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Differential treatment response trajectories in individuals with subclinical and clinical PTSD

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Abstract

Subclinical presentations of posttraumatic stress disorder (PTSD), wherein patients are one or two symptom criteria short of the full disorder, are prevalent and associated with levels of distress and impaired functioning approximating that of full PTSD. Nonetheless, research examining treatment efficacy for this group is in the nascent stage. The purpose of the present study was to examine whether the subclinical PTSD group would: (1) show a greater reduction in PTSD symptoms at pre and post treatment in response to an exposure based treatment and (2) show a greater rate of change over the course of treatment, when compared to the full criteria PTSD group. We also examined whether differences would emerge when examining PTSD symptom clusters. Consistent with predictions, the subclinical PTSD group demonstrated a greater reduction in PTSD symptoms at post-treatment (29%) than those with a PTSD diagnosis (14%). Further, the groups had different treatment trajectories, with the subclinical PTSD group showing a marginally greater rate of change during the course of treatment. Findings also varied by symptom cluster with the subclinical group showing a greater rate of change in the intrusions, hyper-vigilance, and avoidance symptom clusters. There was not a significant between group difference in the numbing symptom cluster. This study provides preliminary evidence that treating PTSD symptoms at the subclinical level may result in a larger, and more rapid symptom reduction, and thus has implications supporting treatment earlier in the developmental trajectory of the disorder.

Keywords

PTSD; Posttraumatic stress disorder; Subclinical; Subthreshold; Military; Veterans; Growth curve modeling

1. Introduction

Post-traumatic stress disorder (PTSD) is one of the most prevalent mental health conditions among veterans seeking treatment at Veterans Affairs Medical Centers (VAMCs) (Seal et al., 2009). PTSD is chronic (Smith et al., 2008; Zlotnick, Franklin, & Zimmerman, 2002), associated with significantly impaired functioning (Breslau, Lucia, & Davis, 2004), and leads to substantial cost and economic burden for patients and society (Kessler, 2000).

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Subclinical PTSD is also prevalent, with approximately 5% of current veterans (Grubaugh et al., 2005) and 21% of lifetime veterans (Weiss et al., 1992) classified as falling within the subclinical range.

Subclinical PTSD, also referred to as partial, subthreshold, or subsyndromal PTSD (Dickstein et al., 2014; Mylle & Maes, 2004; Zlotnick et al., 2002), can be broadly defined as the presence of PTSD related symptoms that are elevated but do not meet full diagnostic criteria for PTSD. Although subclinical PTSD has been conceptualized using a range of approaches, it has often been measured by assessing for the presence or absence of PTSD symptoms (Blanchard, Hickling, Taylor, Loos, & Gerardi, 1994), ranging from a minimum of one symptom on each PTSD symptom cluster (Stein et al., 1997) to more comprehensive definitions wherein at least one re-experiencing symptoms (Criteria B) and at least two symptoms in avoidance (Criteria C) and hyperarousal (Criteria D) domains are required as defined in the *Diagnostic and Statistical Manual for Mental Disorders* DSM-IV TR (American Psychiatric Association 2000; Kilpatrick & Resnick, 1993). A third approach is to designate subclinical PTSD according to the following DSM-IV criteria: (1) Criteria A of experiencing or witnessing a traumatic event, (2) Criteria B of re-experiencing symptoms (e.g, intrusions, flashbacks, nightmares) and (3) ***either*** Criteria C of avoidance and numbing symptoms (e.g., avoiding reminders of the traumatic event, lack of strong emotions) ***or*** Criteria D of hyperarousal symptoms (e.g., exaggerated startle reaction; Blanchard et al., 1994; Grubaugh et al., 2005) is present. Similar definitions exist for the newer DSM-5 criteria for PTSD.

Whereas significant research to improve treatment for full criteria PTSD exist (Bradley, Greene, Russ, Dutra, & Westen, 2005; Foa, Keane, Friedman, & Cohen, 2009), evaluation of treatments targeting subclinical PTSD are still in nascent stages (Steenkamp et al., 2012). Given the high prevalence of subclinical PTSD, examining the effectiveness and course of treatment in these individuals is of significant importance. Specifically, subclinical PTSD is associated with elevated levels of distress, impairment in interpersonal and occupational functioning (Cukor, Wyka, Jayasinghe, & Difede, 2010), and elevated levels of comorbidity (Jakupcak et al., 2007; Zlotnick et al., 2002) compared to individuals without subclinical PTSD. Subclinical PTSD is also associated with increased levels of anger, aggression, and suicidality (Marshall et al., 2001; Jakupcak et al., 2007; Zlotnick et al., 2002). Further, having subclinical PTSD may also place individuals at increased risk for developing full clinical PTSD (Mylle and Maes, 2004). Despite evidence demonstrating impairment and distress associated with subclinical PTSD, and the potential for these symptoms to increase in severity over time eventually resulting in clinical PTSD, this group has typically been neglected, and indeed excluded from PTSD treatment evaluations and randomized controlled trials (Zlotnick et al., 2002). As such, individuals with subclinical PTSD are often excluded from many PTSD treatment settings and fail to receive treatment they need, resulting in a substantial lack of treatment resources for these individuals, and a gap in the literature pertaining to the treatment of this group.

Given the lack of research examining the treatment of subclinical PTSD, there is uncertainty about appropriate approaches for this group. Recent evidence demonstrates efficacy of treating subclinical PTSD using paroxetine (Naylor et al., 2013); however, the preliminary

literature examining trauma-focused psychotherapy has been somewhat equivocal. In particular, Kornfield, Klaus, McKay, Helstrom, and Oslin (2012) cautioned against the use of evidence based psychotherapy targeting avoidance symptoms of PTSD in primary care patients with subclinical PTSD. This recommendation was based on their finding of low levels of avoidance in these individuals. More recently, preliminary evidence has emerged demonstrating efficacy of evidence based psychotherapy approaches for PTSD in individuals with subclinical PTSD. Specifically, Dickstein, Walter, Schumm, and Chard (2013) examined the use of Cognitive Processing Therapy (CPT; Resnick, Monson, & Chard, 2007) to treat subclinical PTSD, finding that individuals with subclinical PTSD had a similar reduction in PTSD symptoms at post-treatment compared to the clinical PTSD group.

Preliminary evidence demonstrating the efficacy of treating subclinical PTSD with evidence-based psychotherapy is encouraging and warrants further investigation for several reasons. First, it is important to replicate these findings to add to the emerging literature on the treatment of subclinical PTSD. Moreover, research is needed examining the treatment trajectory (i.e., rate) of improvement among these groups. Although preliminary research demonstrates the efficacy of treating subclinical PTSD, it is unknown whether individuals with subclinical PTSD show a differential response during the course of treatment than those with a clinical PTSD diagnosis. Given the lower overall level of symptom frequency, and generally shorter duration or chronicity of the symptoms in individuals with subclinical PTSD, it is feasible that this group may actually respond to treatment more quickly and show different rates of symptom reduction than those with clinical PTSD. In particular, it is possible that those with subclinical PTSD may show a greater decline in symptoms from pre to post treatment. Although it could be argued that individuals with a greater severity of symptoms may demonstrate greater reduction in symptoms due to regression to the mean (Barnett, Van der Pols, & Dobson, 2005), the notion that subclinical PTSD may reflect greater reduction in symptoms is consistent with the prevention literature demonstrating benefits of treating less severe symptom presentations with brief evidence-based interventions for subclinical symptoms of anxiety (Aune & Stiles, 2009; Gardenswartz & Craske, 2001). Further, those with subclinical PTSD may also demonstrate a greater rate of symptom change over the course of treatment (session-by-session symptom reduction) than those with clinical PTSD. Finally, it is possible that the PTSD symptoms clusters (i.e., intrusions, hypervigilance, avoidance, and dysphoria) will show differential treatment response patterns in those with subclinical PTSD versus those with clinical PTSD.

The purpose of the present study is to examine differential treatment response in veterans with subclinical vs. clinical PTSD. We hypothesized that: (1) participants with subclinical PTSD will respond more quickly to evidence based psychotherapy for PTSD; and (2) response patterns across PTSD symptom clusters (i.e., intrusions, hyperarousal, avoidance, and numbing) will be different in the subclinical and clinical PTSD groups (i.e., intrusions may reduce more quickly in the subclinical group than the clinical PTSD group). Although exploratory, we expected the subclinical PTSD group to show a greater rate of change in each of the PTSD symptom clusters than the clinical PTSD group.

2. Methods

2.1. Participants

The sample was composed of 238 combat veterans. Participants were recruited through referrals to a PTSD specialty clinic at a large Southeastern VAMC. Eligible participants were required to meet diagnostic criteria for combat-related PTSD (n = 188) or subclinical PTSD (n = 50). Subclinical PTSD was defined as the presence of Criteria A (traumatic event) and Criteria B (re-experiencing), and either Criteria C (avoidance) or Criteria D (hyperarousal; Grubaugh et al., 2005), along with the impairment criterion. Participants with a military related index trauma were eligible to participate in the study. Eligibility was determined via the *Clinician Administered PTSD Scale* (CAPS; Blake et al., 1995) to assess PTSD symptoms and the *Structured Clinical Interview for the DSM-IV* (SCID; First, Spitzer, Gibbon, & Williams, 1996) to assess for comorbid Axis I and Axis II disorders. Individuals who were acutely suicidal, psychotic, or met criteria for a current substance dependence disorder were excluded from participation. Participants with comorbid anxiety or mood disorders, and participants prescribed psychotropic medication were allowed to participate in the study after a 4 week stabilization period. A majority of the participants were male (92.5%), Black (47.0%) or White (46.5%), and had a mean age of 45.0 years (SD = 14.6). A majority of the participants were also employed (45.0%), married (61.5%), and served in the Army (60.0%). Further, approximately 39% of the percent of participants reported being disabled.

2.2. Measures

2.2.1. Clinician administered measures—The CAPS (Blake et al., 1995) was administered to asses for symptoms of PTSD. The CAPS is a semi-structured clinician administered interview used to diagnose current and lifetime PTSD. The CAPS assesses for the frequency and intensity of the 17 PTSD symptoms from the *DSM-IV* (American Psychiatric Association, 2000). Intensity and frequency ratings of each symptom are made on a five-point Likert scale. To meet threshold criteria, current symptoms must have been present for at least one month. The CAPS has been shown to have good internal consistency, test-retest reliability, convergent and discriminant validity (αs ranging from 0.73 to 0.95; Orsillo, Batten, & Hammond, 2001).

2.2.2. Structured clinical interview for the DSM-IV—The SCID (First et al., 1996) was administered to assess for the presence of current or past Axis I diagnoses for eligibility purposes. The SCID is a clinician administered measure and has been shown to have good internal consistency and convergent and divergent validity (First et al., 1996).

2.2.3. PTSD Checklist—Military—The *PTSD Checklist* (PCL-M; Weathers et al., 1993) was used to measure self-report symptoms of PTSD. The PCL is a 17-item measure assessing PTSD symptom severity. Respondents rate "*How much you have been bothered by that problem in the last month*" in reference to 17 symptoms of PTSD. Responses are made on a five-point Likert scale ranging from 1 (*not at all*) to 5 (*extremely*). The PCL has been shown to have excellent internal consistency in a variety of trauma populations including Veterans, sexual assault victims, and survivors of severe motor vehicle accidents (Orsillo et

al., 2001). The PCL items can be separated into four subscales reflecting the PTSD symptoms clusters consistent with the King model of PTSD symptom clusters (King, Leskin, King, & Weathers, 1998) including: (1) intrusions, (2) re-experiencing, (3) hyperarousal, and (4) numbing. Cronbach's alpha for the current study was in the excellent range (α = .93). In addition, a cut-score of 50 is often considered to distinguish clinically significant levels of PTSD symptoms (Weathers et al., 1993).

2.3. Procedures

The data used in the current study were collected as part of a larger investigation examining the comparative effectiveness of Behavioral Activation and Therapeutic Exposure (BA-TE) administered through either in-person or telehealth modalities. An abbreviated presentation of the methodology is given below. See Gros and Haren (2011) for a full description of the study methodology, including the randomization process and treatment protocols. All assessment measures and psychotherapy were administered by masters level clinicians who received extensive training in all procedures.

Participants received eight, 90-min sessions of BA-TE, a trans-diagnostic exposure-based psychotherapy designed specifically to improve treatment outcome in patients with comorbid symptoms of PTSD and depression (Gros et al., 2011; Gros et al., 2012). BA-TE includes behavioral activation, in vivo exposure, and imaginal exposure techniques, the latter of which are consistent with the prolonged exposure model described by Foa, Hembree, and Rothbaum (2007). Participants completed within and between session exposure trials and used a daily planner to monitor their homework completion. Patients also engaged in behavioral activation in which they were asked to schedule and track the completion of personally meaningful and reinforcing activities in their daily planner (Lejuez, Hopko, LePage, Hopko, & McNeil, 2001).

For purposes of the primary research question in the larger study (Gros et al., 2011), participants were randomized into either in-person treatment (n = 117) or treatment that was provided through telehealth technologies (n = 115). Those in the telehealth group received treatment though an internet based video service (e.g., a "Skype" type program) or a videophone (Viterion 500) was used at the participant's discretion. Weekly supervision was provided by the principal investigator (RA) to all providers throughout the duration of the study. All sessions were audiotaped, and treatment fidelity was assessed through random sampling of 20% of therapy session tapes that were rated according to a checklist of session specific procedures that directly corresponded to the BATE treatment manual. Fidelity for all therapists was maintained at or above 90% across and within conditions. Clinician (SCID and CAPS) and self-report measures (PCL) were completed at baseline and post-treatment. Self-report measures including the PCL were administered bi-weekly throughout the course of treatment (baseline and sessions 2, 4, 6, and 8). It is important to note that there were no significant differences in symptom improvements as a function of the two treatment conditions (i.e., telehealth treatment or in-person treatment), which is consistent with the general tele-health literature (Gros et al., 2013; Morland et al., 2015; Yuen et al., 2015).

2.4. Statistical analyses

Of the initial 238 participants, 38 were excluded from analyses due to dropping out of the study after the baseline assessment, leaving 200 participants for the present study (n = 156 clinical PTSD group; n = 44 subclinical PTSD group). Demographic characteristics were compared using chi-square tests for categorical variables and t-tests for continuous variables using SPSS Version 22 (IBM Corp.). Latent growth curve analysis (with the intercept and slope parameters models as latent variables) was then conducted, using Mplus version 7.3 (Muthen & Muthen, 2014), to examine the primary hypothesis that the subclinical PTSD group would demonstrate significantly greater reductions in PTSD symptoms (PCL total score as well as PCL symptom clusters) relative to the clinical PTSD group. Unconditional linear growth curve models were first fit, and then a quadratic growth function was included to determine if fit was improved. Following this, PTSD status (0 = subclinical PTSD, 1 = clinical PTSD) was included as a predictor of slope. Residual covariances were included for adjacent PCL score timepoints.

Latent growth curve models were fit using full information maximum likelihood to account for missing data and the Yuan–Bentler scaled chi-square (Y-B χ^2) to adjust standard errors for nonnormality and nonindependence (Yuan & Bentler, 2000). The Y-B χ^2 statistic and several fit indices (i.e., comparative fit index [CFI; Bentler, 1990], Tucker–Lewis Index [TLI; Tucker & Lewis, 1973], and root mean square error of approximation [RMSEA; Steiger & Lind, 1980]) were used to assess overall model fit. A nonsignificant Y-B χ^2 indicates that the overall test of model fit was excellent. CFI and TLI values greater than .95 indicate good fit (Hu & Bentler, 1999). RMSEA values less than .05 indicate good fit, values less than .08 indicate adequate fit, and values greater than .10 indicate poor fit, although the RMSEA can be sensitive to smaller sample sizes. In addition, 90% confidence intervals (CIs) are provided for the RMSEA. Lower limit values less than .05 indicate that good fit cannot be ruled out and upper limit values greater than .10 indicate that poor fit cannot be ruled out (MacCallum, Browne, & Sugawara, 1996; Browne & Cudeck, 1993). Participants were included if they attended at least one treatment session (a modified intent-to-treat analysis) as FIML is robust to missing data. Models were conducted, centering time on baseline status so that the intercept could be included to control for initial PTSD symptoms.

3. Results

3.1. Demographics

There were no significant differences in demographic variables or in baseline PCL scores in those who were included in the analyses (n = 200) versus those who dropped out (n = 38). Table 1 presents the demographic characteristics for the analyzed sample. There was a significant effect of age, $F(1, 192) = 7.45$, $p < .01$, such that those in the subclinical PTSD group were younger (M age = 40.21, SD = 14.88) than those in the clinical PTSD group (M age = 47.02, SD = 14.30). There was also a significant difference in the percentage of individuals above the PCL clinical cut-score (≥50; $\chi^2 = 21.72$, df = 1, $p < .001$). There were no other between-group differences in demographics.

3.2. PTSD symptoms

Table 2 shows means and standard deviations of the primary outcome measures for subclinical and clinical PTSD groups. The unconditional linear PCL total score model provided adequate fit to the data (Y-B χ^2 = 8.36, df = 6, p = .21, CFI = .99, TLI = .99, RMSEA = .04, 90% CI [.00, .11]). The quadratic function was not significant when included in this, or in the PCL symptom clusters analyses, and therefore, the linear models only were reported. The conditional model including PTSD status also provided adequate fit to the data (Y-B χ^2 = 9.30, df = 9, p = .41, CFI = 1.00, TLI = 1.00, RMSEA = .01, 90% CI [.00, .08]). Model parameters are provided in Table 3. The effect of PTSD status was significant for the intercept, indicating that participants in the clinical PTSD group started treatment 10.10 points (SE = 2.28, p < .001) higher on the PCL compared to PCL levels for the subclinical PTSD group. There was a significant overall slope effect, indicating that, on average, participants PCL symptoms reduced 1.87 points (SE = .36, p < .001) per session. PTSD status marginally influenced this effect (p = .06), indicating a marginally greater slope (B = 2.59 vs. 1.87) for those in the subclinical PTSD group. This corresponds to .72 point greater reduction in symptoms at each timepoint in the subclinical group than the clinical PTSD group.

3.3. PTSD symptom clusters

We also examined the between group differences in the PTSD symptom clusters using the PCL subscales (intrusion subscale, PCL-I; avoidance subscale, PCL-A; numbing; PCL-N; hypervigilance, PCL-H; Palmieri, Weathers, Difede, & King, 2007) during the course of treatment.

3.3.1. Intrusions symptom cluster (PCL-I)—The unconditional linear PCL-I symptoms model provided adequate fit to the data (Y-B χ^2 = 8.19, df = 6, p = .22, CFI = .99, TLI = .99, RMSEA = .04, 90% CI [.00, .11]) as did the conditional model including PTSD status as a predictor (Y-B χ^2 = 8.31, df = 6, p = .50, CFI = 1.00, TLI = 1.00, RMSEA = .00, 90% CI [.00, .08]). Model parameters are provided in Table 3. The effect of PTSD status was significant for the intercept, indicating that participants in the clinical PTSD group started treatment with PCL-I symptoms 1.87 points (SE = .77, p < .05) higher than did participants in the subclinical group. There was a significant overall slope effect, indicating that, on average, participants' PCL-I symptoms decreased .63 points (SE = .12, p < .001) per session. PTSD status significantly influenced this effect (p < .01), indicating a greater rate of symptom reduction (B = .98 vs. .63) for those in the subclinical PTSD group versus those in the clinical PTSD group.

3.3.2. Avoidance symptom cluster (PCL-A)—The unconditional linear PCL-A symptoms model provided adequate fit to the data (Y-B χ^2 = 6.30, df = 6, p = .39, CFI = 1.00, TLI = 1.00, RMSEA = .02, 90% CI [.00, .09]) as did the conditional model including PTSD status as a predictor (Y-B χ^2 = 9.13, df = 9, p = .43, CFI = 1.00, TLI = 1.00, RMSEA = .01, 90% CI [.00, .08]). Model parameters are provided in Table 3. The effect of PTSD status was significant for the intercept, indicating that participants in the clinical PTSD group started treatment with PCL-A symptoms .90 points (SE = .35, p < .01) higher than did participants in the subclinical group. There was a significant overall slope effect, indicating

that, on average, participants' PCL-A symptoms reduced .31 points (SE = .05, $p < .001$) per session. PTSD status significantly influenced this effect ($p < .01$), indicating a greater rate of symptom reduction ($B = .46$ vs. .31) for those in the subclinical PTSD group versus those in the clinical PTSD group.

3.3.3. Numbing symptom cluster (PCL-N)—The unconditional linear PCL-N symptoms model provided adequate fit to the data (Y-B $\chi^2 = 6.31$, df = 6, $p = .39$, CFI = 1.00, TLI = 1.00, RMSEA = .02, 90% CI [.00, .09]) as did the conditional model including PTSD status as a predictor (Y-B $\chi^2 = 7.73$, df = 9, $p = .56$, CFI = 1.00, TLI = 1.00, RMSEA = .00, 90% CI [.00, .07]). Model parameters are provided in Table 3. The effect of PTSD status was significant for the intercept, indicating that participants in the clinical PTSD group started treatment with PCL-N symptoms 6.51 points (SE = 1.19, $p < .001$) higher than did participants in the subclinical group. There was a significant overall slope effect, indicating that, on average, participants' PCL-N symptoms reduced .59 points (SE = .18, $p < .001$) per session. PTSD status did not significantly impact this slope, indicating that rate of change for PCL Numbing symptoms was the same across groups.

3.3.4. Hypervigilance symptom cluster (PCL-H)—The unconditional linear PCL-H symptoms model provided adequate fit to the data (Y-B $\chi^2 = 20.80$, df = 6, $p = .002$, CFI = .95, TLI = .92, RMSEA = .11, 90% CI [.06, .17]) as did the conditional model including PTSD status as a predictor (Y-B $\chi^2 = 20.23$, df = 9, $p = .02$, CFI = .97, TLI = .94, RMSEA = .08, 90% CI [.03, .13]). Model parameters are provided in Table 3. The effect of PTSD status was not significant for the intercept. There was a significant overall slope effect, indicating that, on average, participants' PCL-H symptoms reduced .29 points (SE = .04, $p < .001$) per session. PTSD status significantly influenced this effect ($p < .01$), indicating a greater rate of symptom reduction ($B = .42$ vs. .29) for those in the subclinical PTSD group versus those in the clinical PTSD group.

4. Discussion

The purpose of the present study was to investigate whether there is a differential treatment trajectory in subclinical PTSD and clinical PTSD. Consistent with Dickstein et al. (2013), the present study demonstrated the efficacy of providing an evidence-based therapeutic treatment for subclinical PTSD. Whereas Dickstein et al. (2013) provided support for the use of CPT, the present study provided support for the use of behavioral activation and therapeutic exposure in the treatment of subclinical PTSD.

Significant group differences were evidenced over the course of treatment using behavioral activation and therapeutic exposure for total PTSD symptoms (marginally significant effect) as well as for the majority of PTSD symptom clusters. These findings suggest that veterans with subclinical PTSD demonstrate a greater treatment response trajectory than those with clinical PTSD. Specifically, the subclinical PTSD group demonstrated a marginally greater rate of change over the course of the exposure based treatment than the clinical PTSD group, thereby providing preliminary support for the notion that those with subclinical PTSD may respond better to treatment than those with clinical PTSD.

The present study also showed a greater reduction in PTSD symptoms at post-treatment in the subclinical group, despite a lower initial total score (i.e., the opposite of any regression to the mean propensity). Notably, symptom reduction from pre- to post-treatment in the subclinical group was approximately double that of the full PTSD group (e.g., subclinical group reductions of 35.9% in avoidance symptoms and 22.1% in numbing symptoms vs. clinical PTSD group reductions of 15.7% in intrusion symptoms and 12.0% reduction in numbing symptoms). Although the present study is the first that we are aware of to directly compare the level of symptom reduction at post-treatment in subclinical and clinical groups of PTSD, the findings from this study are consistent with prior research showing that those with lower levels of PTSD symptoms at pre-treatment tend to respond to treatment more quickly, resulting in lower levels of PTSD symptoms at post-treatment than those with higher levels of PTSD symptoms (Van Minnen, Artz, & Keijsers, 2002). Further, these findings may also suggest that those with subclinical PTSD might possibly require fewer treatment sessions, as has been suggested for individuals presenting with less severe cases of PTSD (Van Minnen et al., 2002). These findings underscore the importance of treating subclinical PTSD from cost effectiveness and prevention perspectives. Specifically, treating subclinical PTSD potentially reduces the overall burden associated with PTSD in a manner similar to that demonstrated in terms of cost effectiveness of treating subclinical anxiety and depression using stepped care approaches (Veer-Tazelaar et al., 2010).

Given the tendency for individuals with subclinical PTSD to be overlooked or even excluded from treatment settings (Zlotnick et al., 2002), establishing the utility of treating this symptom presentation is especially important to ensure these individuals receive the treatment they need. This is particularly important given that those with subclinical PTSD are presenting for treatment early in the developmental trajectory before their symptoms potentially worsen and severity and result in threshold levels of PTSD. As such, integrated primary care programs may be the ideal setting to target this population (Gros & Haren, 2011; Zeiss & Karlin, 2008). Further, given the level of impairment observed in individuals with subclinical presentations of anxiety and PTSD (Cukor et al., 2010; Korte, Antonio-Brown, & Schmidt, 2013), this subgroup represents a group of individuals in need of early intervention to potentially prevent the further escalation of symptoms (Mylle & Maes, 2004).

Moreover, the present study demonstrated that not only can we effectively reduce symptoms of PTSD in subclinical individuals, but that the overall symptom reduction and rate of change over the course of treatment using behavioral activation and therapeutic exposure is marginally larger than the symptom reduction observed in the clinical PTSD group. Consistent with prevention literature (Lau & Rapee, 2011), these findings highlight the potential benefit of treating subclinical presentations of mental disorders, such as subclinical PTSD, in that we may expect these individuals to respond better to treatment than those with clinical levels of symptoms. As such, these findings have important implications for clinical settings, particularly in settings such as VA PTSD clinics where patients with subclinical PTSD commonly present for treatment. In such settings, more emphasis should be placed on providing treatment resources for individuals who meet criteria for subclinical PTSD.

Finally, the PTSD symptom clusters (intrusions, avoidance, hyperarousal, and numbing) showed different response patterns over the course of treatment in the subclinical PTSD group. The subclinical PTSD group demonstrated significantly greater reduction in intrusions, avoidance, and hyperarousal symptom clusters throughout the course of treatment, whereas no between group differences emerged for the numbing symptom cluster. Thus, it appears that the majority of the PTSD symptoms respond at a greater rate during the course of treatment in the subclinical PTSD group than the PTSD group. Given the exploratory nature of the symptom cluster analyses, additional research is needed to more fully understand these differences. This is particularly true for the numbing symptoms due to the lack of significant between group differences in this symptom cluster.

As with any investigation, the current study should be considered in light of its limitations. First, the sample used was predominately male (92.5%) and entirely veteran which limit the generalizability of the findings. Future investigations would benefit from examining this effect in a sample with a larger proportion of females as well civilians to discern whether these groups with subclinical PTSD have similar treatment trajectories as in the present study. In addition, the present study was limited by the imbalance in the sample size across the subclinical and clinical PTSD groups. Although significant differences emerged in the latent growth curve models for present study, it is important to replicate these findings to in a sample with roughly equivalent numbers of subclinical and clinical PTSD. Further, it is important to note that although there were significant between group differences observed at the symptom cluster level, the overall effect for total PTSD was marginally significant ($p = .06$), thereby highlighting the importance of replicating the results of the present study.

The present study investigated the treatment trajectories of subclinical and clinical PTSD during the course of evidence-based psychotherapy. The findings are novel, have important implications, and provide several avenues for future investigation. Aside from providing further support for the efficacy of treating subclinical PTSD, perhaps the most interest finding of the present study in the evidence showing that treating subclinical PTSD not only results in a significant reduction of symptoms but that there is a marginally greater rate of symptom change over the course of treatment in subclinical PTSD group than the clinical PTSD group. Based on these findings, it seems that the literature on subclinical PTSD would benefit from examining the efficacy of developing a brief evidence-based treatment for those with emerging PTSD symptoms to assess whether subclinical PTSD could be effectively treated though a brief intervention that is shorter in duration than a typical course of treatment for clinical PTSD. Consistent with the literature on prevention (Rapee, 2008), providing treatment early could have a significant impact on the cost-effectiveness of treating subclinical PTSD and, moreover, may possibly lead to the prevention of the development of more intractable forms of PTSD that can occur when subclinical symptoms go untreated.

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Table 1

Baseline demographic and clinical characteristics among participants in the subclinical PTSD and PTSD groups.

Variable	Subclinical PTSD (*n* = 44) M (SD)	PTSD (*n* = 156) M (SD)	Total (*n* = 200) M (SD)	F
Age	40.21 (14.88)	47.02 (14.30)	45.51 (14.67)	7.45**
Variable	%	%	%	χ^2
Above PCL clinical cut-score (≥50)	55.6%	86.3%	78.0%	21.72***
Gender (Male)	95.8%	88.3%	92.5%	1.12
Race (Caucasian)	56.6%	44.7%	46.5%	2.65
Race (African-American)	34.0%	52.5%	47.0%	3.77
Relationship (Married)	58.0%	63.3%	61.5%	3.32
Education (At Least Some College)	60.5%	47.0%	50.0%	3.53
Unemployed/Retired/Disabled	45.8%	54.1%	42.6%	3.54
Most Common Service Branch				
Army	67.3%	59.3%	60.0%	2.21
Navy	6.1%	11.6%	11.1%	2.32
Marines	8.2%	13.4%	12.6%	1.61
Air Force	18.4%	15.7%	15.3%	.21

Note. $N = 200$.

Table 2

Descriptives of the primary outcome measures among participants in the subclinical PTSD and PTSD groups.

	Treatment session					
Measure	**0**	**2**	**4**	**6**	**8**	**Post**
Subclinical PTSD (*n* = 44)						
PCL Total Score	52.1 (16.8)	48.3 (13.5)	45.7 (14.8)	40.5 (17.2)	38.3 (15.7)	36.9 (15.3)
PCL ≥ 50 (%)	55.6%	54.3%	64.9%	60.0%	67.7%	14.3%
PCL-I	16.1 (7.3)	14.1 (3.9)	13.3 (4.7)	12.0 (5.1)	11.4 (4.9)	11.2 (5.1)
PCL-A	6.4 (2.2)	6.2 (2.5)	5.7 (2.4)	4.5 (2.4)	4.1 (2.5)	4.1 (2.5)
PCL-H	16.9 (4.6)	16.3 (4.6)	14.7 (2.4)	13.0 (5.4)	12.4 (4.8)	11.9 (2.2)
PCL-N	12.2 (5.6)	11.59 (4.8)	11.9 (4.8)	11.0 (5.5)	10.1 (4.7)	9.5 (7.2)
PTSD group (*n* = 156)						
PCL Total Score	61.4 (12.5)	59.2 (11.9)	56.3 (13.8)	52.9 (15.1)	52.3 (16.2)	53.0 (16.7)
PCL ≥ 50 (%)	86.3%	85.1%	83.2%	84.7%	83.0%	56.0%
PCL-I	17.8 (4.7)	16.5 (4.31)	15.8 (4.9)	15.0 (5.0)	15.0 (5.2)	15.0 (5.4)
PCL-A	7.4 (2.0)	7.1 (2.0)	6.8 (2.1)	6.3 (2.2)	6.1 (2.3)	6.3 (2.3)
PCL-H	19.3 (4.2)	19.0 (3.8)	18.3 (4.5)	16.8 (5.1)	16.6 (5.4)	16.8 (2.6)
PCL-N	16.6 (4.5)	16.2 (4.2)	15.4 (4.3)	14.6 (5.0)	14.3 (5.2)	14.6 (8.2)

Note. PCL = PTSD Checklist; PCL-I = PTSD Checklist, Intrusions Subscale; PCL-H = PTSD Checklist, Hyperarousal Subscale; PCL-A = PTSD Checklist, Avoidance Subscale; PCL-N = PTSD Checklist, Numbing Subscale.

Table 3

Model parameter estimates for the effect of PTSD status on PCL total score and symptom clusters.

	PCL Total		PCL Intrusions		PCL Avoidance		PCL Numbing		PCL Hypervigilance	
	Parameters	**SE**	**Parameters**	**SE**	**Parameters**	**SE**	**Parameters**	**SE**	**Parameters**	**SE**
Intercept	51.47***	2.10	15.40***	.65	6.54***	.32	21.94***	1.08	7.23***	.34
Variance^{I}	131.36***	21.97	16.77***	3.00	3.48***	.56	31.09***	5.59	2.89***	.59
Slope	−1.87***	.36	−.63***	.12	−.31***	.05	−.59***	.18	−.29***	.04
Variance^{S}	2.20**	.78	.30**	.11	.05***	.02	.36*	.18	.03	.02
Covariance	−3.91	3.79	−.63	.50	−.20	.09	−.81	.93	.13**	.05
PTSD Status	B	SE	B	SE	B	SE	B	SE	B	SE
Intercept	10.10***	2.28	1.87*	.77	.90**	.35	6.51***	1.19	.73	.38
Slope	.72****	.72	.35**	.13	.15**	.06	.06	.20	.13**	.05

Note. Intercept is centered at Baseline. Slope and intercept parameters are presented as unstandardized parameters (i.e., raw PCL scores). Parameters = Unstandardized Parameters. Variance^{I} = Intercept Variance. Variance^{S} = Slope Variance. PTSD status coded as 0 = subclinical PTSD, 1 = clinical PTSD. SE = Standard error.

* $p \leq .05$.

** $p \leq .01$.

*** $p \leq .001$.

**** $p \leq .10$.