF, Legrand AC, van Stolk-Cooke K, & Acierno R (2018). An examination of social support and PTSD treatment response during prolonged exposure. *Psychiatry*, 81(3), 258–270. 10.1080/00332747.2017.1402569 [PubMed: 30020026]

Rauch SA, Grunfeld TE, Yadin E, Cahill SP, Hembree E, & Foa EB (2009). Changes in reported physical health symptoms and social function with prolonged exposure therapy for chronic posttraumatic stress disorder. *Depression and Anxiety*, 26(8), 732–738. 10.1002/da.20518 [PubMed: 18781660]

Riggs DS, Byrne CA, Weathers FW, & Litz BT (1998). The quality of the intimate relationships of male Vietnam veterans: Problems associated with posttraumatic stress disorder. *Journal of Traumatic Stress*, 11(1), 87–101. 10.1023/A:1024409200155 [PubMed: 9479678]

Schnurr PP, & Lunney CA (2016). Symptom benchmarks of improved quality of life in PTSD. *Depression & Anxiety*, 33(3), 247–255. 10.1002/da.22477 [PubMed: 26882293]

Sherman MD, & Thelen MH (1996). Fear of intimacy scale: validation and extension with adolescents. *Journal of Social and Personal Relationships*, 13(4), 507–521. 10.1177/0265407596134002

Stein DJ, Ipser JC, & Seedat S (2006). Pharmacotherapy for posttraumatic stress disorder (PTSD). Cochrane Database of Systematic Review, 1, Art. No.: CD002795. 10.1002/14651858.CD002795.pub2

Tucker P, Zaninelli R, Yehuda R, Ruggiero L, Dillingham K, & Pitts CD (2001). Paroxetine in the treatment of chronic posttraumatic stress disorder: Results of a placebo-controlled, flexible-dosage trial. *Journal of Clinical Psychiatry*, 62(11), 860–868. 10.4088/JCP.v62n1105

van Minnen A, Zoellner LA, Harned MS, & Mills K (2015). Changes in comorbid conditions after prolonged exposure for PTSD: A literature review. *Current Psychiatry Reports*, 17(3), 1–16. 10.1007/s11920-015-0549-1 [PubMed: 25617038]

Xue C, Ge Y, Tang B, Liu Y, Kang P, Wang M, & Zhang L (2015). A meta-analysis of risk factors for combat-related PTSD among military personnel and veterans. *PloS One*, 10(3), e0120270 10.1371/journal.pone.0120270 [PubMed: 25793582]

Wachen JS, Jimenez S, Smith K, Resick PA (2014). Long-term functional outcomes of women receiving cognitive processing therapy and prolonged exposure. *Psychological Trauma: Theory, Research, Practice, and Policy*, 6(Suppl 1), S58–S65. 10.1037/a0035741

Watts BV, Schnurr PP, Mayo L, Young-Xu Y, Weeks WB, & Friedman MJ (2013). Meta-analysis of the efficacy of treatments for posttraumatic stress disorder. *Journal of Clinical Psychiatry*, 74(6), e541–e550. 10.4088/JCP.12r08225

World Health Organization. (2018). International classification of diseases for mortality and morbidity statistics (11th Revision). <https://icd.who.int/browse11/l-m/en>

Zalta AK, Gillihan SJ, Fisher AJ, Mintz J, McLean CP, Yehuda R, & Foa E (2014). Change in negative cognitions associated with PTSD predicts symptom reduction in prolonged exposure. *Journal of Consulting and Clinical Psychology*, 82(1), 171–175. 10.1037/a003473 [PubMed: 24188512]

Zoellner LA, Roy-Byrne PP, Mavissakalian M, & Feeny NC (2019). Doubly randomized preference trial of prolonged exposure versus sertraline for treatment of PTSD. *American Journal of Psychiatry*, 176(4), 287–296. 10.1002/aj.20588 [PubMed: 30336702]

Open Practices Statement

The preregistration for this clinical trial can be accessed at clinicaltrials.gov, [NCT00127573](https://clinicaltrials.gov/ct2/show/NCT00127573). The study reported in this article was not formally preregistered. Neither the data nor the materials have been made available on a permanent third-party archive. Access of data is limited to qualified researchers; requests for the data or study materials may be sent via email to the study's Principal Investigators at ncf2@case.edu or zoellner@uw.edu

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

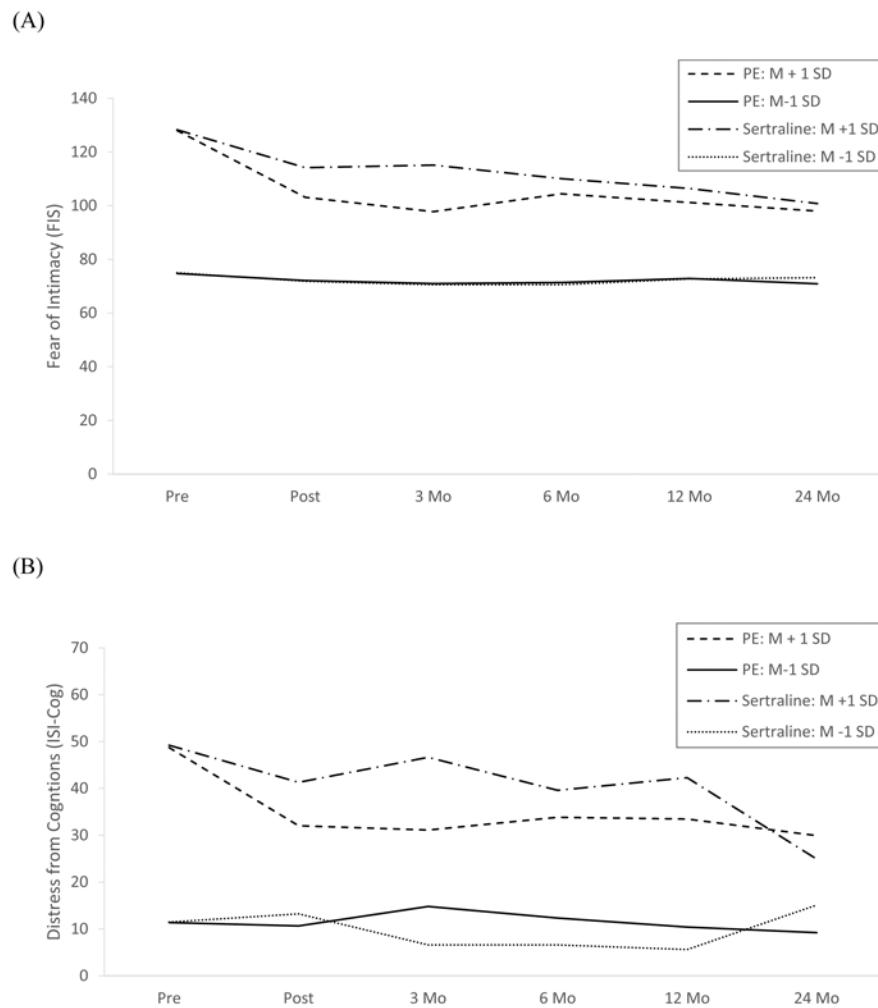


Figure 1. Trajectories of Individuals With Higher and Lower Scores of (A) Fear of Intimacy and (B) Distress From Negative Cognitions in Social Situations, from Pre- to Posttreatment and Through 24-Month Follow-Up

Note. Trajectories are presented for patients with higher (mean score + 1 standard deviation) and lower scores of pretreatment functioning (mean score - 1 standard deviation). Higher scores indicate more severe difficulties. FIS = Fear of Intimacy Scale; Mo = months after treatment; PE = prolonged exposure; Post = posttreatment; Pre = pretreatment; ISI-Cog = Inventory of Social Interactions—Negative Cognitions subscale.

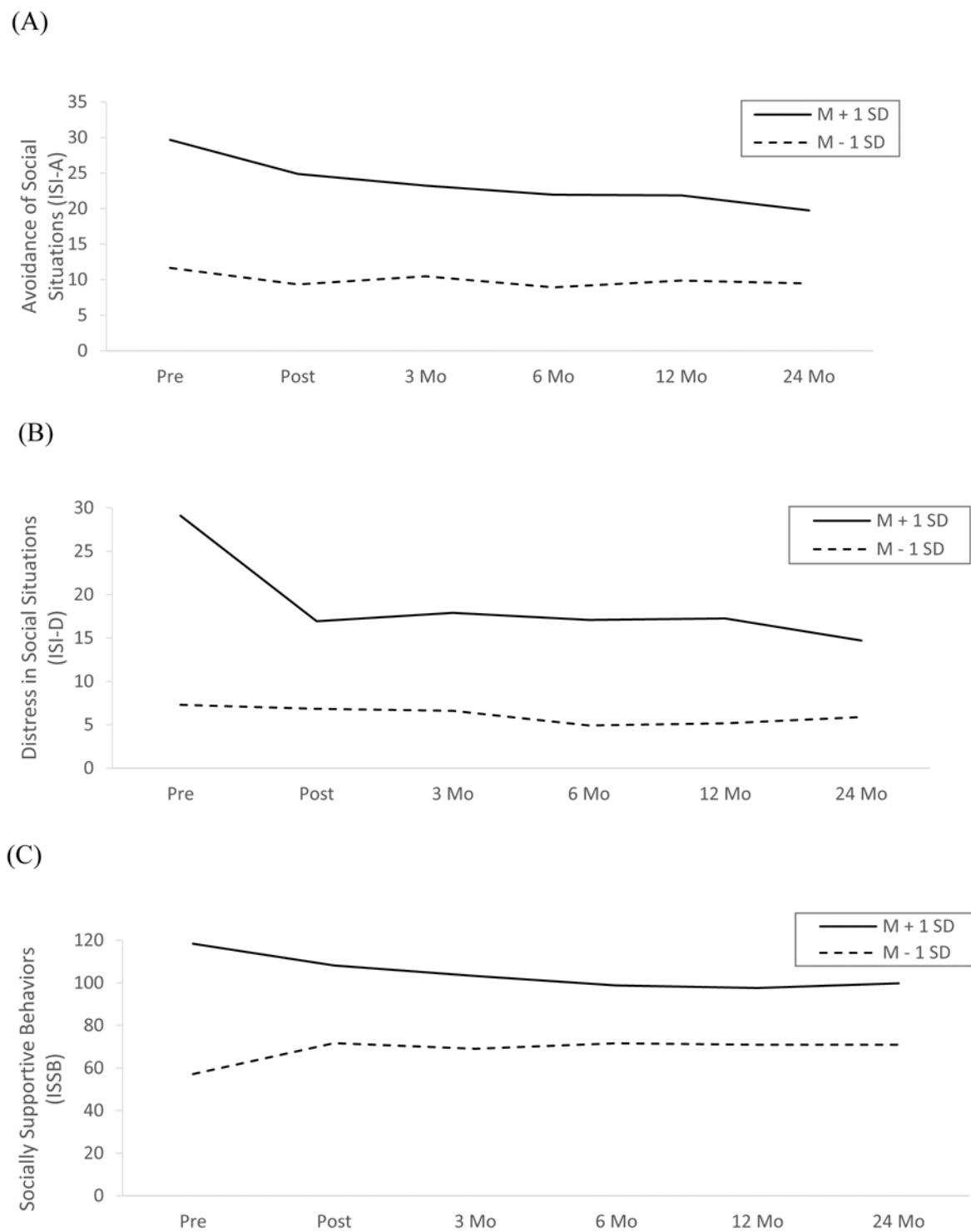


Figure 2. Trajectories of Individuals With Higher and Lower Scores of (A) Avoidance of Social Situations, (B) Distress in Social Situations, and (C) Receipt of Social Support, from Pre- to Posttreatment and Through 24-Month Follow-Up

Note. Trajectories of individuals higher and lower in pretreatment social functioning with regard to avoidance of social situations (Inventory of Social Interactions [ISI] Avoidance

subscale [ISI-A]; Panel A) and distress in social situations (ISI Distress [ISI-D] subscale; Panel B) are presented for patients with higher scores of pretreatment functioning (mean score + 1 standard deviation) and lower scores of pretreatment functioning (mean score - 1 standard deviation). Higher scores indicate more avoidance and distress. Frequency of receiving socially supportive behaviors (Inventory of Socially Supportive Behaviors [ISSB]; Panel C) is presented for patients who received more socially supportive behaviors (mean score + 1 standard deviation) and fewer supportive behaviors (mean score - 1 standard deviation). Higher scores indicate a higher frequency of receiving socially supportive behaviors. Mo = months after treatment; Post = posttreatment; Pre = pretreatment. Treatments are combined due to the lack of group differences.

Patient Characteristics and Baseline Functioning, by Treatment Condition

Characteristic	PE (n = 116)			Sertraline (n = 84)		
	%	M	SD	%	M	SD
Age (years)		36.60	11.30		38.52	11.26
Female gender	75.9			75.0		
Caucasian ethnicity	65.5			64.3		
Relationship status						
Single	50.9			38.1		
Married	16.4			22.6		
Cohabitating	14.7			10.7		
Divorced/separated	14.7			23.8		
Widowed	1.7			1.2		
Other	1.7			3.6		
Time since trauma (years)		12.02	12.24		11.89	13.35
Primary trauma type						
Adult sexual assault	30.2			32.1		
Childhood sexual and/or physical assault	26.7			20.2		
Adult physical assault	20.7			25.0		
Accident (e.g., MVA, natural disaster)	13.8			10.7		
Death/violence to a loved one	6.0			7.1		
Combat/war	2.6			2.4		
PTSD (PSS-1; range: 0–51)		29.41	6.90		29.79	6.42
Fear of intimacy (FIS; range 0–140)		97.78	24.69		103.21	28.73
Inventory of Social Interactions scale						
Negative cognitions (ISI-Cog; range: 0–80)	28.51	18.62		28.84	18.65	
Avoidance (ISI-A; range: 0–52)	19.89	8.50		21.63	9.70	
Distress (ISI-D; range 0–52)	17.04	10.07		19.02	11.85	
Perception of social support (ISSB; range 40–200)	86.65	31.15		84.38	29.92	

Note. As expected, there were no baseline characteristic differences between the treatment groups. CSA = childhood sexual assault; FIS = Fear of Intimacy scale; ISI = Inventory of Social Interactions; ISI-D = ISI Distress subscale; ISI-A = ISI Avoidance subscale; ISI-Cog = ISI Negative Cognitions subscale; ISSB = Inventory of Socially Supportive Behaviors; MVA = motor vehicle accident; PSS-1 = PTSD Symptom Scale—Interview version; PTSD = posttraumatic stress disorder.

Table 2

Baseline Correlations Between Social Functioning Variables and Posttraumatic Stress Disorder Symptom Severity

Variable	1.	2.	3.	4.	5.	6.
Social Functioning						
1. Fear of Intimacy (FIS)	—	.34 *	.48 *	.47 *	-.16 *	-.14 *
2. Negative social cognitions (ISI-Cog)	—	.57 *	.70 *	-.03	.33 *	
3. Avoidance of social interactions (ISI-A)	—		.85 *	-.19 *	.35 *	
4. Distress in social interactions (ISI-D)	—		—	-.15 *	.41 *	
5. Perception of social support (ISSB)	—		—	—	-.08	
PTSD symptoms						
6. Pretreatment PTSD (PSS-I)	—					

Note. FIS = Fear of Intimacy scale; ISI = Inventory of Social Interactions; ISI-Cog = ISI Negative Cognitions subscale; ISI-A = ISI Avoidance subscale; ISI-D = ISI Distress subscale; ISSB = Inventory of Socially Supportive Behaviors; PTSD = posttraumatic stress disorder.

* $p < .05$, based on Holm's step-down procedure for multiple comparisons (Holm, 1979).

Table 3

Basic Descriptions of Social Functioning Variables, by Treatment

Variable	PE			Sertraline		
	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>
FIS						
Pretreatment	111	97.78	24.69	82	103.21	28.73
Posttreatment	90	84.85	25.13	57	92.60	29.38
3 months	70	80.55	26.56	44	87.75	27.79
6 months	74	83.15	25.90	43	86.55	27.79
12 months	73	83.41	27.08	41	87.36	28.24
24 months	68	81.55	25.26	41	85.13	25.64
ISI-Cog						
Pretreatment	99	28.51	18.62	62	28.84	18.65
Posttreatment	64	20.43	17.72	37	27.62	21.86
3 months	50	21.40	18.40	27	29.89	19.12
6 months	51	21.67	18.22	21	23.90	18.32
12 months	47	21.51	17.15	21	19.10	19.35
24 months	38	15.68	16.25	18	16.61	15.51
ISI-A						
Pretreatment	102	19.89	8.50	65	21.63	9.70
Posttreatment	69	15.30	8.60	41	20.39	10.55
3 months	55	15.44	9.60	31	18.45	8.02
6 months	57	15.86	9.50	24	17.58	8.33
12 months	54	16.35	9.15	28	16.25	10.14
24 months	47	12.57	8.66	23	17.09	9.91
ISI-D						
Pretreatment	112	17.04	10.07	81	19.02	11.85
Posttreatment	89	11.02	7.97	59	12.29	10.93
3 months	69	11.39	9.23	44	11.45	9.76
6 months	74	9.84	8.50	44	10.48	10.30
12 months	73	10.56	8.94	42	10.90	10.12
24 months	66	8.85	8.05	41	10.95	10.36
ISSB						
Pretreatment	113	88.65	31.15	81	84.28	29.92
Posttreatment	88	91.55	32.42	59	87.51	31.62
3 months	69	89.92	29.53	44	83.80	33.16
6 months	73	88.78	28.82	44	82.36	31.69
12 months	71	84.72	29.22	40	88.76	37.97
24 months	67	85.27	29.77	41	86.75	35.06

Note. FIS = Fear of Intimacy scale; ISI = Inventory of Social Interactions; ISI-Cog = ISI Negative Cognitions subscale; ISI-A = ISI Avoidance subscale; ISI-D = ISI Distress subscale; ISSB = Inventory of Socially Supportive Behaviors.