Posttraumatic Stress Disorder and Symptom Subclusters as a Mediator of Self-Reported Somatic Health Among Individuals Exposed to Residential Fire

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ABSTRACT

Health outcomes following a traumatic event are an important aspect of recovery from any type of trauma. Further, distress and psychopathology, specifically Posttraumatic Stress Disorder (PTSD), have been shown to have a significant impact on physical health recovery post-trauma. The current study utilized a sample of 56 (48 women, 8 men) residential fire survivors to examine the potential mediating effect of PTSD and PTSD symptom subclusters. Participants were interviewed four months after a residential fire and were assessed on levels of exposure to the fire (Fire Questionnaire & Resource Loss Scale), PTSD symptomology (Anxiety Disorder Interview Schedule), and somatic health complaints (Brief Symptom Inventory). Consistent with previous findings, PTSD was found to mediate the relationship between exposure to a traumatic event and reporting of health symptoms. Further, the increased arousal subcluster was found to mediate the aforementioned relationship; the avoidance symptom subcluster was found to partially mediate the same relationship. Implications of results of the current project are discussed with regard to the impact of trauma on survivors' health, along with recommendations for further research.

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Chapter 1

Introduction

The impact of natural and technological disasters, terrorist attacks, and interpersonal violence has been well studied over the course of the past two decades (Schnurr & Green, 2004a). Research has examined the effects of both physical and psychological repercussions following the event for most everyone involved (Schnurr & Green, 2004b). Given the negative impact of disasters on mental health outcomes, such as Posttraumatic Stress Disorder (PTSD; Ozer, Best, Lipsey, & Weiss, 2003) and physical health outcomes (Wagner, Wolfe, Rotnitsky, Proctor, & Erickson, 2000), it is important to further investigate physical symptoms in survivors of trauma. Given disaster's impact on both physical and psychological symptoms, it is desirable to better understand the relationship of exposure in the role of health outcomes and the development of PTSD, as well as the link between PTSD and health outcomes. Additionally, the possible mediating relationship of PTSD between exposure and health outcomes needs to be explored further.

1.1 Exposure and Health

Exposure to a traumatic event and chronic stress has been shown to have a negative impact on a variety of health concerns, including reduction in immune system functioning and increased blood pressure, which are both linked to elevated risk of myocardial infarctions (Carlson, 2002; Madhavan, Ooi, Cohen, & Alderman, 1994). Such findings point to the importance of a better understanding of health outcomes following trauma.

The research on exposure to traumatic events, particularly violence against women, and health outcomes is lengthy and well-established. Koss and Heslet (1992) reviewed the literature on somatic health outcomes of women exposed to violence. In their review of 110 studies, the

authors found convergent themes of acute issues (e.g. injury, pregnancy, and sexually transmitted diseases), chronic concerns (e.g. chronic pain, negative health behaviors, and gastrointestinal symptoms), and finally, long-term trends of increased medicinal use. Further, Resick, Acierno, and Kilpatrick (1997) performed a similar review, documenting the acute physical and emotional consequences for women exposed to interpersonal violence. They concluded that women exposed to such traumatic events are at greater risk for physical and mental health problems. Indeed, the link between exposure to interpersonal violence and increased frequency of somatic complaints has been well documented (Straight, Harper, & Arias, 2003).

Research with military veterans has also shown that exposure to traumatic events leads to decreased health outcomes. Elder, Shanahan, and Clipp (1997) performed a study of 328 World War II veterans. Through retrospective examination of medical records, analyzed by multivariate logistical regression equations, the study concluded that veterans exposed to combat (as compared to those not serving in a combat role) demonstrated greater risk of decline in physical health.

Though the effect of traumatic events on somatic symptoms is evident (Engel, 2004), disasters have been shown to be one of the best predictors of both somatic and psychological problems because of their indiscriminate nature (North, 2002). One of the first studies to examine the effects of disaster on somatic symptoms was completed by Escobar, Canino, Rubio-Stipec and Braco (1992), who examined 375 flood survivors in Puerto Rico. An English-to-Spanish translation of a structured interview was administered to participants to determine somatic health complaints. The results indicated that exposure to floods was significantly related to increased prevalence of somatic symptoms, in particular, gastrointestinal and pseudoneurological symptoms. In another early study by Phifer (1990), 374 participants (222

males and 152 females) were assessed both before and after a local flood. Participants were assessed via a self-report measure of what the authors deemed functional and specific ailments to determine the incident of somatic health. Results of the study suggest that exposure to flooding was associated with higher somatic symptom levels 18 months post-flood.

Ullman & Siegel (1996) completed an epidemiological study with a national sample, collecting data on trauma history and self-reported health outcomes. Results of the study indicated an increased likelihood of poor health and more chronic health concerns for individuals with at least one lifetime exposure to a traumatic event. Thus, while it is clear that exposure to trauma does indeed have an adverse impact on somatic health, no studies have examined the impact on somatic health resulting from exposure to a residential fire. Therefore, the present study intends to explore this relationship.

1.2 PTSD and Health

Posttraumatic Stress Disorder is the most common disorder endorsed following a traumatic event (Shalev et al., 1998). The Diagnostic and Statistical Manual of Mental Disorders IV (DSM-IV; APA, 1994) includes three subclusters of PTSD; reexperiencing, avoidance, and increased arousal. The negative health implications of PTSD have been extensively studied (Schnurr & Jankowski, 1999). Symptoms of PTSD have been shown to produce health complications and biological abnormalities such as hypothalamic-pituitary-adrenal (HPA) axis dysregulation, reduction in growth hormones, and immune system suppression (Friedman & McEwen, 2004). Cohen et al. (1998) examined heart rate and heart rate variability through the use of spectral analysis in individuals with PTSD as compared to a control group. Nine participants with PTSD and nine without PTSD were exposed to trauma related cues and heart rate/heart rate variability was examined through use of an electrocardiogram. Results of the

study indicated a lack of significant autonomic response activation for those with PTSD when asked to recall their presumed triggering traumatic event, indicating trauma survivors with PTSD may have elevated levels of autonomic activation in a resting state. Further, individuals with PTSD demonstrated a level of autonomic dysregulation in a resting state that was consistent to that seen in control group individuals' reaction to recalling a stressful life event.

Given PTSD can be considered a chronic stressor (Baum, Cohen, & Hall, 1993), it is important to examine the role of chronic stress on health. As indicated earlier, chronic stress is often associated in a reduction in immune cell counts (Dougall & Baum, 2004). Additionally, individuals who have experienced a traumatic event are often primed to overreact to subsequent stressors, creating a cycle of vulnerability to hyperarousal (Kendall-Tackett, 2000).

A review of literature by Rozanski and Kubzansky (2005) examined the relationship between psychological functioning and physical health. Results of the study indicated that vitality and positive emotionality were correlated with energy levels. Chronic stress and negative emotions were linked to diminished vitality. Physiopathologically, negative emotionality and chronic stressors evoked a chronic stress response, characterized by increased stimulation of the sympathetic nervous system and HPA axis, (peripheral effects included elevated heart rate and blood pressure, as well as delayed recovery from stressful stimuli). Results indicated hyperarousal to be a major factor in inflexibility in psychological functioning and/or coping with stressful environmental conditions. Conversely, coping was linked with diminished hyperresponsiveness.

Kiecolt-Glaser et al., (1995) directly demonstrated the effect of stress on healing. The study utilized two groups, one with individuals whom were considered chronically stressed (caretakers of family members with dementia), the other, a control group. Participants received a

3-5 mm punch biopsy wound and were monitored for healing rates. Individuals in the chronic stress group took nine days longer to fully heal from the wound. Additionally, as compared to a control group, chronically stressed individuals' peripheral-blood leucocytes produced significantly less interleukin-1 beta (which regulated the production of metalloproteinase) in response to stimulation.

Pole (2007) performed a meta-analysis of the literature of psychophysiological changes of PTSD. The study examined 58 resting baseline studies, 25 startle studies, 17 standardized trauma cue studies, and 22 idiographic trauma cue studies. All 122 studies utilized control groups and examined such variables as facial electromyography (muscle contraction), heart rate, skin conductance, and blood pressure. Findings from the study revealed significant weighted mean effects between PTSD and control groups for heart rate and skin conductance in resting baseline studies. Facial electromyography, heart rate, and skin conductance exhibited significant habituation slopes in startle studies. Differences in heart rate between groups were found in standardized trauma cue studies. Finally, significant differences in skin conductance were found in idiographic trauma cue studies. Consistent with other findings, the author concluded that PTSD is associated with elevated psychophysiological activity.

This empirical evidence attests to the importance of the need for physicians and clinicians to be able to rule out the physiological effects of psychopathology (for example, hyperarousal's long-term impact) from that of other psychopathologies such as somatoform disorders and other medical conditions. Given the link of exposure to PTSD as well as PTSD to negative health outcomes, it is desirable to better understand the role of PTSD in the relationship between exposure to a traumatic event and adverse health outcomes. Although previous PTSD literature supports the theory that alterations occur within the body when psychopathology is present,

research heretofore fails to examine the specific causal mechanisms that influence the relationship between PTSD and adverse health outcomes (Pole, 2007).

1.3 PTSD as a Mediator between Exposure and Health

Given the relationships between PTSD and physical and psychological outcomes, the need to explore the potential mediational role among these variables is compelling. A review of the literature has shown the dose-response model to be one of the most effective means to study the mediational role of PTSD between exposure to a traumatic event and adverse health outcomes. Though alternative methods of studying this mediational role have been used (i.e., Friedman and Schnurr's [1995] path analysis), most prominent studies utilize the dose response model to examine this relationship (i.e. Kimerling, Clum, & Wolfe [2000] and Wolfe, Schnurr, Brown, & Furey [1994]).

The first study to examine the mediational role of PTSD on trauma exposure and physical health outcomes was completed by Wolfe, Schnurr, Brown, and Furey (1994). The study utilized a sample of 109 female veterans (from service in the Vietnam conflict) and assessed their levels of exposure during military service, current PTSD symptoms, and self-reported health at four different points; pre-Vietnam war, during the war, post-Vietnam war, and at the time of the study. Participants rated their health for each of the four time points on a six point likert scale (the first three time points where rated retrospectively). Through the use of combined regression analysis, results indicated that PTSD and war zone exposure were significantly associated with poorer health outcomes and that PTSD symptoms partially mediated the relationship between war zone exposure and physical health outcomes. However, a limitation of this study is that it failed to examine the role, and the potential influence, of each of the three subclusters of PTSD (reexperiencing, avoidance, and increased arousal) for a more accurate

understanding of the disorders influence. As defined by DSM-IV criteria, the reexperiencing subcluster of PTSD includes symptoms such as intrusive memories of the event, distressing dreams, and intense distress when confronted with traumatic cues. The avoidance subcluster includes symptoms such as efforts to avoid thoughts, feelings, activities, or conversations about the event, social isolation, and a sense of a foreshortened future. Finally, the increased arousal subcluster includes such symptoms as difficulty falling or staying asleep, irritability or anger, hypervigilance, and an exaggerated startle response.

Through use of the same dataset employed in the above study, Friedman and Schnurr (1995) examined the same mediational relationship via path analysis. When they examined health, the authors found that 56% of the effect for the relationship between exposure and respondents' self-reported current health was indirectly mediated through PTSD. There were also significant direct effects for exposure in the model. Again however, a limitation of this investigation was the authors' failure to examine the specific role of the subclusters. A similar study found that PTSD partially mediated the relationship of exposure to toxic gases and health outcomes (Ford et al., 2004).

Hammad, Barsky, & Regestein (2001) found somatic complaints and hyperarousal to be highly correlated, and that, when data were analyzed with logistic regression, hyperarousal accounted for most of the variance of somatic symptomatology. The authors concluded that somatization reporting may involve altered central nervous system processing of somatic stimuli. Wagner, Wolfe, Rotnitsky, Proctor, & Erickson utilized combat exposure to examine how PTSD can influence health outcomes. Results indicate that even after controlling for exposure and gender, PTSD symptoms were predictive of reported health problems such as headaches, aches/pains, stomach disturbances, and dizziness (2000).

Green and Kimerling (2004) reviewed several studies examining PTSD as a mediator between trauma exposure and adverse health outcomes. Physical symptoms associated with PTSD can be consistent with other disorders, and when these other psychological disorders are controlled for, the effects of PTSD are reduced. However, though psychological distress or other psychiatric disorders may influence the relationship between trauma exposure and health complaints, PTSD more strongly impacts this relationship. In addition, Green and Kimerling's (2004) review points to the influential role of PTSD, which influences the relationship between exposure to trauma and adverse health outcomes as the primary pathway of psychological disorders or distress following trauma.

Returning to mediational studies, one study that examined the effects of the three subclusters of PTSD on the physical health of trauma survivors was completed by Kimerling, Clum, and Wolfe (2000). The study utilized a sample of 52 female veterans who had experienced a variety of traumas and were predominantly assessed for combat exposure. Through the use of hierarchical multiple regression, the study demonstrated that PTSD symptoms fully mediated the relationship between exposure to war zone stressors and physical health outcomes. Further, when analyzing each of the subclusters of PTSD independently, only hyperarousal accounted for a significant amount of variance in the association between exposure and health. The authors speculated on the reason for this finding. Explanations provided included neurobiological alterations, mislabeling of symptomology, and poor health habits of those living with PTSD. Limitations of this study were that the sample included only women and DSM-III (APA, 1987) which has four symptom subclusters, rather than DSM-IV (APA, 1994) which has three symptom subclusters, criteria were employed.

While it is clear that hyperarousal has been most highly correlated with somatic outcomes, there are a few studies that have examined the impact of other subclusters affecting somatic outcomes. For example, intrusive memories have been linked to PTSD years after the trauma and are correlated with chronic stress (Baum, Cohen, Hall, 1993). Other studies have linked the reexperiencing subcluster of PTSD to be correlated positively with self-reports of physical health symptoms (McFarlane, Atchison, Rafalowicz, & Papay, 1994; Zoellner, Goodwin, & Foa, 2000).

1.4 Rationale for Thesis

Though the relationship between trauma exposure, PTSD, and health is established, these effects have yet to be examined in a population of residential fire survivors. Given the connection between experiencing a traumatic event and somatic illnesses as well as the association between PTSD and somatic symptoms, the current project will attempt to further explain the understanding of this relationship within the context of residential fires. The role of PTSD and PTSD symptom subclusters on somatic health complaints will be examined in light of DSM-IV (APA, 1994) criteria for PTSD and specific subclusters. More specifically, the current study aims to examine the extent to which PTSD will mediate the relationship between trauma exposure and somatic health reporting in survivors of residential fire. The study aims to understand four relationships specifically:

1. It is hypothesized that PTSD will be found to partially mediate the relationship between exposure and reported somatic symptomatology at Time One (four months post-trauma).

   a. Similarly, it is hypothesized that PTSD will be found to partially mediate the relationship between exposure reported at Time One and somatic health complaints at Time Two (eleven months post-trauma).

2. Given the strength of support from previous findings, it is hypothesized that the increased arousal subcluster of PTSD will partially mediate the relationship between exposure and somatic health complaints at Time One.
   a. Further, it is hypothesized that increased arousal reported at Time One will be found to partially mediate the relationship between exposure reported at Time One and somatic health complaints at Time Two.
3. The study intends to test for possible mediation of the reexperiencing subcluster of PTSD as reported at Time One on somatic health complaints reported at Time One; however, given the lack of support from previous findings, a specific hypothesis will not be offered and the analysis will be conducted in an exploratory fashion.
   a. Additionally, the reexperiencing subcluster of PTSD as reported at Time One will be tested as a mediator of the relationship between exposure as reported at Time One and somatic health complaints reported at Time Two.
4. Finally, the avoidance subcluster of PTSD will be examined as a potential mediator between exposure and somatic health outcomes reported at Time One, but again, given the lack of support from previous research, the analysis will be conducted through exploratory analysis.

a. Further, the avoidance subcluster of PTSD as reported at Time One will be examined as a mediator of the relationship of exposure (reported at Time One) and somatic health complaints at Time Two.

Chapter 2

Methods

2.1 Participants

Participants of the current study were taken from a larger dataset of 167 survivors of residential fires (Jones & Ollendick, 2002). Fifty six adults participated in the current study (48 women and 8 men). Participant's included in the study (after listwise deletion was applied to all applicable cases from the larger dataset) ages ranged from 24-79 and the average age of the participants was 37.84 (SD=9.88). Of these participants, 23 were African-American (41.1%), 29 European-American (51.8%), 1 Hispanic (1.8%), 1 Asian (1.8%), 1 Biracial (1.8%), and 1 "Other" (1.8%).

2.2 Measures

Brief Symptom Inventory (BSI; Derogatis, 1993) is a self-report questionnaire designed to assess nine factors of symptoms including somatization, obsessive compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideations, and psychoticism. The instrument also has three subscales, including global severity, positive symptom total, and a positive symptom distress index. The instrument asked participants to rate their level of distress in response to 56 items on a five point likert scale (Groth-Marnat, 2003). The BSI assesses such physical complaints as chest pains, numbness or weakness in parts of the body, and hot or cold spells. Internal consistency for the present sample was .84. In this study, the somatization scale was used to assess somatic health complaints post-trauma.

Fire Questionnaire (Jones & Ollendick, 2002) is a semi-structured interview designed for the Residential Fire Project. Using a three-point likert response scale, the Fire Questionnaire assessed 16 factors, including characteristics of the fire, whether the participant was at home during the time of the fire, exposure, emotional control, negative thoughts, coping, number of professionals seen and number of visits, quality of professional visits, loss, appraisal (both pre- and post-fire), appraisal attributes, fire prevention, fire guilt, and good things that have happened since the fire. In this study the instrument was used to assess exposure levels to the traumatic event (i.e. as part of the exposure variable).

Resource Loss Scale (Freedy, Shaw, Jarrell, and Masters, 1992) is a 53 item self-report measure of loss sustained during a traumatic event. The scale assessed five types of loss: object loss (e.g. physical property), condition loss (e.g. stability of interpersonal relationships and vocation), personal characteristics loss (e.g. self-esteem or other personal qualities), energy loss (e.g. time for pleasurable activities or motivational drive), and pet loss (e.g. loss of companionship from a pet). Additionally, the instrument can also be summed for a total loss factor. Participants responded to each item on a five point likert scale (0=no loss, 4=extreme amount of loss). Internal consistency for the scale with the present sample was .96. The total impact of loss was calculated by summing across the 53 items for a total loss score which was used as a component of the exposure variable in the current project.

Anxiety Disorders Interview Schedule-IV: Lifetime Version (ADIS; Di Nardo, Brown, & Barlow, 1994) is a structured clinical interview designed to assess for a number of anxiety disorders. Regarding the PTSD module of the ADIS, the interview consists of inclusion criteria questions regarding experience of a traumatic event, symptomology, and frequency, intensity, and interference of the symptoms (necessary for diagnosis). The instrument also assesses for

symptoms of the reexperiencing, avoidance, and hyperarousal subclusters consistent with diagnosis for PTSD within the Diagnostic and Statistical Manuel-IV (APA, 1994). Participants were asked to rate their levels of severity for each symptom on a nine point likert scale, ratings above a four were considered clinically significant. The instrument provides a dichotomous diagnosis result and continuous ratings of symptoms for both frequency and distress. The internal consistency for the three subclusters was: reexperiencing was .81, avoidance was .82, and increased arousal was .78. The continuous rating of PTSD symptomology of the ADIS PTSD module was used to assess PTSD symptomology.

2.3 Procedure

Participants were recruited from five locations: Atlanta, Georgia; Charlotte, North Carolina; Charleston, South Carolina; and Blacksburg and Richmond, Virginia. In order for an individual to be included in the study, the individual must have sustained 15% loss of their home and/or personal belongings. Participants who recently experienced a residential fire in their homes were targeted via incident reports sent to the investigators by fire departments, newspaper or television reports, and information given to the fire survivors about the study by cooperating Red Cross agencies. Potential participants were then contacted and informed about the project through letters and telephone calls. A brief screening survey was first conducted over the telephone. Participants who met inclusion criteria were asked to participate; subsequently, assessments were arranged for the participants who agreed to take part in the study. Approximately one third of potential participants contacted met criteria to participate, and two thirds of acceptable potential participants agreed to take part in the study. Approximately 90 percent of these participants completed the first assessment and the 56 participants used in this project were among this group.

During data collection of the study, all policies and procedures of the study were in accordance with American Psychological Association (APA) ethical guidelines. Informed consent was obtained prior to data collection with each participant. Participants were initially assessed approximately four months post-fire and then again eleven and eighteen months post-trauma. Participants were interviewed independently by graduate students who were enrolled in an APA approved clinical psychology doctoral training program and fully trained in all instruments. Interviews were conducted in a variety of locations, including health clinics, libraries, local churches, and Red Cross offices. Measures listed in this project were part of a larger battery of interviews and self-report instruments which took on average three hours to complete (including measures for both participants themselves and their children). The assessment consisted of an unstructured interview, a structured interview, and several self-report measures in order to target their experiences during the fire, psychopathology following the fire, and a number of other factors including coping, resource loss, and levels of exposure. All participants were debriefed before departing the study and each participant/family received $75 for their participation in the original Jones and Ollendick (2002) study.

Chapter 3

Results

3.1 Measure Description

The exposure factor for the current project was based upon that used by Jones and Ollendick (2002) and includes assessment of proximity to the fire, appraisal of life threat, injury sustained during the fire, resource loss, and concern for a family member being harmed during the fire (as measured by the Fire Questionnaire and Resource Loss Scale). For full information regarding descriptive data on each measure please refer to Table 1. Regarding reporting of

exposure, on a 1-4 scale, the average proximity to the fire was 2.72, injury sustained was reported as 1.24, life threat was reported on average at 1.52, and thought a family member would be injured was reported at 2.44. Finally, total resource loss was entered into the exposure variable, reporting of total loss ranged from 2-103 with an average of 50.1. Each item was converted to a z-score, then summed and again reconverted to a final exposure z-score. Participants were required to report three of five items to remain eligible in the study.

Regarding reporting of PTSD symptomology on the ADIS, in congruence with Kimberling, Clum, and Wolfe (2000), the continuous recurrence rating of PTSD was used. Symptoms in each of the three subclusters were averaged and used in inferential data analysis. Further the average of across symptom subclusters were used as a rating of full PTSD symptomology. The average symptom recurrence rating for the reexperiencing subcluster was 2.38, for the avoidance subcluster, 1.42, the increased arousal subcluster's average was 2.66, and the average symptom reporting of full PTSD was 2.06. Participants were included if missing only one symptom across the subclusters.

Finally, regarding somatic symptom reporting, the BSI was utilized. Seven items were reported on a 0-4 likert scale and averaged for a mean score of somatic symptom reporting. The most commonly endorsed somatic complaints included "feeling weak in parts of your body," "nausea or upset stomach," and "tingling sensations or numbness in parts of your body." Across the study, the average reporting of somatic symptoms at Time One was .77 and .66 at Time Two. Participants were required to respond to six of seven symptoms for inclusion.

3.2 Influences of Demographic Variables

Three different demographic variables were included into the analysis, including age, gender, and ethnicity. Given the inability to run t-tests on age as a continuous variable and

knowledge gained from previous literature about the effect of age on PTSD symptom reporting (Acierno, Ruggiero, Kilpatrick, Resnick, and Galea (2006), the demographic variable was entered into each of the regressions. Age was found to account for significant variances when examining exposure reporting influence on PTSD and all three symptom subclusters (PTSD: $t = -3.36$, $p < .01$; Increased Arousal: $t = -3.07$, $p < .01$; Reexperiencing: $t = -3.29$, $p < .01$; and Avoidance: -2.32, $p < .05$). Results of the t-tests suggest an inverse relationship between age and PTSD symptom reporting, indicating that young adults are more likely to report PTSD symptoms than older adults. No significant gender effects were found for either exposure or somatic symptoms, indicating that men and women reported both equally. For results of independent samples t-tests regarding gender, please refer to Table 2.

Ethnicity was also examined via independent sample t-tests. Given the low frequency of Hispanic, Biracial, Asian-American, and "other" ethnicities, only African-American and Caucasian ethnic groups were compared via the t-tests. For complete results of ethnicities effect on exposure and symptom reporting, please refer to Table 3. Ethnicity was found to have a significant impact on two aspects of the project. Given African-Americans on average reported lower levels of exposure then Caucasians, an ethnic effect between the two groups was found when reporting rates of exposure ($t (50) = -2.14$, $p < .05$). Please refer to Table 4 for results of independent sample t-tests for the five exposure variables. Additionally, a significant difference was found between African-American and Caucasian participants where reporting reexperiencing symptoms of PTSD ($t (50) = -2.017$, $p < .05$) with Caucasians reporting higher levels of reexperiencing symptoms than African-Americans. No differences between groups were found for avoidance or increased arousal symptoms. Given the impact of ethnicity in the

reporting of exposure and reexperiencing information, the variable was be entered as necessary in regressions of mediational tests.

3.3 Relationships Among Variables

The first step in examining the relationship among the aforementioned variables was to assess for potential correlations. Two-tailed correlations were examined amongst the following variables: exposure, PTSD and the three symptom subclusters, and somatic reporting at Time One and Time Two. For full information regarding correlations amongst variables, please refer to Table 5. Regarding important relationships amongst variables, at Time One, exposure was significantly related to PTSD symptoms ($r(56) = .558$, $p<.001$), and the three symptoms subclusters of reexperiencing ($r(56) = .471$, $p < .001$), avoidance ($r(56) = .419$, $p < .01$), and increased arousal ($r(56) = .573$, $p < .001$), as well as somatic health complaints ($r(56) = .426$, $p < .01$). However, exposure failed to correlate significantly with somatic health complaints at Time Two ($r(38) = .155$, $p > .05$).

Posttraumatic Stress Disorder symptoms at Time One was found to correlate significantly with somatic symptom reporting at Time One ($r(56) = .535$, $p < .001$). Additionally, the three symptom subclusters of PTSD as reported at Time One correlated with somatic symptom reporting at Time One: reexperiencing ($r(56) = .371$, $p < .01$), avoidance ($r(56) = .505$, $p < .001$), and increased arousal ($r(56) = .506$, $p < .001$). Finally, somatic symptom reporting at Time Two correlated significantly with PTSD symptoms at Time One ($r(38) = .369$, $p < .05$), and the reexperiencing ($r(38) = .324$, $p < .05$) and avoidance ($r(38) = .350$, $p < .05$) subclusters at Time One. Somatic symptoms at Time Two failed to correlated significantly with the increased arousal subcluster at Time One ($r(38) = .279$, $p > .05$).

3.4 Tests of Mediation

Utilizing Baron and Kenny (1986) and Holmbeck (1997) as models for analysis, the data were analyzed through multiple regression. As a significant correlation was not achieved between exposure and somatic symptom reporting at Time Two, hypotheses for mediators at Time Two (1a, 2a, 3a, and 4a) are not able to be tested. To test for mediation among the four potential mediators, four analyses were performed for each hypothesis (for a total of 16 regressions in the current project). Further, blocking variables were included; age was entered into each of the regressions and ethnicity was entered when necessary as indicated by significant difference amongst aforementioned t-tests (given a lack of significant findings amongst gender, it was not entered into any of the equations). The first step in performing the analyses was to examine the correlations between aforementioned variables (as previously reported). Step two of the analysis was to include in the regression analysis demographic information to account for variance attributable to ethnicity and age. Finally, the independent variable or the independent variable and potential mediating variable were entered into a second regression as to determine the influence of each within the equation.

In each analysis, four regressions were performed for each potential mediating variable. The first regression examined the relationship between exposure and somatic health complaints. Then a regression was performed between exposure and the potential mediating variable. Next, a regression between the potential mediating variable and somatic health complaints was conducted. Finally, a regression of exposure and the potential mediator onto somatic heath complaints was analyzed. For each of the potential mediators examined, demographic information was entered into the regression to control for their effects (per results of aforementioned t-tests) prior to the examination of the relationship of the potential mediator

(please see appropriate Tables 6, 7, 8, and 9 for specific information regarding when demographic variables were entered). Finally, given the number of mediators tested, a Bonferroni adjustment was completed to control for experiment-wise error (Sankoh, Huque, & Dubey, 1997). The adjustment procedure recommends use of critical test statistic of ($t > 2.5825$, $p < .0125$) which was used for determining significance of regressions and in post hoc power analyses. Further, the post hoc Sobel test (Sobel, 1982) was apply to each significant mediational relationship. The Sobel test in this case examined whether the mediator carried the influence of the mediator(s) from exposure to a traumatic event to somatic symptoms. By assessing the unstandardized coefficient and standard error of the relationship from exposure to the mediator and from the mediator to somatic symptoms, the test produces a z-score which recommends a critical test statistic of, $z = 1.96$, $p < .05$ to demonstrate significant influence of the mediator on the relationship between exposure and somatic symptomology.

The first potential mediator to be examined was PTSD symptomology. The first step was to perform a regression of exposure onto somatic symptom reporting which yielded a significant result, ($t (55) = 3.306$, $p < .01$). Next, exposure was regressed onto PTSD symptomology which resulted in significant findings, ($t (55) = 4.745$, $p < .001$). Third, a statistically significant relationship was found in the regression examining PTSD symptomology's effect on somatic health complaints, ($t (55) = 4.422$, $p < .001$). Finally, exposure was entered into an equation first and PTSD second, to examine PTSD as a potential mediator between exposure and somatic health complaints. In this regression, exposure was no longer found to be significant, ($t (55)= 1.343$, $p >.05$), and support was found for PTSD as a mediator, ($t (55)= 2.937$, $p < .01$). A post hoc power analysis was performed to examine for sufficient power (Faul, Erdfelder, Lang, & Buchner, 2007). Results of the power analysis

indicate use of an ($F > 3.17$, $p < .004$), which was obtained ($F = 5.999$, $p = .000$; power = .98) indicating sufficient power. A Sobel test was performed which produced a significant effect, ($z = 3.23$, $p < .01$); further indicating that PTSD is perhaps influential in the relationship between exposure and reported somatic health complaints. For changes in R squared and beta of reporting for PTSD symptoms, please refer to Table 6.

The second set of regressions examined the increased arousal subcluster of PTSD as a potential mediator. The first step was to regress exposure onto somatic symptom reporting, which yielded a significant result, ($t (55) = 3.306$, $p < .01$). Next, a regression was performed to examine exposure's influence on increased arousal symptom reporting, which produced a significant result, ($t (55)= 4.862$, $p < .001$). Third, increased arousal symptomology was regressed onto somatic symptom reporting, again yielding a significant finding, ($t (55) = 4.071$, $p < .001$). Finally, exposure was entered into the final equation first and increased arousal symptoms second, to examine increased arousal symptoms as a potential mediator between exposure and somatic health complaints. In this regression, exposure was no longer found to be significant, ($t (55)= -1.092$, $p >.05$), and support was found for increased arousal symptoms as a mediator, ($t (55)= 2.638$, $p < .05$). Again, a post hoc power analysis was performed to test for sufficient power (Faul, Erdfelder, Lang, & Buchner, 2007). Results of the power analysis indicate use of a critical test statistic of ($F > 3.17$, $p < .007$), which the current analysis does obtain ($F = 5.474$, $p = .000$; power = .96) indicating sufficient power. A Sobel test was performed and again was found to be significant, ($z = 3.11$, $p < .01$), perhaps providing further support that increased arousal is influential in the relationship between exposure and self-reported somatic complaints. R squared, changes in R squared, and beta weights from the regressions examining increased arousal symptoms are further outlined in Table 7.

A third set of regressions was performed to examine the reexperiencing subcluster of PTSD as a potential mediator. The first step was to perform a regression of exposure onto somatic symptom reporting which yielded a significant result, ($t(55) = 3.626$, $p < .01$). Second, a regression was performed examining the influence of exposure on reexperiencing, producing significantly significant results, ($t(55) = 3.602$, $p < .01$). Next, the influence of reexperiencing on somatic symptom reporting was examined through a regression, yielding a statistically significant finding, ($t(550 = 2.591$, $p < .05$). Finally, exposure was entered into an equation (first) and reexperiencing symptoms (second), to examine reexperiencing symptoms as a potential mediator between exposure and somatic health complaints. In this final regression, exposure was still found to be significant, ($t(55) = 2.370$, $p < .05$), and no support was found for reexperiencing symptoms as a mediator, ($t(550 = 1.351$, $p > .05$). No Sobel test or power analysis was performed given the lack of significance for the proposed mediator. For changes in R squared, R squired, and beta figures of reexperiencing symptoms, please refer to Table 8.

The final set of regressions was performed to examine avoidance symptoms of PTSD as a mediator between exposure and somatic symptom reporting. The first step was to perform a regression to examine exposures influence on somatic symptom reporting, yielded a significant result, ($t(55) = 3.626$, $p < .01$). Another regression was completed, examining the influence of exposure on avoidance symptom reporting, which produced a significant result, ($t(550 = 3.178$, $p < .01$). Next, avoidance symptomology was regressed onto somatic symptom reporting, which also produced a significant finding, ($t(55) = 4.030$, $p < .001$). A final regression was performed in which exposure was entered into the equation first and avoidance symptoms second, to examine avoidance symptoms as a potential mediator between exposure and somatic health complaints. In this final regression, exposure was still found to be significant, ($t(55) = 2.074$, $p <$

.05), yielding support for avoidance symptoms as a partial mediator, ($t(55) = 2.820$, $p < .01$). A post hoc power analysis was performed to assess for sufficient power (Faul, Erdfelder, Lang, & Buchner, 2007). Results of the power analysis indicate use of a critical test statistic of ($F > 3.17$, $p < .005$), which the current project does obtain ($F = 5.474$, $p = .000$; power = .98), indicating sufficient power. Finally, a Sobel test was performed which yielded a significant finding ($z = 2.50$, $p < .01$), perhaps furthering support for avoidance symptoms as a partial mediator. For R squared, changes in R squared, and beta figures of the avoidance symptoms, please refer to Table 9.

Chapter 4

Discussion

4.1 Summary of Findings

The purpose of the study was to further examine the role of PTSD symptomology in the link between exposure to traumatic events and adverse health outcomes. The project was developed to further the understanding of the aforementioned relationship through use of a unique trauma, residential fire. Secondly, the project utilized somatic complaints as an outcomes variable which had been examined only on a limited basis in the past. Finally, the current project used DSM-IV (APA, 1994) criteria in defining PTSD symptoms and the three symptom subclusters.

A number of the predictions from the project were supported through the results, while others were not. Those that were not supported included predictions of PTSD symptoms and PTSD symptom subclusters' mediational role at Time Two due to the lack of a significant correlation between exposure as reported at Time One and somatic health complaints at Time Two. This may be attributable to two issues, one, the exposure variable for the current project

does not capture secondary aspects of exposure to a residential fire such as life disruption which would be influential long-term. Secondly, though directly not tested, given PTSD symptoms did correlate significantly with somatic symptoms at Time Two and exposure (as measured at Time One) did not, what may be occurring is simply that PTSD symptoms have a much greater impact on health long-term then exposure. Further, support for the mediational role of PTSD symptomatology and symptoms of the increased arousal and avoidance subclusters between exposure and somatic symptoms at Time One were supported by the initial analyses, and the post hoc Sobel test. However, that the Sobel test is intended for use with large sample sizes and is based upon the assumption of a normal distribution of the data (Shrout & Bolger, 2002 and Sobel, 1982). Data for the current project fails to meet either of these criteria. Though the Sobel tests of mediational effects were performed and PTSD symptoms, as well as increased arousal and avoidance symptoms indeed meet the critical test statistic level, these results should be interpreted with caution.

4.2 Influences Among Demographic Variables

The effects of demographic variables were examined in the current study, including gender, ethnicity, and age. Gender was not found to have a significant effect on the reporting of any of the variables. This is more then likely due to the uneven ratio of women to men in the sample. Nonetheless, this study does lend support to the continuity of findings regarding womens' health post-trauma and is consistent with previous literature (Kimerling, Clum, & Wolfe, 2000). Ethnicity was found to be influential in two variables, exposure and symptoms of the reexperiencing subcluster of PTSD. Regarding the exposure variable, Caucasians reported greater levels of proximity to the fire and sustained injury, thus resulting in the difference between the two ethnicities on the exposure variable. However, findings of Caucasians reporting

greater levels of reexperiencing is in contrast to previous findings which show support for minority groups (including African-Americans specifically) reporting greater levels of PTSD symptomology (Garrison et al., 1995). This is likely an artifact of the relatively small sample size of the data, rather then an effect of the specific nature of the trauma or recovery of any one ethnicity.

There was an inverse relationship between age and PTSD symptom reporting, demonstrating that younger adults tend to report more symptoms then older adults. This finding is consistent with previous literature by Acierno et al. (2006) and Norris (1992) that documents heightened symptom reporting for young adults as compared to older adults. Norris (1992) offers two explanations for age effects; first, research may see a cohort effect of exposure to trauma and that young adults are at greater risk for exposure; second, traumatic memories and symptoms fade over time. However, neither of these conclusions explain the effect seen in the current sample, since each were exposed to a residential fire and assessment for all participants were around four months most trauma. However, the author goes on to describe a resistance to stress across the lifespan which may provide insight into decreased symptomology in older adults. This resistance may develop across the life span due to resiliency from previous difficult situations or traumas, it may also be due to development of more coping strategies.

4.3 Mediation Analyses: The Role of PTSD & Symptom Subclusters

The study examined the potential mediating roles of symptoms of PTSD and PTSD symptom subclusters at four and eleven months post-trauma. As discussed previously, given the lack of association between exposure and somatic complaints at eleven months post-trauma, only Time One hypotheses were examined. Four separate regression analyses were conducted to test the hypothesized mediation relationships. Posttraumatic Stress Disorder symptoms were

hypothesized to partially mediate the relationship between exposure and somatic health complaints at four months post-trauma. The results of the current project found support for this hypothesis, above and beyond the prediction of partial mediation, in that full mediation was obtained. These findings are consistent with those of Kimerling, Clum, and Wolfe (2000), who also found support for full mediation in female veterans. Results of the current project are also similar to that of Wolfe, Schnurr, Brown, and Furey (1994), which found support for partial mediation of PTSD symptoms between the relationship of exposure and negative health outcomes. These findings further support existing literature demonstrating the mediational role of PTSD symptomology in averse health outcomes post-trauma and may have important implications for clinicians and primary care physicians. If an individual is having health complications following a traumatic event, it may be important to assess for PTSD and other disorders prior to treatment. Given the role of PTSD in adverse health outcomes, drug or psychotherapy for PTSD may be instrumental in gaining positive effects to physical health treatments.

Support was found for the second hypothesis stating that symptoms of the increased arousal subcluster of PTSD would partially mediate the relationship between exposure and somatic health complaints at four months post-trauma, in that full mediation was obtained. Results further support the existing literature regarding the influence of increased arousal and extend the results of Kimerling, Clum, and Wolfe (2000) study demonstrating that increased arousal symptoms may be a specific subcluster of PTSD that mediates the relationship between exposure and somatic health complaints. Again, these findings may have important implications for clinicians and primary care physicians as to the potential biological causal factor of increase arousal symptoms and their effects on the body. Individuals seeking medical treatment

following a traumatic event may benefit from PTSD assessment and treatment (if necessary) to complement medical services in targeting etiological factors of health complications post-trauma.

The third set of regressions for the current project was completed to examine to potential mediating effect of symptoms of the reexperiencing subcluster of PTSD. No specific hypothesis was offered for this potential mediator, given that the analyses were carried out in an exploratory fashion. Results of the current study failed to show support for symptoms of the reexperiencing subcluster of PTSD as a mediator between exposure and somatic health complaints. Results of the current project do not necessarily support previous literature, but are in line with existing findings. For example, Kimerling, Clum, and Wolfe (2000) reported an inability to find significant effects for reexperiencing symptoms of PTSD. Reexperiencing symptomology, though perhaps influential in the full model consideration of PTSD, fails to contribute significant variance for somatic symptoms in the current sample of residential fire survivors.

The final proposed mediation analysis for the project intended to examine symptoms of the avoidant subcluster of PTSD as a potential mediator between exposure to a traumatic event and increased somatic health complaints. Similarly in testing the reexperiencing subcluster, no specific hypothesis was provided in analyzing avoidance symptoms. Results of the current project found support for partial mediation of avoidance symptoms in the relationship between exposure and increased somatic health complaints. Previous literature has failed to report symptoms of the avoidance subcluster of PTSD as having a specific influence, thus these results are unique. Results may point to a more important role of avoidance symptoms then previously thought. These findings may be due to the similar nature of the avoidance subcluster's symptoms to general distress and other pathologies that have an impact on health. For example,

the avoidance symptoms of anhedonia and social withdrawal, which are consistent with Major Depressive Disorder and have been shown to have a significant influence in the association between trauma exposure and self-reported health (Clum, Calhoun, & Kimerling, 2000).

4.4 Potential Benefits of the Study

Exposure to a traumatic event has been shown to have an adverse impact on health (Elder, Shanahan, & Clipp, 1997; Ullman & Siegel, 1996). Posttraumatic Stress Disorder has been linked to aversive health outcomes in a number of different traumatized populations (Mohr et al., 2003; Schnurr & Jankowski, 1999). The present study may help to bring light to previous trauma exposure studies. Perhaps, PTSD symptoms have a major influence in the reduction of immune function and increased risks of myocardial infarctions (Carlson, 2002; Ooi et al., 1994) which have be documented post-trauma, as it seems reasonable that the somatic health complaints reported in the current project may be a results of suppressed immune system functioning (Rozanski & Kubzansky, 2005). Posttraumatic Stress Disorder symptoms may also have been influencing the reported relationship between trauma exposure and health such as those found by Elder et al (1997) and Escobar (1992). Finally, again though not directly tested, some of the somatic complaints reported, such as trouble catching one's breath, may be linked to previous findings supporting decreased cardiovascular health (Cohen et al., 1998).

The current project additionally adds to the growing body of literature supporting PTSD symptomology as a primary pathway in which health is impacted post-trauma (Friedman & Schnurr, 1995; Kimerling, Clum, & Wolfe, 2000; Wolfe, Schnurr, Brown, & Furey, 1994). The project furthers the literature in a number of ways. First, it demonstrates that the mediating role of symptoms of PTSD is found with DSM-IV (APA, 1994) criteria as well as DSM-III (APA, 1987) criteria. This may help to provide insight into the reason for finding partial mediation of

the avoidance symptomology with DSM-IV criteria. Given DSM-III criteria used four subclusters of reexperiencing, avoidance, numbing, and hyperarousal, DSM-IV criteria are different, as numbing is part of the avoidance subcluster. This inclusion of numbing in the avoidance subcluster may provide additional power to the avoidance subcluster for the current project. A second strength of the current study is that it provides further support for the importance of examining symptom subclusters. It helps to strengthen the growing body of literature that implicates the increased arousal symptom subcluster as instrumental in exacerbating aversive health outcomes. It also brings light to the idea that perhaps the avoidance symptom subcluster of PTSD may be more influential in this relationship then previously anticipated. Third, the project adds to previous findings that somatic health complaints are not only associated with exposure post-trauma, but that they are also mediated by PTSD symptomology.

4.5 Limitations of the Current Project

The current project has a number of limitations that are important to discuss. First, the study is limited by its relatively small sample size. Though previous studies have found similar effects with similar sample sizes (Kimberling, Clum, & Wolfe, 2000), the effects of the current study may have been strengthened by a larger sample. The small sample size may have prevented the project from carrying out analyses for Time Two hypotheses. This dilemma is not uncommon, as 72% of disaster studies report assessment only once after the disaster (Norris & Elrod, 2006). A larger sample may also have increased the likelihood of finding differences amongst gender and races, as well as had power implications for the mediation regressions.

The study is also limited by the difficulty of defining and measuring the exposure variable, particularly in the case of residential fires. The study could benefit from a further

developed and refined measure of exposure that holistically captures the key elements of exposure to residential fires. The instrument may assess similar factors (in a greater depth) such as those used in this study, including proximity, life-threat, concern for family, sustained injury, and resource loss. Similarly, the current study is limited by reliance on self-reports of heath complaints. Pain tolerance and distress from symptoms is something that is highly personal and varies between all individuals affected (Wise, Price, Meyers, Heft, & Robinson, 2002). Alternative modes of data collection for health (including medical records and physiological measurement) may provide more unbiased insight into health concerns post-trauma.

One of the most significant limitations of the study is that it lacks a mechanistic approach to understanding why PTSD produces adverse health outcomes. Though Schnurr and Green (2004c) make a strong argument for PTSD being the primary pathway through which trauma leads to negative health outcomes, the current study does not explain any biological factors that may aid in the production of negative health outcomes. That is, the current project does not provide insight into biological abnormalities or dysregulation associated with PTSD.

4.6 Future Directions of Research

Schnurr and Green (2004c) developed a theoretical model to explain health outcomes following a traumatic event. The model proposes that PTSD is the primary pathway for which trauma leads to negative health outcomes. It is important to note, however, that PTSD does not fully mediate the relationship between trauma exposure and health outcomes. The model lists eight factors: trauma exposure, PTSD, biological alterations, psychological alterations, attentional processes, health risk behaviors, illness behaviors, and morbidity & mortality. Please see Figure 1 for an explanation of the model of trauma exposure and health outcomes.

For the purpose of the current project, four key aspects of the model are worth mentioning. The first variable which initiates the process is trauma exposure which the authors described as simply witnessing or participating in a traumatic event, regardless of the proximity or involvement. Trauma exposure is hypothesized to directly affect PTSD, which is characterized by a full diagnosis (not any one specific sub-cluster). PTSD then influences biological alterations including such factors as activation of the HPA axis, as well as noradrenergic and immune functioning. The biological alterations lead to the outcome variable of morbidity and mortality including physical health symptoms, official medical diagnoses, and fatalities (Schnurr & Green, 2004c). The model is relevant in that, to date, it provides the most holistic approach to explaining health outcomes post-trauma. After all, the somatic symptoms that were reported in the current project may be a result of these biological abnormalities such as increases in cortisol levels and a suppression of the immune system as a result of PTSD symptomology. Further, other aspects of the model may certainly have an influence on somatic symptom reporting, including attentional processes paid to symptoms and health risk behaviors what may change following the traumatic event (i.e. substance abuse, exercise routines, ect.).

Future steps that need to be taken in reference to this project include first obtaining a larger sample size. Additionally, future efforts could work toward the development of a standardized assessment instrument of exposure to better grasp the important processes and contextual factors that occur during a residential fire. Third, further research may benefit from the utilization of a more objective measure of health outcomes (such as medical record examination or physiological measurements). Finally, future studies could also explore Schnurr and Green (2004c) model discussed above in more depth. More specifically, the examination of the four primary variables (exposure, PTSD, biological abnormalities, and morbidity), followed

by exploration of additional variables of the model to gain a holistic understanding of all factors influencing adverse health outcomes post-trauma, may be in order.

Given the physiological activation/deactivation of survivors both during and following a traumatic event, it is then important to understand the biological alterations the body sustains following such events. Yehuda and McFarlane (1997) and Friedman and McEwen (2004) reviewed the literature regarding biological abnormalities associated with PTSD. The reviews note several aversive alterations, particularly to the HPA axis system, resulting in increases in the corticotropin releasing factor, alterations in cortisol levels, and an increase in glucocorticoid receptors (influencing the regulation of cortisol). The studies also cite influences on the sympathetic nervous system, and a number of PTSD increased arousal symptoms, including increased startle responses and disturbed sleep. When examining the influence of PTSD on the relationship between trauma exposure and health outcomes, researchers should carefully examine these biological underpinnings of PTSD biological abnormalities, particularly within the context of the aforementioned theoretical model. Results from the current project (though directly not studied) suggest that the documented biological abnormalities of PTSD may be leading to adverse somatic health complaints. More specifically, perhaps symptoms of the increased arousal subcluster are leading to the physiological alterations/changes, thus leading to the outcome of the reported health complaints. There is no doubt the need for future examination into the health effects of PTSD.

Despite the limitations of the current project, the study still offers evidence in support of PTSD symptoms serving as a mediator between exposure to traumatic events and adverse health outcomes. A strength of the current study is its utilization of residential fire survivors and DSM-IV (APA, 1994) criterion. Despite the small sample size and reliance on self-report measures,

the study still offers support for the role of PTSD symptomology as a primary pathway in health outcomes. These findings reaffirm the greater role of PTSD serving as a mediator between exposure to a traumatic event and negative health outcomes.

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Table 1.

Measures Range, Means, and Standard Deviations

	Range	Mean	Standard Deviation
Exposure Variable			
Proximity to the fire N=54	1.0-4.0	2.72	1.30
Sustained Injury N=54	1.0-4.0	1.24	.61
Life Threat N=56	1.0-4.0	1.52	.97
Concern for Family member N=56	1.0-4.0	2.44	1.35
Total Loss N=40	2.0-103.0	50.10	24.89
Exposure Total (z-score)	-.98-1.51	-.03	.57
Anxiety Disorder Interview Schedule: Posttraumatic Stress Disorder (averaged across symptoms)			
PTSD Symptoms N=56	0-6.29	2.06	1.60
Reexperiencing Symptoms N=56	0-8.0	2.38	1.90
Avoidance Symptoms N=56	0-5.71	1.41	1.63
Increased Arousal Symptoms N=56	0-7.80	2.66	2.08
Brief Symptom Inventory: Somatic Health Complaints (averaged across symptoms)			
Somatic Complaints Time One N=56	0-3.00	.77	.83
Somatic Complaints Time Two N=38	0-2.86	.66	.71

Table 2.

Comparison of Means and Standard Deviations of the Effects of Gender on Exposure, PTSD symptoms, and Somatic Health Complaints

Measure	Gender	*M*	*SD*	*N*	*t*	*df*	*Significance (Two-tailed)*
Exposure							
	Men	.10	.61	8	-.72	54	.477
	Women	-.06	.57	48			
Anxiety Disorder Interview Schedule							
Total PTSD							
	Men	1.68	2.10	8	.72	54	.474
	Women	2.13	1.52	48			
Avoidance							
	Men	1.07	1.71	8	.64	54	.562
	Women	1.47	1.62	48			
Increased Arousal							
	Men	2.15	2.73	8	.77	54	.457
	Women	2.75	1.97	48			
Reexperiencing							
	Men	2.08	2.33	8	.48	54	.634
	Women	2.43	1.84	48			
Brief Symptom Inventory							
	Men	.70	.91	8	.285	54	.777
	Women	.79	.82	48			

Table 3.

Comparison of Means and Standard Deviations of the Effects of Ethnicity on Exposure, PTSD symptoms, and Somatic Health Complaints

Measure	Ethnicity	*M*	*SD*	*N*	*t*	*df*	*Significance (Two-tailed)*
Exposure							
	African-American	-.21	.50	23	-2.14	50	.037
	Caucasian	.13	.61	29			
Anxiety Disorder Interview Schedule							
Total PTSD							
	African-American	1.72	1.23	23	-1.67	50	.100
	Caucasian	2.45	1.83	29			
Avoidance							
	African-American	1.27	1.53	23	-.963	50	.340
	Caucasian	1.71	1.73	29			
Increased Arousal							
	African-American	2.19	1.70	23	-1.49	50	.141
	Caucasian	3.06	2.35	29			
Reexperiencing							
	African-American	1.87	1.32	23	-2.02	50	.049
	Caucasian	2.92	2.19	29			
Brief Symptom Inventory							
	African-American	.66	.79	23	-1.32	50	1.94
	Caucasian	.96	.93	29			

Note: Only the ethnicities of African-American and Caucasian were analyzed because of the low response rate of other ethnicities (n=1 for each).

Table 4.

Comparison of Means and Standard Deviations of the Effects of Ethnicity Factors Included in the Exposure Variable

Factor	Ethnicity	*M*	*SD*	*N*	*t*	*df*	*Significance (Two-tailed)*
Proximity to Fire							
	African-American	2.22	1.31	23	-2.53	48	.015
	Caucasian	3.11	1.19	27			
Injury Sustained							
	African-American	1.04	.21	23	-2.33	48	.024
	Caucasian	1.44	.80	27			
Life Threat							
	African-American	1.39	.94	23	-.819	50	.417
	Caucasian	1.62	1.05	29			
Concern for family member							
	African-American	2.39	1.27	23	-.422	50	.675
	Caucasian	3.55	1.42	29			
Total Loss							
	African-American	51.15	22.53	13	.126	36	.900
	Caucasian	50.08	25.91	25			

Note: Only the ethnicities of African-American and Caucasian were analyzed because of the low response rate of other ethnicities (n=1 for each).

Table 5.

Reporting of Correlations for One-tailed Pearson's Correlations amongst Variables

	1.	2.	3.	4.	5.	6.	7.
1. Exposure	1						
2. PTSD Symptomology	.558**	1					
3. Reexperiencing Subcluster	.471**	.813**	1				
4. Avoidance Subcluster	.419**	.887**	.546**	1			
5. Increased Arousal Subcluster	.573**	.901**	.615**	.727**	1		
6. Somatic Symptoms at Time 1	.426**	.535**	.371**	.505**	.506**	1	
7. Somatic Symptoms at Time 2	.155	.369*	.324*	.350*	.279	.468**	1

Note: N=56, except for Time 2 Somatic Symptom reporting where N=38.
* indicates correlation significant at the .05 level (2-tailed).
** indicates correlation significant at the .01 level (2-tailed).

Table 6.

Analysis of PTSD Symptoms as a Mediator Four months Post-Trauma

Steps	Predictors	ΔR^2	*p*	R^2	β	*p*
Step 1	Demographics Exposure to Somatic Health Complaints	.167	.002	.205	.599	.002
Step 2	Demographics Exposure to PTSD	.249	.000	.425	1.413	.000
Step 3	Demographics PTSD to Somatic Health Complaints	.262	.000	.290	.292	.000
Step 4	Demographics 1) Exposure and 2) PTSD to Somatic Health Complaints	.282	.000	.320	.272 .231	.185 .005

Note: Age was entered into each equation and due to results of aforementioned t-tests, ethnicity was entered into regressions for steps 1, 2, and 4.

Table 7.

Analysis of the Increased Arousal Subcluster of PTSD as a Mediator at Four months Post-Trauma

Steps	Predictors	ΔR^2	*P*	R^2	β	*p*
Step 1	Demographics Exposure to Somatic Health Complaints	.167	.002	.205	.599	.002
Step 2	Demographics Exposure to Increased Arousal	.264	.000	.420	1.888	.000
Step 3	Demographics Increased Arousal to Somatic Health Complaints	.230	.000	.257	.208	.000
Step 4	Demographics 1) Exposure and 2) Increased Arousal to Somatic Health Complaints	.263	.000	.300	.294 .162	.162 .011

Note: Age was entered into each equation and due to results of aforementioned t-tests, ethnicity was entered into regressions for steps 1, 2, and 4.

Table 8.

Analysis of the Reexperiencing Subcluster of PTSD as a Potential Mediator Four months Post-Trauma

Steps	Predictors	ΔR^2	*P*	R^2	β	*p*
Step 1	Demographics Exposure to Somatic Health Complaints	.167	.002	.205	.599	.002
Step 2	Demographics Exposure to Reexperiencing	.167	.001	.338	1.377	.001
Step 3	Demographics Reexperiencing to Somatic Health Complaints	.110	.012	.384	.158	.012
Step 4	Demographics 1) Exposure and 2) Reexperiencing to Somatic Health Complaints	.195	.003	.232	.477 .089	.022 .183

Note: Age was entered into each equation and due to results of aforementioned t-tests, ethnicity was entered into regressions for steps 1, 2, and 4.

Table 9.

Analysis of the Avoidance Subcluster of PTSD as a Partial Mediator Four months Post-Trauma

Steps	Predictors	ΔR^2	*P*	R^2	β	*p*
Step 1	Demographics Exposure to Somatic Health Complaints	.167	.002	.205	.599	.002
Step 2	Demographics Exposure to Avoidance	.146	.002	.250	1.099	.002
Step 3	Demographics Avoidance to Somatic Health Complaints	.228	.000	.256	.254	.000
Step 4	Demographics 1) Exposure and 2) Avoidance to Somatic Health Complaints	.277	.000	.315	.385 .195	.043 .006

Note: Age was entered into each equation and due to results of aforementioned t-tests, ethnicity was entered into regressions for steps 1, 2, and 4.

Figure 1.

Model of health following a traumatic event

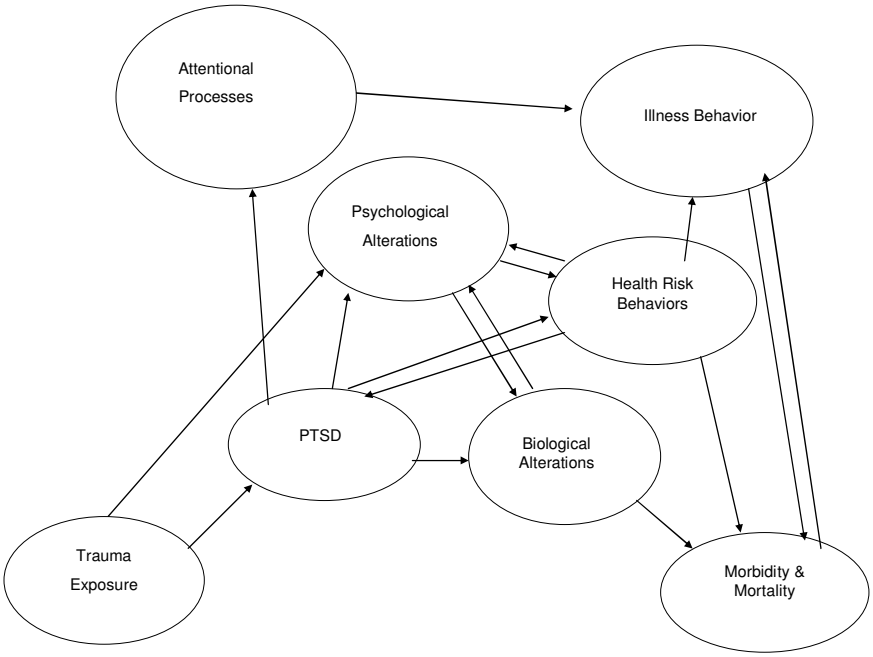

Figure 1. A proposed model of health following exposure to a traumatic event. The influence of exposure, PTSD, attentional processes, psychological alterations, biological alterations, health risk behaviors, illness behavior, on morbidity and mortality.

From "Understanding relationships among trauma, posttraumatic stress disorder, and health outcomes," by P. P. Schnurr and B. L. Green, 2004, In P. P. Schnurr & B. L. Green (Eds.), Trauma and health: Physical health consequences of exposure to extreme stress. (pp.247-275). Washington DC: American Psychological Association.