A MULTI-LEVEL MODELING APPROACH EXAMINING PTSD SYMPTOM REDUCTION
DURING PROLONGED EXPOSURE THERAPY AMONG HIV-POSITIVE ADULTS

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by

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TABLE OF CONTENTS

LIST OF FIGURES

PLOTTED ESTIMATED MEANS ILLUSTRATING THE MODERATING EFFECT OF INDEX TRAUMA BY TIME ON HYPERAROUSAL SYMPTOM CHANGE OVER THE COURSE OF PE.

PLOTTED ESTIMATED MEANS ILLUSTRATING THE MODERATING EFFECT OF TRAUMA HISTORY BY TIME ON PTSD SYMPTOM CHANGE OVER THE COURSE OF PE.

PLOTTED ESTIMATED MEANS ILLUSTRATING THE MODERATING EFFECT OF TRAUMA HISTORY BY TIME ON RE=EXPERIENCING SYMPTOM CHANGE OVER THE COURSE OF PE.

PLOTTED ESTIMATED MEANS ILLUSTRATING THE TREND-LEVEL MODERATING EFFECT OF TRAUMA HISTORY BY TIME ON AVOIDANCE SYMPTOM CHANGE OVER THE COURSE OF PE.

PLOTTED ESTIMATED MEANS ILLUSTRATING THAT TRAUMA HISTORY DOES NOT MODERATE TIME ON HYPERAROUSAL SYMPTOM CHANGE OVER THE COURSE OF PE.

LIST OF TABLES

Acknowledgments

INTRODUCTION

Human immunodeficiency virus (HIV) is a serious health concern in the United States, with approximately 1.1 million people living with HIV(PLWH) today (Centers for Disease Control and Prevention (CDC), CDC, 2013a; CDC, 2013b). Due to recent medical advances, a diagnosis of HIV is less often seen as a terminal diagnosis and more often considered as a chronic disease (CDC, 2007; Erlandson et al., 2012). However, HIV remains a life-threatening illness that is not only associated with mortality, but is also associated with a multitude of negative health, social, and quality of life outcomes (i.e., Ashton et al., 2005; Galantino et al., 2014; van Sighem, Gras, Reiss, Brinkman, & de Wolf, 2010). Besides the health and well-being costs associated with HIV, it also comes with a large financial burden. For example, this year the United States government budget for HIV, including prevention, treatment, and research was $29.7 billion (Kaiser Family Foundation, 2014). Besides being associated with high mortality, negative health and social consequences, and high monetary costs, HIV is also associated with interpersonal traumatic events (i.e., Kelly et al., 1998; Theuninck, Lake, & Gibson, 2010; Whetten, Reif, Whetten, & Murphy-McMillan, 2008).

PLWH experience very high rates of interpersonal trauma, which is highly associated with PTSD (i.e., Alisic et al., 2014; Breslau et al., 1999; Hetzel-Riggin & Roby, 2013; Mauritz, Goossens, Draijer, & Achterberg, 2013; Resnick et al., 1993). A study examining prevalence rates of trauma among PLWH found that 32% of women and 47% of men reported childhood sexual abuse, and 34% of women and 27% of men reported childhood physical abuse (Kalichman et al., 2002). Moreover, 68% of women and 35% of men reported that they had been sexually abused after the age of 15-years-old. Strikingly, 80% of participants reported 2 or more sexual assaults, with a sample mean of 10 sexual assaults. In contrast, an epidemiological study

examining trauma rates in the general population found that 14.7% of women and 4.5% of men reported a history of sexual assault, and 8.1% of women and 2.2% of men reported a history of being raped (Mills et al., 2011).

Due in part to the high levels of interpersonal trauma experienced, PLWH also have high rates of PTSD, with rates of PTSD ranging from 22-60% (Atkinson et al., 1988; Bing et al., 2001). Moreover, since the publication of the Diagnostic and Statistical Manual of Mental Disorders fourth edition (DSM-IV), a diagnosis of a life-threatening illness also has the potential to lead to PTSD (American Psychiatric Association (APA), 1994). Research has found that approximately 30% of people with HIV meet criteria for PTSD at some point during their lives in response to their HIV diagnosis (Stine & Kosten, 1999). In stark contrast to the rates of PTSD among PLWH, rates of PTSD in the general population are much lower and range between 7 and 10% (Kessler at al., 1995; Kessler el al., 2005).

According to the DSM-IV, text revision (DSM-IV TR; APA, 2000), PTSD is classified as an anxiety disorder that may develop in response to a specific traumatic event, such as witnessing or experiencing violence, physical or sexual assault, automobile accident, natural disaster, etc.). As stipulated in the DSM-IV, an individual needs to satisfy the following criteria in order to qualify for a PTSD diagnosis: (A1) an individual must experience, witness, or be confronted with a situation that involved actual or perceived threat of death or serious injury to ones' self or others; and (A2) the individual's response had to involve intense fear, helplessness, and/or horror. As stated previously, with the publication of the DSM-IV, a diagnosis of a terminal disease or life-threatening illness was included as a sufficient A1 criterion for a potentially traumatic event that has the ability to lead to a diagnosis of PTSD (APA, 1994). The DSM-IV separated PTSD symptoms into three clusters: (B) re-experiencing symptoms (such as

intrusive recollections of the event, flashbacks, etc.), (C) avoidance of people, places, or situations that serve as a reminder of the event, and (D) hyperarousal symptoms (such as difficulty falling asleep or maintaining sleep, elevated heart rate, etc.). In order to receive a PTSD diagnosis, symptoms must persist for at least one month (E), and the symptoms must cause clinically significant distress and disruption of functioning (F).

One of the most researched risk factors for developing PTSD following a trauma is having had a history of prior traumatic experiences. For example, Irish et al. (2008) found that number of lifetime trauma types experienced was one of the most meaningful predictors of PTSD development in a sample of motor vehicle accident (MVA) survivors. Similar findings were reported in active-duty military personnel; Agorastos et al. (2014) found that a larger number of different trauma types experienced in childhood were related to the development of more severe adult PTSD and depressive symptoms. Trauma history may also play a role in the development of negative outcomes associated with PTSD symptoms, such as poor physical health. For instance, the number of different trauma types experienced helped to predict worse physical health outcomes 6-months post-MVA (Irish et al., 2012).

In addition to poorer physical health outcomes (i.e., Olatunji, Cisler, & Tolin, 2007; Pietrzak, Goldstein, Southwick, & Grant, 2011), PTSD has been associated with a number of other negative outcomes including reduced quality of life (i.e., Mendlowicz & Stein, 2000), poor health behaviors, such as risky sexual behaviors, tobacco use, and low levels of exercise (i.e.; Breslau, Davis, & Schultz, 2003; Buckley, Mozley, Bedard, Dewulf, & Greif, 2004), and suicidal ideation (i.e., Panagota, Gooding, Taylor & Tarrier, 2014; Smith, Armelie, Boarts, Brazil, & Delahanty, in press). Moreover, PTSD rarely occurs alone, and is frequently comorbid with other disorders, such as depression, anxiety disorders, substance use disorders, and personality

disorders. For example, a meta-analysis of 57 studies found comorbidity of PTSD and major depressive disorder (MDD) to be 52% (Rytwinski, Scur, Feeny, & Youngstrom, 2013). Further, research has found that prevalence rates of alcohol and drug use disorders among people with PTSD is 46% (Pietrzak, Goldstein, Southwick, & Grant, 2011), Moreover, 58% of inpatients and 25% of outpatients with borderline personality disorder also meet diagnostic criteria for PTSD (Barnicot & Priebe, 2013). The high comorbidity of these disorders with PTSD makes effective treatment challenging for clinicians (i.e., Mills, Teeson, Baker, Hopewood, & Back, 2010; Rizvi & Harned, 2013).

PTSD in the context of PLWH is especially concerning because of the negative outcomes associated with PTSD, especially risky sexual behaviors, lower medication adherence, and lower CD4 cell counts (i.e., Brief, Bollinger, & Vielhaur, 2004; Reilly, Clark, Schmidt, Benight, & Kissinger, 2009). For example, one study examining the relationship between PTSD and HIV sexual risk-taking among men who have sex with men found that higher PTSD symptoms were related to more risky sexual behavior (Reisner, Mimiaga, Safren, & Mayer, 2009). Further, another study examining the relationship between PTSD and HIV sexual risk behaviors among women experiencing interpersonal partner violence (IPV) found that women who had PTSD had four times the odds of engaging in risky sexual behaviors than women who experienced IPV but did not have PTSD (Cavenaugh & Hansen, 2010). Another negative outcome associated with PTSD of particular relevance to PLWH is lower medication adherence. For example, Delahanty, Bogart, and Figler (2004) found that PTSD was related to lower levels of reported medication adherence, with medication non-adherence associated with higher HIV viral load. Research has also found that PTSD is associated with lower CD4 cell counts in PLWH (i.e., Brief, Bollinger, & Vielhaur, 2004; Reilly et al., 2009). Lower CD4 counts are directly related to HIV stage

progression and being diagnosed with acquired immune-deficiency syndrome (AIDS), the final

stage of HIV (AIDS.gov, 2014). Lastly, due to the lower immune-functioning associated with

HIV/AIDS (i.e., O'Leary, 1990), the negative health outcomes associated with PTSD may be

especially concerning when PTSD is comorbid with HIV (i.e., Lauterbach, Vora, Rakow, 2005).

Given the myriad of adverse psychiatric, psychosocial, and medical outcomes associated

with PTSD, it is important to utilize efficacious treatments to reduce symptoms and,

consequently, reduce other negative effects associated with these symptoms. Prolonged exposure

(PE) therapy is an evidence-based cognitive-behavioral therapy that has been shown to be

efficacious in the treatment of PTSD symptoms in a variety of different samples exposed to a

range of different index traumas (Foa, 2005; Powers, Halpern, Farenschak, Gillihan, & Foa,

2010). For example, PE has been shown to significantly reduce PTSD symptoms in combat

veterans in a wide variety of wars and conflicts, as well as in sexual assault survivors (i.e.,

Goodson, Lefkowitz, Helstrom, & Gawrysaik, 2013; Powers et al, 2010; Rauch et al., 2009;

Rothbaum, Astin, & Marsteller, 2005) and among PLWH (Pacella et al., 2012). A 2010 meta-

analysis of 13 randomized controlled trials (RCT) concluded that PE was significantly better

than control conditions at reducing PTSD symptoms as well as producing better secondary

outcomes (i.e., reducing depression) both following treatment and at follow-up assessments

(Powers et al., 2010). PE typically ranges from 8 to 15 sessions (Foa, 2005). Sessions one and

two of PE primarily consist of psychoeducation and gathering of information, while the later

sessions focus on repeated applications of imaginal and in-vivo exposure anchored to the index

trauma (Foa, 2005; Powers, et al., 2010). Although PE has not been shown to produce

significantly different outcomes than other trauma focused therapies (i.e., cognitive processing

therapy), it is considered to be the gold-standard for PTSD symptom reduction. The American

Psychological Association concluded that PE is strongly supported by research and is a "well-established" treatment for PTSD (Powers et al., 2010). Moreover, PE was selected or recommended by major healthcare administrations (i.e., Veterans' Administration Office of Mental Health and the Substance Abuse and Mental Health Services Administration) for nationwide dissemination (Powers, 2010).

Parent study

In the parent study to the present research, Pacella and colleagues (2012) conducted a randomized controlled trial (RCT) with a cross-over design to examine the efficacy of PE at reducing PTSD symptoms, depressive symptoms, and post-traumatic cognitions among a sample of PLWH. Participants were randomly assigned to either the PE treatment group or the control group. Thirty-four participants received PE therapy and 24 participants served as the waitlist control. Results indicated that the PE group experienced significant reductions in PTSD symptoms, depressive symptoms, and negative post-traumatic cognitions compared to the control group.

Current study

Although PE has been found to be efficacious in reducing PTSD symptoms as well as secondary outcomes (such as depression), we know very little about how PTSD symptom change occurs during the course of PE. Prior research has exclusively examined symptom reduction pre- to post-treatment. This creates in effect, the illusion that symptom reduction occurs suddenly or all-at-once, and ignores the fact that symptoms change over time during the course of therapy. Empirically examining how symptoms change during the course of PE will provide a better understanding of how to potentially tailor treatments to the individual and maximize efficacy of PE.

In order to better understand how PTSD symptoms change throughout the course of PE, the current study had the following aims:

1)   Examine how PTSD symptoms and the symptom clusters of re-experiencing, avoidance, and hyperarousal symptoms changed during the course of PE.

2)   Examine whether trauma history moderated the slope of PTSD symptoms and the symptom clusters of re-experiencing, avoidance, and hyperarousal during the course of PE.

3)   Examine whether experiencing an HIV diagnosis-related or non-HIV-related index trauma moderated the slope of PTSD symptoms and the symptom clusters of re-experiencing, avoidance, and hyperarousal during the course of PE.

4)   Examine whether age moderated the slope of PTSD symptoms and the symptom clusters of re-experiencing, avoidance, and hyperarousal during the course of PE.

5)   Examine whether gender moderated the slope of PTSD symptoms and the symptom clusters of re-experiencing, avoidance, and hyperarousal during the course of PE.

More specifically, we hypothesized that: 1) PTSD symptoms and the symptom clusters of re-experiencing, avoidance, and hyperarousal symptoms would be significantly reduced over the course of PE; 2) trauma history would moderate the slope of PTSD symptoms and the symptom clusters of re-experiencing, avoidance, and hyperarousal during the course of PE, such that a more substantial trauma history would result in less symptom reduction over the course of PE than a less substantial trauma history; and 3) experiencing an HIV diagnosis-related index trauma would result in less PTSD symptom and PTSD symptom cluster (re-experiencing, avoidance, and hyperarousal) reduction during the course of PE than those whose index trauma was non-HIV-related. We made no a priori hypotheses regarding whether age and gender would

moderate the slope of PTSD symptoms and the symptom clusters of re-experiencing, avoidance, and hyperarousal during the course of PE (aims 4 and 5, respectively).

METHOD

Participants

All participants were recruited from two social service agencies near Cleveland, OH, between 2005 and 2008 as part of the parent study. See Figure 1 for a flow chart of participant progress from the parent study to the current study. Initially, 99 adult PLWH were screened in order to determine eligibility for the study. Inclusion criteria for the study were fluency in English, meeting criteria for a likely diagnosis of PTSD as assessed by the self-report PTSD Diagnostic Scale (PDS; Foa, 1995), and currently taking antiretroviral medications for HIV. Exclusionary criteria for the study were a diagnosis of a psychotic disorder, a current or previous diagnosis of schizophrenia, and current suicidal ideation. Of the 99 PLWH initially screened, 34 did not meet requirements of the study; therefore, 65 eligible PLWH (24 male, 41 female) completed the baseline questionnaires.

Thirty-four participants were randomly assigned to the PE treatment group, and 24 were randomly assigned to the waitlist control (weekly monitoring) group. At the post-intervention assessment, 23 PLWH were retained in the PE treatment group (32% attrition rate), while 24 participants were retained in the waitlist control group (0% attrition rate). At the 3-month follow-up, 19 PE treatment group participants were retained and all waitlist control participants were retained. After completion of the 3-month assessment, participants in the waitlist control group were all offered the opportunity to receive the PE intervention, and 19 participants in the waitlist control group agreed to receive PE. For the current study, we combined all participants who received PE as either part of the PE treatment group (n = 28) or later as part of the waitlist control group (n = 19), for a total of 47 PLWH who received PE.

Study Groups

Prolonged exposure

The PE intervention was conducted individually in a private room within the social service agency by one of two clinical psychology post-doctoral fellows who received extensive training in administering PE. In addition to their PE training, the therapists were given feedback throughout the course of the intervention from experts in PE in order to ensure adherence to the PE therapeutic protocol. Each participant was only seen by one therapist throughout the duration of their PE therapy in order to maintain continuity of treatment. The treatment followed standard PE protocol and it consisted of 10 sessions, which were conducted twice a week for 5 weeks, and each PE session lasted between 90 and 120 minutes. Treatment procedures included education about common reactions to trauma, breathing retraining, prolonged exposure to trauma memories, repeated in-vivo exposure to situations the client was avoiding due to trauma-related fear, and discussion of thoughts and feelings related to exposure exercises. The structure and format of sessions remained consistent with the PE protocol, with content of sessions varying to meet individual client needs. The foci of PE sessions were individually tailored to address the most severe index trauma reported by the participant whether it is HIV diagnosis-related or non-HIV-related trauma.

Weekly monitoring/waitlist control

Participants assigned to the weekly monitoring/waitlist control group continued with their standard visits to the social service agency, but they were also contacted by their case manager once a week for five weeks (the same as the duration of PE therapy) in person or by telephone to ensure that they were not experiencing any symptom exacerbation.

Procedure

The study protocol was approved by the human subjects review boards of Kent State University and the Summa Health System. Potential participants were initially approached about the study by their case manager at the social service agency. Participants who were interested completed the PDS (Foa, 1995) to determine whether they were eligible to participate in the study. Those participants who met likely criteria for PTSD then met with a Ph.D.-level clinical psychology student who described the study in detail to them. Participants who were still interested in the study then provided written informed consent. The Structured Clinical Interview with Psychotic Screen was then administered in order to confirm the presence of diagnostic levels of PTSD, including the endorsement of criterion A, associated functional impairment, and the absence of any psychotic disorder or other exclusionary criteria.

The baseline assessment occurred within one week of the initial screening protocol. All eligible participants were administered the PTSD Symptom Scale-Interview (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993) with regards to both PTSD related to the HIV diagnosis and in regards to the most severe non-HIV-related trauma. They then completed self-report questionnaires including demographic information, the Center for Epidemiological Studies-Depression Scale (CES-D; Radloff, 1997), the Posttraumatic Cognitions Inventory (PTCI; Foa, Ehlers, Clark, Tomlin & Orsillo, 1999), and a self-report scale assessing substance use over the past 30 days. Following the baseline assessment, participants were randomly assigned to either the PE treatment group of the waitlist control group. The principal investigator (DLD) generated the allocation sequence using blocked randomization (4:3 ratio of experimental:control participants), and the graduate student conducting the assessments remained blind to group membership. Unequal numbers of participants were assigned to each group, as it was anticipated

that the PE group would have a higher dropout rate. Consistent with therapy protocol, PE participants completed the PSS-I every other session (e.g., 2, 4, 6, 8, and 10) to track symptom change throughout the course of PE therapy. All follow-up assessments were conducted at the social service agency by the same blind interviewer who conducted the baseline assessment. Participants received $25 for each assessment and $10 for every PE session they completed, with an extra $25 bonus if they completed all 10 PE sessions.

Measures

Sociodemographics

Standard demographic questions about age, gender/sex, race/ethnicity, number of years living with HIV, and income were obtained at baseline.

Trauma History

For the current study, we defined trauma history as the total number of lifetime trauma types experienced. In order to assess this, we asked participants to fill out a modified, self-report version of the Traumatic Stress Schedule (TSS; Norris, 1990). It asked about lifetime exposure to nine different types of traumatic events: Robbery/mugging, physical assault, sexual assault, unexpected loss of a loved one, injury or property damage due to a fire, injury or property damage due to a nature or mane-made disaster, serious MVA, seeing another person seriously injured or killed, and serious injury due to an accident other than an MVA. In addition, an opened-ended question was added for participants to write in any other experiences they thought were traumatic that had not been covered by the nine types of events mentioned above. The overall number of lifetime trauma types experienced was calculated by summing the number of traumas that were endorsed on the checklist.
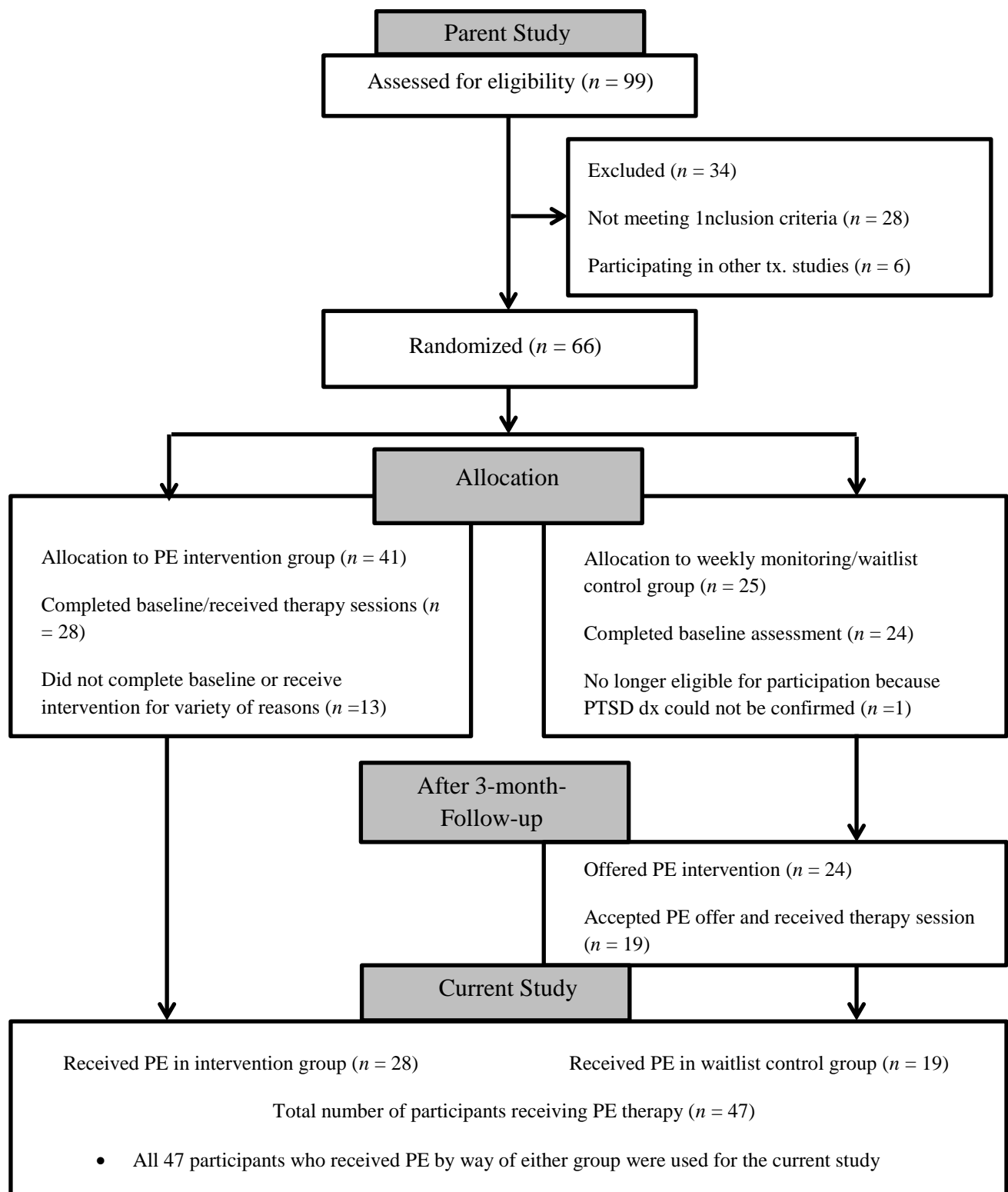
PTSD Symptoms

All participants completed the 17-item Posttraumatic Stress Diagnostic Scale (Foa, 1995) at screening to determine whether they met likely criteria for a PTSD diagnosis. Participants were asked to rate the frequency with which they experienced each of the 17 PTSD symptoms corresponding to the criteria in the DSM-IV (APA, 2000) within the past month. The PDS is a well-validated measure of PTSD and PTSD symptoms (Foa, Cashman, Jaycox, & Perry, 1997). A likely diagnosis of PTSD was considered present if the participant endorsed at least one re-experiencing symptom, three avoidance symptoms, and two hyperarousal symptoms. Diagnoses were verified at screening with the SCID with Psychotic Screen for the DSM-IV (First et al., 1997). The SCID is a gold standard structured interview that allows interviewers to make psychiatric diagnoses based on DSM-IV criteria.

At each assessment, participants completed the PTSD Symptom Scale-Interview (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993). The PSS-I is a 17-item semi-structured interview designed to measure the frequency and severity of PTSD symptoms. The total score is calculated based on the sum of the individual items. Internal consistency for the PSS-I was acceptable in our sample (Baseline alpha = .79, session 2 = .86, session 4 = .85, session 6 = .92, session 8 = .89, and session 10 = .94.)

HIV-related vs. non-HIV-related index trauma

At baseline, we gave the PSS-I (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993) to each participant twice. In the first version participants were instructed to answer the questions in reference to their PTSD symptoms related to their HIV diagnosis, and in the other version they were instructed to answer the questions in reference to the most severe non HIV-related trauma. We then summed each PSS-I and selected the highest PTSD symptom score as the index trauma. We anchored all other PSS-I assessments during the course of PE to their index trauma.

Parent Study

Assessed for eligibility (*n* = 99)

Excluded (*n* = 34)

Not meeting 1nclusion criteria (*n* = 28)

Participating in other tx. studies (*n* = 6)

Randomized (*n* = 66)

Allocation

Allocation to PE intervention group (*n* = 41)

Completed baseline/received therapy sessions (*n* = 28)

Did not complete baseline or receive intervention for variety of reasons (*n* =13)

Allocation to weekly monitoring/waitlist control group (*n* = 25)

Completed baseline assessment (*n* = 24)

No longer eligible for participation because PTSD dx could not be confirmed (*n* =1)

After 3-month-Follow-up

Offered PE intervention (*n* = 24)

Accepted PE offer and received therapy session (*n* = 19)

Current Study

Received PE in intervention group (*n* = 28)   Received PE in waitlist control group (*n* = 19)

Total number of participants receiving PE therapy (*n* = 47)

- All 47 participants who received PE by way of either group were used for the current study

Figure 1. *Flow Diagram of participation progress from parent study to current study.*

DATA ANALYSIS PLAN

Cleaning of data and structuring of data set

      Before any analyses could be conducted, data had to be merged from multiple datasets into one single dataset and restructured. First, select variables were merged from the main dataset that included baseline assessments and various level 2 data (such as trauma history, gender, and age) into a within session data set that contained only level 1 data. After merging these two files, there were a total of six assessments per person: One baseline measure of PTSD symptoms and five follow-up assessments of PTSD symptoms. The follow-up assessments were collected during PE sessions 2, 4, 6, 8, and 10. The final dataset was restructured from a person-level dataset to a person-period dataset. More specifically, after the dataset restructure, each participant had six rows of data (one for each time point) and a time variable that allowed for examination of the outcome variable at each time point. This new data structure facilitated the use of multilevel modeling for data analysis. A PTSD symptom severity variable was also created that contained the PTSD symptom severity sum score for each of the respective time points. Similarly, a re-experiencing symptom total score was created by summing the PSS-I items that corresponded to DSM-IV re-experiencing/intrusions symptoms, an avoidance symptom total score was created by summing the PSS-I items that corresponded to DSM-IV avoidance/numbing symptoms, and a hyperarousal symptom total score was created by summing the PSS-I items that corresponded to DSM-IV hyperarousal symptoms. All missing data were handled using the conservative last assessment forward (LAF) method.

MLM Growth Curve Modeling for Aim One

      All models were conducted using HLM 7.0 student version. The first aim of this study was to examine the effect of number of PE sessions had on PTSD symptom severity over time

and to examine the shape of the curve. Multilevel modeling (MLM) growth curve analysis is ideally suited to modeling change over time. MLM allows for the use of nested models and enables each part of the variance to be divided into its respective level. For repeated measure designs, such as growth models, MLM allows for analyses of time (level 1) nested within persons (level 2). Further, MLM growth curve analyses do not require the large number of participants that structural equation modeling (SEM) techniques require for latent growth curve analyses. Another benefit to MLM growth curve techniques is that MLM models are less sensitive than both repeated measures ANOVA and SEM models to missing data, giving MLM better statistical power, more modeling flexibility, and better chance of convergence than these other longitudinal techniques. Given the relatively small sample size of 47, MLM growth curve analysis was an appropriate choice for the first aim of the current study.

Due to the fact that, when working with real-world data, change over time is often not linear, non-linear/quadratic change was examined by creating squared time variables and adding them incrementally as predictors in the model. The time squared function allows for one bend in the slope line. After running the MLM growth models, the change over time was then graphed using Microsoft Excel 2010.

Examining Moderators of the slope of symptom change over the course of PE: Aims two, three, four, and five

All models were conducted using HLM 7.0 student version. For aims two, three, four, and five, we wanted to examine moderators of the slope of symptoms changes throughout the course of PE. This involved the use of cross-level interactions. For aim 2, we examined the interaction (or multiplicative effect) of trauma history (level 2) and time (level 1) on change of PTSD symptoms and the symptom clusters of re-experiencing symptoms, avoidance symptoms,

and hyperarousal symptoms over the course of PE. For aim 3, we examined the interaction of index trauma type (HIV-related vs. non-HIV-related; level 2) and time (level 1) on change of PTSD symptoms and the symptom clusters of re-experiencing symptoms, avoidance symptoms, and hyperarousal symptoms over the course of PE. For aim 4, we examined the interaction of age (level 2) and time (level 1) on change in PTSD symptoms and the symptom clusters of re-experiencing symptoms, avoidance symptoms, and hyperarousal symptoms over the course of PE. Lastly, for aim 5, we examined the interaction of gender (level 2) and time (level 1) on change in PTSD symptoms and the symptom clusters of re-experiencing symptoms, avoidance symptoms, and hyperarousal symptoms over the course of PE. For trauma history, we created new variables at one standard deviation above and below the mean and used them in the interaction equation.

RESULTS

Descriptive Statistics

　　　　The current sample consisted of 47 PLWH who received PE therapy, of which 57% (n =

27) were male and 43% (n = 20) were female. The age of the sample ranged from 31- to 60-

years-of-age, with a mean age of 46-years (SD = 6.41). Fifty-one percent (n = 24) of participants

identified as African American, 34% (n = 16) identified as Caucasian, 4.3% (n = 2) identified as

Hispanic/Latino, 8.5% (n = 4) identified as mixed race, and 2.1% (n = 1) identified as other.

Further, the participants experienced on average (M) a total of 4.98 (SD = 1.88) lifetime trauma

types. For descriptive statistics of PTSD symptoms, re-experiencing symptoms, avoidance

symptoms, and hyperarousal symptoms, see Table 1.

Table 1. *Illustrating Means and Standard Deviations of PTSD, Re-Experiencing, Avoidance, and Hyperarousal Symptoms.*

	Baseline **Mean (SD)**	**Session 2** **Mean (SD)**	**Session 4** **Mean (SD)**	**Session 6** **Mean (SD)**	**Session 8** **Mean (SD)**	**Session 10** **Mean (SD)**
PTSD Symptoms	32.60 (1.88)	28.11 (9.53)	25.97(9.05)	20.81 (10.78)	17.23 (9.77)	16.04 (11.05)
Re-experiencing	8.81 (2.89)	7.81 (3.27)	7.63 (3.17)	5.60 (3.45)	4.68 (3.38)	4.32 (3.53)
Avoidance Symptoms	14.38 (3.71)	11.62 (4.55)	10.43 (4.61)	8.70 (4.92)	7.04 (4.83)	6.49 (5.00)
Hyperarousal	9.40 (2.95)	8.68 (3.18)	7.91 (3.17)	6.51 (3.97)	5.66 (3.20)	5.32 (3.71)

Note. SD = standard deviations

MLM Growth Curve Analyses: Hypothesis One

First, in order to examine whether there was a significant linear change in PTSD symptoms over the course of PE, the following model was examined:

$$Y = \pi_{0} + \pi_{1}\,(\text{time}) + e$$

$$\pi_{0} = \beta_{00} + r_{0}$$

$$\pi_{1} = \beta_{10}$$

where Y represents PTSD symptom severity, π_{0} represents the intercept, π_{1} (time) represents the slope for time of PTSD symptom assessment, e represents level 1 residual error, and r_{0} represents residual error in intercepts (level 2). Equation one is basically a simple regression equation. Equation two makes this a multilevel model, by allowing a random effect for the intercept (r_{0}). This takes into account the nested nature of time within person by allowing each person to have their own unique intercept. Results of this model revealed significant linear change in PTSD symptoms over the course of PE, $\beta = -3.29$, $t(\sim224) = -13.16$, $p < .001$ (see Figure 2).

Because change in symptoms over time following therapy should not be assumed to be best explained by a straight line, the time squared quadratic model (allowing for one bend in the slope line) was examined:

$$Y = \pi_{0} + \pi_{1}\,(\text{time}) + \pi_{2}\,(\text{time}^{2}) + e$$

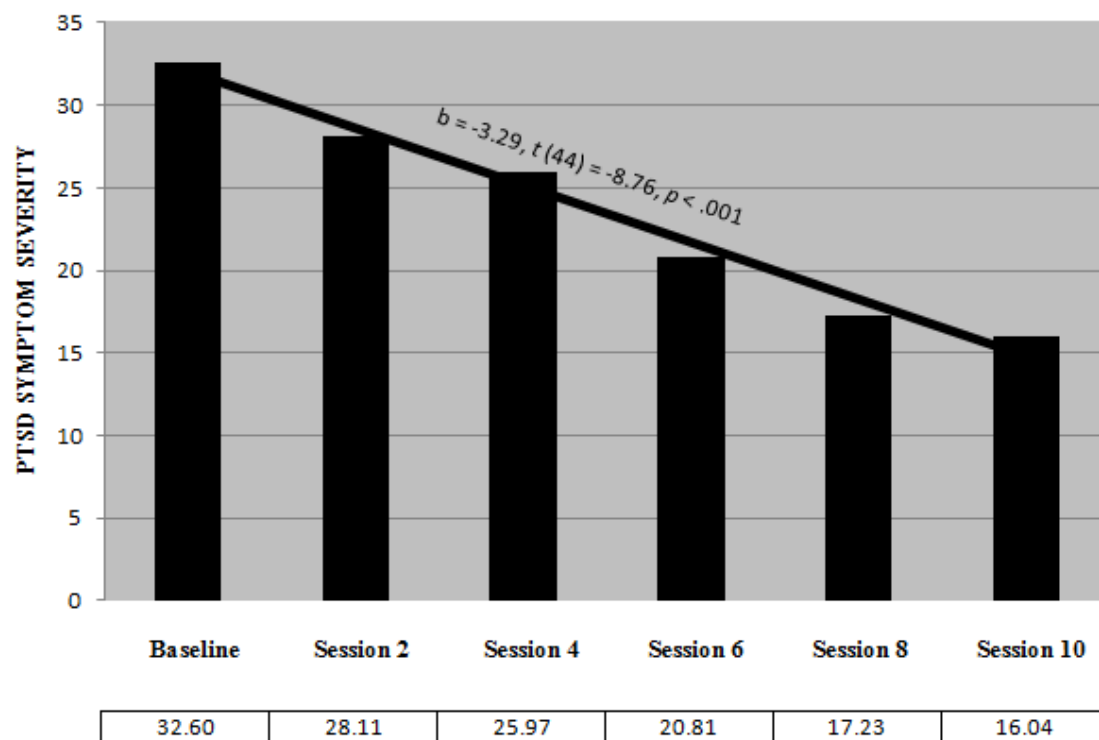$$\pi_{0} = \beta_{00} + r_{0}$$

$$\pi_{1} = \beta_{10}$$

$$\pi_{2} = \beta_{20}$$

where Y represents PTSD symptom severity, π_{0} represents the intercept, π_{1} (time) represents the slope for time of PTSD symptom severity assessment, π_{2} (time^{2}) represents the slope for time of

21

PTSD symptom severity assessment squared, e represents level 1 residual error, and r_0 represents residual error in intercepts (level 2).

Results of this model revealed that the time squared quadratic function was non-significant in the prediction of PTSD symptom change during PE, $\beta = .20$, $t(\sim223) = 1.16$, $p = .247$. These results indicate that PTSD symptom change during the course of PE was best explained by a linear function. In other words, PTSD symptom change throughout the course of PE was best explained as a constant rate of symptom reduction throughout PE.

Figure 2. *Plotted Means Illustrating PTSD Symptom Change over the Course of PE.*

Baseline	Session 2	Session 4	Session 6	Session 8	Session 10
32.60	28.11	25.97	20.81	17.23	16.04

Next, in order to examine the rate of change for each of the PTSD symptom clusters of re-experiencing, avoidance, and hyperarousal, we examined the following models:

Re-experiencing symptom change:

$$Y = \pi_{0} + \pi_{1} \,(\text{time}) + e$$

$$\pi_{0} = \beta_{00} + r_{0}$$

$$\pi_{1} = \beta_{10}$$

where Y represents re-experiencing symptom severity, π_{0} represents the intercept, π_{1} (time) represents the slope for time of re-experiencing symptom assessment, e represents level 1 residual error, and r_{0} represents residual error in intercepts (level 2). Results of this model revealed significant linear change in re-experiencing symptoms over the course of PE, $\beta = -.92$, $t(\sim 224) = -9.88$, $p < .001$ (see Figure 3).

Avoidance symptom change:

$$Y = \pi_{0} + \pi_{1} \,(\text{time}) + e$$

$$\pi_{0} = \beta_{00} + r_{0}$$

$$\pi_{1} = \beta_{10}$$

where Y represents avoidance symptom severity, π_{0} represents the intercept, π_{1} (time) represents the slope for time of avoidance symptom assessment, e represents level 1 residual error, and r_{0} represents residual error in intercepts (level 2). Results of this model revealed significant linear change in re-experiencing symptoms over the course of PE, $\beta = -1.52$, $t(\sim 223) = -9.19$, $p < .001$ (see Figure 4).

Figure 3. *Plotted Means Illustrating Re-Experiencing Symptom Change over the Course of PE.*

| 8.81 | 7.81 | 7.63 | 5.6 | 4.68 | 4.32 |

Figure 4. *Plotted Means Illustrating Avoidance Symptom Change over the Course of PE.*

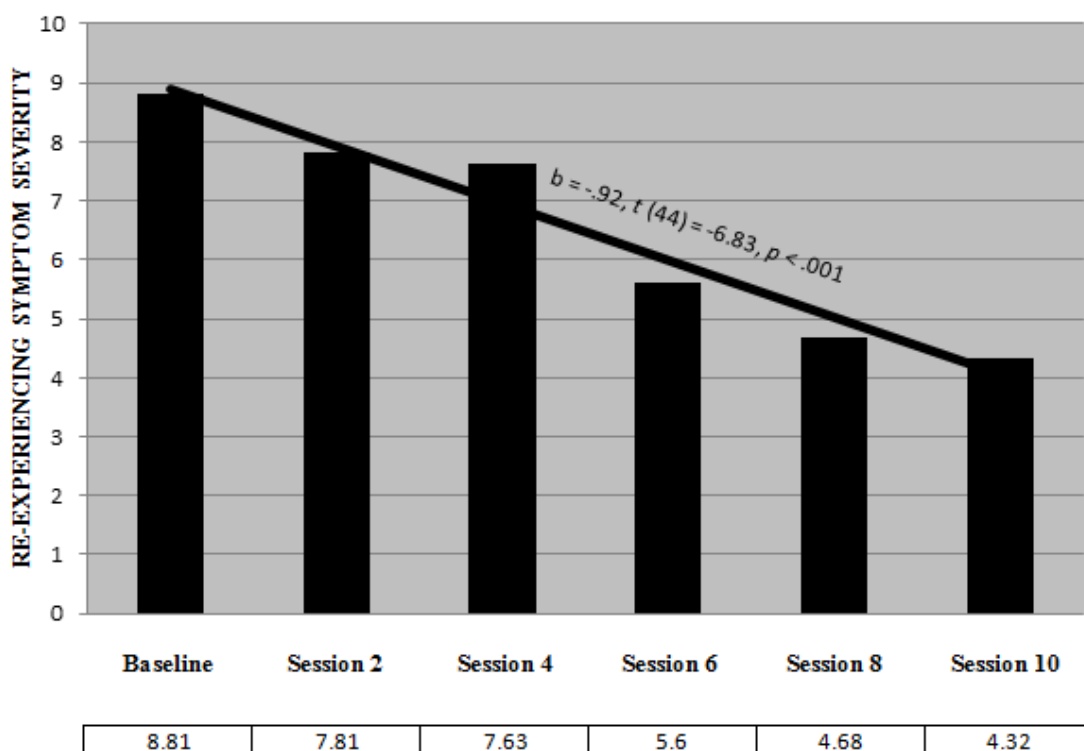| 14.38 | 11.62 | 10.43 | 8.70 | 7.04 | 6.49 |

Hyperarousal symptom change:

$$Y = \pi_{0} + \pi_{1} \, (\text{time}) + e$$

$$\pi_{0} = \beta_{00} + r_{0}$$

$$\pi_{1} = \beta_{10}$$
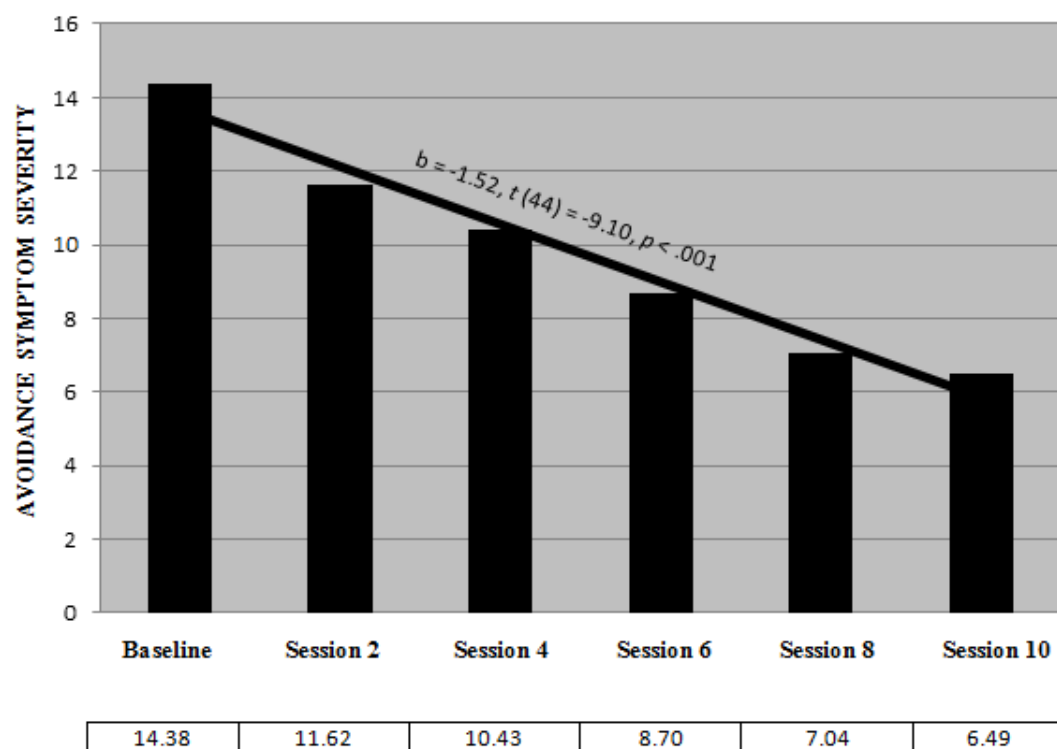
where Y represents hyperarousal symptom severity, π_{0} represents the intercept, π_{1} (time) represents the slope for time of hyperarousal symptom assessment, e represents level 1 residual error, and r_{0} represents residual error in intercepts (level 2). Results of this model revealed significant linear change in re-experiencing symptoms over the course of PE, $\beta = -.84$, $t(\sim224) = -9.79$, $p < .001$ (see Figure 5).

Figure 5. *Plotted Means Illustrating Hyperarousal Symptom Change over the Course of PE.*

Baseline	Session 2	Session 4	Session 6	Session 8	Session 10
9.40	8.68	7.91	6.51	5.66	5.32
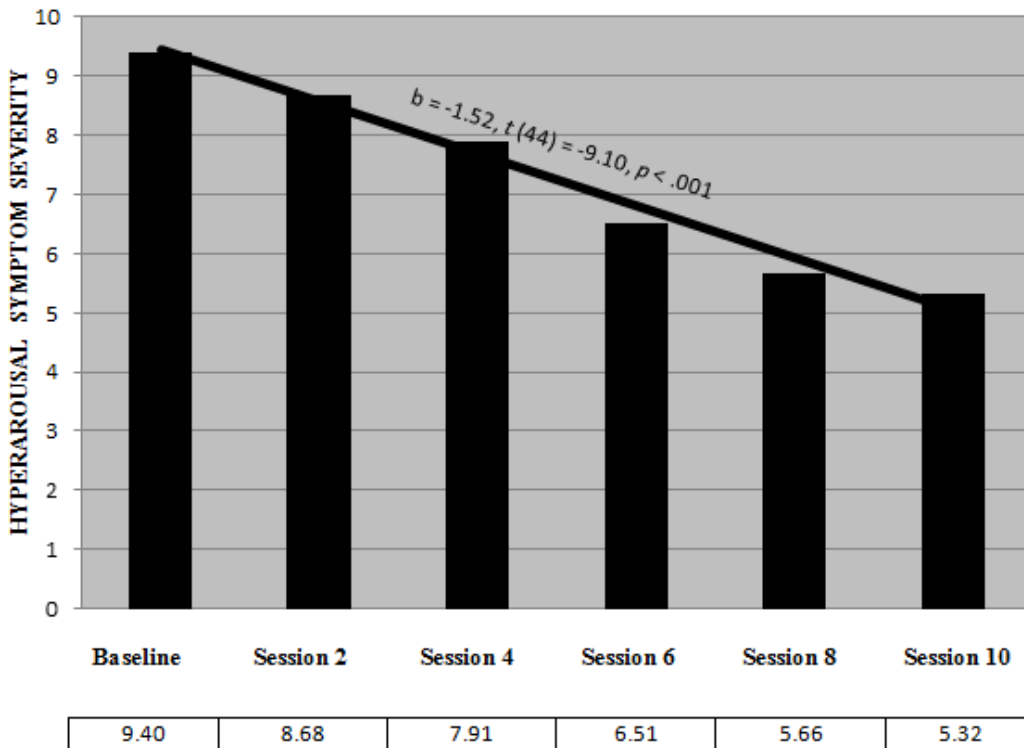
Examining HIV-related vs. non-HIV-related index trauma as a moderator of the slope of symptom change over the course of PE: Hypothesis two

In order to examine whether having an index trauma related to the HIV diagnosis or having an index trauma that was not HIV-related would moderate the rate of symptom reduction over the course of PE, we examined whether the index trauma would interact with time in the prediction of PTSD symptom change:
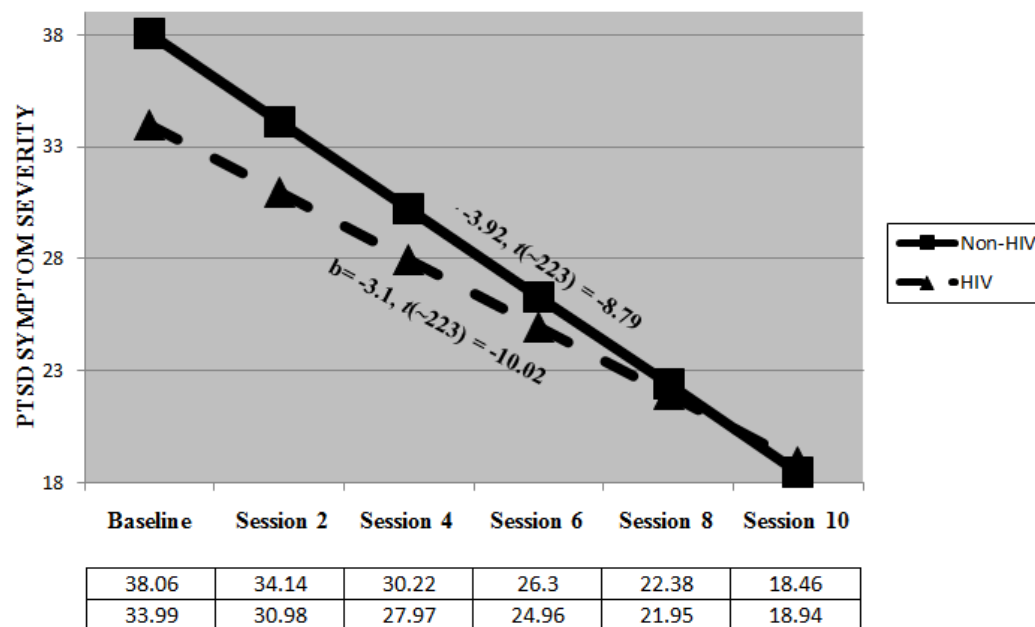
$$Y = \pi_{0 +} \pi_1 \,(\text{time}) + e$$

$$\pi_{0 =} \beta_{00} + \beta_{01} * (\text{Index Trauma}) + r_0$$

$$\pi_{1 =} \beta_{10} + \beta_{11} * (\text{Index Trauma})$$

where Y represents PTSD symptom severity, π_0 represents the intercept plus the main effect of index trauma, π_1 (time) represents the slope for time of PTSD symptom assessment plus the interaction of index trauma and time, e represents level 1 residual error, and r_0 represents residual error in intercepts (level 2). Results of this model revealed that there was not a significant interaction between the index trauma and time in the prediction of PTSD symptom change over the course of PE, $\beta = .59$, $t(\sim223) = 1.33$, $p = .186$ (see Figure 6). Although these results were non-significant, we wanted to examine whether the index trauma would interact with time in predicting the individual symptom clusters of re-experiencing, avoidance, and hyperarousal over the course of PE as opposed to PTSD symptoms as a whole.

Figure 6. *Plotted Estimated Means Illustrating that Index Trauma did not Moderate the effect of Time on Re-Experiencing Symptom Change over the Course of PE.*

Baseline	Session 2	Session 4	Session 6	Session 8	Session 10
38.06	34.14	30.22	26.3	22.38	18.46
33.99	30.98	27.97	24.96	21.95	18.94

Note. Non-HIV = non-HIV-related index trauma; HIV = Index trauma related to HIV diagnosis.
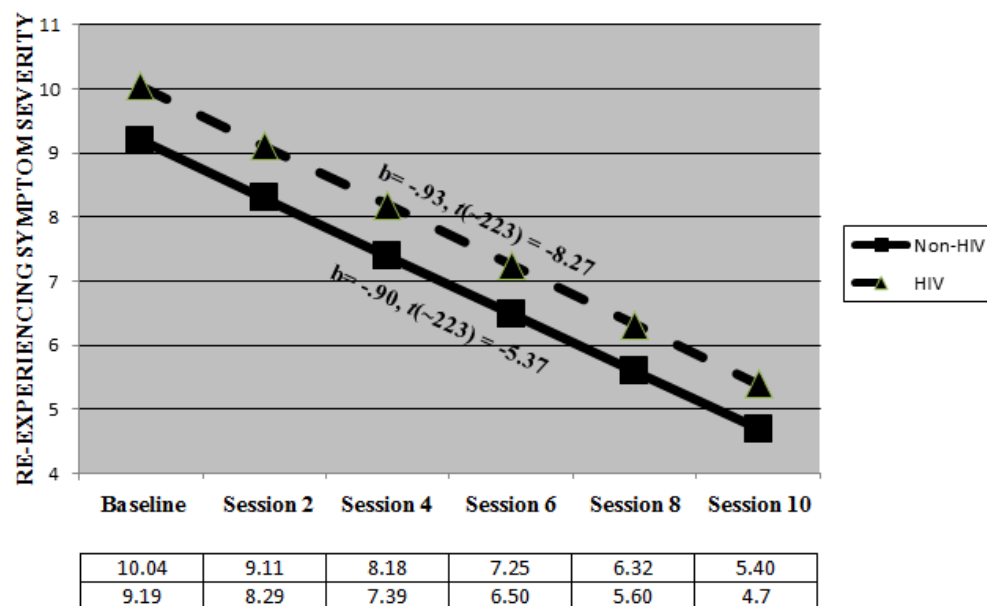
Re-experiencing symptom change:

$$Y = \pi_{0} + \pi_{1} \, (\text{time}) + e$$

$$\pi_{0} = \beta_{00} + \beta_{01} * (\text{Index Trauma}) + r_{0}$$

$$\pi_{1} = \beta_{10} + \beta_{11} * (\text{Index Trauma})$$

where Y represents re-experiencing symptom severity, π_{0} represents the intercept plus the main effect of index trauma, π_{1} (time) represents the slope for time of PTSD symptom severity assessment plus the interaction of index trauma and time, e represents level 1 residual error, and r_{0} represents residual error in intercepts (level 2). Results of this model revealed that there was not a significant interaction between the index trauma and time in the prediction of re-experiencing symptom change over the course of PE, $\beta = -.13$, $t(\sim223) = -.81$, $p = .419$ (see Figure 7).

Figure 7. *Plotted Estimated Means Illustrating that Index Trauma did not Moderate^ Time on Re-Experiencing Symptom Change over the Course of PE.*

	Baseline	Session 2	Session 4	Session 6	Session 8	Session 10
	10.04	9.11	8.18	7.25	6.32	5.40
	9.19	8.29	7.39	6.50	5.60	4.7

Note. Non-HIV = non-HIV-related index trauma; HIV = Index trauma related to HIV diagnosis.

^ $P = .878$

Avoidance symptom change:
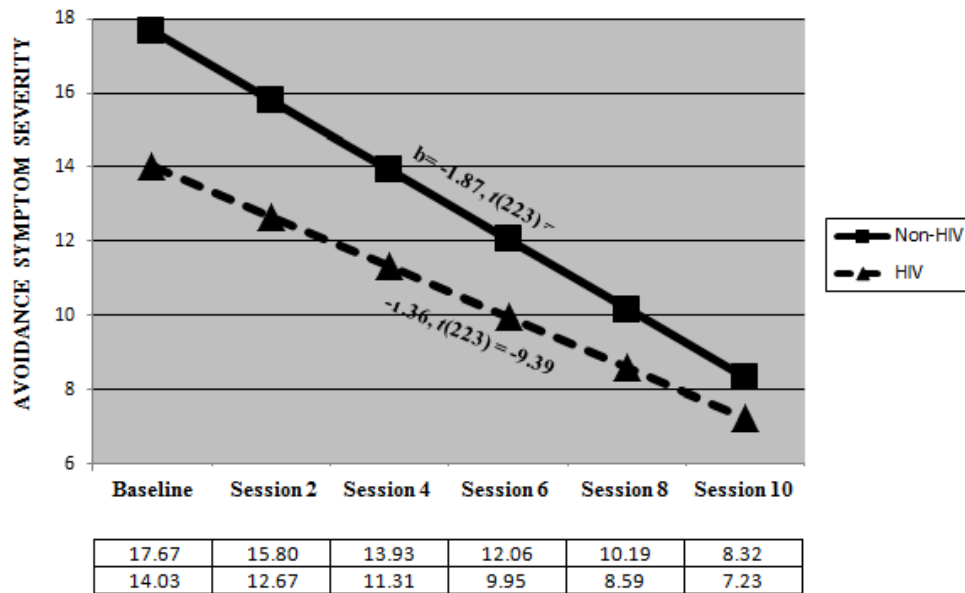
$$Y = \pi_{0+} \pi_1 \,(\text{time}) + e$$

$$\pi_0 = \beta_{00} + \beta_{01} * (\text{Index Trauma}) + r_0$$

$$\pi_1 = \beta_{10} + \beta_{11} * (\text{Index Trauma})$$

where Y represents avoidance symptom severity, π_0 represents the intercept plus the main effect

of index trauma, π_1 (time) represents the slope for time of PTSD symptom severity assessment

plus the interaction of index trauma and time, e represents level 1 residual error, and r_0 represents

residual error in intercepts (level 2). Results of this model revealed a marginally significant

interaction between the index trauma and time in the prediction of avoidance symptom change

over the course of PE, $\beta = .40$, $t(\sim223) = 1.86$, $p = .065$. Decomposition of this interaction

indicated that participants whose index trauma was related to their HIV diagnosis had a

shallower slope of avoidance symptom reduction (b = -1.36) during the course of PE than

participants whose index trauma was not related to their HIV diagnosis (b = -1.87) (see Figure

8).

Figure 8. *Plotted Estimated Means Illustrating the Trend-Level Moderating Effect^ of Index Trauma by Time on Avoidance Symptom Change over the Course of PE.*

	Baseline	Session 2	Session 4	Session 6	Session 8	Session 10
	17.67	15.80	13.93	12.06	10.19	8.32
	14.03	12.67	11.31	9.95	8.59	7.23

Note. Non-HIV = non-HIV-related index trauma; HIV = Index trauma related to HIV diagnosis.

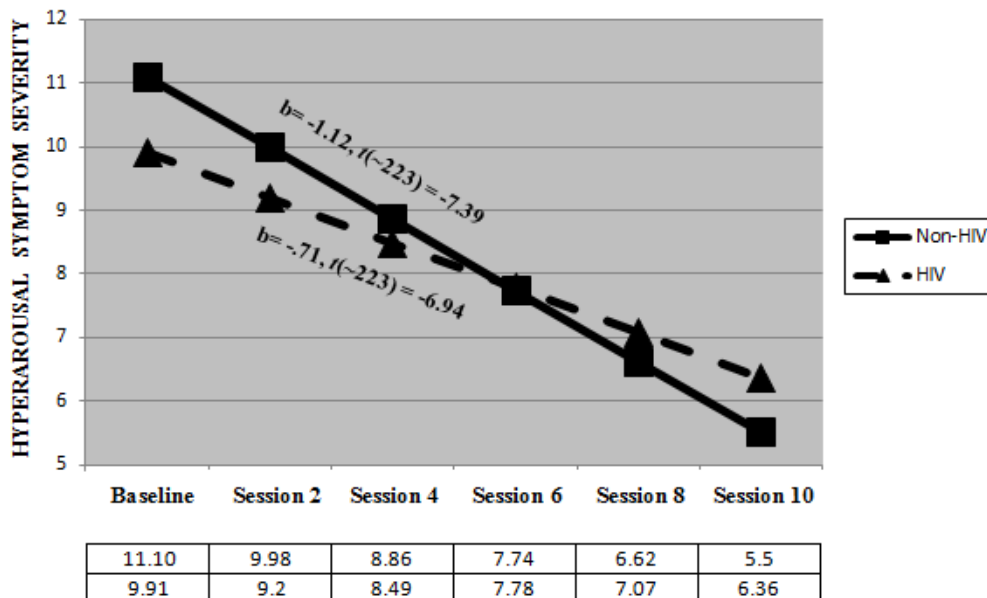^ *P* = .065

Hyperarousal symptom change:

$$Y = \pi_{0} + \pi_{1} \,(\text{time}) + e$$

$$\pi_0 = \beta_{00} + \beta_{01} * (\text{Index Trauma}) + r_0$$

$$\pi_1 = \beta_{10} + \beta_{11} * (\text{Index Trauma})$$

where Y represents hyperarousal symptom severity, π_0 represents the intercept plus the main effect of index trauma, π_1 (time) represents the slope for time of PTSD symptom severity assessment plus the interaction of index trauma and time, e represents level 1 residual error, and r_0 represents residual error in intercepts (level 2). Results of this model revealed a significant interaction between the index trauma and time in the prediction of arousal symptom change over the course of PE, $\beta = .33$, $t(\sim223) = 2.21$, $p = .028$. Decomposition of this interaction indicated that participants whose index trauma was related to their HIV diagnosis had a shallower slope of hyperarousal symptom reduction (b = -1.12) during the course of PE than participants whose index trauma was not related to their HIV diagnosis (b = -.71) (see Figure 9).

Figure 9. *Plotted Estimated Means Illustrating the Moderating effect* of Index Trauma by Time on Hyperarousal Symptom Change over the Course of PE.*

	Baseline	Session 2	Session 4	Session 6	Session 8	Session 10
	11.10	9.98	8.86	7.74	6.62	5.5
	9.91	9.2	8.49	7.78	7.07	6.36

Note. Non-HIV = non-HIV-related index trauma; HIV = Index trauma related to HIV diagnosis.

* $p < .05$

Examining trauma history as a moderator of the slope of symptom change over the course of PE:

Hypothesis three

In order to examine whether trauma history would moderate the rate of symptom reduction over the course of PE, we examined whether the trauma history would interact with time in the prediction of PTSD symptom change:
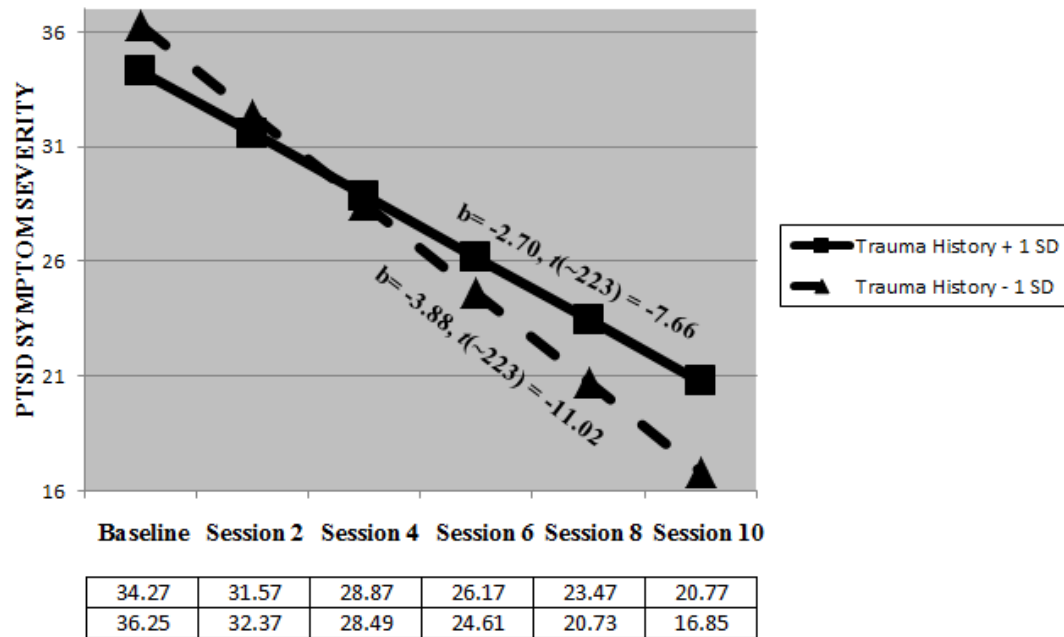
$$Y = \pi_{0 +} \pi_1 \text{(time)} + e$$

$$\pi_{0 =} \beta_{00} + \beta_{01} * \text{(trauma history)} + r_0$$

$$\pi_{1 =} \beta_{10} + \beta_{11} * \text{(trauma history)}$$

where Y represents PTSD symptom severity, π_0 represents the intercept plus the main effect of trauma history, π_1 (time) represents the slope for time of PTSD symptom severity assessment plus the interaction of trauma history and time, e represents level 1 residual error, and r_0 represents residual error in intercepts (level 2). Results of this model revealed a significant interaction between the trauma history and time in the prediction of PTSD symptom change over the course of PE, $\beta = .31$, $t(\sim223) = 2.36$, $p = .019$. Decomposition of this interaction indicated that participants who had experienced a greater number of lifetime trauma types had a shallower slope of PTSD symptom reduction (b = -2.70) during the course of PE than participants who experienced a lesser number of lifetime trauma types (b = -3.88) (see Figure 10). In order to examine whether this moderating effect of trauma history would be consistent across the symptom clusters of re-experiencing, avoidance, and hyperarousal symptom change over the course of PE, we examined the following models.

Figure 10. *Plotted Estimated Means Illustrating the Moderating effect* of Trauma History by Time on PTSD Symptom Change over the Course of PE.*

Baseline	Session 2	Session 4	Session 6	Session 8	Session 10
34.27	31.57	28.87	26.17	23.47	20.77
36.25	32.37	28.49	24.61	20.73	16.85

Note. Trauma History + 1 SD = Trauma History at 1 SD above the mean; Trauma History - 1 SD = Trauma History at 1 SD below the mean

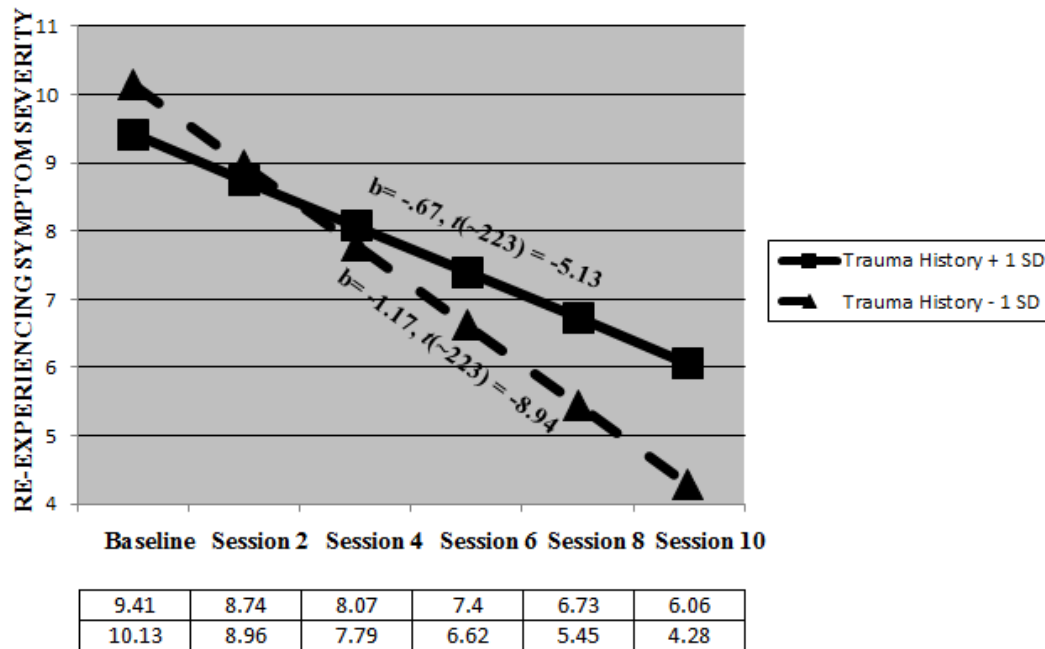* *p* < .05

Re-experiencing symptom change:

$$Y = \pi_{0} + \pi_{1}\,(\text{time}) + e$$

$$\pi_{0} = \beta_{00} + \beta_{01} * (\text{trauma history}) + r_{0}$$

$$\pi_{1} = \beta_{10} + \beta_{11} * (\text{trauma history})$$

where Y represents re-experiencing symptom severity, π_{0} represents the intercept plus the main effect of trauma history, π_{1} (time) represents the slope for time of PTSD symptom severity assessment plus the interaction of trauma history and time, e represents level 1 residual error, and r_{0} represents residual error in intercepts (level 2). Results of this model revealed a significant interaction between trauma history and time in the prediction of re-experiencing symptom change over the course of PE, $\beta = .13$, $t(\sim223) = 2.67$, $p = .008$. Decomposition of this interaction indicated that participants who had experienced a greater number of lifetime trauma types (b = -.67) had a shallower slope of re-experiencing symptom reduction during the course of PE than participants who had experienced a lesser number of lifetime trauma types (b = -1.17) (see Figure 11).

Figure 11. *Plotted Estimated Means Illustrating the Moderating effect** of Trauma History by Time on Re-Experiencing Symptom Change over the Course of PE.*

Baseline	Session 2	Session 4	Session 6	Session 8	Session 10
9.41	8.74	8.07	7.4	6.73	6.06
10.13	8.96	7.79	6.62	5.45	4.28

Note. Trauma History + 1 SD = Trauma History at 1 SD above the mean; Trauma History - 1 SD = Trauma History at 1 SD below the mean

* $p < .01$

Avoidance symptom change:
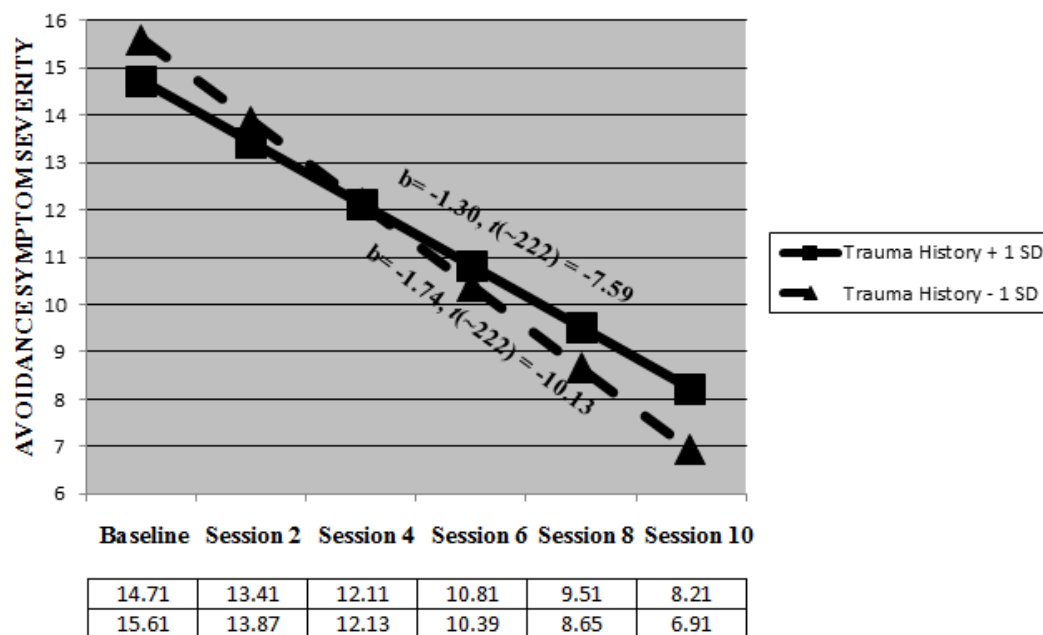
$$Y = \pi_{0} + \pi_{1} \, (\text{time}) + e$$

$$\pi_{0} = \beta_{00} + \beta_{01} * (\text{trauma history}) + r_{0}$$

$$\pi_{1} = \beta_{10} + \beta_{11} * (\text{trauma history})$$

where Y represents avoidance symptom severity, π_{0} represents the intercept plus the main effect

of trauma history, π_{1} (time) represents the slope for time of PTSD symptom severity assessment

plus the interaction of trauma history and time, e represents level 1 residual error, and r_{0}

represents residual error in intercepts (level 2). Results of this model revealed a trend-level

significant interaction between the trauma history and time in the prediction of avoidance

symptom change over the course of PE, $\beta = .12$, $t(\sim222) = 1.81$, $p = .072$. Decomposition of this

interaction indicated that participants who had experienced a greater number of lifetime trauma

types (b = -1.30) had a shallower slope of avoidance symptom reduction during the course of PE

than participants who had experienced a lesser number of lifetime trauma types (b = -1.74) (see

Figure 12).

Figure 12. *Plotted Estimated Means Illustrating the Trend-Level^ Moderating effect of Trauma History by Time on Avoidance Symptom Change over the Course of PE.*

14.71	13.41	12.11	10.81	9.51	8.21
15.61	13.87	12.13	10.39	8.65	6.91

Note. Trauma History + 1 SD = Trauma History at 1 SD above the mean; Trauma History - 1 SD = Trauma History at 1 SD below the mean

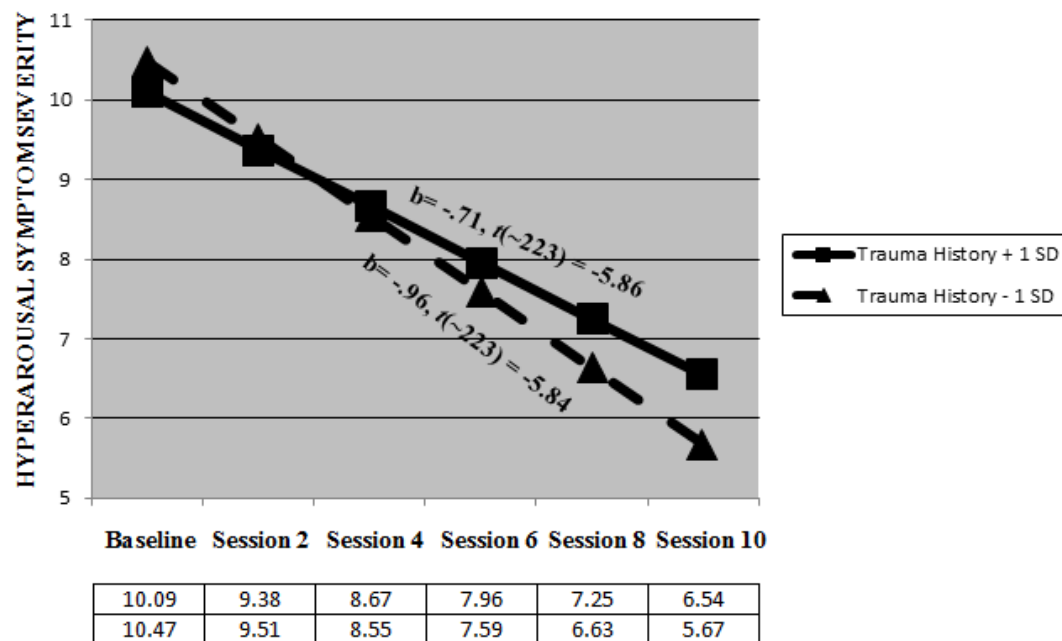^ *p* = .072

Hyperarousal symptom change:

$$Y = \pi_0 + \pi_1 \,(\text{time}) + e$$

$$\pi_0 = \beta_{00} + \beta_{01} * (\text{trauma history}) + r_0$$

$$\pi_1 = \beta_{10} + \beta_{11} * (\text{trauma history})$$

where Y represents hyperarousal symptom severity, π_0 represents the intercept plus the main

effect of trauma history, π_1 (time) represents the slope for time of PTSD symptom severity

assessment plus the interaction of trauma history and time, e represents level 1 residual error, and

r_0 represents residual error in intercepts (level 2). Results of this model revealed that there was

not a significant interaction between the trauma history and time in the prediction of

hyperarousal symptom change over the course of PE, $\beta = .06$, $t(\sim 223) = 2.67$, $p = .151$ (see

Figure 13).

Figure 13. *Plotted Estimated Means Illustrating that Trauma History does not^ Moderate Time on Hyperarousal Symptom Change over the Course of PE.*

	Baseline	Session 2	Session 4	Session 6	Session 8	Session 10
	10.09	9.38	8.67	7.96	7.25	6.54
	10.47	9.51	8.55	7.59	6.63	5.67

Note. Trauma History + 1 SD = Trauma History at 1 SD above the mean; Trauma History - 1 SD = Trauma History at 1 SD below the mean

^ *p* = .151

Examining age as a moderator of the slope of symptom change over the course of PE: Aim four

We next examined, whether age would interact with time in the prediction of PTSD symptom change:

$$Y = \pi_{0} + \pi_1 \text{ (time)} + e$$

$$\pi_0 = \beta_{00} + \beta_{01} * \text{(age)} + r_0$$

$$\pi_1 = \beta_{10} + \beta_{11} * \text{(age)}$$

where Y represents PTSD symptom severity, π_0 represents the intercept plus the main effect of age, π_1 (time) represents the slope for time of PTSD symptom severity assessment plus the interaction of age and time, e represents level 1 residual error, and r_0 represents residual error in intercepts (level 2). Results of this model revealed that there was not a significant interaction between age and time in the prediction of PTSD symptom change over the course of PE, $\beta = .01$, $t(\sim223) = 2.36$, $p = .712$.

Examining gender as a moderator of the slope of symptom change over the course of PE: Aim five

In order to examine whether age would moderate the rate of symptom reduction over the course of PE, we examined whether age would interact with time in the prediction of PTSD symptoms:

$$Y = \pi_{0} + \pi_1 \text{ (time)} + e$$

$$\pi_0 = \beta_{00} + \beta_{01} * \text{(gender)} + r_0$$

$$\pi_1 = \beta_{10} + \beta_{11} * \text{(gender)}$$

where Y represents PTSD symptom severity, π_0 represents the intercept plus the main effect of gender, π_1 (time) represents the slope for time of PTSD symptom severity assessment plus the interaction of gender and time, e represents level 1 residual error, and r_0 represents residual error

in intercepts (level 2). Results of this model revealed that there was not a significant interaction between gender and time in the prediction of PTSD symptom change over the course of PE, $\beta = .56$, $t(\sim223) = 1.10$, $p = .271$.

DISCUSSION

In the parent study, we demonstrated that PE therapy was efficacious in reducing PTSD symptoms among PLWH. The current study analyzed our previous data in order to examine PTSD symptom change over the course of PE therapy. Further, the current study also examined symptom change within the PTSD symptom clusters of re-experiencing, avoidance, and hyperarousal during PE. Moreover, because our sample consisted entirely of PLWH, we sought to examine whether having an index trauma related to the HIV diagnosis versus having a non-HIV-related index trauma would moderate the rate of symptom change over the course of PE. We examined this in PTSD symptoms as a whole and in the PTSD symptom clusters. Further, we wanted to examine whether having a more extensive trauma history would moderate the rate of PTSD symptom change as a whole and in the PTSD symptom clusters over the course of PE. Lastly, we wanted to examine whether age and gender would moderate the rate of PTSD symptom change over the course of PE.

Results of the current study indicated that PTSD symptom change over the course of PE was linear. In other words, PTSD symptom reduction was best described by a constant rate of symptom reduction as opposed to fluctuating symptom change over time. This constant rate of symptom reduction held for re-experiencing, avoidance, and hyperarousal symptom clusters as well as for PTSD symptoms as a whole.

Results also indicated that type of index trauma (HIV or non-HIV) significantly moderated the rate of hyperarousal symptom reduction and trended toward significantly moderating the rate of avoidance symptom reduction over the course of PE. More specifically, participants whose index trauma was related to their HIV diagnosis had less hyperarousal symptom reduction and avoidance symptom reduction over the course of PE than participants

whose index trauma was not related to their HIV diagnosis. Due to the fact that an HIV diagnosis

is an ever-present, chronic threat that has the potential to lead to death (Luszczynska et al., 2012),

it is understandable why PTSD symptom reduction may be less for an index trauma that is

related to the HIV diagnosis than for another trauma that is non-HIV-related. Especially in the

context of an HIV-related index trauma, hyperarousal symptoms might be harder to reduce,

because of the constant threat of a HIV diagnosis, and the necessity to be aware of immune

system compromising threats (i.e. contact with people who have a cold) which may evoke a

physiological threat response (Marris, E, 2010). Based on Pavlovian conditioning and fear

extinction models, it is plausible that avoidance symptoms might also be difficult to reduce,

because mortality threat reminders may never fully extinguish due to the chronicity of an HIV

diagnosis (Vanelzakker, Dahlgren, Davis, Dubois, & Shin, 2014).

Results of the current study also indicated that trauma history moderated the rate of

PTSD symptom reduction over the course of PE. More specifically, participants with more

lifetime trauma types experienced had less PTSD symptom reduction throughout PE compared to

participants with fewer lifetime trauma types experienced. Moreover, this pattern of symptom

reduction held for re-experiencing symptoms and showed a trend towards a similar pattern for

avoidance symptoms, but this pattern of symptom reduction did not hold for hyperarousal

symptoms. These results imply that re-experiencing symptoms may be driving this pattern of

symptom reduction.

Prolonged or repeated exposure to traumatic stress is associated with significant mood

disturbances and functional impairment (i.e., Cerda, et al., 2013; Tracy, Morgenstern, Zivin,

Aiello, & Galea, 2014). Individuals with a trauma history and/or PTSD are at risk of

experiencing disruptions in the organization and integration of the trauma memories into their

autobiographical memory (i.e., Jelinek, Randjbar, Seifert, Kellner, & Moritz, 2009; Lorenzzoni, Gacia-Silva, Poletto, Kristensen, & Gauer, 2014). These disruptions may provide an explanation for the role re-experiencing symptoms play in diminishing the effectiveness of PE in this sample. For example, the function of the imaginal exposure component of PE is to target re-experiencing symptoms by processing the trauma memory through repeated verbal exposure to the trauma narrative (i.e., Hagenaars, van Minnen, & de Rooij, 2010). Therefore, it is possible that individuals who have experienced more traumas during their lifetime may need more time to process their trauma narrative, or require more sessions to target multiple trauma narratives in therapy. Lastly, we did not find any moderating effects of PTSD symptom reduction for age and gender, which indicates that PE was just as efficacious at reducing PTSD symptoms regardless of whether the participant was male or female and regardless of the age of the participant.

There were some limitations to the current study. First, we used DSM-IV criteria to assess PTSD symptoms. This limits our knowledge of how PTSD symptom reduction (and the various symptoms of PTSD) may behave when using the new DSM-5 (American Psychiatric Association, 2013) criteria for PTSD. Secondly, our study used a sample of entirely HIV-positive individuals, which provides insight into how PTSD symptom reduction takes place in this at-risk and under-researched population. However, having an entirely HIV-positive population limits the ability of the study's findings to generalize to other samples and populations. Lastly, we did have a relatively small sample, with a total of 47 participants, which limited the power of our statistical analyses; Therefore, to address these short-comings, future studies should ty to replicate the current study using 1) DSM 5 criteria for PTSD, 2) should examine whether these findings hold in other samples of non-HIV-positive participants, and 3) should try to use larger samples to increase statistical power and, subsequently, increase the ability to detect significant

effects. Further, future directions should examine whether adding additional PE sessions or providing different presentations of PE, such as altering the number of sessions per week or making other temporal changes to the PE protocol would benefit those with a an HIV-related index trauma as well as those with a more substantial trauma history.

Conclusion

Despite its limitations, the current study provided empirical evidence regarding PTSD symptom change throughout the course of PE. We found that individuals who had an index trauma related to their HIV diagnosis had a lower rate of hyperarousal and avoidance symptom reduction over the course of PE. We also found that individuals with a more substantial trauma history had a lower rate of PTSD symptom reduction during PE. Moreover, this effect seemed to be driven by re-experiencing symptoms. Age and gender did not affect the rate of symptom reduction during PE. In sum, these results indicate that reductions in PTSD symptoms are not the same for all participants and are not the same for all types of symptoms, indicating that further understanding of the symptom reduction process during PE is warranted in order to personalize PE to the individual needs of the client and in order to maximize PTSD symptom reduction during the therapeutic process.

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