2014-07-30

Brief Psychological Intervention for Acute Posttraumatic Stress: Individual and Trauma Factors Affecting Recovery in Low-SES Minorities.

Lindsay M. Bira
University of Miami, lindsaybira@gmail.com

UNIVERSITY OF MIAMI

BRIEF PSYCHOLOGICAL INTERVENTION FOR ACUTE POSTTRAUMATIC STRESS: INDIVIDUAL AND TRAUMA FACTORS AFFECTING RECOVERY IN LOW SES MINORITIES

By

Lindsay M. Bira

A DISSERTATION

Submitted to the Faculty
of the University of Miami
in partial fulfillment of the requirements for
the degree of Doctor of Philosophy

Coral Gables, Florida

August 2014

UNIVERSITY OF MIAMI

A dissertation submitted in partial fulfillment of
the requirements for the degree of
Doctor of Philosophy

BRIEF PSYCHOLOGICAL INTERVENTION FOR ACUTE POSTTRAUMATIC STRESS: INDIVIDUAL AND TRAUMA FACTORS AFFECTING RECOVERY IN LOW SES MINORITIES

Lindsay M. Bira

Approved:

_____	_____
Gail Ironson, M.D., Ph.D.	Neil Schneiderman, Ph.D.
Professor of Psychology	Professor of Psychology
_____	_____
Rick Stuetzle, Ph.D.	Blanche Freund, Ph.D
Lecturer of Psychology	Lecturer of Psychology
_____	_____
Kent Burnett, Ph.D.	M. Brian Blake, Ph.D.
Associate Professor Emeritus	Dean of the Graduate School

BIRA, LINDSAY M. (Ph.D., Psychology)
Brief Psychological Intervention for Acute Posttraumatic Stress: Individual and Trauma Factors Affecting Recovery in Low SES Minorities. (August 2014)

Abstract of a dissertation at the University of Miami.

Dissertation supervised by Professor Gail Ironson.
No. of pages in text. (153)

INTRODUCTION: Low-socioeconomic status minorities in urban areas experience higher rates of trauma and resulting distress with a greater need for treatment than the general population. In addition, individual factors and trauma factors may determine who benefits most from which kind of treatment and brief intervention as well as group format may be particularly helpful in low SES minorities in order to fully utilize minimal resources and maximize treatment gain for a majority of individuals. For the study detailed in this dissertation, which is part of a larger NIH funded study, we worked together with a community health center in an underserved area to provide three types of previously supported, brief posttraumatic stress oriented psychological interventions for individuals with a recent trauma. OBJECTIVES: This dissertation compares three brief interventions (Psychological First Aid – PFA: group format, control; Stress Management Therapy – SMT: group format, expressive trauma writing component; and Eye Movement Desensitization and Reprocessing – EMDR: individual format) to determine the impact of the interventions in a low SES sample and to determine whether certain interventions are better for certain types of people and trauma. METHODS: A total of 87 low SES, minority participants were randomized to receive 4 active sessions of either PFA, SMT or EMDR, with 31, 29 and 27 participants in each group, respectively. Follow-up assessments were conducted at one, three and six months post-intervention

and outcome measures include PTSD symptoms, depressive and physical symptoms. Individual factors are PTSD severity, gender, substance abuse, childhood trauma, borderline personality disorder and trauma discussion. Trauma factors include time since trauma and trauma type. HLM analyses were used to explore the relationships and test the hypotheses. RESULTS: For the entire sample, EMDR worked best for reducing depressive symptoms, PFA worked best for reducing PTSD symptoms, and SMT worked best for reducing physical symptoms. However, when looking within group at individual and trauma factors, EMDR worked best for those high in baseline PTSD and for those endorsing borderline personality characteristics. SMT worked best for those who reported using marijuana and for those with a trauma of bereavement, whether violent or non-violent in nature. PFA worked best for individuals with a history of childhood sexual abuse and those with a trauma that was violent in nature. Limitations and future directions are discussed. CONCLUSIONS: Treatment type may be selected based on individual and trauma factors after a traumatic event. In addition, brief treatments may have great utility, particularly when the need for treatment is high and time and/or resources are low. These findings in an underserved population contribute to previous literature on post-trauma interventions and provide new evidence for individual and trauma factors that may influence recovery.

Acknowledgements

This project, and the author's graduate experience as a whole, would not have been possible without the continued support from dedicated committee members. Dr. Gail Ironson, the author's primary graduate advisor, provided a rich training environment and communicated faith in the author's abilities. She guided the author in finding her clinical and research areas of interest and supported her in her endeavors, which were acts of superb mentorship that will never be forgotten. Dr. Rick Stuetzle's continuous efforts in providing the author with years of guidance in statistical methods and analysis support as well as mentorship when needed were vital in the development of this dissertation as well as the author's master's thesis. In addition, the experience working with Dr. Freund is highly valued, as her rich clinical skills in the area of trauma-focused treatment were motivating and helpful during conceptualization of the project and in the author's professional development. Lastly, it is important to note that Dr. Neil Schneiderman, director of the Clinical Health Psychology doctoral program, played an important role in helping the author find her area of interest and expertise through his continued support and focus on individual student training needs. Much appreciation goes to these committee members, as they were integral to the author's clinical, research, and professional development, and ultimately, the completion of her Ph.D.

TABLE OF CONTENTS

LIST OF CHARTS

Page

LIST OF TABLES

Page

LIST OF FIGURES

Page

LIST OF ABBREVIATIONS

AIP	Adaptive information processing
BDI	Beck Depression Inventory – 2nd Edition
BPD	Borderline personality disorder
CBT	Cognitive behavioral therapy
CSA	Childhood Sexual Abuse
CISD	Critical Incident Stress Debriefing
CPT	Cognitive processing therapy
DSM	Diagnostic Statistical Manual of Mental Disorders
EMDR	Eye movement desensitization and reprocessing
HLM	Hierarchical Linear Modeling
PE	Prolonged Exposure
PILL	Pennebaker Inventory of Limbic Languidness
PFA	Psychological First Aid
PTCI	Posttraumatic Cognitions Inventory
PTSD	Posttraumatic Stress Disorder
SCID	Structured Clinical Interview for the DSM-IV-TR
SES	Socioeconomic status
SMT	Stress Management Therapy
TFCBT	Trauma-focused cognitive behavioral therapy

CHAPTER 1. INTRODUCTION

According to data collected by the Bureau of Justice regarding criminal victimization in the United States from 1996 – 2005, minorities are at a much higher risk for being victims of crime than are whites (Bureau of Justice Statistics, 2005). In 2005, the last year for which this data is made available, the violent crime (aggravated assault, sexual violence, robbery and homicide) rate per 1,000 people in the U.S. was 40.2 for Caucasians while it was more than doubled for U.S. minorities at rate of 104.6. Comparing this overall rate to urban areas specifically, the rate for both of these groups is even higher, at 56.2 for Caucasians and 136.1 for minorities. Within minorities in urban areas, African Americans had the highest risk overall, with a rate of 76.6 per 1000 people in 2005 compared to a rate of 59.5 in Hispanics. This makes minorities living in urban areas (particularly African Americans) the most at-risk group for being victims of violent crime. This is supported in a review by Alim, Charney and Mellman (2006), who concluded that low socioeconomic status (SES) and urban dwelling placed African Americans at an increased risk for experiencing a violent trauma.

Witnessing or being a direct victim of crime with or without physical assault can be a traumatic event, and trauma, by definition, is an emotional stressor or physical injury that causes short-term and sometimes long-term psychological and/or behavioral distress (Merriam-Webster, 2012). Experiencing such an event can lead to adverse psychological states, one of which is Posttraumatic Stress Disorder (PTSD). According to the Diagnostic & Statistical Manual for Mental Disorders (DSM, 4th ed., text revised; American Psychiatric Association, 2000), an individual meets diagnostic criteria for PTSD if they were confronted with, experienced or witnessed an event that threatened the

safety of or caused injury to themselves or others. In addition, the individual must also report feelings of re-experiencing the event (such as flashbacks, intrusive thoughts, nightmares, and physiological reactivity to cues that represent the trauma), avoidance and numbing (avoiding cues of the trauma, feelings of detachment, restricted affect range, inability to remember important aspects of the trauma) and hyper-arousal (sleep difficulties, anger outbursts, difficulty concentrating, heightened startle response, hyper-vigilance). Lastly, these symptoms must cause functional disturbance in important areas of life and must be present for more than one month. An individual may not meet all criteria necessary for full diagnosis but can still be considered to be experiencing posttraumatic stress. According to the National Comorbidity Survey Replication, PTSD affects 14-24% of people exposed to trauma and this rate has been found to be even higher for violent crime victims, specifically sexual crime victims (Kessler et al., 2005; Foa, 1997). Since minority individuals are at a high risk for experiencing crime trauma, they are also at high risk for experiencing adverse psychological states following a trauma, including PTSD and general posttraumatic stress (Alim et al., 2006).

According to data analyzed from the 2004-2005 National Epidemiologic Survey on Alcohol and Related Conditions, lifetime prevalence of PTSD has been found to be highest among African Americans. Interventions that focus on trauma processing in low-SES minorities could help decrease this disparity in PTSD development (Alim et al., 2006). However, it has also been found that minorities are much less likely than Caucasians to seek and receive treatment for their symptoms (Roberts et al., 2011). Furthermore, high rates of undiagnosed and untreated PTSD have been found in low SES, urban African Americans (Davis, Ressler, Schwartz, Stephens and Bradley, 2008;

Schwartz, Bradley, Sexton, Sherry and Ressler, 2005). Several reasons for low treatment seeking and high rates of untreated PTSD have been found, including the separation of mental health services from other medical services, a lack of mental health services overall, and also the stigma that is often associated with treatment for mental illness, which are all especially prevalent in low income communities (Lawrence & Kisely, 2010).

Chronic PTSD can lead to other co-morbid psychological and physical problems such as depression, anxiety, drug/alcohol abuse and dependence, somatization, obesity, physical health problems (possibly increasing with PTSD severity), and more costs spent on health care (Lazar and Offenkrantz, 2010). Therefore, intervention for PTSD is not only beneficial for improving mental health, it can also help prevent further problems, such as physical symptoms and health difficulties. Based on research in this area, early treatment intervention in the face of severe initial traumatic response has been suggested due to the high likelihood of functional disturbance and the potential to develop chronic health problems if left untreated (U.K. National Institute for Clinical Excellence, 2005). Swift, accessible and effective post-trauma intervention for individuals experiencing post-traumatic distress is important, particularly in minority-populated, underserved urban areas.

One such minority-populated, underserved area in the northwestern portion of Miami is an urban neighborhood named Model City, or more commonly referred to as Liberty City. As of 2010, Liberty City had a population of 22,749 and it holds more than half of Miami's total African American population: 83.89% of citizens in Liberty City are African American, versus only 16.27% of Miami's citizens as a whole (U. S. Census,

2010). As of 2009, citizens of Liberty City had a median income of $21,529 compared to $28,999 in Miami overall (UrbanMapping, 2011), which indicates higher levels of poverty. In 2010, the violent crime rate per 1,000 people was 4.03 for the U.S., 5.42 for Florida, 7.33 in Miami-Dade County, and 12.34 in the city of Miami (Federal Bureau of Investigation, 2011a; Federal Bureau of Investigation, 2011b). Although the exact crime statistics could not be obtained for Liberty City as this information requires purchase (K. Gil, personal communication, May 24, 2012), this area has a well-known reputation for being an unsafe place of residence as the violent crime rate is high compared to surrounding Miami neighborhoods. This can be confirmed by comparing crime activity in Miami's neighborhoods during the 90 days preceding May 25, 2012. Data shows that Liberty City had 202 violent crime reports in a one-mile radius from the center of the neighborhood, compared to 90 in Little Havanna, 60 in Coconut Grove and 56 in Brickell, all of which are other nearby neighborhoods in Miami (CrimeMapping, 2012). Citizens of Liberty City are at high risk for violent crime victimization as well as trauma-related PTSD and negative mood states which could affect functioning and physical health.

The disparities in trauma prevalence, PTSD development and psychological treatment among low SES minorities are clear. It is important that integrated services and more psychological interventions after trauma are provided in underserved communities like Liberty City so that accessible and effective PTSD treatment is available. However, it is also important when designing such services to be aware of

previous literature regarding efficacious interventions as well as individual and trauma factors affecting treatment outcomes so that the most beneficial interventions are implemented.

POST-TRAUMA INTERVENTIONS

Critical Incident Stress Debriefing (CISD) is a single-session intervention that was the prior post-trauma treatment of choice, often times provided on-site immediately after traumatic event exposure (e.g. a crime or natural disaster; Mitchell, 1983). It was designed to help people cope with adverse psychological and physiological symptomatology resulting from trauma by helping them process, reflect and ventilate. Although early literature showed it to be effective for trauma victims and response workers (Everly, Flannery & Mitchell, 2000), it has since been shown through meta-analyses to be largely ineffective, possibly even detrimental to some individuals, due to re-traumatization from ventilation of emotions (Rose, Bisson, Churchill and Wessely, 2002; van Emmerik, Kamhuis, Hulsbosch and Emmelkamp, 2002).

Psychological First Aid (PFA), which is offered by the American Red Cross, has replaced various forms of psychological debriefing. This intervention utilizes a brief, supportive post-trauma protocol for victims groups of traumas such as natural disasters, large acts of violence, etc. (National Center for PTSD, 2006). It is composed of eight components ("Contact and Engagement," "Safety and Comfort," "Stabilization," "Information Gathering," "Practical Assistance," "Connection with Social Support," "Coping," and "Linkage with Collaborative Services"), which were developed based on reviews of previous research regarding effective supportive and brief response

intervention for disaster and trauma. These eight components can be used together or singularly to aid survivors as necessary after a traumatic event. Trained volunteers offer support and psychoeducation to people who may be experiencing stress in the hours or days after a traumatic event while also remaining aware of and supporting natural resiliency (Uhernik and Husson, 2009). PFA is endorsed by guidelines from many organizations, including Federal guidelines in the 2008 National Response Framework (U.S. Department of Homeland Security, 2008) and guidelines from the World Health Organization (Kondro, 2011).

Emotional disclosure about a trauma is another helpful technique to facilitate posttraumatic growth and reduce psychological and physical symptoms. When compared to control groups writing about a neutral subject, individuals who engage in emotional disclosure through trauma writing have shown to experience a reduction in physical symptoms, health care visits, depression, and pain after trauma (Gidron et al., 2002; Smyth, 1998; Smyth, Stone, Hurewitz, and Kaell, 1999; Pennebaker and Beall, 1986; Frisina, Borod and Lepore, 2004). Meta-analyses investigating the effect of emotional disclosure trauma writing have been conflicting, ranging from showing no effect (Frisina, et al., 2004) to moderate effects (Frattaroli, 2006) to large effects (Smyth, 1998). Emotional disclosure through trauma writing can also be added as a component in other interventions to include an exposure/processing feature, as will be described later in this dissertation.

There is an abundance of literature regarding more intensive psychological interventions for PTSD. In a 2008 report, the Institute of Medicine examined literature on various treatments for PTSD, concluding that exposure-based therapies, such as

Prolonged Exposure (PE; Foa and Rothbaum, 1998) and Cognitive Processing Therapy (CPT; Resnick and Schnicke, 1992), have a high degree of efficacy (Institute of Medicine, 2008). They reported that there was insufficient evidence to determine efficacy of eye movement desensitization and reprocessing (EMDR; Shapiro, 1999), although EMDR is now often considered an exposure therapy as has it has components of trauma memory exposure. The report also concluded that there is insufficient evidence to determine efficacy of group therapy, early intervention and optimal treatment length in the treatment of PTSD.

EMDR and PE were compared as brief treatments for PTSD in a previous study from our lab (Ironson, Freund, Strauss and Williams, 2002). We found that at least three sessions of preparatory trauma treatment and three sessions of active EMDR or PE significantly reduced PTSD and depressive symptoms at 3 month follow-up, but that the EMDR treatment group showed reductions faster, experienced treatment as less painful (as indicated by SUDS), and had less dropout than the PE group. This suggests that although the two might be equally effective in reducing PTSD symptoms, EMDR may have an advantage in that it has the potential to be easier to endure with a higher likelihood of treatment completion. It is important to note that in-vivo exposure homework was assigned in the EMDR group as well as the PE group and that participants had a total of 6 sessions, only 3 of which were active treatment sessions.

Seidler and Wagner (2006) conducted a review of 8 studies that compared the effectiveness of EMDR to trauma-focused CBT in the treatment of PTSD. The results suggested that both are effective PTSD treatments, which supports the findings of our previous study and earlier meta-analyses (Ironson et al., 2002; van Etten and Taylor,

1998; Davidson and Parker, 2001; Bradley et al., 2005). However, in this review, superiority of one treatment over the other could not be demonstrated and the two approaches were concluded to be equally efficacious. This lack of treatment differences is further shown in another meta-analysis of 15 studies which failed to find treatment differences and concluded that PTSD-focused treatments are all beneficial (Benish, Imel and Wampold, 2008).

Similar findings regarding equal treatment outcomes have been found in still other meta-analyses. Bisson and Andrew (2007) conducted a review of 33 studies in which they investigated efficacious PTSD treatments. They found that individualized trauma-focused treatments, specifically trauma-focused cognitive behavioral therapy (TFCBT), eye movement desensitization and reprocessing (EMDR) and stress management, as well as group TFCBT were all significantly better at reducing PTSD symptoms than waitlist/usual care and other less intensive interventions. In addition, TFCBT, EMDR and stress management were all found to be equally effective.

In another large review and meta-analyses of 38 randomized controlled trials by Bisson et al. (2007), trauma-focused CBT, EMDR, stress management and group CBT were all found to be efficacious in the reduction of PTSD symptoms compared to waitlist or usual care. No efficacy difference was found between trauma-focused CBT and EMDR, but both were superior to stress management, which was superior to other therapies. The authors suggest that trauma-focused CBT and EMDR should be the most utilized treatments for PTSD. Trauma-focused CBT has also been found to be the best approach within reviews of more specific populations, such as refugees (Nickerson, Bryant, Silone & Steel, 2011) and veterans (Goodson and Helstrom, 2001). The findings

that trauma-focused CBT may be the best line of approach are supported by published PTSD treatment guidelines (Foa, Keane, Friedman and Cohen, 2009; U.K. National Institute for Clinical Excellence, 2005).

Similarly, in a recent meta-analysis of 13 studies regarding PTSD treatments, Powers, Halpern, Ferenschak, Gillihan and Foa (2010) investigated first the efficacy of PE and found that PE out performs control conditions on both primary and secondary outcome measures and this effect persisted from one to 12 month follow-up. However, when comparing PE to other active treatments (EMDR, CPT, cognitive therapy and stress inoculation training [similar to stress management]), there was no significant difference between treatments on outcome measures, meaning that EMDR and other active treatments seem to be just as effective in treating PTSD as is PE.

Finally, some reviews have suggested that novel treatments for PTSD, such as interpersonal therapy, acceptance and commitment therapy and psychodynamic therapy, have preliminary data supporting their PTSD treatment efficacy, although the data is insufficient to draw a full conclusion and should be used only when evidence-based treatments fail to yield improved outcomes (Bomyea and Lang, 2012; Cukor, Spitalnick, Difede, Rizzo and Rothbaum, 2009).

Existing literature has identified several trauma-focused treatment approaches as effective in treating PTSD and it seems that the supported interventions are all equal in efficacy. In regards to this dissertation, the interventions of focus will be Psychological

First Aid (PFA; as a post-trauma standard-of-care), Trauma-focused Stress Management Treatment (SMT) and EMDR, all of which are supported in the literature as effective and useful post-trauma interventions.

Stress Management Treatment

Research described earlier in this literature review points to PE, EMDR and other trauma-focused CBT approaches as the most supported interventions for PTSD. Stress management (SMT; similar to Stress Inoculation Training – SIT) is a cognitive-behavioral intervention aimed at reducing experienced distress and teaching individuals how to be aware of as well as decrease current and future stress using cognitive and behavioral methods (Litz and Roemer, 1996). It is important to note that SMT is a flexible CBT intervention that can be augmented to have a trauma focus and can even include aspects of exposure, although research is lacking regarding this type of format. In the few studies that did combine other CBT techniques with SMT, combined treatments were not superior over the constituents and SMT alone continues to be recommended (Cahill and Foa, 2007). Although it is unclear how SMT can be beneficially augmented, this flexibility is useful in that it can easily be adjusted to fit a specific population and can be used in group format, something that may be difficult to do in other interventions.

SMT techniques include coping skills, cognitive restructuring, problem-solving and relaxation training and can be used as a treatment within itself, although components of SMT have been included in more intensive post-trauma treatments (EMDR and PE).

Several meta-analyses have shown benefits of using SMT as an intervention in PTSD populations (Bisson and Andrew, 2007; Bisson et al., 2007; Bolton et al., 2004; Powers et al., 2010). In a study by McKibben, Britt, Hoge and Castro (2009), post-Iraqi combat soldiers were surveyed and it was found that those who received SMT (educational in nature) after returning from deployment reported lower levels of PTSD and physical symptoms in the following year compared to those who did not receive SMT. Those who rated the program as most helpful after completion reported the lowest PTSD and physical symptoms in the following year and those who did not report having SMT reported the highest PTSD and physical symptoms in the following year. Previous research has also shown this relationship between SMT and physical symptoms (Berger and O'Brien, 1998).

Eye-Movement Desensitization and Reprocessing

EMDR is frequently listed as one of the highest recommended trauma-based interventions for PTSD. It is currently considered by some to be an exposure-based PTSD intervention (Goodson et al., 2011), though proponents of the approach believe that EMDR is a comprehensive treatment involving beneficial elements of psychodynamic, CBT, experiential, interpersonal, and physiological therapies and its clinical effects are due to mechanisms other than exposure (Schubert and Lee, 2009). Although there is an abundance of support for EMDR's efficacy, controversy has surrounded its use and a closer look at EMDR development and research is warranted.

EMDR was developed by Francine Shapiro (Shapiro, 1999) for post-trauma treatment and is based on the Adaptive Information Processing (AIP) model for PTSD (Shapiro, 2001). According to this model, PTSD may occur when memories of an experience are not properly processed and become unintegrated and dysfunctionally stored (Schubert and Lee, 2009). This storage also contains emotions, images and sensations related to the event, which can become reactivated by a trigger in the present and can then produce PTSD symptoms. EMDR theory posits that by targeting avoided memories, identifying pathology-maintaining beliefs, and implementing bilateral stimulation (eye tracking, sounds, or taps on each side of the body to activate both hemispheres of the brain), the brain is given the time and tools necessary to process the memory and store it in a more adaptive way. Sessions follow an eight-phase format, which is described in Appendix 3.

According to the AIP model, information is adaptively processed through deconditioning with relaxation skills, neurologically activating and strengthening weak associations, and factors associated with the client's dual focus on both the bilateral stimulation and memory (Shapiro, 2001). The mechanisms behind and contribution of bilateral stimulation to EMDR's therapeutic outcome remain unclear (for meta-analysis, see Davidson and Parker, 2001), but research has pointed to the association between eye-movements alone and decreased vividness/emotionality of autobiographical memories, enhanced retrieval of episodic memories, increased cognitive flexibility, changed frontal lobe inter-hemispheric coherence and psychophysiological de-arousal during the recall of distressing memories (for complete review, see Schubert and Lee, 2009). Rather than through exposure and extinction of conditioned responses, proponents of the AIP model

suggest that EMDR facilitates the integration of the dysfunctionally stored trauma memory with preexisting and healthy memory networks to produce favorable outcomes (Shapiro, 2001; Schubert and Lee, 2009).

Even in the face of controversy surrounding mechanisms underlying EMDR processes, recent reviews and meta-analyses have concluded that EMDR is efficacious in the treatment of PTSD (Spates et al., 2009; Nathan and Gorman, 2007; Ponniah and Hollon, 2009; Bisson and Andrew, 2007; Bisson et al., 2007; Seidler and Wagner 2006). In fact, EMDR for the treatment of PTSD has been rated in the highest effectiveness category in the International Society of Stress Studies practice guidelines (Foa, Keane, Friedman, & Cohen, 2009), the American Psychiatric Association (2004) practice guidelines and by the U.S. Department of Veterans Affairs and Department of Defense (2010). Several international guidelines also recommend EMDR for the treatment of PTSD (U.K. National Institute for Clinical Excellence, 2005; Australian Centre for Posttraumatic Mental Health, 2007; Bleich, Kotler, Kutz and Shalev, 2002; CREST, 2003; INSERM, 2004).

EMDR research has pointed to its specific efficacy in adult single-trauma populations (Schubert and Lee, 2009), with recent studies showing desirable outcomes with accident witnesses and assault victims (Högberg et al., 2007) as well as adult sexual trauma victims (van der Kolk et al., 2007; Rothbaum, Astin and Marstellar, 2005). An advantage that EMDR may hold is that it has been shown to be effective in reducing PTSD and depressive symptoms in as little as three active sessions following a single, acute trauma (i.e., Ironson et al., 2002; Marcus, Marquis, & Sakai, 1997, 2004; Rothbaum, 1997; PTSD symptoms only: Wilson, Becker, & Tinker, 1995, 1997), which

make it an ideal brief, post-trauma intervention when time, funds and/or therapist availability are low. In addition, as noted earlier, Ironson et al. (2002) found that their EMDR treatment group showed PTSD reductions faster, experienced treatment as less painful (as indicated by SUDS), and had less dropout than the PE treatment group. This suggests that EMDR has the potential to be easier to endure with a higher likelihood of treatment completion. In sum, despite controversy and lack of knowledge surrounding the mechanisms of bilateral stimulation, one thing is clear: EMDR has the research and agency support necessary to make it one of the top efficacious treatments for PTSD, especially in single-trauma cases, and may even have advantages over other forms of equally efficacious treatments.

Brief Intervention and Group versus Individual Format

Based on a review of the literature, the Institute of Medicine (2008) concluded that there is insufficient evidence to determine optimal treatment length and efficacy of group versus individual therapy in the treatment of PTSD. However, there are some supporting articles for brief intervention in populations with PTSD. Bloom (2002) conducted a literature review on brief treatment of anxiety disorders. Regarding PTSD, his review indicates that brief intervention in a single-session format (specifically psychological debriefing) is not effective and more intensive CBT-based treatment in several sessions is more beneficial. Effective treatments ranged from two three-hour

sessions to sixteen one-hour sessions (Bloom, 2002). Significant CBT elements included psychoeducation, relaxation and breathing training, cognitive restructuring, imagery rehearsal and exposure.

Foa, Hearst-Ikeda and Perry (1995) compared a brief, four session CBT intervention for female rape victims with PTSD to a waitlist control and collected follow-up data over 5-months post-trauma. The investigators found that the CBT group (which received psychoeducation about common trauma reactions and general cognitive and behavioral skills training) had significantly less PTSD symptoms at two months post-assault than the control group and only 10% met criteria for PTSD compared to 70% of the control group. This was maintained at five and a half months post-assault, with the CBT group showing significantly less depression and less PTSD symptoms than the control.

As described earlier, EMDR and PE were compared as brief treatments for PTSD in a previous study from our lab (Ironson, et al., 2002). We found that at least three sessions of preparatory trauma treatment and three sessions of active EMDR or PE significantly reduced PTSD and depressive symptoms by 70 percent at 3 month follow-up, but that the EMDR treatment group showed reductions faster, experienced treatment as less painful (as indicated by SUDS), and had less dropout than the PE group. This means that EMDR may be the treatment of choice when offering brief treatment, and 3-4 sessions may be the ideal length for reduction in PTSD and depressive symptoms.

Başoğlu, Livanou, Şalcioğlu and Kalender (2003) conducted a trial to examine whether CBT could be conducted in a minimal number of sessions while maintaining

efficacy in 231 survivors with PTSD after an earthquake in Turkey. Intervention largely focused on behavior therapy and treatment was terminated after "significant clinical improvement as rated by both the therapist and survivor" was attained. It is reported that 76% of cases improved significantly on levels of PTSD and depression after one treatment session and 88% improved after two sessions with survivors overall receiving a mean of 4.3 sessions. It is important to note however that no control group was used and participants were not randomized due to the difficulty of dealing with the aftermath of the earthquake.

Cigrang, Peterson and Schobitz (2005) conducted a brief intervention study on three post-combat veterans who were experiencing symptoms of PTSD but not the full disorder in hopes of preventing full, chronic PTSD. Each veteran was given four active sessions of repeated imaginal and in-vivo exposure (prolonged exposure; PE) over a period of five weeks. After the four sessions, PTSD symptoms had reduced by an average of 56% and PTSD Checklist scores were within normal limits (they met criteria for full PTSD at baseline). This study, although very small in sample size and lacking in follow-up data, indicates significant improvements in little time and may provide some evidence for PE being an effective rapid individual intervention for PTSD.

We do not know if group or individual format is better at reducing PTSD symptoms and we also do not know if brief treatment (e.g., four active therapy sessions) can be as sufficient as longer term treatment. In sum, considering the three interventions of focus in this dissertation, PFA is a supportive, standard-of-care intervention that is often administered in minimal sessions (as few as one) and is effective in group or individual format (National Center for PTSD, 2006). SMT techniques can be taught in

few sessions and can be augmented as necessary, but comparative effectiveness of group or individual format is unclear (IOM, 2008). EMDR, as stated previously, has been shown to be effective in as little as three active sessions and is mostly investigated in an individual format (i.e., Ironson et al., 2002; Marcus, Marquis, & Sakai, 1997, 2004; Rothbaum, 1997; Wilson, Becker, & Tinker, 1995, 1997). Although brief SMT could be beneficial in a group format, brief EMDR is most likely most effective when administered individually, due to its more intense trauma focus and the difficult nature of disclosing details of a traumatic experience to strangers.

Although the existing literature on brief intervention and group format for the treatment of PTSD is lacking, both may be necessary with a traumatized population in order to fully utilize minimal resources and maximize treatment gain for a majority of individuals (particularly in low SES minorities). Further research on these specified post-trauma interventions in brief, group and individual format will contribute to the existing literature on PTSD treatment.

INDIVIDUAL AND TRAUMA FACTORS

It has been suggested that since efficacy of the detailed trauma interventions in the treatment of PTSD symptoms seems to be high, what may be important to also investigate at this time is which trauma patients are more likely to benefit from one treatment over the other (i.e., the investigation of individual and trauma factors affecting treatment outcomes; Seidler and Wagner, 2006; Bomyea and Lang, 2012). It is possible that individual variation within PTSD symptom sufferers may mask some treatment

differences, and it is important to determine for whom each kind of treatment works best, so that the most efficacious treatment can be applied in the future.

Individual Factors

Although current literature investigating specific individual factors affecting recovery after PTSD intervention is lacking, there is some evidence regarding which factors may influence who benefits from certain interventions. In a review chapter by Lumley, Tojek and Macklem (2002), authors investigated which individuals seems to most benefit from emotional writing about a stressful event. The authors concluded that individuals who tend to inhibit negative emotions and have repressive coping styles do not seem to benefit from disclosure about a stressful event compared to individuals who were disinhibited in expression and who were not repressive in coping. In addition, for individuals who may be alexithymic (i.e., who have a deficit in emotional awareness and understanding), it seems that disclosure alone does not bring the same benefits, especially in bereaved individuals and individuals with PTSD. It may be that individuals who are less able to access, disclose and understand negative emotions would benefit from more intensive and guided psychotherapy along with disclosure tasks than from a disclosure task alone.

It has also been shown that in individuals who have experienced a trauma, PTSD severity can affect recovery outcomes. Level of experienced PTSD symptoms varies between individuals and may be influential in intervention engagement and recovery response. In a recent and large web-based questionnaire study by Ehring and Quack

(2010), which assessed 616 trauma survivors, authors found that PTSD symptom severity was related to lower levels of clarity and emotional awareness, higher levels of avoidance, higher levels of emotion suppression, difficulties engaging in goal-directed behavior when stressed, and impaired emotion regulation. Although causation cannot be implied due to the cross-sectional nature of this study, high levels of PTSD symptoms have the potential to make it more difficult for an individual to engage in treatment and a more intensive, directed intervention may be beneficial when baseline PTSD symptoms are high.

In a recent literature review by Blain, Galovski and Robinson (2010), gender differences in psychotherapy for PTSD outcomes were investigated. There is some evidence in randomized controlled trials with mixed trauma samples (using cognitive therapy, imaginal exposure or EMDR intervention) that females experience more treatment gains than males. Gender differences were not found in other studies and mixed findings were reported regarding attrition, so the exact role of gender in PTSD recovery after treatment still remains unclear. It is important to note that most of the studies available for review had a small number of male participants and a small overall sample size, which may have provided limited power to detect gender effects. The authors also discussed the confounding issue that females are more prevalent in PTSD populations, as they are exposed to severe traumas more frequently than men.

Regarding gender differences in emotional expression, Smyth (1998) conducted a meta-analyses in which he found that males seem to benefit more from emotional expression writing than females. It was suggested that this difference could be because males are less likely to express overall, but after given a medium through which to do so,

they experience more gains than women, who express more by nature. Although literature is lacking regarding how this relates to more evidence-based PTSD treatment, it could be that men benefit more from more intensive, trauma-oriented and therapist-guided interventions.

It has been shown in epidemiological studies of trauma that individuals with PTSD also often experience other comorbid psychiatric disorders, such as substance abuse (Breslau, 2002). It could be that substance abuse increases the risk for PTSD as drug/alcohol users may expose themselves to more dangerous situations, or it could be that PTSD increases the risk for substance abuse as individuals attempt to self-medicate. Comorbid substance abuse could affect treatment outcome in several ways: 1) increasing the risk for subsequent traumas that maintain PTSD symptoms; 2) making the participant less dependable and more likely to miss appointments; and/or 3) numbing physical and emotional systems so that necessary activation during trauma-oriented treatment is attainable. In the latest review of substance abuse and PTSD treatment literature, van Dam, Vedel, Ehring and Emmelkamp (2012) suggested that non-trauma-focused CBT, coping skills training and twelve-step programs are the most efficacious in treating substance abuse disorders alone, and there is not enough evidence to determine if adding a trauma-oriented intervention to an efficacious substance abuse intervention leads to increased benefits for those with comorbid difficulties. The review by van Dam et al. (2012) also reported that strictly trauma-focused interventions for comorbid PTSD and substance use diagnosed individuals has been associated with higher levels of drop out, which could be due to the intensity of the treatment, but again, more evidence is needed. In addition, it is suggested that substance abuse treatment before treating PTSD can be

ineffective, as withdrawal symptoms increase the severity of PTSD symptoms and therefore dropout rate is high (van Dam, et al., 2012). The authors suggest that more evidence regarding EMDR treatment approaches as well as comparing trauma-focused versus non-trauma-focused interventions are necessary to determine treatment outcomes for individuals with PTSD and concurrent substance abuse.

Another individual factor, the experience of childhood abuse (sexual or physical), can have a profound impact in later years, possibly affecting recovery from subsequent adulthood traumas. One study found that the prevalence of childhood abuse (sexual and/or physical) was a significant factor that predicted PTSD in an inner-city sample of primary care patients after experiencing an adverse event (Wrenn et al., 2011). In a cross-sectional study including 616 trauma survivors, Ehring and Quack (2010) found that survivors of childhood chronic trauma (sexual and/or physical abuse) showed higher levels of PTSD and more difficulty with emotion regulation when compared to survivors of adulthood trauma with no childhood traumatic events. Neurobiological differences in the brains of childhood trauma survivors compared to adulthood trauma survivors and healthy controls have also been found, which may underly the experience of PTSD and depressive symptoms by childhood trauma survivors and may make treatment more difficult (Murrough et al., 2011). In a study by van der Kolk et al. (2007) which focused on outcome differences after intervention (eight sessions of EMDR) between childhood-onset trauma victims and adulthood-onset trauma victims experiencing PTSD and depressive symptoms, it was found that adulthood-onset trauma victims showed a reduction in both types of symptoms after treatment while childhood-onset trauma victims showed no improvement.

In further complications as a result of childhood trauma, a strong relationship has been identified between many types of childhood abuse/trauma and the development of borderline personality disorder (BPD), according to several review studies (Gunderson and Sabo, 1993; Goodman and Yehuda, 2002; Huang, Yang and Wu, 2010; Keinänen, Johnson, Richards and Courtney, 2012) and in other recent studies (Sansone, Hahn, Dittoe and Wiederman, 2011; van Dijke et al., 2012). Borderline personality disorder is a severe mental disorder marked by emotional dysregulation, impulsivity, and instability of interpersonal relationships as well as self image (DSM-IV TR, APA, 2000). BPD has been found in a recent epidemiological study to increase the likelihood for comorbid adulthood full or partial PTSD (Pietrzak, Goldstein, Southwick and Grant, 2011) and has also been shown to be associated with complex PTSD in adulthood (van Dijke et al., 2012). Dissociation is one symptom commonly experienced by individuals with BPD (Gershuny and Thayer, 1999; Panzer and Viljoen, 2004; Jiang, Chen, Tang and Zhang, 2010) that makes it difficult for them to remain grounded and stable in intensive, trauma-focused therapy. Current evidence-based treatments for BPD are lengthy and complex due to the complex nature of the disorder (Zanarini, 2009) and it has even been suggested in a literature review by Vignarajah & Links (2009) that intensive, PTSD-focused treatment without BPD-focused treatment may be detrimental to individuals experiencing both disorders. Having facets of BPD, especially dissociation, can majorly influence how an individual experiences not only life in general, but also subsequent traumas and interventions for PTSD so it is important to consider this when designing interventions for traumatic distress.

Lastly, there has also been a long history of research regarding common factors that predict outcomes across all psychotherapies. Lambert (1992) reviewed the literature and identified four main common factors that predict therapeutic outcomes: extratherapeutic factors, the therapeutic relationship, specific therapeutic techniques and expectancy or placebo effects. Extratherpeutic factors center around the client individually, and include the client's environment, accessibility to services, social support, motivation/readiness for change, and ability to tolerate and manage affect, to name a few (Drisko, 2004). This highlights the importance of the client's engagement in treatment for maximum therapeutic outcome. Within post-trauma interventions, although specific techniques or approaches have been identified as more efficacious than others, participant factors such as attendance and overall involvement remain as important factors in predicting outcomes.

Trauma Factors

There are also trauma factors that may affect psychological and physical recovery over time, including time since trauma and trauma type. Several studies have shown that symptoms of PTSD and/or depression after a trauma decrease for some people over time without intervention (e.g., Williams, Burke, McDevitt-Murphy and Neimeyer, 2012; Rothbaum, Foa, Riggs and Murdock, 1992; McLaughlin et al., 2011). Therefore, time since trauma is an important factor that may affect trauma recovery, as a shorter time window between traumatic experience and assessment of symptoms would most likely be correlated with higher psychological symptoms than a longer time window, and this may be more pronounced with intervention. However, this could look different in different

types of interventions, such as psychoeducational interventions versus trauma-oriented interventions. It may be that people experiencing higher posttraumatic distress at study entry may benefit more from one type of treatment over another, and since the research points to more intensive, trauma-focused interventions (such as EMDR) as the most supported treatments for PTSD, these therapies could be more beneficial than psychoeducational approaches (such as PFA).

Type of trauma may also impact trauma recovery. Trauma type is often categorized in the literature as assaultive violence, injury or shocking experience, serious trauma to a loved one or sudden death of a loved one. Within these categories, epidemiological studies have shown that the subsets of trauma carrying the highest risk for PTSD include sexual assault, being attacked and injured, serious accidents and sudden unexpected death of a loved one (Breslau, 2009; Kessler, 1995; van Ameringen, Mancini, Patterson and Boyle, 2008).

The loss of a loved one can be further broken down into natural and non-natural/violent causes (suicide, homicide or accidents). Grief is a complicated process and it becomes more so when individuals lose a loved one suddenly and unexpectedly (Sanders, 1993). The distress experienced after a violent death of a loved one has been shown to be compounded and more severe than death from natural circumstances (Lehman, Wortman and Williams, 1987; Amick-McMullan, Kilpatrick and Resnick, 1991; Kaltman and Bonanno, 2003). The grief process encompasses many emotions, cognitions and behaviors that change over time (Shear, 2012). Many times people experience yearning, sadness, an aching void, disorientation, guilt, remorse, and/or anxiety. There can even be feelings of bitterness, anger and resentment surrounding the

death. Some people have difficulty moving on towards a more adaptive and healthy stage of bereavement due to continuing maladaptive cognitions and avoidance behavior, which maintain acute grief and distress (Shear, 2012). These states may be even more pronounced and intertwined with symptoms of posttraumatic stress when the loss of a loved one happens under traumatic circumstances, such as knowing a loved one was attacked, maimed, or something of the sort.

In a review of the bereavement literature, Kristensen, Weisaeth and Heir (2012) concluded that sudden and violent loss puts an individual at significantly higher risk for experiencing elevated PTSD and depressive symptoms with a longer recovery time than an individual experiencing loss due to natural death. This may be in part due to the media involvement and criminal justice procedures that arise following violent death in particular, which can make the experienced more difficult for the bereaved.

Williams, Burke, McDevitt-Murphy and Neimeyer (2012) looked specifically at changes in psychological and physical consequences of homicidal bereavement in 47 African Americans over six months without intervention. Results indicated that there was a significant decrease in depressive symptoms and aspects of complicated grief over time but no changes in PTSD symptoms or health functioning, indicating the potential benefit of intervention. As far as treatments, Kristensen et al. (2012) concluded in their review that CBT, grief-focused therapies and exposure techniques are efficacious in reducing mental health problems as a result of loss for bereaved individuals showing clear distress. Therapists can intervene to help grieving individuals process the incident and arrive at more adaptive cognitions, especially when the individuals are experiencing anxiety in addition to grief (Shear, 2012). Efficacious trauma intervention may help

decrease PTSD and physical health symptoms in African Americans and other populations after traumatic loss which may not ameliorate otherwise, especially treatments that focus on the trauma and use CBT as well as exposure techniques.

This Dissertation

In order to address the literature regarding efficacious post-trauma intervention and individual/trauma factors affecting recovery in this study, we selected three types of treatments to investigate 1) which intervention is the most efficacious, and 2) who benefits most from which intervention over time in a low-SES sample. Each of the three interventions were offered in a brief, four-session/four-week format which utilize both group and individual structure so that the effect of brief intervention and group versus individual approaches can be investigated for the different treatments. In addition, a range of treatments (from standard-of-care to intensive, trauma-focused therapy) was chosen so that individual and trauma factors affecting recovery could be explored.

Our control group, Psychological First Aid (PFA) was offered in a group format and we selected four of what we thought would be the most useful of the eight total components ("Stabilization," "Safety and Comfort," "Coping," and "Linkage with Collaborative Services"). Our first experimental group, Stress Management Treatment, was also offered in group format and was augmented to include a trauma focus and written disclosure/emotional expression component during which participants wrote for twenty minutes about their trauma each session. Our second experimental group, eye movement desensitization and reprocessing (EMDR), is an intensive, trauma-focused

treatment which we offered in individual format. See the "Method" section (pg. 32) and Appendices 1-3 for a more detailed description of interventions and procedures.

SUMMARY

In sum, statistics show that minorities, especially in urban areas, experience higher rates of trauma than the population at large and are therefore more susceptible to resulting distress. Previous literature shows that certain post-trauma psychological interventions are effective at reducing posttraumatic distress and associated physical symptoms and these interventions are recommended by several organizations for the treatment of PTSD. Psychological First Aid (PFA), the standard-of-care offered by the American Red Cross, is one such intervention that has shown to be effective in the aftermath of a crisis to reduce distress. Stress Management Treatment (SMT), with or without a trauma focus, has also been shown to be beneficial and can be augmented to incorporate other aspects of evidence-based post-trauma treatment. Eye-movement Desensitization and Reprocessing (EMDR), which is trauma-focused and has been shown to be effective especially in single-trauma populations, is one of the most supported treatments for symptoms of PTSD and can facilitate clinically significant improvements in minimal sessions.

Minorities living in urban areas may not have access to psychological treatment and may not be able to afford long term services. Brief intervention and group format may be particularly helpful with this population in order to fully utilize minimal resources and maximize treatment gain for a majority of individuals. Furthermore, it would be useful to know which type of individual can benefit from brief treatment and

who may need more intensive intervention. We will be examining individual and trauma factors that have been related to recovery and some factors that have not yet been investigated. It is of value to investigate these factors further in order to determine who can benefit most from which type of treatment.

In the underserved, highly minority-populated community of Liberty City, citizens' medical and health-related needs are largely served by a well-established health center, the Jessie Trice Community Health Center (JTCHC), which provides comprehensive care on a reduced-cost basis. However, as trauma is highly prevalent among these citizens, more readily available, psychological-oriented services are needed to help individuals cope and move forward in a positive direction following a traumatic event.

This proposed study, led by project leader Dr. Gail Ironson, is part of a larger study funded by the National Institute on Minority Health and Health Disparities (NIMHHD) and has several objectives. For the study outlined in this dissertation, we worked together with JTCHC to provide three types of brief, posttraumatic stress oriented psychological intervention (Psychological First Aid, Stress Management Treatment, and eye movement desensitization and reprocessing) for the underserved citizens of Liberty City. This dissertation will attempt to identify the most effective interventions for brief post-trauma treatment in a minority, low socioeconomic sample as well as identify who might benefit best from which treatment.

CHAPTER 2. OBJECTIVES AND HYPOTHESES

Objective 1: First, we will determine the main effect of treatment type on preventing the worsening of posttraumatic stress and depressive symptoms.

Hypothesis: We hypothesize that EMDR (Eye Movement Desensitization and Reprocessing) will be the most effective brief treatment for acute traumatic stress (measured by posttraumatic stress and depressive symptoms) in our sample, with group SMT (stress management) being better than the control, group PFA (Psychological First Aid).

Objective 2: Our second aim is to determine the main effect of treatment type on physical symptoms experienced after trauma.

Hypothesis: We hypothesize that EMDR (Eye Movement Desensitization and Reprocessing) will be the most effective brief treatment for physical symptoms experienced after a trauma in our sample, with group SMT (stress management) being better than the control group PFA (Psychological First Aid).

Objective 3: Thirdly, we will focus on individual factors and trauma factors that predict psychological recovery, by determining the effect of these factors on posttraumatic stress and depressive symptoms by intervention and which intervention works best for which factor. Individual factors to be examined include severity of baseline PTSD score, gender, substance use, past trauma history (prevalence or absence of childhood sexual abuse), borderline personality disorder (number of borderline traits), and participation level (attendance and discussion of trauma) to predict recovery on the

outcome variables. Trauma factors to be examined are the nature of trauma (violent, bereavement or violent bereavement trauma) and time since trauma.

Hypotheses: Concerning individual factors and psychological recovery, we believe that higher baseline PTSD score, substance abuse, childhood abuse and prevalence of borderline personality disorder will predict overall worse recovery as indicated by worsening of PTSD symptoms or mood states. Within groups, we hypothesize that 4 sessions of EMDR will work best with people who have not experienced childhood abuse, and who do not have borderline personality disorder or substance abuse. For more severe cases (i.e. more baseline PTSD symptoms, prevalence of childhood abuse and/or borderline personality disorder, substance abuse), it may be better not to touch the trauma and that group PFA and/or group stress management might be best (except at reducing PTSD symptoms). We hypothesize that the SMT group (and even PFA) may be more effective for females than males because of the group format, the large number of females, and the likelihood that females may be more involved in discussions than males. In addition, we hypothesize that those individuals who participated more than others (i.e. those who attended all sessions and discussed their trauma) benefit more that those who do not. Concerning trauma factors, we are unsure how type of trauma (violent, bereavement, or violent bereavement trauma) and time since trauma will affect psychological recovery.

Objective 4: Our fourth aim is to determine which individual factors and trauma factors predict physical recovery (the same individual and trauma factors as listed in Aim 3 will be evaluated), as well as which intervention works best, taking each factor into account.

Hypotheses: Concerning individual factors and physical symptoms, we believe that higher baseline PTSD score, substance abuse, childhood abuse and prevalence of borderline personality disorder will predict worse recovery as indicated by increased or stable physical symptoms for participants who endorsed them at the baseline assessment. Within groups, we hypothesize that 4 sessions of EMDR will work best with people who have not experienced childhood abuse, and who do not have borderline personality disorder or substance abuse. For more severe cases (i.e. more baseline PTSD symptoms, prevalence of childhood abuse and/or borderline personality disorder, substance abuse), it may be better not to touch the trauma and that group PFA and/or group stress management might be best. We hypothesize that the SMT group (and even PFA) may be more effective for females than males because of the group format, the large number of females, and the likelihood that females may be more involved in discussions than males. In addition, we hypothesize that those individuals who participated more than others (i.e. those who attended all sessions and discussed their trauma) benefit more that those who do not. Concerning trauma factors, we are unsure how type of trauma (violent, bereavement or violent bereavement trauma) and time since trauma will affect physical symptoms.

CHAPTER 3. METHOD

Sample: Our sample is predominantly low-income African American, largely from the underserved community of Liberty City, Florida. All participants were enrolled on a paid volunteer basis and were monetarily compensated for their time. After screening, eligible participants were randomly assigned to one of three intervention groups. For the parent grant, we had a total of 357 participants screened and of those, 104 were randomized. By group, 33, 37 and 34 participants were assigned to the EMDR, group SMT and group PFA groups, respectively (see Chart 1 for study consort flow chart). We had 88 completers at one month follow up assessment, 85 completers at the three month follow up assessment and 82 completers at the 6 month follow up assessment. For the sample used in this study, we included all participants with follow-up data from at least one time point. This brought the total sample to 88 participants. However, one individual was removed from EMDR analyses due to repeated and compounding traumas during and after treatment that rendered follow-up data for the original trauma unreliable. This brought the total sample for this study to 87, with 27, 29 and 31 participants in the EMDR, SMT and PFA groups, respectively (note that Chart 1 identifies 28 people completing F1 assessment, but this number includes the individual that was removed as mentioned above). Demographics for the sample used in this dissertation (total and by group) can be found in Table 1. Descriptives for outcome variables at baseline can be found in Table 2, and descriptives for predictors be found in Tables 3 and 4.

Screening: We began by recruiting participants who had a trauma in the last 6 months from the Jessie Trice Community Health Center, physicians' offices, churches

and other social service agencies (see Table 5 for recruitment breakdown). Initial phone interviews were conducted to screen for exclusion criteria and this was further assessed during the baseline visit. We excluded people who were psychotic, drug dependent, who dissociate, or who would not be appropriate for short-term trauma treatment. If the trauma was more than one month prior to the phone screen, the participant was required to have symptoms from at least two of the PTSD clusters. When people did not meet eligibility requirements for our study or needed immediate treatment for more severe issues, they were given the numbers for community organizations where they could seek the necessary services. These referrals included sliding scale clinics within the individual's community as well as crisis centers and telephone hotlines.

Design Overview: The study began with a baseline visit, then random group assignment if eligible, four weeks of intervention, and then follow-up assessments at one, three and six months post-intervention. At baseline, appropriate research participants signed an informed consent form and any questions they had were answered. Self-report and interview measures were administered at this time as well and can be viewed by time of administration in Table 6. At the end of the baseline visit, participants were randomly assigned to one of three treatment groups, either four sessions of individual trauma treatment using EMDR, four sessions of group stress management with a trauma focus (referred to as SMT; CBT with relaxation skills and an expressive trauma writing component), or four sessions of selected modules from the "Psychological First Aid" (PFA) manual of the Red Cross (elaborated below). All group and individual sessions were 90-minutes to 2-hours long and were audio-taped for the purposes of supervision and fidelity checks.

Therapists, Supervisors and Assessors: The therapists for the EMDR, group SMT and group PFA interventions were master's level individuals and were trained on all three treatment approaches. All therapists (three total) completed training in level 1 and 2 EMDR, were trained in PFA by the American Red Cross and completed SMT training. Ethnicities of therapists varied (one African American, one Caribbean American, and one Caucasian) and each therapist provided each treatment approach. They were supervised by Drs. Gail Ironson and Blanche Freund, both of whom are trained in PFA, SMT, levels 1 and 2 of EMDR and fidelity checked. Carol Crow, who is a certified EMDR supervisor, provided additional supervision for EMDR including a workshop in the use of the cognitive interweave (an EMDR technique). Follow-up assessments were also done by master's level individuals and the assessor was blind to the group assignment.

Brief Description of Interventions: Our control intervention, Psychological First Aid (PFA), included selected modules from the "Psychological First Aid" manual of the American Red Cross (specifically "Stabilization," "Safety and Comfort," "Coping," and "Linkage with Collaborative Services"). This is intended as a "standard of care" support control group and was not as integrated with each individual's traumatic experience as were the other two experimental groups. Instead, psychoeducation surrounding stress reactions was provided and discussed with the group according to the protocol (see Appendix 1 for PFA protocol by session for this study). If a participant began discussing their individual trauma, they were redirected to the more general topics and identified ways in which they noticed the topic at hand in their lives.

Our first experimental intervention, Stress Management Treatment (SMT), was a group-administered intervention where the traumatic event was discussed, as were other

topics that are relevant to disaster recovery. As a framework, we used materials that were previously developed for a stress management program for HIV+ people (Antoni, Ironson, & Schneiderman, 2007), and augmented this material with more trauma-focused information. Topics in the four sessions included: awareness of stress/trauma symptoms, cognitive restructuring, coping, and using resources such as social support and spirituality (see Appendix 2). Relaxation techniques were also taught, including deep breathing, muscle relaxation, imagery, and mindful meditation. In each of the four sessions, to add a trauma-focus and exposure component, the participants wrote for twenty minutes about the details and emotions surrounding their individual trauma experience. The group facilitators tracked each participant's involvement in the group over time (e.g., being active in discussion, taking home the manual for review, etc.).

Our second experimental intervention was Eye-Movement Desensitization and Reprocessing (EMDR). Participants randomized to the EMDR treatment group received 4 individual sessions of active EMDR, which followed the protocol suggested by Francine Shapiro. The eight phases were included in the sessions and are described in detail in her book (Shapiro, 1995) as well as in the EMDR manual from level 1 training and briefly in Appendix 3. In EMDR sessions, patients are asked to briefly visualize their trauma and identify a negative core cognition relating to the trauma. They are then subtly guided by the therapist to process their experience, which finally helps them arrive at a more realistic positive cognition. During the course of treatment, associated emotions and physical sensations are also processed as needed.

Assessments Overview: One and three month follow-up assessments were funded by the original NIH grant for the study and the six month follow-up assessments were

funded by an additional grant that was obtained from EMDRIA. Instruments given during the baseline visit only included the SCID psychotic screen (Unusual symptoms), the SCID screening questions for drug/alcohol dependence, which was followed by the module for those people who said "yes" to the screener questions (to rule out drug dependence), the SCID borderline personality disorder screener (BPD), and the dissociative experiences questionnaire (DES). These were used for further screening of participants. Also given at baseline was the trauma history questionnaire (brief version), used to identify individuals with extensive childhood sexual or physical trauma.

Outcome measures were given at baseline and then one month, three months, and six months post-intervention (see Table 5 for administration schedule). They cover the following variables: a) PTSD symptoms (Davidson PTSD Scale and Post Traumatic Cognitions Inventory [PTCI]), b) depressive symptoms (Beck Depression Inventory-II and Hamilton Depression Scale), c) physical symptoms (The PILL), d) substance use (Addiction Severity Index; ASI/Drug Use). Other data and demographics were gathered at the phone screen and baseline interview, such as type of trauma, time since trauma and gender. All of the interviewer-administered measures were administered by an interviewer who was blind to the group assignment. In addition, as mentioned above, level of participation in group will also be looked at as an outcome measure in the SMT group and was recorded for each participant by the therapists after each SMT intervention session.

Screening Measures:

Screening form. This form was used for potential participants who called with interest in the study. Basic demographic information, contact information, type and date

of trauma, PTSD symptoms, basic medical and psychiatric history, and substance use were recorded. If a caller seemed to fit requirements, a baseline assessment session was then scheduled.

Structured Clinical Interview for DSM-IV for Diagnosis and Screening (First et al., 2002). We administered selected modules of the SCID for screening and diagnostic purposes. All participants were administered the full PTSD module for diagnosis. To assess criteria for exclusion, participants also received the SCID psychotic screen as well as screening questions for current drug or alcohol dependence. To screen for suicidality, we used scores on the BDI-II questions 2 (hopelessness) and 9 (suicidal ideation) and followed-up with a clinical interview. Patients who were excluded were referred to appropriate mental health programs in the community including the Psychological Services Center at University of Miami (a low cost clinic for clients from the community) as well as other community centers and telephone hotlines. The SCID module for PTSD diagnosis was repeated at the one, three and six months follow-up visits.

Dissociative Experiences Scale (DES). (Bernstein & Putnam, 1986; van Ijzendoorn & Schuengel, 1996) The DES is a 28 item self-report scale (each scored on a 0 to 100 scale) with demonstrated reliability and validity, which is used to measure the degree to which participants experience dissociation. While dissociation occurs in normal individuals, it is elevated in individuals with major mental illnesses, especially those with borderline personality disorder. The overall score is the average rating. We used it to screen out individuals for whom a brief trauma treatment would not be sufficient (DES>30).

Structured Abuse and Trauma Interview: In order to obtain standardized questions that assess trauma including sexual and physical abuse history, we used a previously developed structured interview (Leserman, et al. 1996; Leserman, et al. 1997; Leserman, et al. 1998; Leserman, et al. 1998; Leserman, et al. 1995). This measure of sexual and physical abuse was originally adapted from other research (Kilpatrick, et al. 1993; Badgley, et al. 1984; Koss, et al. 1985) and it correlates highly with many indicators of poor health (Leserman, et al. 1996; Leserman, et al. 1998). This measure defines sexual abuse to include the following experiences where force or threat of harm is used: 1) touching the participant's breasts, pubic area, vagina or anus with hands, mouth or objects, 2) making the participant touch the perpetrator's pubic area or anus with hands, mouth, or objects, and 3) making the participant have vaginal or anal intercourse. To meet criteria for sexual abuse, there must be clear force or threat of harm, however, in children (<13 years) threat of force is implied by a 5 year age differential between the victim and perpetrator. We define physical abuse as incidents separate from sexual abuse that include: 1) life threat (being physically attacked with or without a weapon, with the intent to kill or seriously injure), and 2) other physical abuse (being beat up, hit, kicked, bit, or burned by another person, incidents outside the range of normal "spanking" or kids fighting). The Structured Abuse Interview also includes questions concerning possible abuse risk factors (e.g., age at onset, number of episodes, serious injury, and number of perpetrators).

The interview also includes questions concerning a range of other childhood and adult traumas such as: 1) problems with primary caretakers (e.g., being mentally ill or alcoholic, going to prison, parental divorce, and seeing mother beat up or life threatened,

2) major events happening to participants (e.g., placed in orphanage or reform school, put in prison, life threatening illness, natural disaster, witnessing violence), and 3) trauma to close relative or friend (e.g., murder, death of family member). This list of traumas was adapted from other research (Felitti, et al. 1998; Kilpatrick, et al. 1993), where more traumatic events have been associated with later poor health outcome (Felitti, et al. 1998).

Background & Outcome Measures:

Demographic Questionnaire. This brief self-report questionnaire was given to all patients prior to entering the study. This questionnaire was used to obtain demographic variables (age, race, education, SES), weight, height, health habits (e.g., exercise, smoking) and medication use.

Davidson PTSD Symptom Scale (Davidson, et al. 1997; Zlotnick, et al. 1996). This 17-item measure is based on the PTSD symptom clusters defined by DSM-IV. Each item is rated from 0 to 4 for both frequency and severity during the past week. Items are summed for a total score, and subscales of re-experiencing, avoidance, and arousal are computed. The total scale has demonstrated good test-retest reliability (r=.86) and internal consistency (r=.99). The subscales also have high reliability (alpha ≥ .83). In diagnosing PTSD compared to the *Structured Clinical Interview for the DSM-IV* (SCID), (using a cutoff score of 40) the positive predictive value was 92% and the negative predictive value was 79%, with a diagnostic accuracy of .83 (Davidson et al., 1996). The Davidson scale was used to screen patients to determine if participants meet study inclusion/exclusion criteria. Participants did not have to meet criteria for PTSD (identified as a score of >32; still obtaining this reference from Dr. I) to be enrolled in the study but had to show symptoms in two of the symptom clusters, so that a range of

posttraumatic stress is represented in our sample. In our data from a previous study (Ironson et al., 2013), women with elevated PTSD symptom scores (the cut off of 20 and 40) who were in a post Trauma Writing group showed significant improvement compared to the Daily Events Writing control.

The Pennebaker Inventory of Limbic Languidness. This brief self-report questionnaire (see Appendix 4) assesses frequency of common symptoms or bodily sensations recently experienced by the participant (Pennebaker, 1982) and has demonstrated high validity and reliability. It consists of 54 items and total scores can range from zero to 216 (112.7 is the mean found in college students by Pennebaker, 1982). In follow-up assessments, this measure was adjusted to target symptoms experienced in the time since the last assessment (e.g. the last month or last three months).

Beck Depression Inventory-II (BDI-II). The BDI-II is a 21-item instrument that assesses cognitive, affective and somatic symptoms of depression (Beck. 1978; Beck, et al. 1961). The items consist of statements that are scored on a range of 0 to 3. The BDI-II has acceptable test-retest reliability (r=.79) in non-clinical populations and demonstrates concurrent validity (range from .67 to .79) in both clinical and non-clinical populations.

Hamilton Depression Scale (HAM-D). The HAM-D is an interviewer administered, 17-item scale designed to measure depression and has high demonstrated validity and reliability (Hamilton 1960, 1969). The items consist of statements that are scored on a range of 0 to 2 or 0 to 4 and has a total score range of 0 to 52.

Post Traumatic Cognitions Inventory (PTCI). The PTCI is a 33-item questionnaire (scoring ranges from 0-63) that measures trauma-related thoughts and beliefs, including three factors: negative cognitions about self, negative cognitions about world, and self-blame (Foa et al., 1999). The three factors show excellent internal consistency, test–retest reliability, and convergent validity.

Addiction Severity Index (ASI). This questionnaire assesses a participant's lifetime alcohol and drug use, use in the past 30 days, and route of administration of substance (McLellan et al., 1980). It also assesses amount of treatment for abuse or addiction received by the participant and any negative effects their substance use has caused them.

ANALYSES

Hierarchical linear modeling (HLM; Bryk & Raudenbush, 2002) was used to explore all four objectives and hypotheses. HLM was chosen because it allows us to predict slope over time instead of a predicting a single time point. It also allows us to control for important variables such as baseline PTSD, depressive and physical symptoms that may vary for each participant at different time points. Outcome variables are PTSD symptoms (measured by the Davidson and PTCI), depressive symptoms (measured by the BDI and Hamilton) and physical symptoms (measured by the PILL). Each outcome was modeled from its baseline value, time, group assignment, and interaction of predictor and group. For the first and second aims, HLM was used to explore associations between treatment type and psychological and physical outcomes over a follow-up period of six months post-intervention (four time points: baseline, one month, three months and six

months). We also used HLM analyses to determine if individual/trauma factors listed previously predict emotional/physical recovery over time. Within these analyses, it was determined which treatment type works best for which individuals and which trauma factors.

Analyses were performed on HLM software version 6.06. Both Level 1 and Level 2 datasets were prepared using SPSS version 17.0. To reduce data scoring and entry error, data was double-scored and double-entered by two separate individuals and the entries were then compared. Discrepancies were investigated and corrected for by returning to the original file data. Outlier data on each variable was identified (i.e., flagged if more than 3.0 standard deviations above the mean) and winsorized for accurate analysis. Winsorizing involved replacing outlier values with the value of 3 standard deviations above the mean for the respective variable, except for one participant outlier on the Hamilton at F3; if winsorized in this way, this participant's score would have fallen into another diagnostic category, so we replaced this outlier value with the lowest value of the diagnostic bracket in which it originally fell, which was only 1 point different from the 3rd standard deviation value. In total, only four to six individual scores were altered at each time point, with the lowest number on the PTCI outcome (one individual at baseline only) and the highest number on the PILL outcome (seven altered scores total; one at baseline, and two at F1, F3 and F6). Once all data was clean and adjusted as needed, the analyses to test the hypotheses were performed. Due to the large number of analyses necessary to explore the associations, linear regression analyses and ANOVAS were performed first to determine existing patterns and relationships in the

data. Results of these analyses can be found in Appendix 5. The following HLM analyses were performed only for relationships that were found significant using these preliminary analyses.

See Table 7 for basic HLM equations for evaluating the direct and indirect (interaction with individual/trauma factors) effects of group assignment on continuous study outcomes. Level 1 includes the *Time* variable. The time structure, what is defined as months since baseline, was coded as 0 for baseline, 2 for the one month follow-up assessment, 4 for the three month follow-up assessment, and 7 for the six month follow-up assessment and were held constant within subjects. Base models were run to explore slope of an outcome over time, and then standard covariates were added into the model (i.e., age, gender, ethnicity & education). Then group effects were explored. The Level 2 equation used between person characteristics (grouping variables), such as randomized treatment condition, gender and the interaction of the two in order to predict the slope, which represents change in the dependent variables over time. Individual variation at study entry was controlled for in the Level 2 models by including the baseline value of the dependent variable as a covariate. All Level 2 dichotomous variables were coded as a 0 or 1 to facilitate meaningful and accurate evaluation of parameter estimates and continuous variables were centered around their group means. The treatment groups in Level 2 were dummy coded in order to evaluate effect differences. Each group was compared to the other (EMDR vs. group PFA; group SMT vs. group PFA; EMDR vs. group SMT) for the first two objectives (main effects) and then for the second two objectives (individual and trauma factors affecting recovery). In addition, treatment versus control was dummy coded (TXvsCTRL) and was run for main effects only to see

if being in any treatment group (SMT or EMDR) was better than being in the control (PFA). Chi-square analyses were also performed in HLM to determine if being in a treatment group (SMT or EMDR) added significantly to the predictive ability of the base models. Treatment effects and individual/trauma factor effects were determined in the Level 2 model by examining beta coefficient significance that is associated with treatment condition.

To explore gender participation differences as a potential confound, four of the recorded sessions from the PFA group were evaluated. It was concluded that the conversation across sessions was mixed and not necessarily dominated by one gender. Therefore, gender differences in group discussion participation does not seem to be an issue and this factor alone is not believed to affect treatment outcomes.

Demographic data and baseline outcome averages per group are displayed in Tables 1 and 2. The PTSD variable is continuous in nature. On the depressive symptoms outcome variable, scores on the BDI-II and the HAM-D were converted to z-scores for accurate comparisons and were correlated to determine if a composite score was possible. If the correlation was .6 or above, a composite score would have been made; however, the correlation was less than .6, so the measures were analyzed separately. Depressive and physical symptoms outcome variables are also continuous in nature and are defined as number of symptoms.

Data regarding individual and trauma factors for each group is displayed in Tables 3 and 4. Distributions were examined to determine appropriate cut-offs for these variables. Baseline Davidson PTSD severity is dichotomous, defined as ≤ 32 or > 32, to

identify individuals who met criteria for PTSD at baseline (still obtaining this reference from Dr. I). Borderline personality disorder (BPD) was also dichotomous, identifying those who reported experiencing < 5 or ≥ 5 symptoms, which is the cutoff identified in the DSM-IV-TR (APA, 2000). Childhood sexual abuse (CSA) was dichotomous as well, identifying those with a self-reported history of forced or unwanted sexual interaction by someone five years older before the age of 18 or no history. For substance abuse, the ASI was used to identify drug categories in which at least 10% of the sample reported use. Of the categories, only alcohol and marijuana use met this requirement, so these two substances were explored in analyses. Alcohol use was defined as < 10 days or ≥ 10 days of drinking in the past month (i.e., the cutoff for the DSM-IV-TR alcohol screen; APA, 2000) and marijuana use was defined as any or no use in the past month (due to the low number of reporters), both dichotomous variables. The proposed attendance variable was removed from analyses because all participants attended or made-up all four treatment sessions (this was a requirement of study participation). Discussion of trauma within sessions was dichotomous, coded as whether or not the individual discussed their particular trauma during treatment. For trauma factors, time since trauma (defined as ≤ 5 weeks or > 5 weeks, based on the DSM-IV-TR cutoff for acute or recent trauma; APA, 2000) and trauma type (i.e., violent, bereavement, or violent bereavement traumas) are dichotomous in nature.

Analyses were run as appropriate to evaluate whether groups were matched at baseline on outcome variables and individual/trauma factors (Tables 3 and 4). Groups were matched on all variables except discussion of trauma (X^2 (2, N = 87) = 24.944, p = .000), with the highest number of discussers in group EMDR (27 people discussed;

discussion was mandatory in this group) and the lowest number of discussers in group PFA (12 people discussed; discussion was discouraged in this group). In addition, it should be noted that a trend was found for group differences in violent bereavement trauma type (X^2 (2, N = 87) = 4.836, p = .089), with group SMT reporting the highest violent bereavement trauma type (12 people endorsed) and group PFA reporting the lowest (5 people endorsed). Partial correlation analyses were performed for both of these variables to determine if they were related to the outcome values at the 6-month follow-up when controlling for baseline value of the outcome. Violent death was not correlated with any outcome, but the discussed trauma predictor was significantly correlated with the PTCI outcome. After further investigation, it was determined that comparisons between group do not make sense on this variable, as trauma discussion was not allowed in PFA (immediately redirected to the subject at hand), somewhat discussed verbally in SMT (verbal discussion was voluntary but all participants wrote about their trauma), and always discussed in EMDR (this was the focus of this approach). In order to still use this data, HLM analyses were run to consider PFA and SMT separately, in order to see if within group, discussers of the trauma benefitted more than non-discussers.

CHAPTER 4. RESULTS

Sample Characteristics

Our sample (n = 87) is predominantly low-income African American, largely from the underserved community of Liberty City, Florida. Demographic information for the total sample as well as each treatment group can be found in Table 1. Participants were of diverse gender, ethnicity and sexual orientation. Groups varied significantly on levels of education, with individuals in the SMT group being less likely to have education beyond high school. Descriptive information for participant scores on outcome measures can be found in Table 2. Participants varied on level of individual and trauma factors, as shown by the descriptive information for these measures in Tables 3 and 4. Figures 1-6 depict the change in means over time by group for each outcome.

Testing of the Hypotheses

The basic equations for the HLM models as well as the explanation of equation terms can be found previously in the "Objectives and Analyses" section beginning on page 29. Main effects of treatment type on outcomes as well as individual and trauma factors that influence recovery were explored. Analyses were run to compare each treatment group to another, resulting in six separate group runs (EMDR versus other, SMT versus other, EMDR and SMT versus PFA, SMT versus PFA, EMDR versus SMT, and EMDR versus PFA). In addition, TXvsCTRL was run for main effects only to see if being in any treatment group (SMT or EMDR) was better than being in the control (PFA).

Objective 1: First, we will determine the main effect of treatment type on preventing the worsening of posttraumatic stress and depressive symptoms.

Hypothesis: We hypothesize that EMDR (Eye Movement Desensitization and Reprocessing) will be the most effective brief treatment for acute traumatic stress (measured by posttraumatic stress and depressive symptoms) in our sample, with group SMT (stress management) being better than the control, group PFA (Psychological First Aid).

Objective 2: Our second aim is to determine the main effect of treatment type on physical symptoms experienced after trauma.

Hypothesis: We hypothesize that EMDR (Eye Movement Desensitization and Reprocessing) will be the most effective brief treatment for physical symptoms experienced after a trauma in our sample, with group SMT (stress management) being better than the control group PFA (Psychological First Aid).

Objective 3: Thirdly, we will focus on individual factors and trauma factors that predict psychological recovery, by determining the effect of these factors on posttraumatic stress and depressive symptoms by intervention and which intervention works best for which factor. Individual factors to be examined include severity of baseline PTSD score, gender, substance use, past trauma history (prevalence or absence of childhood sexual abuse), borderline personality disorder (number of borderline traits), and discussion of trauma to predict recovery on the outcome variables. Trauma factors to be examined are the nature of trauma (violent, bereavement or violent bereavement trauma) and time since trauma.

Hypotheses: Concerning individual factors and psychological recovery, we believe that higher baseline PTSD score, substance abuse, childhood abuse and

prevalence of borderline personality disorder will predict overall worse recovery as indicated by worsening of PTSD symptoms or mood states. Within groups, we hypothesize that 4 sessions of EMDR will work best with people who have not experienced childhood abuse, and who do not have borderline personality disorder or substance abuse. For more severe cases (i.e. more baseline PTSD symptoms, prevalence of childhood abuse and/or borderline personality disorder, substance abuse), it may be better not to touch the trauma and that group PFA and/or group stress management might be best (except at reducing PTSD symptoms). We hypothesize that the SMT group (and even PFA) may be more effective for females than males because of the group format, the large number of females, and the likelihood that females may be more involved in discussions than males. In addition, we hypothesize that those individuals who discussed their trauma benefit more that those who do not. Concerning trauma factors, we are unsure how type of trauma (violent, bereavement, or violent bereavement trauma) and time since trauma will affect psychological recovery.

Objective 4: Our fourth aim is to determine which individual factors and trauma factors predict physical recovery (the same individual and trauma factors as listed in Aim 3 will be evaluated), as well as which intervention works best, taking each factor into account.

Hypotheses: Concerning individual factors and physical symptoms, we believe that higher baseline PTSD score, substance abuse, childhood abuse and prevalence of borderline personality disorder will predict worse recovery as indicated by increased or stable physical symptoms for participants who endorsed

them at the baseline assessment. Within groups, we hypothesize that 4 sessions of EMDR will work best with people who have not experienced childhood abuse, and who do not have borderline personality disorder or substance abuse. For more severe cases (i.e. more baseline PTSD symptoms, prevalence of childhood abuse and/or borderline personality disorder, substance abuse), it may be better not to touch the trauma and that group PFA and/or group stress management might be best. We hypothesize that the SMT group (and even PFA) may be more effective for females than males because of the group format, the large number of females, and the likelihood that females may be more involved in discussions than males. In addition, we hypothesize that those individuals who discussed their trauma benefit more that those who do not. Concerning trauma factors, we are unsure how type of trauma (violent, bereavement, or violent bereavement trauma) and time since trauma will affect physical symptoms.

Prediction to DAV Change Over Time

Basic Model

Davidson PTSD slope, representing change in PTSD symptomatology, significantly decreased over time when controlling only for time since baseline (γ_{10} = -3.57, $p < .001$) and maintained significance when controlling for the other covariates (γ_{10} = -4.53, $p < .01$) as well as group assignment (γ_{10} = -4.61, $p < .01$; See Table 8). Table 9 exhibits the basic model results and significance tests for the change in Davidson PTSD over time, controlling for time since baseline, the standard covariates (age, gender, ethnicity, and education), and baseline Davidson score. Table 10 exhibits the same but

with the additional control for group assignment. The average Davidson PTSD score for participants upon study entry is 44.11, which decreased at a rate of 4.53 points per month. This decrease was found to occur above and beyond the effects of age, gender, ethnicity, and education. There is no significant individual variation in the change in Davidson PTSD over time when controlling for standard covariates (χ^2 (81) = 68.21, $p > .500$).

Covariates

All covariates were in the level 2 model. At level 2, higher Davidson PTSD at study entry was significantly related to a faster decline in Davidson PTSD scores over time (see Table 9).

The Contribution of Group Assignment and Individual and Trauma Factors

Significance tests for the predictive ability of group assignment on the change in Davidson PTSD over time are displayed in Table 11. In terms of group assignment, receiving treatment in the SMT group (β = 1.64, $p = .089$; trend) predicted a slower DAV decrease over time when compared to other groups. Specifically, when comparing the groups to each other, being in SMT predicted a slightly slower decrease in Davidson scores when compared to PFA (β = 1.70, $p = .102$; approaching significance on one-tailed test). Results of chi-square analyses indicate that being in any treatment group (SMT or EMDR) did not add significantly to the predictive ability of the base models (Table 12).

Significance tests for the predictive ability of individual and trauma factors on the change of Davidson PTSD over time are displayed in Table 13. Those with a history of CSA in the SMT group experienced a slower decline in Davidson scores over time (β = 3.87, $p = .048$) than did those in other groups. Specifically, when comparing the groups

to each other, SMT was significantly different than PFA (β = 4.04, *p* = .049) but not significantly different than EMDR in terms of change in Davidson scores over time for those with CSA, and EMDR and PFA were also not significantly different than each other. Individuals in the SMT group with a trauma categorized as a "death" of someone close to them experienced a faster decline in Davidson scores (β = -4.26, *p* = .044) than those in other groups. Specifically, when comparing the groups to each other, SMT was better than EMDR (β = 3.92, *p* = .095; trend) and PFA (β = -4.22, *p* = .091; trend) but EMDR and PFA were not significantly different than each other in terms of reducing Davidson scores over time for those who identified a "death" trauma. Similarly, individuals with a "violent death" trauma in the SMT group experienced a significantly faster decline in Davidson scores (β = -4.97, *p* = .012) than those in other groups while individuals in the EMDR group experienced a slower decline in Davidson scores then those in the other two groups (β = 4.28, *p* = .086; trend). Specifically, when comparing the groups to each other, SMT was significantly better than PFA (β = -3.18, *p* = .056; trend) and EMDR (β = 6.07, *p* = .026) in terms of Davidson reduction over time but EMDR and PFA were not significantly different from each other. No significant relationships or trends were found for the other individual or trauma factors.

Interpretation

For the sample as a whole, Davidson PTSD scores significantly decreased over time and this decrease maintained significance when controlling for covariates and group assignment (Table 8). When comparing treatments, being assigned EMDR or PFA was better than being in SMT in terms of Davidson PTSD score reduction over time in this sample and PFA was better than SMT (trend; Table 14). These findings do not support

the hypothesis that EMDR would be the most effective brief treatment for post traumatic stress in this sample and that SMT would be better than the control group PFA. However, although SMT was the worst treatment (trend) for the reduction of Davidson scores, there were individual factors that indicate which treatment was significantly better for which population (Tables 15 and 16). For individuals with CSA, SMT was the worst treatment in terms of Davidson PTSD score reduction over time and PFA was significantly better. However, these findings were different when considering trauma types. For individuals with a "death" or "violent death" trauma, SMT was the best treatment for reducing Davidson scores, while PFA and EMDR performed similarly to each other. These findings for individual and trauma factors support the conclusion that for the reduction of Davidson PTSD scores over time, PFA is the most effective treatment if CSA is present and that SMT is the most effective if the trauma is a death or violent death.

Prediction to PTCI Change Over Time

Basic Model

PTCI (Post-traumatic Cognitions Inventory) slope significantly decreased over time when controlling only for time since baseline (γ_{10} = -4.24, $p < .001$) and maintained significance when controlling for the other covariates (γ_{10} = -4.35, $p < .05$) as well as group assignment (γ_{10} = -4.11, $p < .05$; Table 8). Table 17 exhibits the basic model results and significance tests for the change in PTCI scores over time, controlling for time since baseline, the standard covariates (age, gender, ethnicity, and education), and baseline Davidson score. Table 18 exhibits the same but with the additional control for

group assignment. The average PTCI score for participants upon study entry is 92.97, which decreased at a rate of 4.35 points per month. This decrease was found to occur above and beyond the effects of age, gender, ethnicity, and education. There is no significant individual variation in the change in PTCI scores over time when controlling for standard covariates (χ^2 (81) = 78.27, $p > .500$).

Covariates

All covariates were in the level 2 model. At level 2, higher PTCI score at study entry was significantly related to a faster decline in PTCI scores over time (see Table 17).

The Contribution of Group Assignment and Individual and Trauma Factors

Significance tests for the predictive ability of group assignment on the change in PTCI scores over time are displayed in Table 11. In terms of group assignment, no particular group was significantly related to PTCI score change over time, indicating the importance of exploring other factors that may influence recovery within group. Results of chi-square analyses indicate that being in any treatment group (SMT or EMDR) did not add significantly to the predictive ability of the base models (Table 12).

Significance tests for the predictive ability of individual and trauma factors on the change in PTCI scores over time are displayed in Table 19. Those with self-reported marijuana use (any use in the past month) in the SMT group experienced a faster decline in PTCI scores over time ($\beta = -4.36$, $p = .083$; trend) than did those in other groups. Specifically, when comparing the groups to each other for those with marijuana use, SMT was significantly different than PFA ($\beta = -6.32$, $p = .002$) but not EMDR in terms of change in PTCI scores over time, and EMDR and PFA performed similarly to each

other. For individuals with features of borderline personality, a trend was found that indicated those in the EMDR group experienced a faster decrease of PTCI scores over time (β = -3.82, *p* = .080; trend) when compared to the SMT group, but neither EMDR and PFA nor SMT and PFA were significantly different from each other. Within group, individuals in the PFA treatment who voluntarily discussed their trauma experienced a faster decrease in PTCI scores over time than those who chose not to discuss their trauma (β = -3.13, *p* = .050; Table 20) but no differences was found between discussers and non-discussers in the SMT group. No significant relationships or trends were found for the other individual or trauma factors.

Interpretation

For the sample as a whole, PTCI scores significantly decreased over time and this decrease maintained significance when controlling for covariates and group assignment (Table 8). When comparing treatments, no particular treatment was better than another in terms of PTCI score reduction (Table 14). These findings do not support the hypothesis that EMDR would be the most effective brief treatment for post traumatic stress in our sample, followed by SMT and lastly PFA. However, although no treatment was significantly better than the control for the reduction of PTCI scores, there were individual factors (no trauma factors reached significance) that indicate which treatment was significantly better for which population (Tables 15 and 16). In terms of PTCI score reduction, SMT worked best for individuals with self-reported marijuana use (trend), while EMDR and PFA were similar in performance. Conversely, EMDR worked better than SMT for individuals with borderline features (trend), but neither EMDR nor SMT were significantly different from PFA. It also seems that within PFA only, those who

discussed their trauma faired better in terms of PTCI reduction that those who chose not to discuss their trauma (Table 20). These findings for individual and trauma factors support the conclusion that EMDR would not be best for those who use marijuana and provide evidence that EMDR may work best for individuals with borderline features.

Prediction to BDI Change Over Time

Basic Model

BDI slope, representing change in depression symptomatology, significantly decreased over time when controlling only for time since baseline (γ_{10} = -1.02, $p < .001$) but did not maintain significance when controlling for the other covariates (γ_{10} = -0.47, $p = .254$) or group assignment (γ_{10} = -0.36, $p < .386$; Table 8). Table 21 exhibits the basic model results and significance tests for the change in BDI over time, controlling for time since baseline, the standard covariates (age, gender, ethnicity, and education), and baseline Davidson score. Table 22 exhibits the same but with the additional control for group assignment. The average BDI score for participants upon study entry is 12.33, which decreased, but not significantly, at a rate of 0.47 points per month. While there was a significant decrease found when just controlling for time since baseline, this decrease was not found to occur above and beyond the effects of age, gender, ethnicity, and education. There is no significant individual variation in the change in Davidson PTSD over time when controlling for standard covariates (χ^2 (81) = 66.28, $p > .500$).

Covariates

All covariates were in the level 2 model. At level 2, a higher BDI score at study entry was significantly related to a faster decline in BDI score over time (see Table 21).

The Contribution of Group Assignment and Individual and Trauma Factors

Significance tests for the predictive ability of group assignment on the change in BDI over time are displayed in Table 11. In terms of group assignment, receiving treatment in the EMDR group (β = -0.42, p = .086; trend) predicted a faster BDI decrease over time, but no relationship was found for any of the other group combinations. These results indicate that EMDR was better at reducing BDI than the other two groups but SMT and PFA were not significantly different than each other in terms of BDI score reduction in this sample. Results of chi-square analyses indicate that being in any treatment group (SMT or EMDR) did not add significantly to the predictive ability of the base models (Table 12).

Significance tests for the predictive ability of individual and trauma factors on the change of BDI scores over time are displayed in Table 23. Those with a baseline Davidson score of 32 or above (indicating clinically significant levels of PTSD at study entry) in the EMDR group experienced a faster decline (β = -1.25, p = .021) in BDI scores over time than did those in other groups. Specifically, when comparing the groups to each other, EMDR was significantly better than SMT (β = -1.57, p = .013) and PFA in terms of change in BDI scores over time (β = -1.30, p = .104; trend, significant on a one-tailed test). This indicates that EMDR performed best at reducing BDI scores over time for individuals with clinically significant PTSD at baseline assessment, followed by PFA and lastly SMT, which were not significantly different from each other. Within group, individuals in the PFA treatment who voluntarily discussed their trauma experienced a slower decrease in BDI scores over time than those who chose not to discuss their trauma (β = 0.65, p = .015; Table 20) but no differences was found between discussers and non-

discussers in the SMT group. In terms of trauma types, individuals in the EMDR group with a "violent" trauma experienced a slower decline (β = -0.84, *p* = .058; trend) in BDI scores than those in the PFA group, meaning PFA was the best at reducing BDI for those with a "violent" trauma, especially when compared to EMDR, but EMDR and SMT were not significantly different than each other. In addition, individuals in the SMT group with a "violent death" of someone close to them experienced a faster decline (β = -1.31, *p* = .007) in BDI scores than those in other groups. Specifically, when comparing the groups to each other, those with a "violent death" trauma in EMDR experienced a significantly slower decline (β = 1.25, *p* = .029) in BDI compared to those in SMT, and those in SMT experienced a significantly faster decline in BDI compared to those in PFA (β = -1.38, *p* = .014). EMDR was not significantly different than PFA in terms of BDI reduction over time for those with a "violent death" trauma. These results indicate that SMT is the best at reducing BDI scores over time for those with a "violent death" trauma. No significant relationships or trends were found for the other individual or trauma factors.

Interpretation

For the sample as a whole, depression symptomatology as measured by BDI significantly decreased over time but only when controlling for time since baseline alone, as the decrease did not maintaining significance when controlling for covariates and group assignment (Table 8). When comparing treatments effects overall, being assigned to EMDR was most effective at BDI depression symptoms overtime (trend; Table 14). These findings support the hypothesis that EMDR would be the most effective brief treatment for depressive symptoms in this sample, although neither treatment group was

significantly different from PFA when compared directly. While EMDR was overall most effective, there were individual and trauma factors that indicate which treatment was significantly better for which population (Tables 15 and 16). For participants who began treatment with clinically significant PTSD, EMDR was the best intervention for reducing BDI scores over time. It also seems that within PFA only, those who did not discuss their trauma faired better in terms of BDI reduction than those who did (Table 20). For trauma type, PFA was the best at reducing depression symptoms for those with a "violent" trauma (trend), but for those with a "violent death" trauma, SMT performed the best. These findings for individual and trauma factors do not support the hypothesis that EMDR would be harmful for more severe cases (i.e., higher baseline PTSD), as it was shown by these results to be the most helpful. In addition, these findings provide evidence for how trauma types may influence recovery with treatment, which will be discussed further in the next chapter.

Prediction to Hamilton Change Over Time

Basic Model

Hamilton slope, representing change in depression symptomatology, decreased over time when controlling only for time since baseline (γ_{10} = -0.21, p = .095; trend) but did not maintain significance when controlling for the other covariates (γ_{10} = -0.29, p = .396) or group assignment (γ_{10} = -0.23, p < .502; Table 8). Table 24 exhibits the basic model results and significance tests for the change in Hamilton over time, controlling for time since baseline, the standard covariates (age, gender, ethnicity, and education), and baseline Hamilton score. Table 25 exhibits the same but with the additional control for

group assignment. The average Hamilton score for participants upon study entry is 8.47, which decreased, but not significantly, at a rate of 0.29 points per month. This decrease was not found to occur above and beyond the effects of age, gender, ethnicity, and education. There is significant individual variation in the change in Hamilton over time when controlling for standard covariates (χ^2 (81) = 147.11, p = .000).

Covariates

All covariates were in the level 2 model. At level 2, a higher Hamilton score at study entry was significantly related to a slower decline in Hamilton score over time (see Table 24).

The Contribution of Group Assignment and Individual and Trauma Factors

Significance tests for the predictive ability of group assignment on the change in Hamilton over time are displayed in Table 11. In terms of group assignment, results indicate that no group was significantly better than another in terms of reduction in Hamilton scores over time. Results of chi-square analyses indicate that being in any treatment group (SMT or EMDR) did not add significantly to the predictive ability of the base models (Table 12).

Significance tests for the predictive ability of individual and trauma factors on the change of Hamilton scores over time are displayed in Table 26. Those with a baseline Davidson score of 32 or above (indicating clinically significant levels of PTSD) in the SMT group experienced a slower decline (β = 1.06, p = .020) in Hamilton scores over time than did those in other groups. Specifically, when comparing the groups to each other, EMDR was significantly better than SMT (β = -1.40, p = .018) in terms of

reducing Hamilton scores over time but was not significantly different than PFA. In addition, SMT had a slower decline in Hamilton scores when compared to PFA (β = 0.89, p = .059; trend). This indicates that SMT performed worst at reducing Hamilton scores over time for individuals with clinically significant PTSD at baseline assessment, with PFA and EMDR not being significantly different from each other. In terms of trauma types, individuals in the SMT group with a "violent death" trauma experienced a faster decline (β = -0.78, p = .090; trend) in Hamilton scores than those in the other two groups, meaning SMT was the best at reducing Hamilton scores for those with a "violent death" trauma. No significant relationships or trends were found for the other individual or trauma factors.

Interpretation

For the sample as a whole, depression symptomatology as measured by Hamilton significantly decreased over time but only when controlling for time since baseline alone, as the decrease did not maintain significance when controlling for covariates and group assignment (Table 8). When comparing treatments effects overall, group assignment made no significant difference in terms of change in Hamilton over time (Table 14). These findings do not support the hypothesis that EMDR would be the most effective brief treatment for depressive symptoms stress in this sample, followed by SMT and lastly PFA. However, although no treatment was significantly better than the control for the reduction of Hamilton scores, there were individual factors that indicate which treatment was significantly better for which population (Tables 15 and 16). For individuals who began treatment with clinically significant PTSD, SMT performed worst at reducing Hamilton scores over time. For trauma type, SMT performed the best for

those with a "violent death" trauma (trend). These findings for individual and trauma factors do not support the hypothesis that EMDR would be harmful for more severe cases (i.e., higher baseline PTSD), as it was shown to be more helpful than SMT. In addition, these findings provide evidence for how trauma types may influence recovery with treatment, which will be discussed further in the next chapter.

Prediction to PILL Change Over Time

Basic Model

PILL slope, representing change in physical symptoms, decreased over time when controlling only for time since baseline (γ_{10} = -1.63, p = .001) but did not maintain significance when controlling for the other covariates (γ_{10} = -1.56, p = .392) or group assignment (γ_{10} = -1.06, p < .550; Table 8). Table 27 exhibits the basic model results and significance tests for the change in PILL over time, controlling for time since baseline, the standard covariates (age, gender, ethnicity, and education), and baseline PILL score. Table 28 exhibits the same but with the additional control for group assignment. The average PILL score for participants upon study entry is 104.96, which decreased at a rate of 1.56 points per month. While there was a significant decrease found when just controlling for time since baseline, this decrease was not found to occur above and beyond the effects of age, gender, ethnicity, and education. There is no significant individual variation in the change in PILL over time when controlling for standard covariates (χ^2 (81) = 92.72, p = .176).

Covariates

All covariates were in the level 2 model. At level 2, a higher PILL score at study entry was significantly related to a faster decline in PILL score over time (see Table 27).

The Contribution of Group Assignment and Individual and Trauma Factors

Significance tests for the predictive ability of group assignment on the change in PILL over time are displayed in Table 11. In terms of group assignment, results indicate that receiving treatment in the SMT group ($\beta = 1.64$, $p = .076$; trend) predicted a faster PILL decrease over time when compared to other groups. Specifically, when comparing the groups to each other, SMT treatment predicted a significantly faster decline in PILL over time when compared to PFA ($\beta = -2.21$, $p = .042$). It was also shown that individuals in any treatment group (SMT or EMDR) experienced faster declines in physical symptoms over time than those in the control (PFA; $\beta = -1.77$, $p = .047$), although this relationship can be explained by the strength of the relationships found for SMT. These results indicate that SMT was the best at reducing PILL symptoms over time, especially when compared to PFA. Results of chi-square analyses indicate that being in any treatment group (SMT or EMDR) did not add significantly to the predictive ability of the base models (Table 12).

Significance tests for the predictive ability of individual and trauma factors on the change of PILL scores over time are displayed in Table 29. Those with a history of childhood sexual abuse in the EMDR group experienced a slower decline ($\beta = 3.84$, $p = .035$) in PILL scores over time than did those in other groups. Specifically, when comparing the groups to each other, EMDR predicted a significantly slower decline in PILL for those with childhood sexual abuse than did PFA ($\beta = -1.40$, $p = .018$) and SMT

had a slower decline in PILL scores when compared to PFA (β = 3.00, p = .080; trend). This indicates that PFA performed best at reducing PILL scores over time for individuals with a history of childhood sexual abuse, followed by SMT and lastly EMDR, although these latter two groups were not significantly different from each other. No significant relationships or trends were found for the other individual or trauma factors.

Interpretation

For the sample as a whole, physical symptoms as measured by the PILL significantly decreased over time but only when controlling for time since baseline alone, as the decrease did not maintaining significance when controlling for covariates and group assignment (Table 8). When comparing treatments effects overall, SMT predicted the fastest decline in physical symptoms, especially when compared to PFA (trend; Table 14). These findings do not support the hypothesis that EMDR would be the most effective brief treatment for physical symptoms in this sample. However, although SMT performed the best for the reduction of PILL scores, there was an individual factor (but no significance was found for trauma factors that indicate which treatment was significantly better for which population (Tables 15 and 16). For those with a history of childhood sexual abuse, PFA was the best at reducing physical symptoms and EMDR performed the worst. No significant differences were found for trauma factors. These findings support the hypothesis that EMDR could be harmful for those with a complex trauma history (i.e., childhood sexual abuse), and that PFA may have the most treatment impact for reducing physical symptoms after a trauma.

CHAPTER 5. DISCUSSION

Overall, the results of this dissertation provide evidence that individual and trauma factors influence recovery with brief treatment after an acute trauma, and that it may be possible to determine what kind of treatment will be most effective given who the person is and the type of trauma experienced for individuals in a low-SES, primarily African American sample.

The results of main effects provide evidence that certain brief treatment approaches may be more effective than others, depending on the outcomes of interest in a recently traumatized, low-SES population. PFA was the best at reducing PTSD symptoms, individually administered EMDR was the treatment of choice for reducing depressive symptoms, and SMT was the treatment of choice for reducing physical symptoms (Table 28). These findings support previous literature showing that each of these three intervention types is effective and recommended as a posttraumatic treatment (America Psychiatric Association, 2004; Foa, Keane, Friedman, & Cohen, 2009; Kondro, 2011; U.K. National Institute for Clinical Excellence, 2005; U.S. Department of Homeland Security, 2008; U.S. Department of Veterans Affairs and Department of Defense, 2010). In fact, these relationships to specific outcomes by group support previous literature. Ho & Lee (2010) performed a meta-analyses comparing EMDR and trauma-focused CBT (TFCBT; similar to our trauma-focused SMT) and found that although there were no differences in performance between EMDR and TFCBT on outcome measures of PTSD (also supported in other meta-analyses by Bisson, et al., 2007 and Davidson and Parker, 2001), EMDR held an advantage over TFCBT in reducing depression and these results were maintained over time. The National Institute for

Clinical Excellence (2005) has also noted the effectiveness of EMDR for reducing symptoms of depression. In addition, SMT may have worked best at reducing physical symptoms in our sample because of the multi-faceted relaxation techniques and the suggestions to utilize them outside of session. Ho & Lee's findings support this result, as it was shown in their meta-analysis that TFCBT with a homework component reduces physical symptoms and health care visits compared to TFCBT with no homework component.

However, it is unclear why PFA worked best for reducing PTSD in our sample (i.e., no experimental treatment was clearly better than PFA), as this is in conflict with findings of Bisson and Andrew (2007) and Bisson et al. (2007), whose review and meta-analyses found that EMDR and TFCBT therapies were all significantly better at reducing PTSD symptoms that usual care and other less intensive interventions. PFA is considered a less intensive intervention, but it is important to note that the approach has shown its own effectiveness and has been endorsed as a recommended intervention after an acute trauma (National Center for PTSD, 2006). It is an important improvement upon Critical Incident Stress Debriefing (CISD; Mitchell, 1983), which was found to be harmful and is no longer used (Rose, Bisson, Churchill and Wessely, 2002; van Emmerik, Kamhuis, Hulsbosch and Emmelkamp, 2002). PFA adds more than the discussion and emotion ventilation alone that CISD provided. The psychoeducational style, simple relaxation techniques (i.e., breathing relaxation) and non-trauma focused approach (often provided in multiple sessions as opposed to sometimes only one session of CISD) benefits survivors of trauma in a valuable way, without the risk of retraumatization, as was previously found with CISD. Therefore, it could be said that our findings support

previous findings that have led to PFA being regarded as an effective brief treatment after an acute trauma. Even still, we know from previous literature that trauma-focused, intensive interventions such as EMDR and SMT post-trauma are among the highest recommended, so it is important to ask what may be happening in the EMDR and SMT groups that is preventing these individuals from showing treatment gains larger than PFA participants on our measures of PTSD. Is it possible that four sessions of these more intensive, trauma-focused treatments (particularly EMDR) are increasing rater response on certain scales of these measures (such as the intrusions/re-experiencing scale but not avoidance/numbing or hyperarousal) so that true clinical improvement is not portrayed? The increased scores on these scales would maintain overall high scores over time in these groups, but may not truly be reflective of psychological distress and impairment. This could make sense, especially as specific trauma discussion is avoided in PFA. In addition, it is important to note that this finding does not match the change depicted in Figure 1. This is due to differential drop out and the lack of covariates when looking at means only. Figure 2 displays the means when these factors are taken into account, and reflects what was found in the HLM analyses.

Along another vein, individual differences and trauma type help explain our main effect findings. When we performed additional analyses to investigate individual and trauma factors that may influence treatment response, the main effect relationships became clearer and other relationships reached significance, indicating the importance of considering who the client is when implementing post-trauma intervention.

Individual Factors

Our results provide evidence that certain brief treatment approaches may be more effective than others, depending on the individual factors of interest (Table 29). Specifically, for individuals reporting borderline personality features, EMDR was best in terms of reducing PTSD symptoms over time. Borderline personality disorder has been shown to be one of the most common personality disorders for individuals with PTSD (Zanarini et al., 1998) and this comorbid disorder can make it more difficult for therapists to engage individuals in treatment, but even so, these individuals have been shown to tolerate and benefit from trauma-focused treatment (Bolton and Mueser, 2009). However, it has been suggested in other literature that intensive, PTSD-focused treatment without BPD-focused treatment may be detrimental to individuals experiencing both disorders (for review, see Vignarajah and Links, 2009). In our study, it is important to note that we classified individuals dichotomously as having enough borderline features to be flagged on the SCID or not, so individuals in this sub-sample did not necessarily meet criteria for the full disorder. This could partially explain why EMDR was found to be most helpful for this group. The one-on-one, four-session format may have provided the individual attention these participants needed and may not have been enough time for treatment-impeding, detrimental patterns to emerge (e.g., patterns of idealizing/devaluing, emotional instability, etc.; Bolton and Mueser, 2009), providing a stable situation for EMDR technique effectiveness. Due to even mild impairments in interpersonal functioning, patterns of distrust and difficulty with emotional regulation

(Bolton and Meuser, 2009), these individuals may have been disruptive or disengaged in the group scenerios of SMT and PFA, which would have prevented treatment gain and would help explain our finding.

For individuals who reported using marijuana, SMT performed best at reducing PTSD symptoms over time. We believe that these individuals do not benefit most from EMDR because the substance may be numbing their physical and emotional systems, making necessary activation in trauma-focused, intensive treatment unattainable. Recently, clinicians have shied away from intensive exposure-based treatments (e.g., Prolonged Exposure and EMDR) for individuals with comorbid substance abuse issues due to client drop out, non-compliance and fear of iatrogenic effects, but a technique that circumvents these concerns and has been found particularly useful is trauma exposure through written disclosure (Bragdon and Lombardo, 2012). Although these individuals in our study did not meet criteria for abuse, this may partially explain our finding, as our SMT treatment was skills-based, administered in a group context (meaning decreased overall intensity), and trauma-focused with a written emotional disclosure task, while EMDR implements intensive exposure and PFA entails no exposure. Van Dam, Vedel, Ehring and Emmelkamp (2012) reviewed the substance abuse and PTSD treatment literature and concluded that it is unclear if adding a trauma-focused component to CBT/skills training interventions leads to increased benefit for individuals with substance abuse and PTSD, and our findings may help shed light to this question. Another possibility is that the in-depth relaxation component of SMT is what helped those that use marijuana the most, due to the possibility that they may use marijuana to self-medicate (a means to relax) and learning other relaxation skills provided them the most benefit.

For individuals who met criteria for PTSD at study entry, EMDR performed best at reducing depressive symptoms over time. This finding is supported by previous literature detailing EMDR's ability to decrease depression faster than other approaches after an acute trauma for those with a diagnosis of PTSD (Ironson et al., 2002) and that these differences are maintained over follow-up (Ho and Lee, 2010; Marcus, Marquis, & Sakai, 1997, 2004). In addition, higher PTSD symptom severity has been found to be related to lower levels of clarity and emotional awareness, higher levels of avoidance, higher levels of emotion suppression, difficulties engaging in goal-directed behavior when stressed, and impaired emotion regulation (Ehring and Quack, 2010), and it seems that more intensive, one-on-one intervention, such as EMDR may help engage these individuals in successful treatment.

For individuals with a history of childhood sexual abuse, PFA performed best at reducing both PTSD and physical symptoms over time. This is supported by previous literature, in which childhood-onset trauma victims (not only sexual in nature) showed no improvements after eight sessions of EMDR while adulthood-onset trauma victims showed significant reduction in PTSD symptoms and depression (van der Kolk, et al., 2007). However, our participants with CSA experienced both childhood- and adulthood-onset traumas. Existing literature regarding individual factors as predictors of recovery with treatment after a trauma is lacking and our findings from this study may significantly contribute to this area.

Lastly, those who discussed their trauma in the PFA group experienced faster decreases in PTSD symptoms and slower decreases in depression symptoms than those who did not discuss their trauma, but no relationship was found for individuals in the

SMT group. There were some problems with this variable in terms of SMT, as 9 cases were missing data and all participants wrote verbally about their trauma in this group. This reduction in sample size and the confound of writing in the whole sample could explain why no relationships were found within SMT individuals. However, it is unclear why discussers in PFA experienced faster decreases in PTSD symptoms but slower decreases in depression symptoms. It is important to note that when an individual in PFA voluntarily discussed their trauma, they were quickly redirected to the general topics at hand and how they could apply them to their situation. It is possible that this made them feel invalidated and alone in their experiences (possibly maintaining depressive symptomatology more than those who made no effort to discuss). PTSD symptomatology, on the other hand, may have decreased faster for discussers because they better understood how to redirect their thoughts and engage in positive coping.

Trauma Factors

Results also indicate that it is important to consider trauma type when selecting a brief treatment approach after an acute trauma (Table 30). Specifically, for those with a trauma classified as "violent" in nature, PFA performed best at reducing depressive symptoms. Violent traumas have been considered among the most severe of all trauma types with the highest risk for PTSD development (Breslau, 2009; Kessler, 1995; van Ameringen, Mancini, Patterson and Boyle, 2008), so it might be assumed that these individuals in our study may have been experiencing much higher distress than those with a non-violent trauma. It could be that their posttraumatic cognitions and beliefs about the self, others and world were so unrealistic and threatening, that the trauma-focused interventions were too intensive and therefore disruptive, and the low-intensity

information/relaxation-based structure of PFA is what provided them the most benefit. However, this is just speculation, and it would be beneficial to examine these individuals within group to gain an understanding of the unique presentation of their symptoms (e.g., Under what circumstance would intensive trauma-focused treatment be most valuable for this group? Does more time need to pass between trauma event and intervention? Are relaxation techniques and psychoeducation key in preparing this group for more intensive treatment?).

Along with traumas that are violent in nature, existing literature also considers traumas involving the sudden and unexpected death of a loved one to be among the most severe of all trauma types with the highest risk for PTSD development (Breslau, 2009; Kessler, 1995; van Ameringen, Mancini, Patterson and Boyle, 2008), and those whose loss was violent in nature (suicide, homicide or accident) fall twice into this severe trauma category, making their trauma even more compounded and severe (Lehman, Wortman and Williams, 1987; Amick-McMullan, Kilpatrick and Resnick, 1991; Kaltman and Bonanno, 2003).

In our study, for individuals with a trauma classified as a death of a loved one, SMT was the best at reducing PTSD symptoms. When looking at only deaths that were violent in nature, these relationships became stronger and in addition to symptoms of PTSD, SMT was also found to be best at reducing depressive symptoms for this group. These results are supported by a recent review concluding that CBT and exposure techniques are effective treatments for bereavement with posttraumatic stress after sudden and violent losses (for review, see Kristensen, et al., 2012) and also contribute to the authors' stated need for more intervention research in this area. Conceptualizing

these results, EMDR may be too intensive for traumas involving loss and the same questions as asked above for violent traumas should be asked about individuals experiencing traumatic loss. In addition, it is interesting to note that without intervention, the trajectory of recovery for individuals with violent loss traumas has been shown to be slower than those experiencing natural loss (Kristensen, et al., 2012), but our post-treatment findings are reversed. It is unclear why this may be, especially because PFA was found to be most effective for violent traumas, but there seems to be something about a traumatic loss, particularly if violent in nature, that is most responsive to our brief, group-based, trauma-focused CBT intervention. It may be that those experiencing traumatic bereavement need and use the hands-on tools of trauma-focused CBT (coping, cognitive restructuring, multiple relaxation techniques and the trauma-focused emotional writing task) in a different way than non-bereavement traumas, leading to greater gains than the techniques in PFA (only informational with light breathing relaxation) and EMDR (less skills-based and more therapist directed).

Multiple Measures

One strength of our study is that we utilized two separate measures of both PTSD (Davidson and PTCI) and depressive symptoms (BDI and Hamilton), one self-report and one administered by clinical interview, in order to accurately assess symptoms. However, it is interesting to note that most of our results did not match on both measures of the same disorder. In fact, the only findings that matched on both measures for an outcome were EMDR being best for individuals meeting a diagnosis of PTSD and SMT being best for individuals with a violent death trauma, both for the reduction of depressive symptoms. An obvious reason for incongruency is that the two measures are

administered differently (self-report versus clinical interview). On self-report inventories, symptoms can be exaggerated or minimized easily and this can influence final score and ultimately statistical relationships with other variables. In addition, different inventories tap into different sets of symptoms (e.g., the Hamilton highlights somatic and behavioral symptoms while the BDI focuses on subjective experience as described in Steer, Beck, Riskind and Brown, 1987). It has been noted dual measures such as the BDI and Hamilton inventories have a modest correlation and the discrepancy is related to individual factors like personality and demographics (Schneibel, Brakemeier, Wilbertz, Dykierek, Zobel and Schramm, 2012), making them useful as complementary measures only. Even still, relationships were found on all measures in our analyses, indicating value in using more than one inventory to measure individuals on an outcome variable.

Group versus individual treatment

Our results may also provide some evidence for the effectiveness of group versus individually administered treatment after a trauma given that in some instances EMDR was better than both other approaches and in others, both other approaches performed better than EMDR. However, due to our design (SMT and PFA were administered in group format while EMDR was individual), it is unclear to what extent the treatment techniques versus therapist to participant ratio affects treatment gain, but patterns of results may still be discussed in terms of participant preference.

Overall, no strict preference for group or individual treatment was found for main outcomes of PTSD, the Hamilton measure of depression or physical symptoms,

indicating that it was most likely specific treatment techniques that account for any relationships. Depressive symptoms may have responded better to the individual treatment modality given that being in EMDR was better for the reduction of BDI scores over time than was being in other treatments. Considering individual and trauma factors, no strict preference for group or individual treatment was found for those with a history of CSA, self-reported marijuana use, death or violent death trauma, again indicating that it was most likely specific treatment techniques that account for the observed relationships. A preference for individual treatment was shown for those who met criteria for PTSD or had borderline features, and group preference was shown by those who had experienced a violent trauma. Overall, the impact of group versus individual treatment remains unclear, but it could be that each is better for a different subset of individuals or that the treatment techniques utilized are most important for beneficial changes over time.

Treatment length

Being that our design and sample size did not allow us to compare our brief, four-session format with shorter- or longer-term treatment, strong conclusions cannot be drawn regarding what length of treatment is best. However, we did find that four sessions reduced symptoms and that some treatments performed better than others for certain types of people. Therefore, it can be concluded that a four-session format holds benefit, especially if a need for treatment is high but resources and time are low.

Overall contribution of these findings

Our study was unique in that we used brief treatment (four sessions) and compared a range of treatment types in an under-represented population that experiences high rates of trauma and PTSD and demonstrates a need for treatment. Many studies only offer longer-term treatment using college student or veteran samples. In addition, within treatment groups, literature is lacking regarding which types of people and which types of traumas benefit most from which type of treatment. Our results address this deficit. In addition, we created our own combination of PFA modules and trauma-focused CBT techniques (including an emotional writing task), which were each shown to be more effective than the other treatments in certain circumstances. Brief treatment after a trauma is an important area of research, as it could benefit a greater amount of individuals while maximizing resources and time than longer-term treatment.

To the author's knowledge, our study is the first of its kind to look at individual and trauma factors within these brief treatments for low-SES, primarily African American individuals and our results regarding treatment recommendations by individual factor and trauma type help provide clarity to the existing trauma and PTSD treatment literature.

CHAPTER 6. CLINICAL IMPLICATIONS

Overall, our findings showed that certain brief treatment approaches may be more effective than others, depending on the outcomes of interest and individual/trauma factors in a low-SES population with acute trauma. Individual treatment may not necessarily be better than group treatment, as group PFA and group SMT performed better in certain areas, but again, these findings depended on the type of person and trauma. For the sample as a whole, PFA is recommended for reducing PTSD symptoms over time, EMDR is recommended for reducing depressive symptoms over time, and SMT is recommended for reducing physical symptoms (Table 28). Regarding individual factors, EMDR is recommended for individuals who meet criteria for PTSD at study entry, PFA is recommended for individuals with a history of childhood sexual abuse, and SMT is recommended for individuals who report using marijuana (Table 29). In terms of trauma factors, PFA is recommended for those with a violent trauma, and SMT is recommended if the trauma is a death of a loved one, violent or non-violent in nature (Table 30). Regarding the findings about death traumas, practitioners should be particularly aware that a trauma involving bereavement, especially if the loss was in a violent context, could have greater implications in terms of which type of treatment would be beneficial. Clinicians may be wise to consider being trained in using multiple approaches to trauma treatment, rather than just one. In addition, practitioners should remain aware that brief treatments may have great utility (particularly in underserved populations) and should consider who the individual is and what type of trauma they experienced when selecting a treatment approach to implement.

Funding, insurance and time limitations in the clinical setting may make these implications all the more important. If a clinician is only reimbursed for a small number of sessions, he or she may shy away from treating someone with trauma, due to the previously thought need for long-term care. However, these results suggest that four sessions of a trauma treatment (selected after considering the type of individual) may indeed be effective in providing decreases in symptoms. In addition, community centers and hospitals serving low-SES, minority areas may especially benefit from the suggestions presented here, as funding is most likely low and the need for quick, effective treatment is high.

It is wise for practitioners to consider the relationships found in this study and other literature when providing services to patients similar to those in this sample. However, it is important to note that the small sample size used in this study limited our power and limits generalizability of findings, so that more exploration of these relationships would inform practice further.

CHAPTER 7. LIMITATIONS AND FUTURE DIRECTIONS

Limitations to the interpretation of our results include several. The lack of a waitlist control removed our ability to determine if being in any intervention is better than not enrolling in treatment. Thus, it would benefit this area of research if these interventions were compared to a waitlist control in future studies. Additional analyses using our dataset could be run to compare each group's slope alone to the sample slope as a whole to determine if being in that treatment contributed significantly to the model. Also, as this was a pilot study, the sample size is small, and some group differences may have not been detected or could have been exaggerated for this reason. It would be beneficial for a future study to investigate these factors in an increased sample size. Along the same lines, additional analyses could not be run to explore significant relationships (e.g., investigating the predictive ability of PTSD severity within those with a violent trauma) because of significant statistical power loss, and it would be of benefit to explore these relationships in future studies with a larger sample. It is also important to note that having a PTSD diagnosis was not a criterion for study entry, and our sample represents a range of posttraumatic symptom severity. This range of posttraumatic symptom severity is also a strength, as most previous studies used only samples with diagnosed PTSD and it allows us to compare variables across the dimension of PTSD symptom severity. In addition, our sample was primarily low-SES and African American with a history of trauma before and after the acute trauma for which we recruited. We selected this population to test the effectiveness of brief, post-trauma interventions in

underserved, limited resource individuals. Although generalizability to the greater population was not a goal of the study, it remains unclear how these findings would vary for different SES and/or ethnicities, strictly acute traumas, and purely those with a diagnosis of PTSD.

Pertaining to the non-significant or unexpected relationships between treatment type and some of the outcomes as well as individual and trauma factors, it would be beneficial to further explore these variables to determine why no relationships were found. A cross-sectional analysis with these outcomes and predictors could give a better picture of possible relationships, and additional HLM analyses could also be conducted to investigate individual contribution of predictors, including covariates. It may also be beneficial to consider subscales of the outcome measures in additional analyses, to determine if slopes are different relative to different areas of item response and how this relates to group assignment and individual/trauma factor predictors. It could be found that one treatment type drastically decreases responses on one subscale of a measure but increases responses on the other subscales, consequently maintaining a high overall score and preventing a better understanding of treatment gains unless subscales are considered separately. In addition, there may be a better way to operationalize or combine these variables (e.g., three levels of PTSD: low, moderate and high; additional trauma types: accident trauma versus other trauma; etc.) and this could also be explored in future analyses.

Low-SES, African American individuals are at risk for experiencing more severe and repeated traumas compared to the general population. This disparity is important to consider when interpreting the results of this study and others using this population, as

multiple traumas can maintain distress and render treatment less effective. Although an acute trauma within the last six months was a requirement for study entry, many participants experienced chronic trauma before study entry and additional trauma through the follow-up sessions. Participants were reminded of the trauma of focus as part of study protocol, but there is no way for us to determine how much, if any, other traumas influenced their engagement in treatment or report of symptoms throughout the study. Future studies could assess and control for multiple traumas pre- and post-study entry to account for this factor.

In addition, our group (SMT and PFA) and individual (EMDR) treatment design represents a confound, in that it is unclear to what extent the treatment techniques versus therapist to participant ratio affects treatment gain. It would be beneficial in a larger sample size to provide group and individual treatment for each approach, in order to effectively compare modalities and draw accurate conclusions. Similarly, being that our design and sample size did not allow us to compare our brief, four-session format with shorter- or longer-term treatment, strong conclusions cannot be drawn regarding what length of treatment is best, and it would be of benefit to compare a range of treatment lengths for each approach to determine optimum treatment length. In doing so, even if longer-term treatment is determined to be more effective, it is important to note that brief intervention and group format may be particularly helpful with underserved populations in order to fully utilize minimal resources and maximize treatment gain for a majority of individuals.

Concerning group SMT, it is unclear how much only four sessions and group-based administration dilutes the CBT treatment techniques and content, if any. We

selected modules based on the anticipated needs of the population, but there is no way to know if treatment techniques were negatively affected by the group administration versus an individual administration. Therefore, it may be difficult to conclude that SMT is better or worse than another treatment, because we had fewer sessions than normal administration of SMT and we had group administration which may have diluted the CBT. In addition, the expressive writing component is often not a part of SMT or CBT.

Lastly, it would be of benefit to look more closely at one-month and three-month follow-up treatment outcomes, as some group difference may converge at the six-month follow-up, representing a diluted depiction of who benefitted most from which treatment. Conversely, additional follow-up assessments after six months would be of value to investigate if relationships were maintained or changed. In future studies, it would be of benefit to compare results by time point as well as add additional time points for a more complete picture of treatment outcomes.

The information obtained from this dissertation could help lead to a larger trial and eventually lead to the issuance of guidelines for who may benefit from brief EMDR treatment, and who should get stress management or just supportive treatment for posttraumatic stress.

CHAPTER 8. CONCLUSION

In summary, these results provide evidence that treatment type for the mitigation of traumatic stress can be selected based on both individual and trauma factors after a traumatic event. For the entire sample, EMDR worked best for reducing depressive symptoms, PFA worked best for reducing PTSD symptoms, and SMT worked best for reducing physical symptoms. However, when looking within group at individual and trauma factors, EMDR worked best for those high in baseline PTSD and for those endorsing borderline personality characteristics. SMT worked best for those who reported using marijuana and for those with a trauma of bereavement, whether violent or non-violent in nature. PFA worked best for individuals with a history of childhood sexual abuse and a trauma that was violent in nature. In addition, brief treatments may have great utility and certain brief treatments may be recommended more than others when considering who the individual is and what type of trauma they experienced. These findings contribute to previous literature on post-trauma interventions and provide new evidence for individual and trauma factors that may influence recovery. It would be beneficial for future studies to further explore these relationships. Overall, treatment type may be selected based on individual and trauma factors after a traumatic event.

REFERENCES

Alim, T. N., Charney, D. S., & Mellman, T. A. (2006). An overview of posttraumatic stress disorder in African Americans. *Journal of Clinical Psychology*, *62*(7), 801-813. Doi:10.1002/jclp.20280

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

American Psychiatric Association. (2004). *Practice guideline for the treatment of patients with acute stress disorder and posttraumatic stress disorder*. Arlington, VA: American Psychiatric Association Practice Guidelines.

Amick-McMullan, A., Kilpatrick, D. G., Resnick, H. S. (1991). Homicide as a risk factor for PTSD among surviving family members. *Behavior Modification, 15*, 545–559.

Antoni, M., Ironson, G., & Schneiderman, N. (2007). *Cognitive behavioral stress management for people with HIV*. Oxford University Press.

Australian Centre for Posttraumatic Mental Health. (2007). *Australian guidelines for the treatment of adults with acute stress disorder and posttraumatic stress disorder.* Melbourne: Author.

Badgley, R., Allard, H., McCormick, N., Proudfood, P., Fortin, D., Ogilvie, D., Rae-Grant, Q., Gelinas, P., Pepin, L., & Sutherland, S. (1984). Occurrence in the population. In Anonymous, *Sexual offences against children (Vol. 1)*. (pp. 175-193). Ottawa: Canadian Government Publishing Centre.

Başoğlu, M., Livanou, M. M., Şalcioğlu, E. E., & Kalender, D. D. (2003). A brief behavioural treatment of chronic post-traumatic stress disorder in earthquake survivors: Results from an open clinical trial. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences*, *33*(4), 647-654. Doi:10.1017/S0033291703007360

Beck, A. T., Ward, C. H., Mendelson, M. M., Mock, J. J., & Erbaugh, J. J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, 4561-571. Retrieved from EBSCO*host*.

Beck, A.T. (1978). *Beck Depression Inventory.* Philadelphia: Center for Cognitive Therapy.

Benish, S. G., Imel, Z. E., & Wampold, B. E. (2008). The relative efficacy of bona fide psychotherapies for treating post-traumatic stress disorder: A meta-analysis of direct comparisons. *Clinical Psychology Review*, *28*(5), 746-758. Doi:10.1016/j.cpr.2007.10.005

Berger, J. A., & O'Brien, W. H. (1998). Effect of a cognitive-behavioral stress management intervention on salivary IgA, self-reported levels of stress, and physical health complaints in an undergraduate population. *International Journal of Rehabilitation & Health 4*(3), 129–152.

Bernstein, E.M., Putnam, F.W. (1986). Development, reliability, and validity of a dissociation scale. *Journal of Nervous and Mental Disease, 174*(12)*,* 727-735.Berry, D.S., & Pennebaker, J.W. (1993). Nonverbal and verbal emotional expression and health. *Psychotherapy and Psychosomatics*, *59,* 11-19.

Bisson, J., Andrew, M. (2007). Psychological treatment of post-traumatic stress disorder (PTSD). *Cochrane Database of Systematic Reviews, 3*, Art. No.: CD003388. DOI: 10.1002/14651858.CD003388.pub3

Bisson, J. I., Ehlers, A., Matthews, R., Pilling, S., Richards, D., & Turner, S. (2007). Psychological treatments for chronic post-traumatic stress disorder: Systematic review and meta-analysis. *British Journal of Psychiatry*, *190*(2), 97-104. Doi:10.1192/bjp.bp.106.021402

Blain, L. M., Galovski, T. E., & Robinson, T. (2010). Gender differences in recovery from posttraumatic stress disorder: A critical review. *Aggression and Violent Behavior*, *15*(6), 463-474. Doi:10.1016/j.avb.2010.09.001

Bleich, A., Kotler, M., Kutz, I., & Shalev, A. (2002). *Guidelines for the assessment and professional intervention with terror victims in the hospital and in the community. Jerusalem, Israel.* Unpublished manuscript, Israeli National Council for Mental Health.

Bloom, B. L. (2002). Brief interventions for anxiety disorders: Clinical outcome studies. *Brief Treatment and Crisis Intervention*, *2*(4), 325-339. Doi:10.1093/brief-treatment/2.4.325

Bolton, E.E., Lambert, J.F., Wolf, E.J., Raja, S., Varra, A.A., Fisher, L.M. (2004). Evaluating a cognitive-behavioral group treatment program for veterans with posttraumatic stress disorder. *Psychological Services, 1*(2), 140-146. Doi: 10.1037/1541-1559.1.2.140

Bolton, E. E., & Mueser, K. T. (2009). Borderline personality disorder. In *Treatment of posttraumatic stress disorder in special populations: A cognitive restructuring program* (pp. 225-238). Washington, DC US: American Psychological Association. doi:10.1037/11889-011

Bomyea, J., & Lang, A. J. (2012). Emerging interventions for PTSD: Future directions for clinical care and research. *Neuropharmacology*, *62*(2), 607-616. Doi:10.1016/j.neuropharm.2011.05.028

Bradley, R., Greene, J., Russ, E., Dutra, L., & Westen, D. (2005). A multidimensional meta-analysis of psychotherapy for PTSD. *The American Journal of Psychiatry*, *162*(2), 214-227. Doi:10.1176/appi.ajp.162.2.214

Bragdon, R. A., & Lombardo, T. W. (2012). Written disclosure treatment for posttraumatic stress disorder in substance use disorder inpatients. *Behavior Modification*, *36*(6), 875-896. doi:10.1177/0145445512451273

Breslau, N. (2002). Epidemiologic studies of trauma, posttraumatic stress disorder, and other psychiatric disorders. *The Canadian Journal of Psychiatry / La Revue Canadienne de Psychiatrie*, *47*(10), 923-929.

Breslau, N. (2009). The epidemiology of trauma, PTSD, and other posttrauma disorders. *Trauma, Violence, and Abuse*, *10*(3), 198-210. Doi:10.1177/1524838009334448

Bryk, A., & Raudenbush, S. (1992). *Hierarchical linear models: Applications and data analysis methods*. Thousand Oaks, CA US: Sage Publications, Inc. Retrieved from PsycINFO database.

Bureau of Justice Statistics. (2005). Table 54. In *Criminal Victimization in the United States – Statistical Tables Index*. Retrieved May 15, 2012, from http://bjs.ojp.usdoj.gov/content/pub/html/cvus/ethnicity721.cfm.

Cahill, S. P., & Foa, E. B. (2007). PTSD: Treatment Efficacy and Future Directions. *Psychiatric Times*, *24*(3), 32-34.

Cigrang, J. A., Peterson, A. L., & Schobitz, R. P. (2005). Three American troops in Iraq: Evaluation of a brief exposure therapy treatment for the secondary prevention of combat-related PTSD. *Pragmatic Case Studies in Psychotherapy*, *1*(2), 1-25.

CREST. (2003). *The management of posttraumatic stress disorder in adults* . Belfast: Clinical Resource Efficiency Team of the Northern Ireland Department of Health, Social Service and Public Safety.

CrimeMapping (Version 5.2.0b) [Software]. (2012). San Diego, CA: The Omega Group. Retrieved May 15, 2012 from http://www.crimemapping.com/map/fl/86iami

Cukor, J., Spitalnick, J., Difede, J., Rizzo, A., & Rothbaum, B. O. (2009). Emerging treatments for PTSD. *Clinical Psychology Review*, *29*(8), 715-726. Doi:10.1016/j.cpr.2009.09.001

Davidson J. (1996) *Davidson trauma scale*. New York: Multi-Health Systems.

Davidson, J.R.T., Book, S.W., Colket, J.T., Tupler, L.A., Roth, S., David, D., Hertzberg, M., Mellman, T., Beckham, J.C., Smith, R.D., Davison, R.M., Katz, R., & Feldman, M.E. (1997). Assessment of a new self-rating scale for post-traumatic stress disorder. *Psychological Medicine*, *27,* 153-160.

Davidson, P. R., & Parker, K. H. (2001). Eye movement desensitization and reprocessing (EMDR): A meta-analysis. *Journal of Consulting and Clinical Psychology*, *69*(2), 305-316. Doi:10.1037/0022-006X.69.2.305

Davis, R. G., Ressler, K. J., Schwartz, A. C., Stephens, K., & Bradley, R. G. (2008). Treatment barriers for low-income, urban African Americans with undiagnosed posttraumatic stress disorder. *Journal of Traumatic Stress*, *21*(2), 218-222. Doi:10.1002/jts.20313

Drisko, J. W. (2004). Common factors in psychotherapy outcome: Meta-analytic findings and their implications for practice and research. *Families in Society*, *85*(1), 81-90.

Ehring, T., & Quack, D. (2010). Emotion regulation difficulties in trauma survivors: The role of trauma type and ptsd symptom severity. *Behavior Therapy*, *41*(4), 587-598. Doi:10.1016/j.beth.2010.04.004

Everly, G. r., Flannery, R. r., & Mitchell, J. T. (2000). Critical incident stress management (CISM): A review of the literature. *Aggression and Violent Behavior*, *5*(1), 23-40. Doi:10.1016/S1359-1789(98)00026-3

Federal Bureau of Investigation. (2011a). Crime in the United States – Table 10. In *Uniform Crime Reports*. Retrieved May 15, 2012, from http://www.fbi.gov/about-us/cjis/ucr/crime-in-the-u.s/2010/crime-in-the-u.s.-2010/tables/10tbl01.xls.

Federal Bureau of Investigation. (2011b). Crime in the United States – Table 6. In *Uniform Crime Reports*. Retrieved May 15, 2012, from http://www.fbi.gov/about-us/cjis/ucr/crime-in-the-u.s/2010/crime-in-the-u.s.-2010/tables/10tbl01.xls.

Felitti, V.J., Anda, R.F., Nordenberg, D., Williamson, D.F., Spitz, A.M., Edwards, V., Koss, M.P., & Marks, J.S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *American Journal of Preventive Medicine*, *14,* 245-258.

First, M.B., Spitzer, R.L., Gibbon, M., & Williams, J.B.W. Structured Clinical Interview DSM-IV–TR. Axis I Disorders, Research Version, Patient Edition. (2002). New York: Biometrics Research, N.Y. State Psychiatric Institute. Nov 2002

Foa, E. (1997). Trauma and women: course, predictors, and treatment. *Journal of Clinical Psychiatry, 58*(9), 25-28.

Foa, E. B., & Hearst-Ikeda, D., Perry, K. (1995). Evaluation of a brief cognitive-behavioral program for the prevention of chronic PTSD in recent assault victims. *Journal of Consulting and Clinical Psychology*, *63*(6), 948-955. doi: 10.1037/0022-006X.63.6.948

Foa, E. B., & Rothbaum, B. 0. (1998). Treating the trauma of rape: Cognitive behavioral therapy for PTSD. New York: Guilford Press.

Foa, E. B., Keane, T. M., Friedman, M. J., & Cohen, J. A. (Eds.). (2009). *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies* (2nd ed.). New York: Guilford Press.

Foa, E. B., Keane, T.M., Friedman, M.J., & Cohen, J. (2009). *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies* (1-388). New York: Guilford.

Foa, E.D., Ehlers, A., Clark, D.M., Tolin, D.F. & Orsillo, S.M. (1999). The post-traumatic cognition inventory (PTCI): Development and validation. *Psychological Assessment, 11,* 303-314.

Frattaroli, J. (2006). Experimental disclosure and its moderators: A meta-analysis. *Psychological Bulletin, 132*(6), 823-865. doi:10.1037/0033-2909.132.6.823

Frisina, P. G., Borod, J. C., & Lepore, S. J. (2004). A meta-analysis of the effects of written emotional disclosure on the health outcomes of clinical populations. *Journal of Nervous and Mental Disease, 192*(9), 629-634. doi:10.1097/01.nmd.0000138317.30764.63

Gershuny, B. S., & Thayer, J. F. (1999). Relations among psychological trauma, dissociative phenomena, and trauma-related distress: A review and integration. *Clinical Psychology Review*, *19*(5), 631-657. doi:10.1016/S0272-7358(98)00103-2

Gidron, Y., Duncan, E., Lazar, A., Biderman, A., Tandeter, H., & Shvartzman, P. (2002). Effects of guided written disclosure of stressful experiences on clinic visits and symptoms in frequent clinic attenders. *Family Practice, 19*(2), 161-166. doi:10.1093/fampra/19.2.161

Goodman, M., & Yehuda, R. (2002). The relationship between psychological trauma and borderline personality disorder. *Psychiatric Annals*, *32*(6), 337-345.

Goodson, J., Helstrom, A., Halpern, J. M., Ferenschak, M. P., Gillihan, S. J., & Powers, M. B. (2011). Treatment of posttraumatic stress disorder in U.S. combat veterans: A meta-analytic review. *Psychological Reports*, *109*(2), 573-599. Doi:10.2466/02.09.15.16.PR0.109.5.573-599

Gunderson, J. G., & Sabo, A. N. (1993). The phenomenological and conceptual interface between borderline personality disorder and PTSD. *The American Journal of Psychiatry*, *150*(1), 19-27.

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery and Psychiatry*, *23,* 56-62.

Hamilton, M. (1969). Standardized assessment and recording of depressive symptoms. *Psychiatria, Neurologia, Neurochirurgia, 72*(2), 201-205.

Ho, M. K., & Lee, C. W. (2012). Cognitive behaviour therapy versus eye movement desensitization and reprocessing for post-traumatic disorder—Is it all in the homework then?. *European Review of Applied Psychology / Revue Européenne de Psychologie Appliquée*, *62*(4), 253-260. doi:10.1016/j.erap.2012.08.001

Högberg, G., Pagani, M., Sundin, Ö., Soares, J., Åberg-Wistedt, A., Tärnell, B., & Hällström, T. (2007). On treatment with eye movement desensitization and reprocessing of chronic post-traumatic stress disorder in public transportation workers – A randomized controlled trial. *Nordic Journal of Psychiatry*, *61*(1), 54-61. Doi:10.1080/08039480601129408

Huang, J., Yang, Y., & Wu, J. (2010). Relationship of borderline personality disorder and childhood trauma. *Chinese Journal of Clinical Psychology*, *18*(6), 769-771.

INSERM. (2004). *Psychotherapy: An evaluation of three approaches.* Paris: French National Institute of Health and Medical Research.

Institute of Medicine (2008). Treatment of posttraumatic stress disorder: An assessment of the evidence. Washington, DC: The National Academies Press.

Ironson, G., Freund, B. B., Strauss, J. L., & Williams, J. J. (2002). Comparison for two treatments for traumatic stress: A community-based study of EMDR and prolonged exposure. *Journal of Clinical Psychology*, *58*(1), 113-128. Doi:10.1002/jclp.1132

Ironson G, O'Cleirigh C, Leserman, J, Stuetzle R, Fordiani J, Schneiderman N, Fletcher MA. (2013). Gender specific effects of an augmented written emotional disclosure intervention on posttraumatic, depressive and HIV-disease related outcomes: A randomized, controlled trial. *Journal of Consulting and Clinical Psychology*, 81, no. 2: 284-298. Doi: 10.1037/a0030814

Jiang, W., Chen, J., Tang, J., & Zhang, H. (2010). Research on relationship between trauma and dissociation experience with patients suffering from borderline personality disorder. *Chinese Journal of Clinical Psychology*, *18*(3), 329-330.

Kaltman, S., & Bonanno, G. A. (2003). Trauma and bereavement: Examining the impact of sudden and violent deaths. *Journal of Anxiety Disorders*, *17*(2), 131-147. Doi:10.1016/S0887-6185(02)00184-6

Keinänen, M. T., Johnson, J. G., Richards, E. S., & Courtney, E. A. (2012). A systematic review of the evidence-based psychosocial risk factors for understanding of borderline personality disorder. *Psychoanalytic Psychotherapy*, *26*(1), 65-91. Doi:10.1080/02668734.2011.652659

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 62*(6), 593-602.

Kessler, R. C., Sonnega, A., Bromet, E., & Hughes, M. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, *52*(12), 1048-1060. Doi:10.1001/archpsyc.1995.03950240066012

Kilpatrick, D.G., & Resnick, H.S. (1993). A description of the posttraumatic stress disorder field trial. In *Journal of Clinical Psychology*.

Kondro, W. (2011). WHO unveils psychological first aid guide. *Canadian Medical Association Journal*, *183*(13), E1014.

Koss, M.P., & Gidycz, C.A. (1985). Sexual experiences survey: reliability and validity. *Journal of Consulting and Clinical Psychology*, *53,* 422-423.

Kristensen, P., Weisæth, L., & Heir, T. (2012). Bereavement and mental health after sudden and violent losses: A review. *Psychiatry: Interpersonal and Biological Processes*, *75*(1), 76-97. Doi:10.1521/psyc.2012.75.1.76

Lambert, M. J. (1992). Psychotherapy outcome research: Implications for integrative and eclectical therapists. In J. C. Norcross, M. R. Goldfried (Eds.) , *Handbook of Psychotherapy Integration* (pp. 94-129). New York, NY US: Basic Books.

Lawrence, D., & Kisely, S. (2010). Inequalities in healthcare provision for people with severe mental illness. *Journal of Psychopharmacology*, *24*(11, Suppl 4), 61-68. Doi:10.1177/1359786810382058

Lazar S, Offenkrantz W. Psychotherapy in the treatment of posttraumatic stress disorder. *Psychotherapy is worth it: A comprehensive review of its cost-effectiveness* [e-book]. Arlington, VA US: American Psychiatric Publishing, Inc.; 2010:87-102. Available from: PsycINFO, Ipswich, MA. Accessed June 8, 2012.

Lehman, D. R., Wortman, C. B., and Williams, A. F. (1987). Long term effects of losing a spouse or child in a motor vehicle crash. *Journal of Personality and Social Psychology, 52*, 218-231.

Leserman, J., Drossman, D.A., & Li, Z. (1995). The reliability and validity of a sexual and physical abuse history questionnaire in female patients with gastrointestinal disorders. *Behavioral Medicine*, *21,* 141-150.

Leserman, J., Drossman, D.A., Li, Z., Toomey, T.C., Nachman, G., & Glogau, L. (1996). Sexual and physical abuse history in gastroenterology practice: How types of abuse impact health status. *Psychosomatic Medicine*, *58,* 4-15.

Leserman, J., Li, Z., Drossman, D.A., & Hu, Y.J.B. (1998a). Selected symptoms associated with sexual and physical abuse history among female patients with gastrointestinal disorders: The impact on subsequent health care visits. *Psychological Medicine*, *28,* 417-425.

Leserman, J., Li, Z., Drossman, D.A., Toomey, T.C., Nachman, G., & Glogau, L. (1997). Impact of sexual and physical abuse dimensions on health status: Development of an abuse severity measure. *Psychosomatic Medicine*, *59,* 152-160.

Leserman, J., Li, Z., Hu, Y.J.B., & Drossman, D.A. (1998b). How multiple types of stressors impact on health. *Psychosomatic Medicine*, *60,* 175-181.

Litz, B. T. (2008). Early intervention for trauma: Where are we and where do we need to go? A commentary. *Journal of Traumatic Stress*, *21*(6), 503-506. Doi:10.1002/jts.20373

Litz, B. T., & Roemer, L. (1996). Post-traumatic stress disorder: An overview. *Clinical Psychology and Psychotherapy*, *3*(3), 153-168.

Lumley, M. A., Tojek, T. M., & Macklem, D. J. (2002). Effects of written emotional disclosure among repressive and alexithymic people. In S. J. Lepore, J. M. Smyth (Eds.) , *The writing cure: How expressive writing promotes health and emotional well-being* (pp. 75-95). Washington, DC US: American Psychological Association. Doi:10.1037/10451-004

Marcus, S. V., Marquis, P., & Sakai, C. (1997). Controlled study of treatment of PTSD using EMDR in an HMO setting. *Psychotherapy: Theory, Research, Practice, Training*, *34*(3), 307-315. Doi:10.1037/h0087791

Marcus, S., Marquis, P., & Sakai, C. (2004). Three- and 6-month follow-up of EMDR treatment of PTSD in an HMO setting. *International Journal of Stress Management*, *11*(3), 195-208. Doi:10.1037/1072-5245.11.3.195

McKibben, E. S., Britt, T. W., Hoge, C. W., & Castro, C. (2009). Receipt and rated adequacy of stress management training is related to PTSD and other outcomes among Operation Iraqi Freedom veterans. *Military Psychology*, *21*68-81. Doi:10.1080/08995600903249172

McLaughlin, K. A., Berglund, P., Gruber, M. J., Kessler, R. C., Sampson, N. A., & Zaslavsky, A. M. (2011). Recovery from PTSD following Hurricane Katrina. *Depression and Anxiety*, *28*(6), 439-446. Doi:10.1002/da.20790

McLellan, A.T., Luborsky, L., O'Brien, C.P. & Woody, G.E. (1980). An improved diagnostic instrument for substance abuse patients: The Addiction Severity Index. *Journal of Nervous and Mental Diseases, 168,* 26-33.

Merriam-Webster. (2012). Definition of TRAUMA. In *Encyclopedia Britannica Company*. Retrieved May 15, 2012, from http://www.merriam-webster.com/medical/trauma.

Mitchell, J. T. (1983). When disaster strikes . . . the critical incident stress debriefing process. *Journal of Emergency Medical Services*, *8,* 36-39.

Murrough, J. W., Huang, Y., Hu, J., Henry, S., Williams, W., Gallezot, J., & ... Neumeister, A. (2011). Reduced amygdala serotonin transporter binding in posttraumatic stress disorder. *Biological Psychiatry*, *70*(11), 1033-1038. doi:10.1016/j.biopsych.2011.07.003

Nathan, P. E., & Gorman, J. M. (2007). *A guide to treatments that work (3rd ed.)*. New York, NY US: Oxford University Press.

National Center for PTSD. (2006). *Psychological first aid: field operations guide* (2nd ed.). U.S. Department of VeteransAffairs. Retrieved May 28, 2012 from http://www.ptsd.va.gov/professional/manuals/psych-first-aid.asp

National Institute for Clinical Excellence. (2005). *Post-traumatic stress disorder: the management of PTSD in adults and children in primary and secondary care.* Great Britain: Glaskell and the British psychological society.

Nickerson, A., Bryant, R. A., Silove, D., & Steel, Z. (2011). A critical review of psychological treatments of posttraumatic stress disorder in refugees. *Clinical Psychology Review*, *31*(3), 399-417. Doi:10.1016/j.cpr.2010.10.004

Panzer, A., & Viljoen, M. (2004). Dissociation: A developmental psychoneurobiological perspective. *South African Psychiatry Review*, *7*(3), 11-14.

Pennebaker, J.W. (1982). *The psychology of physical symptoms*. New York: Springer-Verlag.

Pennebaker, J. W., & Beall, S. K. (1986). Confronting a traumatic event: Toward an understanding of inhibition and disease. *Journal of Abnormal Psychology, 95*(3), 274-281. doi:10.1037/0021-843X.95.3.274

Pietrzak, R. H., Goldstein, R. B., Southwick, S. M., & Grant, B. F. (2011). Personality disorders associated with full and partial posttraumatic stress disorder in the U.S. population: Results from Wave 2 of the National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Psychiatric Research*, *45*(5), 678-686. Doi:10.1016/j.jpsychires.2010.09.013

Ponniah, K., & Hollon, S. D. (2009). Empirically supported psychological treatments for adult acute stress disorder and posttraumatic stress disorder: a review. *Depression and Anxiety (1091-4269)*, *26*(12), 1086-1109. Doi:10.1002/da.20635

Powers, M. B., Halpern, J. M., Ferenschak, M. P., Gillihan, S. J., & Foa, E. B. (2010). A meta-analytic review of prolonged exposure for posttraumatic stress disorder. *Clinical Psychology Review*, *30*(6), 635-641. Doi:10.1016/j.cpr.2010.04.007

Resnick, P. A., & Schnicke, M. K. (1992). Cognitive processing therapy for sexual assault victims. *Journal of Consulting and Clinical Psychology*, *60*(5), 748.

Roberts, A. L., Gilman, S. E., Breslau, J. J., Breslau, N. N., & Koenen, K. C. (2011). Race/ethnic differences in exposure to traumatic events, development of post-traumatic stress disorder, and treatment-seeking for post-traumatic stress disorder in the United States. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences*, *41*(1), 71-83. Doi:10.1017/S0033291710000401

Rose, S., Bisson, J., Churchill, R., Wessely, S. Psychological debriefing for preventing posttraumatic stress disorder (PTSD). Cochrane Database of Systematic Reviews, 2002, Issue 2. ART#.: CD000560. DOI:10.1002/14651858.CD000560.

Rothbaum, B. (1997). A controlled study of eye movement desensitization and reprocessing in the treatment of posttraumatic stress disordered sexual assault victims. *Bulletin of the Menninger Clinic*, *61*(3), 317-334.

Rothbaum, B., Astin, M. C., & Marsteller, F. (2005). Prolonged exposure versus eye movement desensitization and reprocessing (EMDR) for PTSD rape victims. *Journal of Traumatic Stress, 18*(6), 607-616. Doi: 10.1002/jts.20069

Rothbaum, B. O., Foa, E. B., Riggs, D. S., & Murdock, T. (1992). A prospective examination of post-traumatic stress disorder in rape victims. *Journal of Traumatic Stress*, *5*(3), 455-475. Doi:10.1002/jts.2490050309

Sanders, C. M. (1993). Risk factors in bereavement outcome. In M. S. Stroebe, W. Stroebe, R. O. Hansson (Eds.) , *Handbook of Bereavement: Theory, Research, and Intervention* (pp. 255-267). New York, NY US: Cambridge University Press. Doi:10.1017/CBO9780511664076.018

Sansone, R. A., Hahn, H. S., Dittoe, N., & Wiederman, M. W. (2011). The relationship between childhood trauma and borderline personality symptomatology in a consecutive sample of cardiac stress test patients. *International Journal of Psychiatry in Clinical Practice*, *15*(4), 275-279. Doi:10.3109/13651501.2011.593263

Schneibel, R., Brakemeier, E., Wilbertz, G., Dykierek, P., Zobel, I., & Schramm, E. (2012). Sensitivity to detect change and the correlation of clinical factors with the Hamilton Depression Rating Scale and the Beck Depression Inventory in depressed inpatients. *Psychiatry Research*, *198*(1), 62-67. doi:10.1016/j.psychres.2011.11.014

Schubert, S., & Lee, C. W. (2009). Adult PTSD and its treatment with EMDR: A review of controversies, evidence, and theoretical knowledge. *Journal of EMDR Practice and Research*, *3*(3), 117-132. Doi:10.1891/1933-3196.3.3.117

Schwartz, A. C., Bradley, R. L., Sexton, M., Sherry, A., & Ressler, K. J. (2005). Posttraumatic stress disorder among African Americans in an inner city mental health clinic. *Psychiatric Services*, *56*(2), 212-215. Doi:10.1176/appi.ps.56.2.212

Seidler, G. H., & Wagner, F. E. (2006). Comparing the efficacy of EMDR and trauma-focused cognitive-behavioral therapy in the treatment of PTSD: A meta-analytic study. *Psychological Medicine: A Journal of Research in Psychiatry and the Allied Sciences*, *36*(11), 1515-1522. Doi:10.1017/S0033291706007963

Shapiro, F. (1995). Eye movement desensitization and reprocessing: Basic principles, protocols, and procedures. New York: Guilford Press.

Shapiro, F. (1999). Eye movement desensitization and reprocessing (EMDR): Clinical and research implications of an integrated psychotherapy treatment. *Journal of Anxiety Disorders, 13,* 35-67.

Shapiro, F. (2001). *Eye movement desensitization and reprocessing: Basic principles, protocols, and procedures* (2nd ed.). New York: Guilford Press.

Shear, M. K.. (2012). Getting straight about grief. *Journal of Depression and Anxiety*, *29,* 461-464.

Smyth, J. M. (1998). Written emotional expression: Effect sizes, outcome types, and moderating variables. *Journal of Consulting and Clinical Psychology*, *66*(1), 174-184. Doi:10.1037/0022-006X.66.1.174

Smyth, J. M., Stone, A. A., Hurewitz, A., & Kaell, A. (1999). Effects of writing about stressful experiences on symptom reduction in patients with asthma or rheumatoid arthritis. *Journal of the American Medical Association*, *281*(14), 1304.

Spates, C., Koch, E., Cusack, K., Pagoto, S., & Waller, S. (2009). Eye movement desensitization and reprocessing. In E. B. Foa, T. M. Keane, M. J. Friedman, J. A. Cohen (Eds.) , *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies (2nd ed.)* (pp. 279-305). New York, NY US: Guilford Press.

Steer, R. A., Beck, A. T., Riskind, J. H., & Brown, G. (1987). Relationships between the Beck Depression Inventory and the Hamilton Psychiatric Rating Scale for Depression in depressed outpatients. *Journal of Psychopathology and Behavioral Assessment*, *9*(3), 327-339. doi:10.1007/BF00964561

U.K. National Institute for Clinical Excellence. (2005). *Post traumatic stress disorder (PTSD): The management of adults and children in primary and secondary care.* London: NICE Guidelines.

U.S. Census. (2010). Miami Census Information. In *City of Miami Planning Department.* Retrieved May 15, 2012, from http://www.miamigov.com/Planning/pages/services/Census2010.asp.

U.S. Department of Homeland Security. (2008) National Response Framework. Retrieved May 2008, from www.fema.gov/emergency/nrf/

U.S. Department of Veterans Affairs and Department of Defense (2010). *VA/DoD Clinical practice guideline for the management of post-traumatic stress.* Washington, DC. Veterans Health Administration, Department of Veterans Affairs and Health Affairs, Department of Defense.

Uhernik, J. A., & Husson, M. A. (2009). Psychological first aid: An evidence-informed approach for acute disaster behavioral health response. In G. R. Walz, J. C. Bleuer, & R. K. Yep (Eds.), *Compelling counseling interventions: VISTAS 2009* (pp.271-280). Alexandria, VA: American Counseling Association.

UrbanMapping. (2011). Liberty City Neighborhood in Miami, Fl Detailed Profile. In *City-Data*. Retrieved May 15, 2012, from http://www.city-data.com/neighborhood/Liberty-City-Miami-FL.html.

Van Ameringen, M., Mancini, C., Patterson, B., & Boyle, M. H. (2008). Post-traumatic stress disorder in Canada. *Neuroscience and Therapeutics*, *14*(3), 171-181. Doi:10.1111/j.1755-5949.2008.00049.x

van Dam, D., Vedel, E., Ehring, T., & Emmelkamp, P. G. (2012). Psychological treatments for concurrent posttraumatic stress disorder and substance use disorder: A systematic review. *Clinical Psychology Review*, *32*(3), 202-214. Doi:10.1016/j.cpr.2012.01.004

van der Kolk, B. A., Spinazzola, J., Blaustein, M. E., Hopper, J. W., Hopper, E. K., Korn, D. L., & Simpson, W. B. (2007). A randomized clinical trial of eye movement desensitization and reprocessing (EMDR), fluoxetine, and pill placebo in the treatment of posttraumatic stress disorder: Treatment effects and long-term maintenance. *Journal of Clinical Psychiatry*, *68*(1), 37-46. Doi:10.4088/JCP.v68n0105

van Dijke, A., Ford, J. D., van der Hart, O., van Son, M., van der Heijden, P., & Buhring, M. (2012). Complex posttraumatic stress disorder in patients with borderline personality disorder and somatoform disorders. *Psychological Trauma: Theory, Research, Practice, and Policy*, *4*(2), 162-168. Doi:10.1037/a0025732

van Emmerik, A. P., Kamphuis, J. H., Hulsbosch, A. M., & Emmelkamp, P. G. (2002). Single session debriefing after psychological trauma: A meta-analysis. *The Lancet*, *360*(9335), 766-771. Doi:10.1016/S0140-6736(02)09897-5

Van Etten, M. L. & Taylor, S. (1998). Comparative efficacy of treatments for post-traumatic stress disorder: a meta-analysis. *Clinical Psychology and Psychotherapy 5,* 126–144.

Van Ijzendoorn, M.H., & Schuengel, C. (1996). The measurement of dissociation in normal and clinical populations: Meta-analytic validation of the Dissociative Experiences Scale (DES). *Clinical Psychology Review, **16***, 365–382.

Vignarajah, B., & Links, P. S. (2009). The clinical significance of co-morbid post-traumatic stress disorder and borderline personality disorder: Case study and literature review. *Personality and Mental Health*, *3*(3), 217-224. Doi:10.1002/pmh.89

Williams, J. L., Burke, L. A., McDevitt-Murphy, M. E., & Neimeyer, R. A. (2012). Responses to loss and health functioning among homicidally bereaved African Americans. *Journal of Loss and Trauma*, *17*(3), 218-235. Doi:10.1080/15325024.2011.616826

Wilson, S. A., Becker, L. A., & Tinker, R. H. (1995). Eye movement desensitization and reprocessing (EMDR) treatment for psychologically traumatized individuals. *Journal of Consulting and Clinical Psychology*, *63*(6), 928-937. Doi:10.1037/0022-006X.63.6.928

Wilson, S. A., Becker, L. A., & Tinker, R. H. (1997). Fifteen-month follow-up of eye movement desensitization and reprocessing (EMDR) treatment for posttraumatic stress disorder and psychological trauma. *Journal of Consulting and Clinical Psychology*, *65*(6), 1047-1056. Doi:10.1037/0022-006X.65.6.1047

Wrenn, G. L., Wingo, A. P., Moore, R., Pelletier, T., Gutman, A. R., Bradley, B., & Ressler, K. . (2011). The effect of resilience on posttraumatic stress disorder in trauma-exposed inner-city primary care patients. *Journal of the National Medical Association*, *103*(7), 560-566.

Zanarini, M. C. (2009). Psychotherapy of borderline personality disorder. *Acta Psychiatrica Scandinavica*, *120*(5), 373-377. Doi:10.1111/j.1600-0447.2009.01448.x

Zlotnick, C., Davidson, J., Shea, M.T., & Pearlstein, T. (1996). Validation of the Davidson Trauma Scale in a sample of survivors of childhood sexual abuse. *Journal of Nervous and Mental Disease*, *184,* 255-257.

Chart 1. Study Consort Flow Chart

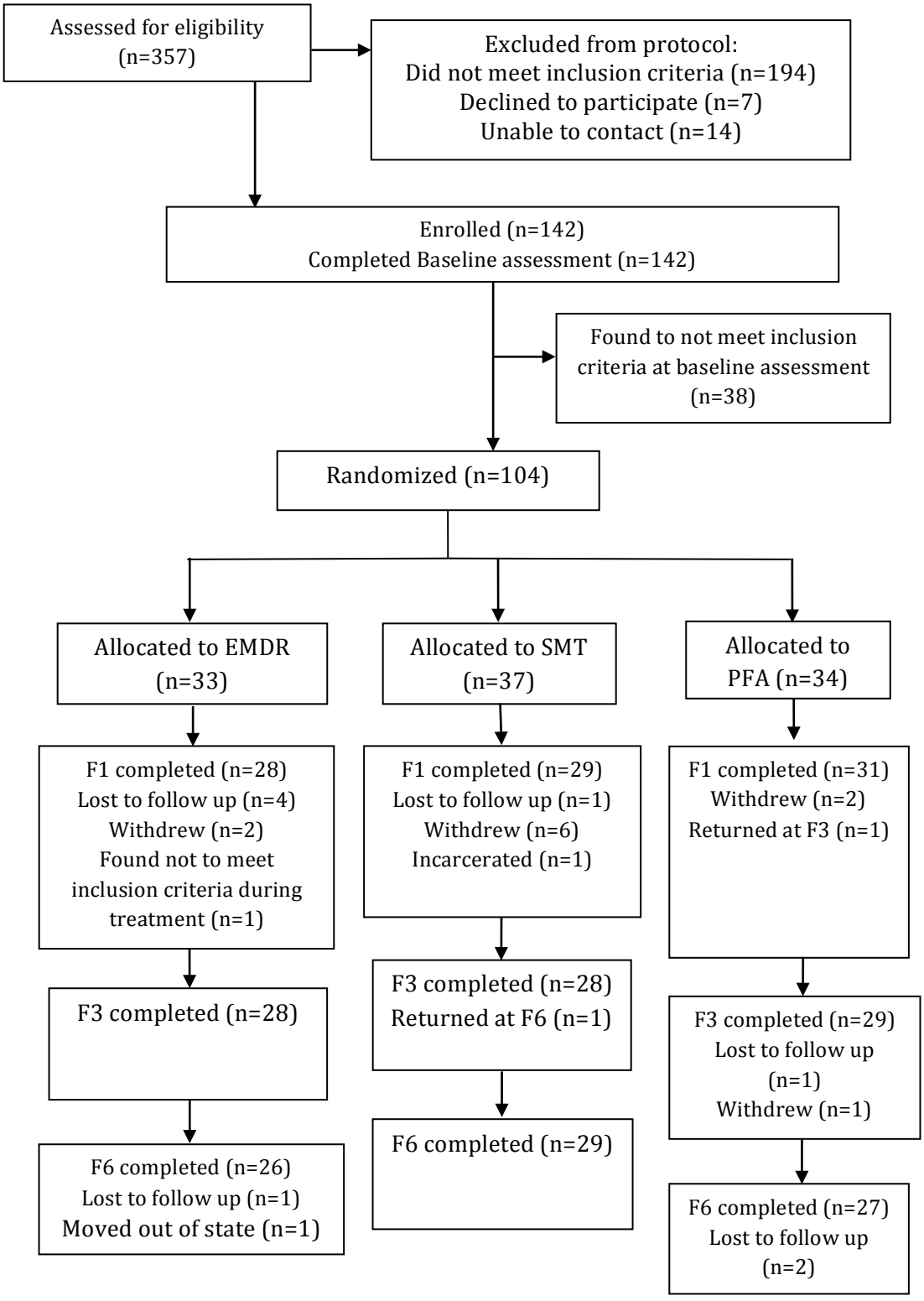


Table 1. Demographic characteristics for control group, treatment conditions and total sample

		PFA	SMT	EMDR	Total
n		31	29	27	87
Age - mean (SD)		49.4 (7.13)	47.3 (9.73)	45.9 (10.97)	47.6 (9.33)
Gender %	Men	38.7	31.0	37.0	35.6
	Women	61.3	69.0	63.0	64.4
Ethnicity %	African American	71.0	96.6	66.7	78.2
	Caucasian (Non-Hispanic)	9.7	0.0	3.7	4.6
	Hispanic	16.1	0.0	18.5	11.5
	Other	3.2	3.4	11.1	5.7
Education %	<High School	32.3	10.3	22.2	21.8
	High School	25.8	58.6	22.2	35.6
	Some College/Trade School	38.8	24.1	40.7	34.5
	College Graduate	3.2	6.8	14.8	8.0
Income %	<= $5000/yr	51.6	48.3	55.6	51.7
	$5001-10,000/yr	22.6	13.7	18.5	18.3

	$10,001-20,000/yr	16.1	34.5	18.5	23.0
	> $20,000/yr	9.7	3.4	7.4	6.9
Employment %	Full time	0	6.9	7.4	4.6
	Part time	9.7	17.2	14.8	13.8
	Unemployed	41.9	20.7	44.4	35.6
	Disability	22.6	34.5	14.8	24.1
	Other	25.8	20.7	18.6	21.9

Table 2. Baseline means on outcome variables for control group, treatment conditions and total sample

Outcomes:

		PFA	**SMT**	**EMDR**	**Total**
n		31	29	27	87
PTSD – mean	Davidson	55.00	54.48	58.82	56.01
(SD)		(22.61)	(25.31)	(29.27)	(25.48)
	PTCI	100.36	97.69	105.96	101.21
		(38.72)	(28.64)	(31.51)	(33.19)
Depression	BDI	14.36	13.31	17.44	14.97
		(8.13)	(8.41)	(9.23)	(8.65)
	HAM-D	10.81	11.35	11.22	11.12
		(5.00)	(5.67)	(6.45)	(5.64)
Physical Sxs	PILL	110.77	101.69	110.48	107.66
		(36.28)	(27.69)	(24.32)	(30.10)

Table 3. Ns and means for all individual factors

		PFA	SMT	EMDR	Total	Tests of group differences
Baseline PTSD	N <= 32	6	8	6	20	X^2 (2, N = 87) = .586, p = .746
	N >32	25	21	21	67	
	% <=32	19.4	27.6	22.2	23.0	
	% >32	80.6	72.4	77.8	77.0	
BPD	Mean	6.84	5.83	6.22	6.31	X^2 (2, N = 87) = 1.474, p = .478
	N < 5	8	10	11	29	
	N >= 5	23	19	16	58	
	% < 5	25.8	34.5	40.7	33.3	
	% >= 5	74.2	65.5	59.3	66.7	
CSA	N yes	12	11	9	32	X^2 (2, N = 87) = .204, p = .903
	N no	19	18	18	55	
	% yes	38.7	37.9	33.3	36.8	
	% no	61.3	62.1	66.7	63.2	
Substance use	(Days in the last 30)					
Alcohol use	Mean days	3.68	3.62	3.82	3.70	X^2 (2, N = 87) = 2.120, p = .346
	N <10 days	27	22	24	73	
	N >=10	4	7	3	14	
	% < 10	87.1	75.9	89.9	83.9	
	% >= 10	12.9	24.1	11.1	16.1	

Cannabis	N any use	2	5	4	11	X^2 (2, N = 87) = 1.746, p = .418
	N no use	29	24	23	76	
	% any use	6.5	17.2	14.8	12.6	
	% no use	93.5	82.8	85.2	87.4	
Discussed Trauma	N yes	12	14*	27	53*	X^2 (2, N = 87) = 24.944, p = .000
	N no	19	6*	0	25*	
	% yes	38.7	48.3*	100	60.9*	*missing 9 cases from SMT
	%no	61.3	20.7*	0	28.7*	

Table 4. Ns for Trauma Factors

		PFA	SMT	EMDR	Total	Tests of Group Differences
Time since trauma	Mean (weeks)	11.55	9.83	12.22	11.18	X^2 (2, N = 87) = 1.235, p = .539
	N <= 5wks	7	10	9	26	
	N > 5wks	24	19	18	61	
	% <= 5wks	22.6	34.5	33.3	29.9	
	% > 5wks	77.4	65.5	66.7	70.1	
Type of	**Trauma**					
Loss of Family	N	14	16	10	40	
	%	45.2	55.2	37.0	46.0	
Loss of friend	N	4	3	4	11	
	%	12.9	10.3	14.8	12.6	
Physical Assault	N	2	3	2	7	
	%	6.5	10.3	7.4	8.0	
Robbery weapon	N	2	1	2	5	
	%	6.5	3.4	7.4	5.7	
Robbery no weapon	N	2	1	0	3	
	%	6.5	3.4	0	3.4	
Car accident	N	1	4	1	6	
	%	3.2	13.8	3.7	6.9	
Sexual crime	N	2	0	1	3	
	%	6.5	0	3.7	3.4	
Shooting of others	N	1	0	1	2	
	%	3.2	0	9.7	2.3	

Other	N	3	1	6	10	
	%	9.7	3.4	22.2	11.5	
						Tests of group differences
Violent Trauma	N yes	17	21	16	54	X^2 (2, N = 87) = 2.097, p = .350
	% yes	54.8	72.4	59.3	62.1	
Bereave-ment Trauma	N yes	18	20	15	53	X^2 (2, N = 87) = 1.221, p = .543
	% yes	58.1	69.0	55.6	60.9	
Violent bereave-ment	N yes	5	12	7	24	X^2 (2, N = 87) = 4.836, p = .089
	% yes	16.1	41.4	25.9	27.6	

Table 5. Recruitment Breakdown

		PFA	SMT	EMDR	Total
Physician/Clinic	N	1	6	2	9
	%	3.2	20.7	7.4	10.3
Persons related to study*	N	14	9	10	33
	%	45.2	31.0	37.0	37.9
Other	N	16	14	15	45
	%	51.6	48.2	55.5	51.7

*This subcategory represents those who were recruited by participants who were already enrolled in the study.

Table 6. Administration of measures

Administration of Measures:

	Name	Screen	Baseline	1, 3 & 6 Months
I	Screening Form	**X**		
I	Drug/Alcohol SCIDS		**X**	
I	Psychotic Screener (Unusual sxs)		**X**	
S	Borderline Screen (BPD)		**X**	
S	Dissociative Screen (DES)		**X**	
I	Trauma History (Brief Version)		**X**	
S	Demographics		**X**	
I	PTSD Scale (Davidson)		**X**	**X**
S	The PILL		**X**	**X**
S	Depression (BDI)		**X**	**X**
I	Hamilton (Depression)		**X**	**X**
S	PTCI		**X**	**X**
I	Addiction Severity Index (ASI/Drug Use)		**X**	**X**

I = Interviewer Administered
S = Self-Report

Table 7. Basic Equations for evaluating the direct and indirect (interaction with individual/trauma factors) effects of group assignment on continuous and dichotomous study outcomes.

Level 1 Model (Continuous Outcomes)

$$Y_{ti} = \beta_{0i} + \beta_{ti}*(\text{Time}) + e$$

Level 1 Model (Dichotomous Outcomes)

$$\text{Prob } Y_{ti} (Y = 1/b) = \beta_{ti}$$

$$\text{Log}[\beta_{ti}/(1- \beta)] = \beta_{0i} + \beta_{ti}*(\text{Time})$$

Level-2 Model (*Main Effects*)

$$\beta_{0i} \text{ (intercept)} = \gamma_{00} + \mu_0$$

$$\beta_{ti} \text{ (slope)} = \gamma_{01} + \gamma_{11}*(\text{ Group1}) + \gamma_{12}*(\text{Group2}) + \gamma_{13} (Y_{\text{baseline}}) + \mu_1$$

Level-2 Model (*Gender Moderated Effects – or other individual/trauma factors*)

$$\beta_{0i} \text{ (intercept)} = \gamma_{00} + \mu_0$$

$$\beta_{ti} \text{ (slope)} = \gamma_{01} + \gamma_{11}*(\text{Group1}) + \gamma_{12}*(\text{Group2}) + \gamma_{13} (\text{Gender}) + \gamma_{14} *(\text{Group1 x Gender}) + \gamma_{15} + *(\text{Group2 x Gender}) + \gamma_{16} (Y_{\text{baseline}}) + \mu_1$$

Level 2 Model for Objectives 3 & 4 (*Comparison of two groups to investigate individual/trauma factors; illustrated with gender as the factor being investigated*)

β_{0i} (intercept) = $\gamma_{00} + \mu_0$

β_{ti} (slope) = $\gamma_{01} + \gamma_{11}$*(Group1) + γ_{12} (Gender) + γ_{13} *(Group1 x Gender) + γ_{18} ($Y_{baseline}$) + μ_1

Please note: 1) Group1 will represent EMDR vs. PFA and will be replaced by Group2 (SMT vs. PFA) and then Group3 (EMDR vs. SMT) so that all groups can be compared to each other. 2) "Gender" will be replaced in subsequent models with the other individual and trauma factor variables one at a time, with each group comparison, so that all effects can be investigated.

Table 8. Base models: Slopes by outcome with time only, time + covariates, and time + covariates + treatment group versus control

* = $p<.05$; ** = $p<.001$

Outcome/Model	**γ**	***t*-rat.**	***p***
DAV + mobasli	-3.57	-7.04	.000**
+ cov	-4.53	-3.09	.003**
+ EDSD	-4.61	-3.085	.003**
PTCI + mobasli	-4.14	-7.18	.000**
+ cov	-4.35	-2.24	.028*
+ EDSD	-4.11	-2.14	.036*
BDI + mobasli	-1.02	-7.36	.000**
+ cov	-0.47	-1.15	.254
+ EDSD	-0.36	-0.87	.386
HAM + mobasli	-0.21	-1.67	.095†
+ cov	-0.29	-0.85	.396
+ EDSD	-0.23	-.066	.502
PILL + mobasli	-1.63	-3.42	.001**
+ cov	-1.56	-0.86	.392
+ EDSD	-1.06	-0.60	.550

† = trend

Table 9. Basic Model including Coefficients and Significance Tests for Level 1 and Level 2 Covariates in Prediction of DAV Slope over 6 months

	Coefficient	Standard Error	*t* Ratio	*df*	*p*
Fixed effects					
DAV intercept, β_0					
Avg. initial DAV, y_{00}	44.114	2.413	18.279	86	.000
DAV slope (per month), β_1					
Average slope, γ_{10}	-4.527	1.464	-3.092	81	.003
Baseline DAV, y_{11}	-0.051	0.018	-2.887	81	.005
Age, γ_{12}	0.013	0.057	0.221	81	.826
Gender, γ_{13}	-0.333	0.932	-0.358	81	.721
Ethnicity, γ_{14}	1.527	1.052	1.451	81	.151
Education, γ_{15}	-0.021	0.432	-0.049	81	.961
Random effects	**SD**	**Variance**	**df**	χ^2	***p* Value**
Intercept, U_0	17.441	304.170	86	180.633	.000
Slope, U_1	1.062	1.128	81	68.213	>.500
Error, *R*	20.565	422.920			

* = $p<.05$; ** = $p<.001$

Table 10. Basic Model including Coefficients, Treatment versus Control and Significance Tests for Level 1 and Level 2 Covariates in Prediction of DAV Slope over 6 months

	Coefficient	Standard Error	*t* Ratio	*df*	*p*
Fixed effects					
DAV intercept, β_0					
Avg. initial DAV, y_{00}	44.128	2.417	18.254	86	.000
DAV slope (per month), β_1					
Average slope, γ_{10}	-4.605	1.493	-3.085	79	.003
Baseline DAV, y_{11}	-0.046	0.017	-2.618	79	.011
Age, γ_{12}	0.019	0.059	0.315	79	.753
Gender, γ_{13}	-0.419	0.920	-0.456	79	.650
Ethnicity, γ_{14}	0.922	1.078	0.855	79	.395
Education, γ_{15}	-0.012	0.434	-0.027	79	.979
EMDR dummy, γ_{16}	0.039	1.051	0.037	79	.971
SMT dummy, γ_{17}	1.648	1.001	1.647	79	.103

Random effects	SD	Variance	df	χ^2	*p* Value
Intercept, U_0	17.313	299.748	86	178.992	.000
Slope, U_1	0.957	0.916	79	67.071	>.500
Error, *R*	20.659	426.803			

* = $p<.05$; ** = $p<.001$

Table 11. Significance tests: Group by outcome (each model includes standard covariates)

Outcome/Model	β	*t*-rat.	*p*
DAV - *ED*	-0.73	-0.73	.470
- *SD*	1.64	1.72	.089'
- *ED, SD*	0.04, 1.65	0.04, 1.65	.971, .103'
- *TXvCTRL*	0.88	1.05	.299
- *EvS*	-1.40	-1.17	.250
- *SvP*	1.70	1.66	.102'
- *EvP*	-0.09	-0.09	.930
PTCI - *ED*	-0.76	-0.77	.446
- *SD*	0.45	0.46	.646
- *ED, SD*	-0.69, 0.13	-0.59, 0.12	.556, .997
- *TXvCTRL*	-0.25	-0.25	.800
- *EvS*	-0.91	-0.81	.419
- *SvP*	-0.28	-0.25	.807
- *EvP*	-0.51	-0.44	.664
BDI - *ED*	-0.42	-1.74	.086'
- *SD*	0.40	1.30	.198
- *ED, SD*	-0.29, 0.27	-1.17, 0.80	.247, .424
- *TXvCTRL*	0.00	0.01	.992
- *EvS*	-0.53	-1.54	.131
- *SvP*	0.39	1.09	.282
- *EvP*	-0.34	-1.32	.192
HAM - *ED*	-0.21	-0.91	.366
- *SD*	0.18	0.80	.427
- *ED, SD*	-0.16, 0.11	-0.65, 0.47	.519, .638
- *TXvCTRL*	-0.01	-0.07	.948
- *EvS*	-0.21	-0.73	.470
- *SvP*	0.17	0.72	.477
- *EvP*	-0.21	-0.87	.388
PILL - *ED*	-0.19	-0.23	.822
- *SD*	-1.64	-1.79	.076'
- *ED, SD*	-1.27, -2.24	-1.34, -2.11	.183, .037*
- *TXvCTRL*	-1.77	-2.02	.047*
- *EvS*	0.92	0.92	.364
- *SvP*	-2.21	-2.08	.042*
- *EvP*	-1.53	-1.61	.114

* = $p<.05$; ** = $p<.001$

† = trend

Table 12. Chi-square tests of model comparisons: Contribution of EDSD and Treatment vs. Control to the model (each model includes standard covariates)

Outcome/Model			**Chi-square**	**df**	***p***
DAV	-	*EDSD*	6.51	0	>.500
	-	*TXvCTRL*	0.80	0	>.500
PTCI	-	*EDSD*	4.74	0	>.500
	-	*TXvCTRL*	0.17	0	>.500
BDI	-	*EDSD*	1.66	0	>.500
	-	*TXvCTRL*	2.55	0	>.500
HAM	-	*EDSD*	1.02	0	>.500
	-	*TXvCTRL*	3.03	0	>.500
PILL	-	*EDSD*	7.91	0	>.500
	-	*TXvCTRL*	3.25	0	>.500

* = $p<.05$; ** = $p<.001$

† = trend

Table 13. Significance of predictors for DAV (each model includes standard covariates)[a]

	CSA			Violent			Death			Violent Death		
Group	β	T-rat.	*p*	β	T-rat.	*p*	β	T-rat.	*p*	β	T-rat.	*p*
EMDR Dummy	-1.17	-0.60	.553	0.72	0.36	.722	1.79	0.93	.359	4.28	1.74	.086†
SMT Dummy	3.87	2.00	.048*	-0.45	-0.21	.835	-4.36	-2.05	.044*	-4.97	-2.59	.012*
EMDR & SMT	1.10 4.38	0.56 2.18	.574 .032*	1.19 0.13	0.60 0.06	.548 .954	-1.17 -5.01	-0.60 -2.14	.548 .036*	2.10 -3.87	0.85 -2.27	.397 .026*
EMDR vs. SMT	-3.11	-1.33	.192	0.73	0.29	.772	3.92	1.69	.095†	6.07	2.31	.026*
SMT vs. PFA	4.04	2.01	.049*	0.13	0.06	.955	-4.22	-1.72	.091†	-3.18	-1.96	.056†
EMDR vs. PFA	1.17	0.60	.551	1.65	0.89	.377	-2.60	-1.25	.217	0.67	0.26	.795

* = $p < .05$; ** = $p < .01$

Table 14. Summary of Best Treatments by Outcome*

Outcome	**Treatment Effectiveness**
Davidson	Other > SMT†
	PFA > SMT†
PTCI	X
BDI	EMDR > Other†
Hamilton	X
PILL	SMT > Other†
	SMT > PFA

† = trend
X = No one treatment is significantly better than another

*See Table 11 for details

Table 15. Summary of Best Treatments for Individual Factors by Outcome*

Outcome	DAV Baseline	CSA	BPD	Marijuana
Davidson	X	Other groups > SMT PFA > SMT	X	X
PTCI	X	X	EMDR > SMT†	SMT > Other† SMT > PFA
BDI	EMDR > Other EMDR > SMT EMDR > PFA†	X	X	X
Hamilton	Other > SMT EMDR > SMT PFA > SMT†	X	X	X
PILL	X	Other > EMDR PFA > EMDR PFA > SMT†	X	X

† = trend; X = No one treatment is significantly better than another
*See Tables 13, 19, 23, 26 and 29 for details

Table 16. Summary of Best Treatments for Trauma Factors by Outcome*

Outcome	Violent	Death	Violent Death
Davidson	X	SMT > Other SMT > EMDR† SMT > PFA†	SMT > Other Other > EMDR† SMT > EMDR SMT > PFA
PTCI	X	X	X
BDI	PFA > EMDR†	X	SMT > Other SMT > EMDR SMT > PFA
Hamilton	X	X	SMT > Other†
PILL	X	X	X

† = trend

X = No one treatment is significantly better than another

*See Tables 13, 19, 23, 26 and 29 for details

Table 17. Basic Model including Coefficients and Significance Tests for Level 1 and Level 2 Covariates in Prediction of PTCI Slope over 6 months

	Coefficient	Standard Error	*t* Ratio	*df*	*p*
Fixed effects					
PTCI intercept, β_0					
Avg. initial PTCI, y_{00}	92.965	3.121	29.788	86	.000
PTCI slope (per month), β_1					
Average slope, γ_{10}	-4.349	1.945	-2.236	81	.028
Baseline PTCI, y_{11}	-0.065	0.016	-4.211	81	.000
Age, γ_{12}	0.045	0.046	0.985	81	.328
Gender, γ_{13}	-1.193	1.009	-1.182	81	.241
Ethnicity, γ_{14}	1.678	1.187	1.413	81	.161
Education, γ_{15}	-0.206	0.531	-0.387	81	.699

Random effects	SD	Variance	df	χ^2	*p* Value
Intercept, U_0	25.068	628.400	86	288.349	.000
Slope, U_1	0.701	0.491	81	78.266	>.500
Error, *R*	20.844	434.480			

Table 18. Basic Model including Coefficients, Treatment versus Control and Significance Tests for Level 1 and Level 2 Covariates in Prediction of PTCI Slope over 6 months

	Coefficient	Standard Error	*t* Ratio	*df*	*p*
Fixed effects					
PTCI intercept, β_0					
Avg. initial PTCI, y_{00}	92.965	3.123	29.766	86	.000
PTCI slope (per month), β_1					
Average slope, γ_{10}	-4.106	1.923	-2.136	79	.036
Baseline PTCI, y_{11}	-0.064	0.016	-4.082	79	.000
Age, γ_{12}	0.045	0.046	0.979	79	.331
Gender, γ_{13}	-1.233	1.009	01.222	79	.226
Ethnicity, γ_{14}	1.452	1.215	1.195	79	.236
Education, γ_{15}	-0.148	0.553	-0.267	79	.790
EMDR dummy, γ_{16}	-0.685	1.160	-0.590	79	.556
SMT dummy, γ_{17}	0.134	1.138	0.118	79	.907

Random effects	SD	Variance	df	χ^2	*p* Value
Intercept, U_0	25.016	625.815	86	286.314	.000
Slope, U_1	0.713	0.508	79	77.558	>.500
Error, *R*	20.918	437.568			

* = $p<.05$; ** = $p<.001$

Table 19. Significance of predictors for PTCI (each model includes standard covariates)[a]

	Dav. Baseline			Alcohol			Marijuana			CSA			BPD			Death		
Group	β	T-rat.	*p*	β	T-rat.	*p*	β	T-rat.	*p*	β	T-rat.	*p*	β	T-rat.	*p*	β	T-rat.	*p*
EMDR Dummy	1.72	0.81	.422	2.88	0.99	.326	1.71	0.51	.612	-0.72	-0.34	.734	-2.94	-1.59	.115	-2.01	-0.98	.331
SMT Dummy	-1.75	-0.75	.454	-2.20	-0.88	.384	-4.36	-1.76	.083†	0.88	0.46	.646	2.49	1.36	.179	-0.60	-0.29	.776
EMDR & SMT	0.98 -1.28	0.39 -0.47	.700 .643	2.14 -1.13	0.65 -0.39	.515 .696	-2.69 -6.20	-0.84 -3.55	.402 .001**	-0.36 0.75	-0.15 0.34	.886 .737	-2.28 1.35	-1.16 0.69	.250 .495	-3.36 -2.23	-1.30 -0.86	.296 .392
EMDR vs. SMT	2.32	0.88	.384	3.46	1.11	.274	3.78	1.14	.262	-1.31	-0.57	.569	-3.82	-1.78	.080†	-0.90	-0.39	.701
SMT vs. PFA	-1.19	-0.43	.671	-1.57	-0.51	.611	-6.32	-3.37	.002**	0.60	0.37	.788	1.51	0.81	.423	-1.79	-0.69	.492
EMDR vs. PFA	0.60	0.24	.809	2.09	0.64	.523	-2.51	-0.79	.434	-0.15	-0.06	.954	-2.87	-1.44	.157	-4.39	-1.63	.110

* = $p < .05$; ** = $p < .01$

[a] = only includes runs where linear regression/ANOVA was significant

Table 20. Significance tests for discussion of trauma within group

Outcome/Model			**β**	***t*-rat.**	***p***
DAV	-	*SMT*	1.47	0.56	.587
	-	*PFA*	0.73	0.69	.495
PTCI	-	*SMT*	-0.42	-0.17	.871
	-	*PFA*	-3.13	-2.06	.050*
BDI	-	*SMT*	-0.32	-0.39	.703
	-	*PFA*	0.65	2.63	.015*
HAM	-	*SMT*	0.60	1.17	.263
	-	*PFA*	0.05	0.18	.856
PILL	-	*SMT*	-0.33	-0.13	.903
	-	*PFA*	-0.23	-0.16	.887

* = $p<.05$; ** = $p<.001$
† = trend

Table 21. Basic Model including Coefficients and Significance Tests for Level 1 and Level 2 Covariates in Prediction of BDI Slope over 6 months

	Coefficient	Standard Error	*t* Ratio	*df*	*p*
Fixed effects					
BDI intercept, β_0					
Avg. initial BDI, y_{00}	12.334	0.796	15.502	86	.000
BDI slope (per month), β_1					
Average slope, γ_{10}	-0.466	0.405	-1.150	81	.254
Baseline BDI, y_{11}	-0.033	0.014	-2.326	81	.023
Age, γ_{12}	0.014	0.012	1.180	81	.242
Gender, γ_{13}	-0.135	0.242	-0.559	81	.578
Ethnicity, γ_{14}	-0.127	0.332	-0.382	81	.703
Education, γ_{15}	-0.170	0.106	-1.592	81	.115

Random effects	SD	Variance	df	χ^2	*p* Value
Intercept, U_0	6.220	38.690	86	242.618	.000
Slope, U_1	0.083	0.007	81	66.277	>.500
Error, *R*	5.803	33.678			

* = $p<.05$; ** = $p<.001$

Table 22. Basic Model including Coefficients, Treatment versus Control and Significance Tests for Level 1 and Level 2 Covariates in Prediction of BDI Slope over 6 months

	Coefficient	Standard Error	*t* Ratio	*df*	*p*
Fixed effects					
BDI intercept, β_0					
Avg. initial BDI, y_{00}	12.225	0.796	15.488	86	.000
BDI slope (per month), β_1					
Average slope, γ_{10}	-0.359	0.412	-0.872	79	.386
Baseline BDI, y_{11}	-0.027	0.013	-2.010	79	.048
Age, γ_{12}	0.015	0.012	1.188	79	.239
Gender, γ_{13}	-0.171	0.235	-0.728	79	.469
Ethnicity, γ_{14}	-0.399	0.373	-0.804	79	.424
Education, γ_{15}	-0.145	0.108	-1.347	79	.182
EMDR dummy, γ_{16}	-0.294	0.252	-1.168	79	.247
SMT dummy, γ_{17}	0.265	0.329	0.804	79	.424

Random effects	SD	Variance	df	χ^2	*p* Value
Intercept, U_0	6.225	38.746	86	242.592	.000
Slope, U_1	0.076	0.006	79	64.525	>.500
Error, *R*	5.804	33.682			

* = $p<.05$; ** = $p<.001$

Table 23. Significance of predictors for BDI (each model includes standard covariates)[a]

	Dav. Baseline			BPD			Time			Violent			Violent Death		
Group	β	T-rat.	*p*	β	T-rat.	*p*	β	T-rat.	*p*	β	T-rat.	*p*	β	T-rat.	*p*
EMDR Dummy	-1.25	-2.37	.021*	-0.29	-0.66	.513	0.17	0.33	.740	0.63	1.42	.160	0.79	1.58	.117
SMT Dummy	0.45	0.72	.475	-0.20	-0.39	.697	-0.02	-0.04	.971	-0.24	-0.40	.689	-1.31	-2.77	.007**
EMDR & SMT	-1.50 -0.23	-2.01 -0.31	.048* .760	-0.63 -0.49	-1.30 -0.84	.199 .405	0.16 0.10	0.24 0.14	.808 .893	0.71 0.04	1.54 0.06	.128 .954	-0.04 -1.36	-0.06 -2.58	.950 .012*
EMDR vs. SMT	-1.57	.2.58	.013*	-0.20	-0.39	.699	-0.09	-0.16	.875	0.52	0.82	.415	1.25	2.25	.029*
SMT vs. PFA	-0.30	-0.39	.695	-0.42	-0.72	.474	0.17	0.22	.828	0.17	0.26	.797	-1.38	-2.57	.014*
EMDR vs. PFA	-1.30	-1.65	.104[†]	-0.34	-0.68	.497	0.48	0.83	.411	0.84	1.94	.058[†]	-0.03	-0.05	.959

* = p <.05; ** = p<.01

[a] = only includes runs where linear regression/ANOVA was significant

[†] = trend

Table 24. Basic Model including Coefficients and Significance Tests for Level 1 and Level 2 Covariates in Prediction of HAM Slope over 6 months

	Coefficient	Standard Error	*t* Ratio	*df*	*p*
Fixed effects					
HAM intercept, β_0					
Avg. initial HAM, y_{00}	8.472	0.491	17.270	86	.000
HAM slope (per month), β_1					
Average slope, γ_{10}	-0.286	0.335	-0.853	81	.396
Baseline HAM, y_{11}	0.067	0.023	2.963	81	.004
Age, γ_{12}	0.004	0.010	0.380	81	.704
Gender, γ_{13}	0.147	0.206	0.714	81	.477
Ethnicity, γ_{14}	0.281	0.214	1.314	81	.193
Education, γ_{15}	-0.114	0.093	-1.230	81	.223
Random effects	**SD**	**Variance**	**df**	χ^2	***p* Value**
Intercept, U_0	3.002	9.010	86	153.773	.000
Slope, U_1	0.923	0.852	81	147.105	.000
Error, R	4.659	21.703			

* = $p<.05$; ** = $p<.001$

Table 25. Basic Model including Coefficients, Treatment versus Control and Significance Tests for Level 1 and Level 2 Covariates in Prediction of HAM Slope over 6 months

	Coefficient	Standard Error	*t* Ratio	*df*	*p*
Fixed effects					
HAM intercept, β_0					
Avg. initial HAM, y_{00}	8.474	0.491	17.248	86	.000
HAM slope (per month), β_1					
Average slope, γ_{10}	-0.231	0.343	-0.675	79	.502
Baseline HAM, y_{11}	0.073	0.022	3.369	79	.002
Age, γ_{12}	0.004	0.010	0.383	79	.702
Gender, γ_{13}	0.138	0.204	0.676	79	.501
Ethnicity, γ_{14}	0.193	0.223	0.858	79	.394
Education, γ_{15}	-0.102	0.095	-1.072	79	.287
EMDR dummy, γ_{16}	-0.155	0.239	-0.647	79	.519
SMT dummy, γ_{17}	0.112	0.238	0.471	79	.638

Random effects	SD	Variance	df	χ^2	*p* Value
Intercept, U_0	3.015	9.088	86	154.114	.000
Slope, U_1	0.974	0.949	79	152.437	.000
Error, *R*	4.653	21.655			

* = $p<.05$; ** = $p<.001$

Table 26. Significance of predictors for HAM (each model includes standard covariates)[a]

	Dav. Baseline			Alcohol			BPD			Violent			Violent Death		
Group	β	T-rat.	*p*	β	T-rat.	*p*	β	T-rat.	*p*	β	T-rat.	*p*	β	T-rat.	*p*
EMDR Dummy	-0.70	-1.40	.166	-0.38	-0.42	.679	-0.09	-0.21	.839	0.56	1.33	.187	0.68	1.25	.215
SMT Dummy	1.06	2.38	.020*	0.69	1.05	.296	0.13	0.29	.773	-0.35	-0.67	.503	-0.78	-1.72	.090†
EMDR &	-0.24	-0.44	.661	0.15	0.17	.866	-0.06	-0.11	.913	0.55	1.27	.207	0.29	0.52	.207
SMT	0.96	1.96	.053	0.78	1.36	.179	0.12	0.24	.809	-0.12	-0.22	.825	-0.64	-1.47	.825
EMDR vs. SMT	-1.40	-2.45	.018*	-0.71	-0.73	.469	-0.23	-0.43	.668	0.50	0.82	.417	0.88	1.52	.135
SMT vs. PFA	0.89	1.93	.059†	0.92	1.54	.130	-0.03	-0.06	.956	-0.21	-0.42	.677	-0.69	-1.53	.132
EMDR vs. PFA	0.04	0.07	.946	0.31	0.35	.722	0.03	0.07	.948	0.56	1.32	.193	0.17	0.32	.748

* = $p < .05$; ** = $p < .01$

[a] = only includes runs where linear regression/ANOVA was significant

Table 27. Basic Model including Coefficients and Significance Tests for Level 1 and Level 2 Covariates in Prediction of PILL Slope over 6 months

	Coefficient	Standard Error	*t* Ratio	*df*	*p*
Fixed effects					
PILL intercept, β_0					
Avg. initial PILL, γ_{00}	104.958	2.975	35.277	86	.000
PILL slope (per month), β_1					
Average slope, γ_{10}	-1.562	1.813	-0.862	81	.392
Baseline PILL, γ_{11}	-0.076	0.016	-4.662	81	.000
Age, γ_{12}	-0.041	0.060	-0.674	81	.502
Gender, γ_{13}	0.030	0.924	0.033	81	.974
Ethnicity, γ_{14}	0.004	1.174	0.004	81	.997
Education, γ_{15}	-0.017	0.447	-0.039	81	.970
Random effects	**SD**	**Variance**	**df**	**χ^2**	***p* Value**
Intercept, U_0	24.925	621.271	86	422.516	.000
Slope, U_1	1.659	2.751	81	92.724	.176
Error, *R*	16.774	281.352			

* = $p<.05$; ** = $p<.001$

Table 28. Basic Model including Coefficients, Treatment versus Control and Significance Tests for Level 1 and Level 2 Covariates in Prediction of PILL Slope over 6 months

	Coefficient	Standard Error	*t* Ratio	*df*	*p*
Fixed effects					
PILL intercept, β_0					
Avg. initial PILL, y_{00}	104.938	2.974	35.280	86	.000
PILL slope (per month), β_1					
Average slope, γ_{10}	-1.055	1.758	-0.600	79	.550
Baseline PILL, y_{11}	-0.077	0.016	-4.820	79	.000
Age, γ_{12}	-0.058	0.059	-0.991	79	.325
Gender, γ_{13}	0.131	0.899	0.146	79	.885
Ethnicity, γ_{14}	0.522	1.124	0.464	79	.643
Education, γ_{15}	0.070	0.431	0.161	79	.873
EMDR dummy, γ_{16}	-1.273	0.948	-1.343	79	.183
SMT dummy, γ_{17}	-2.243	1.061	-2.114	79	.037

Random effects	SD	Variance	df	χ^2	*p* Value
Intercept, U_0	24.908	620.385	86	422.512	.000
Slope, U_1	1.522	2.318	79	87.905	.231
Error, *R*	16.774	281.350			

* = $p<.05$; ** = $p<.001$

Table 29. Significance of predictors for PILL (each model includes standard covariates)[a]

	CSA		
Group	β	T-rat.	*p*
EMDR Dummy	3.84	2.14	.035*
SMT Dummy	0.19	0.12	.908
EMDR &	5.30	2.68	.009**
SMT	2.68	1.54	.128
EMDR vs. SMT	2.59	1.40	.168
SMT vs. PFA	3.00	1.79	.080†
EMDR vs. PFA	4.80	2.47	.017*

* = $p < .05$; ** = $p < .01$

[a] = only includes runs where linear regression/ANOVA was significant

† = trend

Table 30. Treatment Recommendations by Outcome*

Outcome	**Treatment Effectiveness**
Davidson	PFA
PTCI	X
BDI	EMDR
Hamilton	X
PILL	SMT

X = No one treatment is significantly better than another

*See Table 14 for details

Table 31. Treatment Recommendations for Individual Factors by Outcome*

Outcome	DAV Baseline	CSA	BPD	Marijuana
Davidson	X	PFA	X	X
PTCI	X	X	EMDR	SMT
BDI	EMDR	X	X	X
Hamilton	EMDR	X	X	X
PILL	X	PFA	X	X

X = No one treatment is significantly better than another

*See Table 15 for details

Table 32. Treatment Recommendations for Trauma Factors by Outcome

Outcome	**Violent**	**Death**	**Violent Death**
Davidson	X	SMT	SMT
PTCI	X	X	X
BDI	PFA	X	SMT
Hamilton	X	X	SMT
PILL	X	X	X

X = No one treatment is significantly better than another

*See Table 16 for details

Table 33. Dropout over time by treatment group

	Baseline	**F1**	**F3**	**F6**
PFA	31	30	29	27
SMT	29	29	28	29
EMDR	27	25	27	25

Figure 1. Change in Davidson PTSD scores through six-month follow-up assessment

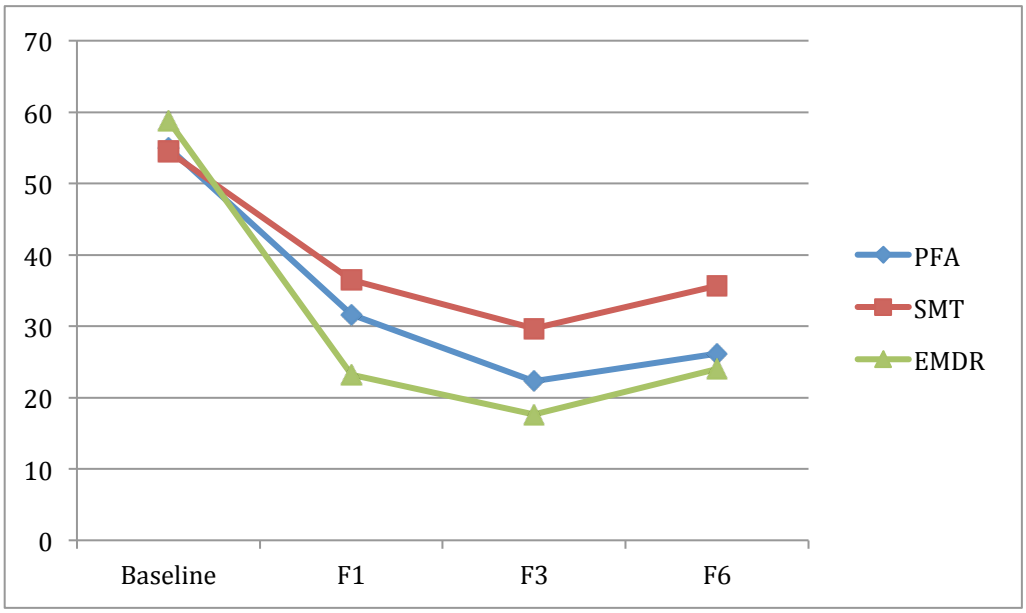


Figure 2. Change in Davidson PTSD scores through six-month follow-up assessment, accounting for covariates and differential dropout over time

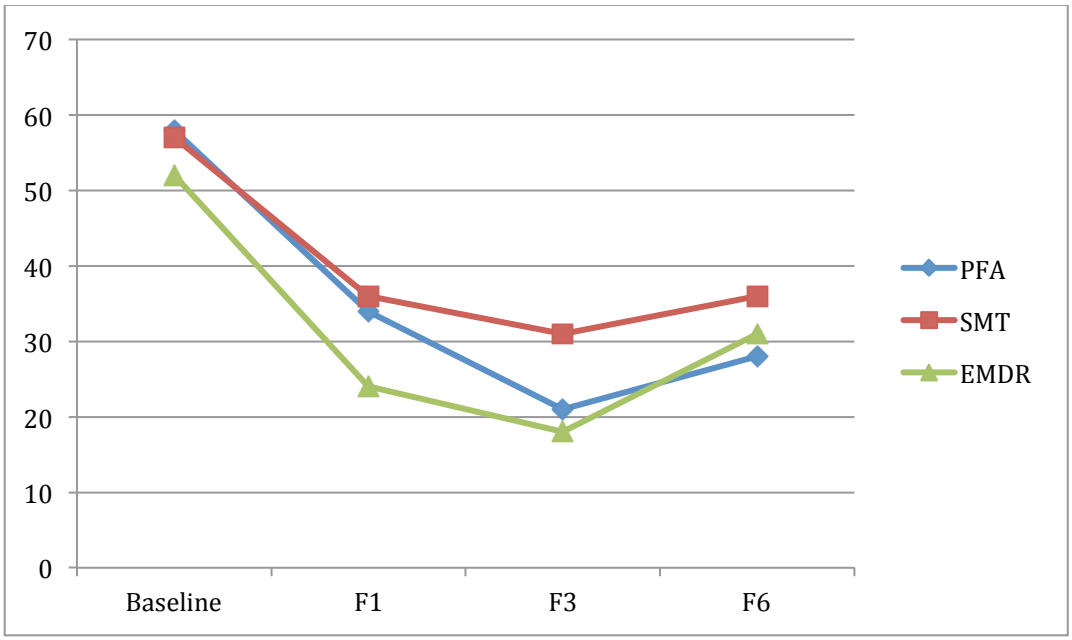


Figure 3. Change in PTCI scores through six-month follow-up assessment

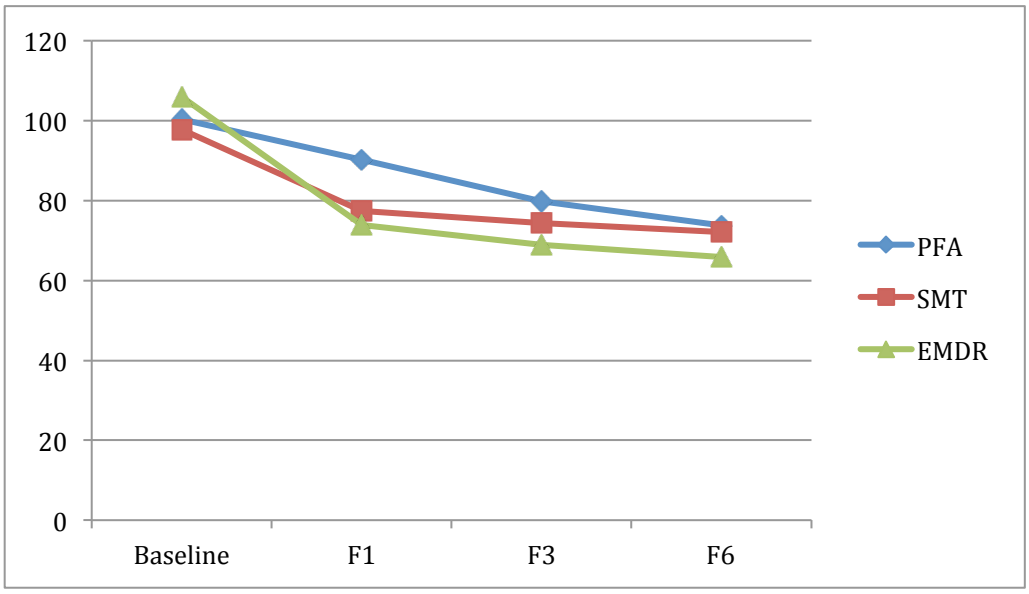


Figure 4. Change in BDI scores through six-month follow-up assessment

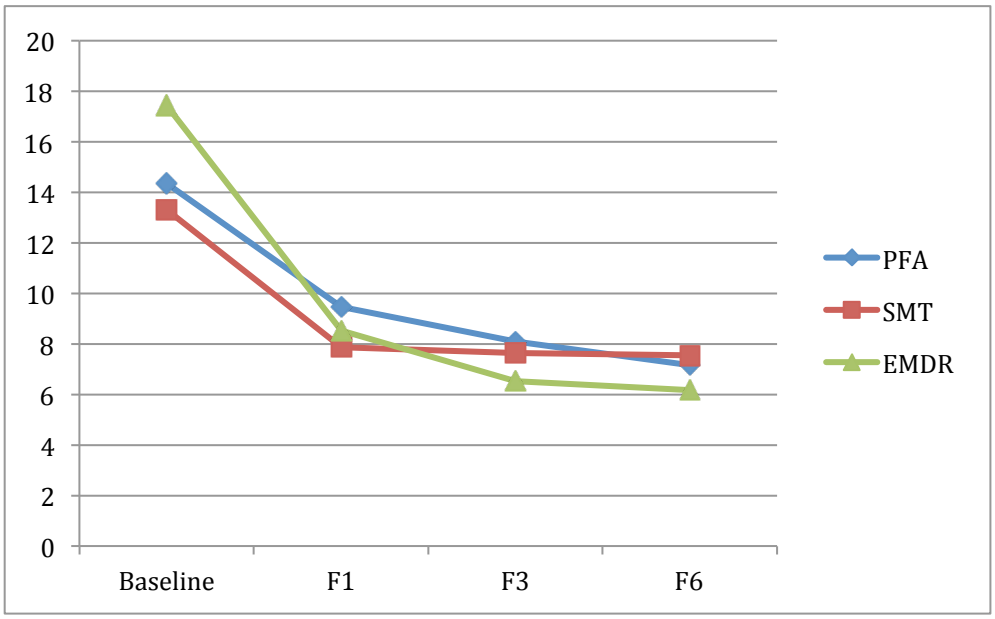


Figure 5. Change in Hamilton scores through six-month follow-up assessment

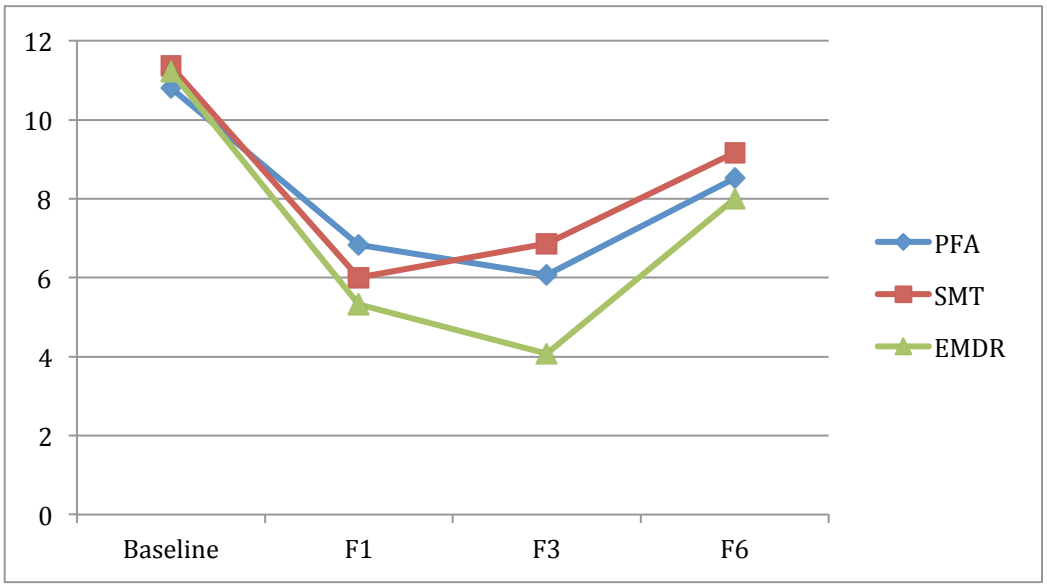
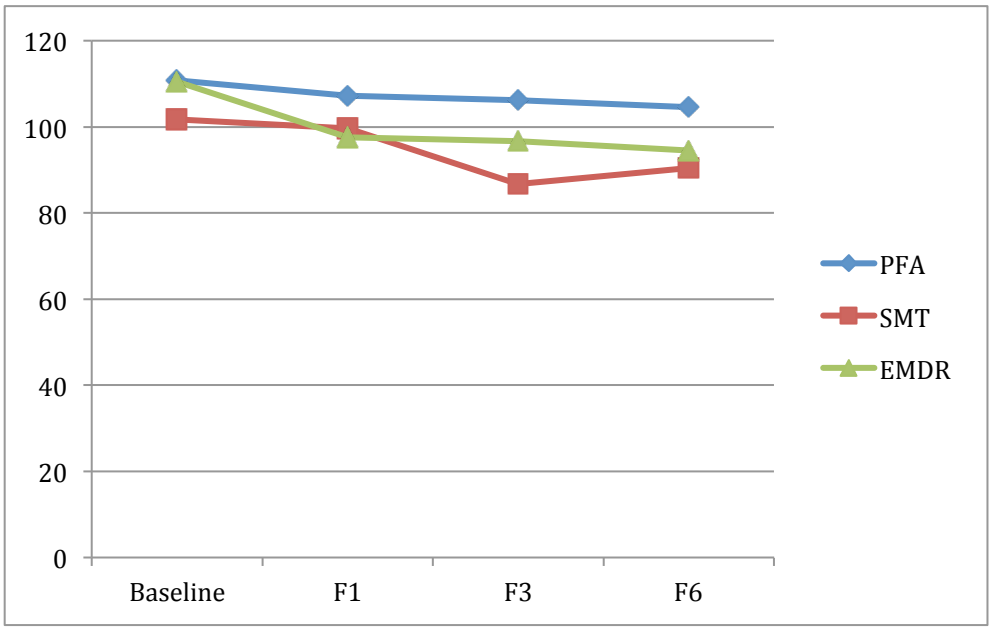


Figure 6. Change in PILL physical symptoms through six-month follow-up assessment



APPENDIX 1

PFA Outline

1. **Session 1 – Introduction to Stress**
    a. Check in
    b. Introduction
        i. Introduction of group leaders
        ii. Group rules, confidentiality, etc.
        iii. Introduction of individuals in group
        iv. Brief introduction about the group
            1. What is PFA?
                a. Stabilization
                b. Brief information on coping
    c. Normalizing Stress
        i. What is trauma? (Group participation)
        ii. What is stress? (Group participation)
        iii. Stress and Trauma Symptoms Checklist
        iv. "When Terrible Things Happen – What You May Experience" handout
    d. Breathing Exercise
    e. Wrap Up

2. **Session 2 – Reactions to Trauma and Stress**
    a. Check in
    b. Introduction
        i. Did anything come up during the last week?
        ii. Review last session (Group participation)
        iii. Brief description of this session
    c. Common Reactions to Trauma and Loss
        i. Symptoms of Stress and PTSD (Group participation)
            a. Feelings
            b. Thoughts
            c. Physical Effects
            d. Behaviors
            e. Spiritual
            2. Modified handout from provider worksheets
            3. PTSD Stress Reactions
        ii. How do you react to stress? (Group participation)
        iii. Common reactions to stress and trauma
        iv. Positive reactions and negative reactions
    d. Breathing Exercise
    e. Wrap Up

3. **Session 3 - Coping**
    a. Check in
    b. Introduction
        i. Did anything come up during the last week?
        ii. Review last session
        iii. Brief description of this session
    c. Reactions to Trauma and Loss
        i. Loss of loved one
    d. Guilt and Shame
    e. Anger Management
    f. Sleep Problems
    g. Coping
        i. "Alcohol, Medication, and Drug Use after Trauma and Disaster" handout
    h. Breathing Exercise
    i. Wrap Up

4. **Session 4 – Resources**
    a. Check in
    b. Introduction
        i. Did anything come up during the last week?
        ii. Review last session
        iii. Brief description of this session
    c. Social Support: "Connecting with Others" handouts
    d. Resources
        i. "Tips for Adults" handout
        ii. Community centers
        iii. Hotlines
        iv. Other
    e. Breathing Exercise
        i. Wrap Up

Synopsis of Stress Management Group Therapy

As used in the Mitigation of Effects of Acute Traumatic Stress Study

For our purposes, the Stress Management was used in group format over four sessions in four weeks. Participants were taught relaxation skills, how to identify stress reactions, basic tools for cognitive restructuring and healthy coping. They were also asked to write emotionally about their recent trauma for twenty minutes in each session. Participants were guided through these processes in session and were asked to put the techniques to practice at home so that they could begin to incorporate them in their daily lives. Participation levels were recorded weekly for each participant.

Session One: Introduction and Overview

After orienting the participants to the treatment and addressing confidentiality, the stress response was discussed. Participants were educated to identify symptoms of stress so that they would be able to recognize when they were becoming stressed. Each participant filled out a stress symptoms checklist and the group discussed how each individual responds to stress. Symptoms were identified as falling into one of the following five categories: cognitive, emotional, behavioral, physical, or social. Excessive worry and sleep difficulties were addressed and participants were taught techniques to help them gain control over these problems. Participants then engaged in the emotional trauma-writing task and were guided through the Relaxation #1, which is deep breathing and muscle relaxation.

Session Two: Thoughts and Feelings – ABC Approach

This session began by quickly reviewing the last session and discussing events of the last week. Participants were then educated about the relationship between distressing thoughts and feelings (emotional or physical). They were guided interactively through the ABC Thought Process, where 'A' is an event, which leads to 'B', a thought/appraisal/self-talk, which leads to 'C', a feeling/emotion/physical state. Automatic thoughts and examples of distorted thinking were discussed. Patients were introduced to cognitive restructuring for distorted thinking and were engaged in challenging their automatic, distorted thoughts. Participants then completed the emotional trauma-writing task and were guided through Relaxation #2, beach scene imagery.

Session Three: Coping and Managing Your Moods

After reviewing contents of the previous session, the concept of coping was introduced and controllable versus uncontrollable situations were identified. Problem-focused versus emotion-focused ways of coping were also identified, and using these

concepts, the participants were taught how to best match a coping technique to a situation. Active (healthy) coping versus passive (unhealthy) coping was discussed. Various moods were also discussed and healthy coping techniques which may manage negative mood states were identified. Patterns of anger as well as overall anger awareness was discussed and basic assertiveness training was implemented. Participants then completed the emotional trauma-writing task and were guided through Relaxation #3, which was mindful meditation.

Session Four: Social Support and Spirituality

After reviewing contents of the previous session, benefits of social support were discussed. Members of participants' social support network were identified and social support was taught as being divided into three categories: psychological/emotional, informational and tangible. Participants engaged in identifying which category their social supporters fall into, and it was emphasized that different people in their life can provide different types of social support. Spirituality was also discussed and questions of, "Why me?" were addressed. Benefits of spirituality were identified, which included providing meaning to an event and providing the sense of support. Participants then completed the emotional trauma-writing task and were guided through Relaxation #4, which combined progressive muscle relaxation, breathing and imagery. At the end of the session, material that was covered over the last few weeks was briefly reviewed and ways to maintain the benefits of the treatment were discussed (e.g. review handbook, practice relaxation).

APPENDIX 3

8 Phases of EMDR

Phase 1 of the EMDR protocol is the History-Taking phase. In this phase, the therapist helps the client identify presenting issues and trauma-related previous experiences that he or she would like to process in treatment. It is important to first assess whether or not the patient is stable enough to confront his or her issues. After this is established, a treatment plan is agreed upon. The treatment plan is a targeting sequence for memory reprocessing, which includes a presenting issue and an associated negative cognition about the self, as well as previous memories associated with the negative cognition (usually a first and worst memory). A positive cognition that the client would like to believe about him or herself is also identified.

Phase 2, the Preparation phase, consists of rapport building, psychoeducation about EMDR, signing of the informed consent, and developing and enhancing affect management skills to ensure that the client is able to effectively shift emotional states. The client is taught a relaxation technique and bilateral stimulation is introduced to install the relaxation.

In Phase 3, the Assessment phase, the focus is on the agreed upon memory or the target memory. The client is asked to identify the current components of the experience, including current emotions and physical sensations that it may evoke. This process helps access the memory further and helps emotionally activate the client. Once the client is fully activated, the clinician takes baseline measurements of discomfort using the Subjective Units of Discomfort Scale (SUDS).

Phase 4 is the primary phase in EMDR and focuses on Desensitization, or the reprocessing of a target memory using bilateral stimulation. During this reprocessing, other channels of related memory are activated and reprocessed. This process is continued until the target incident is successfully resolved and the client's discomfort is minimal.

Once the target incident seems to have been processed and the client's SUDS are reported to be low, the treatment can move into Phase 5, the Installation phase. In this phase, the client is asked to reevaluate the previously selected positive cognition to determine if it still suits the incident. If so, the positive cognition is linked with the target incident and is strengthened using bilateral stimulation until the Validity of Cognition (VOC) reported by the client is high.

In Phase 6, the Body Scan phase, the client is guided to reprocess any residual physical/somatic manifestation of memory. He or she is asked to access the memory and hold the positive cognition in mind, scan the body, and identify any discomfort. Any uncomfortable body sensations are reprocessed using bilateral stimulation until the disturbance is cleared.

Phase 7 is the Closure phase, in which the client is stabilized and reoriented to the present situation in order to bring closure to the reprocessing. The client is debriefed from the session, reinforced for their work, and asked to notice any changes within themselves over the next week so that it can be discussed in the next session.

After all memories have been processed and the targeting sequence is completed over several treatment sessions, it is time for Phase 8, the Reevaluation phase. In this phase, the clinician checks for treatment effects and for any other associations that may require targeting. The clinician reprocesses with the client as needed. Treatment is considered complete when all channels of memory have been processed and the client experiences improvements in functioning.

The PILL

Several common symptoms or bodily sensations are listed below. Most people have experienced most of them at one time or another. We are currently interested in finding out how prevalent each symptom is among various groups of people. On the page below, write how frequently you experience each symptom. For all items, use the following scale:

A	B	C	D	E
Have never or almost never experienced the symptom	Less than 3 or 4 times per year	Every month or so	Every week or so	More than once every week

For example, if your eyes tend to water once every week or two, you would answer "D" next to question #1.

___1. Eyes water
___2. Itchy eyes or skin
___3. Ringing in ears
___4. Temporary deafness or hard of hearing
___5. Lump in throat
___6. Choking sensations
___7. Sneezing spells
___8. Running nose
___9. Congested nose
___10. Bleeding nose
___11. Asthma or wheezing
___12. Coughing
___13. Out of breath
___14. Swollen ankles
___15. Chest pains
___16. Racing heart
___17. Cold hands or feet even in hot weather
___18. Leg cramps
___19. Insomnia or difficulty sleeping
___20. Toothaches
___21. Upset stomach
___22. Indigestion
___23. Heartburn or gas
___24. Abdominal pain
___25. Diarrhea
___26. Constipation
___27. Hemorrhoids
___28. Swollen joints
___29. Stiff or sore muscles
___30. Back pains
___31. Sensitive or tender skin
___32. Face flushes
___33. Tightness in chest
___34. Skin breaks out in rash
___35. Acne or pimples on face
___36. Acne/pimples other than face
___37. Boils
___38. Sweat even in cold weather
___39. Strong reactions to insect bites
___40. Headaches
___41. Feeling pressure in head
___42. Hot flashes
___43. Chills
___44. Dizziness
___45. Feel faint
___46. Numbness or tingling in any part of body
___47. Twitching of eyelid
___48. Twitching other than eyelid
___49. Hands tremble or shake
___50. Stiff joints
___51. Sore muscles
___52. Sore throat
___53. Sunburn
___54. Nausea

Since the beginning of the semester, how many:

_____ Visits have you made to the student health center or private physician for illness
_____ Days have you been sick

APPENDIX 5

Regression *p* values for Group, Gender, & GroupxGender

D.V.	SvP/G/Int	EvS/G/Int	EvP/ G/ Int	Edum/ G/ Int	Sdum/ G/Int
DavF3	.094/.74/.37	**.023**/.83/.77	.32/.41/.54	**.048**/.67/.84	**.022**/.73/.47
DavF6	.11/.83/.17	.14/.99/.36	.76/.26/.96	.26/.67/.57	.072/.61/.20
PTCIF3	.45/.98/.39	.20/.88/.37	**.044**/.39/.989	.06/.80/.67	.77/.83/.32
PTCIF6	.82/.92/.86	.34/.22/.30	.25/.29/.24	.22/.32/.21	.69/.34/.66
BDIF3	.96/.45/.48	.10/.33/.63	**.042**/.68/.76	**.034**/.37/.93	.31/.39/.47
BDIF6	.80/.82/.78	.32/.25/.57	.21/.20/.19	.21/.32/.29	.39/.33/.84
HamF3	.41/.12/.86	**.007**/.72/.26	**.015**/.45/.089	**.004**/.28/.099	**.036**/.32/.56
HamF6	.68/.82/.74	.62/.99/.99	.83/.68/.89	.69/.82/.85	.59/.84/.88
PILLF3	**.024**/.23/.55	.46/.47/.81	.14/.28/.62	.70/.27/.77	.08/.21/.84
PILLF6	.17/.45/.72	.92/.87/.77	.21/.66/.55	.52/.63/.63	.38/.55/.97

RED = $p<.05$ S = SMT; P = PFA, E = EMDR

BLUE = $p<.1$ G = Gender; Int = Gender by group interaction term

ANOVA results: Directionality & *p* values for all groups, gender & PTSD severity

D.V.	3 Groups	Female	Male	DavB>32	DavB<=32
DavF3	E>P>S .084, .492	E>P>S .104, .148	E=P>S .628, .359	E>P>S .052, .558	P>E>S .865, .830
DavF6	E>P>S .265, .138, .636	E>P>S .143, .156, .332	E=P=S .946, .742, .078	E=P>S .263, .120, .754	E=P=S .887, .900, .755
PTCIF3	E>S>P .055, .651	E>S>P .030, .724	E>S>P .408, .517	E>S>P .238, .268	E>P=S .071, .327
PTCIF6	E>S>P .510, .112, .828	E>S>P .102, .302, .954	P>E=S .773, .175, .641	E>S>P .750, .245, .270	E>P>S .476, .053, .620
BDIF3	E>P=S .033, .659	E>S>P .057, .439	E>P>S .534, .634	E>P>S .016, .513	S>P>E .488, .850
BDIF6	E>S=P .126, .287, .663	E>S>P .088, .346, .282	P>E>S .946, .799, .283	E>P=S .041, .201, .532	S>P>E .576, .643, .890
HamF3	E>P>S .351, .301	E>P>S .115, .413	E>P>S .385, .686	E>P>S .613, .401	E>P>S .588, .033
HamF6	E>P=S .885, .511, .126	E=S>P .759, .330, .099	P=E>S .517, .952, .903	E>P>S .652, .812, .379	S>P>E .535, .068, .037
PILLF3	S>E>P .429, .221	S>E>P .619, .388	E>S>P .440, .490	S>E>P .756, .351	S>P>E .072, .510
PILLF6	S>E>P .538, .598, .186	S>E>P .651, .544, .437	E>P>S .348, .964, .295	S>E>P .851, .482, .364	S>E=P .420, .385, .406

Directionality= ___ > (is more effective than) ____
p values: Linear, Quadratic, Cubic (for F6)

RED = $p<.05$
BLUE = $p<.1$

ANOVA results: Directionality & *p* values for alcohol & weed use, and childhood sexual abuse

D.V.	>=10 drinks/mo	<10/mo	Any weed use	No weed use	CSA YES
DavF3	P>E>S .261, .847	E>P>S .253, .582	E>P>S .988, .678	E>P>S .053, .341	E>P>S .211, .827
DavF6	P=E>S .449, .507, .867	E>P>S .303, .198, .658	S>E>P .726, .623, .682	E=P>S .114, .162, .488	P>E>S .107, .297, .378
PTCIF3	P>E>S .967, .333	E>S>P .058, .418	E>S>P .853, .593	E>S>P .046, .304	E>S>P .505, .678
PTCIF6	P>S>E .778, .048, .499	E>S>P .384, .084, .786	E=S>P .396, .424, .685	E>S>P .419, .063, .479	E>S>P .667, .185, .673
BDIF3	P>E>S .459, .838	E>S>P .080, .551	E>P>S .798, .251	E>S>P .032, .590	E>P>S .202, .823
BDIF6	S>E>P .414, .224, .491	E>P>S .228, .178, .788	E>P>S .959, .747, .218	E=S>P .108, .258, .749	E>P>S .176, .772, .648
HamF3	P>E>S .887, .620	E>P>S .304, .117	E>P>S .466, .259	E>P=S .167, .573	E>P>S .660, .289
HamF6	E>P>S .829, .888, .602	S>E=P .867, .345, .023	E>S>P .219, .463, .123	P>E=S .724, .293, .451	E>P>S .832, .994, .201
PILLF3	P=E>S ..763, .229	S>E>P .167, .445	E>S>P .581, .902	S>E>P .491, .154	S>E>P .601, .360
PILLF6	E>S>P .993, .378, .271	S>E>P .481, 429, .331	P>E>S .901, .740, .567	S>E>P .498, .725, .111	S>P>E .559, .066, .512

Directionality= ___ > (is more effective than) ___ **RED** = $p<.05$

p values: Linear, Quadratic, Cubic (for F6) BLUE = $p<.1$

ANOVA results: Directionality & *p* values for c. sexual abuse, borderline char., & time since trauma

D.V.	CSA NO	BPD>=5	BPD<5	TIME SINCE >5wks	TIME SINCE <=5 wks
DavF3	E>P>S .178, .268	E>P>S .386, .820	E=P>S .176, .471	E>P>S .084, .913	E>S>P .614, .053
DavF6	S=P>E .878, .074, .095	P=E>S .521, .422, .987	E=S>P .469, .299, .261	P=E>S .280, .417, .737	E>P=S .718, .366, .275
PTCIF3	E>S>P .085, .532	E>S>P .593, .686	E>P=S .014, .489	E>S>P .179, .755	E>S>P .355, .916
PTCIF6	E>S>P .676, .093, .805	E>S=P .372, .403, .734	P>S>E .126, .085, .993	E>S>P .566, .521, .844	E>S>P .827, .206, .818
BDIF3	E>P=S .161, .383	E>P>S .280, .734	E>S=P .003, .265	P>E>S .360, .638	E>S>P .041, .087
BDIF6	S>E>P .446, .372, .435	E=S>P .360, .776, .449	P>E>S .212, .002, .901	P>E>S .494, .826, .480	E>S>P .298, .065, .578
HamF3	E>P>S ..406, .373	E>P>S .874, .133	E>S=P .348, .690	E>P>S .486, .212	E>S>P .686, .153
HamF6	S=P>E .552, .415, .505	E>P>S .938, .930, .071	S>E=P .732, .402, .941	E=P>S .782, .867, .122	S>E>P .999, .538, .425
PILLF3	S>E>P .664, .402	S>E>P .545, .331	S>E>P .825, .724	S>E>P .524, .676	S>E>P .487, .079
PILLF6	E>S>P .125, .699, .127	E>S>P .921, .646, .121	S>E>P .224, .492, .764	S>E>P .357, .758, .637	S>E>P .469, .729, .140

Directionality= ___ > (is more effective than) ___ **RED** = $p<.05$

p values: Linear, Quadratic, Cubic (for F6) BLUE = $p<.1$

ANOVA results: Directionality & *p* values for discussed trauma & viol. Trauma

D.V.	DISCUSSED T	NO DISC T	VIOLENT
DavF3	E>P>S .250, .168	S=P (no E) .201, .785	E=P>S .464, .905
DavF6	E>P>S .545, .175, .457	S>P .649, .069, .665	P>E>S .403, .681, .993
PTCIF3	E>P>S .384, .474	S>P .931, .136	E>S>P .020, .413
PTCIF6	P>E>S .871, .551, .099	S>P .687, .843, .111	E>S=P .130, .168, .667
BDIF3	E>P>S .038, .327	S=P .025, .451	P=E>S .603, .346
BDIF6	E>S>P .165, .131, .253	P>S .032, .691, .512	P>S>E .793, .962, .328
HamF3	E>P=S .617, .754	S>P .102, .189	E>P>S .969, .039
HamF6	P>E>S .544, .186, .694	S>P .995, .126, .002	P>S>E .570, .909, .023
PILLF3	S>E>P .413, .090	S>P .947, .992	S>E>P .246, .531
PILLF6	E>S>P .917, .255, .013	S>P .850, 934, 935	S>E>P .458, .719, .398

Directionality= ___ > (is more effective than) ____ **RED** = $p<.05$

p values: Linear, Quadratic, Cubic (for F6) BLUE = $p<.1$

D.V.	NONVIOLENT	DEATH	NONDEATH	VIOLDEATH	NON VIODEATH
DavF3	E>P>S .212, .153	E>P=S .425, .055	P>E>S .127, .482	E>S>P .764, .318	E>P>S .055, .823
DavF6	E>S=P .604, .096, .373	E>S>P .707, .130, .115	P>E>S .083, .818, .474	S>P=E .160, .396, .483	E>P>S .071, .448, .890
PTCIF3	E>S>P .565, .902	E>S>P .091, .638	P>S>E .253, .841	E>S>P .130, .309	E>S>P .161, .942
PTCIF6	E>S>P .775, .236, .890	E>S>P .655, .069, .960	P>S=E .568, .701, .743	E>S>P .469, .157, .540	E>P=S .457, .424, .900
BDIF3	E>S>P .013, .051	E>S>P .130, .133	P>E>S .214, .515	E>S>P .918, .290	E>P>S .013, .650
BDIF6	E>S>P .020, .117, .180	S>E>P .341, .152, .215	P>E>S .199, .998, .388	S>E>P .844, .542, .264	E>P>S .014, .423, .700
HamF3	E>S>P .109, .208	E>S=P .399, .187	E>S=P .399, .187	E>S>P .735, .059	E>P>S .116, .991
HamF6	E>S>P .334, .329, .640	S>E>P .804, .289, .208	E=P>S .931, .953, .382	S>P>E .085, .638, .111	E>P>S .433, .524, .670
PILLF3	E>P>S .743, 432	S>E>P .827, .546	S>E>P .182, .263	S>E>P .977, .934	S>E>P .467, .246

ANOVA results: Directionality & *p* values for trauma type

Directionality= ___ > (is more effective than) ____
p values: Linear, Quadratic, Cubic (for F6)

RED = $p<.05$
BLUE = $p<.1$