SUBTYPES OF DISORDER COURSE IN VIETNAM VETERANS WITH WAR-RELATED POSTTRAUMATIC STRESS DISORDER

BY

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A dissertation submitted to the Graduate Faculty in the

Doctoral Subprogram of Clinical Psychology,

in partial fulfillment of the requirements for the degree of Doctor of Philosophy,

The Graduate Center of City University of New York

July 2010

This manuscript has been read and accepted by the

Graduate Faculty in Clinical Psychology in satisfaction of the dissertation requirement

for the degree of Doctor of Philosophy.

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Abstract

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Despite that war-related posttraumatic stress disorder is known to persist for years in many afflicted individuals, few studies have examined the changes in symptoms that occur over the disorder's course. A review of relevant publications found evidence for a variety of trajectories of symptom change including gradual diminution of symptoms over time, persistence of or increases in symptoms over time, and intermittent exacerbations of symptoms punctuated by temporary symptomatic abatements. Theoretical models also suggest the disorder may have a varied course. In this dissertation, the continuous course of war-related PTSD was examined retrospectively in 84 Vietnam veterans by analyzing graphs of symptom change over more than a decade. The goals of this study were to classify and describe different disorder course profiles; to investigate the relationship between course profile and other PTSD outcomes; and to examine the relationships between PTSD course profiles and pre-war, war-zone, and post-war risk factors for PTSD. Results: Three subtypes of PTSD course were identified and these were significantly associated with PTSD outcomes. No associations were found between pre-war risk factors and PTSD course profile. Course profile was significantly associated with one exposure variable— receipt of a purple heart-- and trends were identified with other, though not all, exposure variables. Course profile was also related to co-morbid psychiatric disorders beginning during or after the war, and self-reported use of mental health services. Lastly, course profile was found to moderate the relationship between combat severity and persistence of PTSD between one and two decades after military service. Implications and areas for further research are discussed.

Acknowledgements

Support for this dissertation was provided by the B. Altman Foundation Dissertation Fellowship granted through the Graduate Center of the City University of New York, and by a grant from the FAR Fund provided through the Doctoral Subprogram in Clinical Psychology at The City College of New York.

This dissertation was made possible by the support and mentorship of Dr. Bruce P. Dohrenwend who extended to me the use of data from the National Veterans Readjustment Study and resources at the Department of Social Psychiatry and Columbia University Medical Center. Working with such an accomplished and knowledgeable expert on stress, adversity, and psychopathology has been inspiring and instrumental to my development as a researcher.

Dr. Steven Tuber has been a stalwart advocate of both my clinical and research endeavors, and his faith in my potential as a student and as a psychologist has been palpable at the most critical of times. Steve's reliable presence, his comfort with my traversing varied terrains to arrive at my own professional identity, his confidence in my ideas, and his profound influence on my clinical work will continue to influence me throughout my career, for which I am both fortunate and extremely grateful.

I would like to acknowledge Ms. Jacqueline (Yvonne) Williams Mose whose attention to all the important details of this process was instrumental in moving me towards completion of my degree. Thanks also to Drs. Margaret Rosario, Venezia Michalsen, Tom Yager, Sarai Batchelder, and Nathan Hansen for the time, technical support, teaching, and help in developing my ideas that each provided me at separate

times over the course of my graduate career. Dr. Rosario provided essential feedback at points along the way and I am very grateful for this, her honesty, and her sense of humor.

I have received exceptional support from my colleagues, supervisors, and directors of the fellowship at Counseling and Psychological Services at Columbia University. Drs. Janice Bennett and Richard Eichler have been extraordinarily understanding and flexible while providing superlative training and warm mentorship. Thanks to Drs. Alice Shepard and Maia Miller for the examples you have been for me, and for your advice, encouragement, and friendship. I want to thank my loved ones and close friends for being with me, for accepting my means of connection, and for the constant reminders of what not to lose sight of. Stephen, Anna, Suzy, and Candace: thank you each for the influence you have had in who I have and will become. I love you guys.

To my parents, Lynda Tyrrell and Brian West, for your unflagging love and support, for all your time and energies, and for remaining ever willing to learn from my experiences as I've learned from them myself. To my partner in many marathons, Mum, thanks for showing me how to always keep going, being there to make sure I did, and for engaging critically to strengthen and shape my perspectives. To Dad, thanks for your even presence, for reminding me to take it all in stride, and for constantly managing to provide thoughtful, unselfish consideration of multiple sides of any situation. Lastly, I thank my husband, co-pilot, and best friend Elio, for patiently embracing this journey, for being a source of nuance, optimism, and inspiration, and for making this a truly joint success. In memory of Cindy Grabarek, with whom I first hatched a plan to pursue a PhD in clinical psychology, and of my dearest Tadhg, who managed always to dislodge me from thoughts and obligations and bring me back to the sweetness of now.

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Chapter I

The term *trauma* comprises both the experience of being hurt by a noxious agent as well as the physical and/ or psychological response to that hurt. These two aspects of trauma are also reflected in the criteria for Posttraumatic Stress Disorder (PTSD): It is the only psychopathological diagnosis whose criteria require the presence of an etiological traumatic event in addition to a series of specific symptoms. As such, this disorder captures the crossover of life events and the subjective experience of these events.

In the U.S. there is a lifetime prevalence rate of 7.8% for PTSD in the general population, despite that more than half of all adults are exposed to traumatic stress as defined in the DSM-III-R, and measured by a checklist of stressful events, (American Psychiatric Association, 1987; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Military samples have higher rates of exposure to trauma than people from non-military samples, though it depends on the particular demands of deployment (Dohrenwend, Turner, Turse, Adams, Koenen, & Marshall, 2006; Hoge, Castro, Messer, McGurk, Coting, & Koffman, 2004; Kang, Natelson, Mahan, Lee, & Murphy, 2003), and there are significantly higher rates of PTSD onset and persistence in people exposed to war-related traumas (Blank, 1993; Brewin, Andrews, & Valentine, 2000a; Dirkzwager, Bramsen, & van der Ploeg, 2001; Kulka, Schlenger, Fairbank, Hough, Jordan, Marmar, & Weiss, 1990) than in those exposed to other traumas. Dohrenwend and colleagues (2006) found that even though the majority of Vietnam veterans studied did not develop war-related PTSD, between 18.7% and 22.5% developed war-related PTSD, and between 9.1% and 12.2% continued to have PTSD over a decade after the war. Some of the highest rates of lifetime onset of PTSD occur in samples of prisoners of war, many of whom suffered

severe abuse and deprivation for months or even years. In studies of ex-prisoners of war from WWII carried out several decades after the war, Kluznik and colleagues (Kluznik, Speed, Van Valkenburg, & Magraw, 1986) found a 67% rate of lifetime onset of PTSD, of whom less than one third had fully recovered at the time of the study. Similarly, Zeiss and Dickman (1989) found that 55.7% of their sample of 442 ex-prisoners of war described symptoms "consistent with a clinical diagnosis of PTSD" (p. 83).

A syndrome of re-experiencing, numbing, avoiding, and hyperarousal symptoms, PTSD was not officially recognized as a diagnostic category until 1980 (American Psychiatric Association, 1980), despite that links between traumatic experience and psychological reactions had been made as far back as the mid 19th Century. The APA classification was based in large part upon the work of Abram Kardiner, an analysand of Freud's, who compiled and published years of his clinical observations of U.S. WWI veterans in *The Traumatic Neuroses of War* (Kardiner, 1941). He noted that people with traumatic neuroses experienced an altered conception of themselves in relation to the world due to their focus on the trauma, a capacity for traumatic memories to be triggered by an array of experiences, chronic irritability, an inclination towards withdrawal, detachment, and aggressive reactions, and enduring vigilance to threats in the environment—manifested in conditioned biological responses (van der Kolk, 2007). Before Kardiner, however, generations of European psychiatrists including Jean-Martin Charcot and Pierre Janet had described relationships between trauma, intolerable memories, dissociative states, and the relegation of unthinkable experiences to sensory memories that replayed in the minds of the traumatized (van der Kolk, 2007).

Theories about the pernicious effects of traumatic events have long been met by vociferous arguments against rooting stress reactions in trauma: When French psychiatrist, Briquet, described an association between sexual trauma and psychological reactions, critics blamed false memories rather than actual events; later theorists argued similarly that psychological reactions were rooted in "simulation" and suggestibility rather than the traumatic experience (van der Kolk, 2007). The backlash against attributing the cause of some psychological problems to traumatic events had profound implications for French and German soldiers with stress reactions during World War I who were administered painful treatments and rigorous physiological exercises in efforts to restore what was understood as their failed willpower (van der Kolk, 2007).

In the 20th Century, as it became increasingly clear that traumatic events were undeniably related to human response, debates shifted from disputes over the validity of claims that trauma created stress reactions, to disputes over whether resultant psychopathology was due to the severity of the stressor or to a preexisting vulnerability in the traumatized individual. This controversy has centered on the question of whether the syndrome represents a normal, albeit pathological, response to abnormally stressful situations (e.g. Horowitz, 1978; Herman, 1992; Green, Wilson, & Lindy, 1985), or whether it is better understood as a significant departure from normal stress responses (e.g. LeardMann, Smith, Smith, Wells, & Ryan, 2009; Yehuda & McFarlane, 1995).

Life events literature and studies that measure the effects of stressful events draw attention to the *dose-response relationship*, or the relationship between the severity of the trauma and later PTSD (e.g. Dohrenwend et al., 2006). The dose-response relationship is corroborated by numerous studies on combat veterans that prove that the frequency and

intensity of combat exposure is strongly associated with the risk of developing PTSD and with the severity and chronicity of the PTSD that develops (Green, Grace, Lindy, Gleser, & Leonard, 1990a; Green, Grace, Lindy, & Gleser, 1990b; Hoge et al., 2004; King, King, Foy, Keane, & Fairbank, 1999; King, King, Gudanowski, & Vreven, 1995; Smith et al., 2008; Yehuda, Southwick, & Giller, 1992), and in the most severe traumatic experiences, PTSD prevalence can increase well above 50%, thereby becoming the norm rather than the exception (Dohrenwend et al., in preparation; Kluznik et al., 1986; Speed, Engdahl, Schwartz, & Eberly, 1989; Zeiss and Dickman, 1989). That an increase in trauma severity is directly related to increased risk of PTSD strengthens the argument that the disorder may in fact be a normative, albeit severe, response to abnormal situations.

Those who argue that the PTSD is *not* a normative response cite discrepancies between rates of traumatic exposure and rates of PTSD prevalence to argue that most people exposed to traumatic stress do *not* develop persistent PTSD (e.g. Yehuda & McFarlane, 1995). They also cite findings on pre-military risk factors and vulnerabilities that predispose the individual to developing a psychiatric disorder in the face of adversity (e.g. McNally & Shin, 1995) and on distinct, non-normative biological changes in individuals with PTSD that they claim are associated with the diagnosis rather than the trauma per se (Pitman et al., 2006; Yehuda & McFarlane, 1995). These researchers emphasize that trauma alone does not cause psychopathology without the influence of genetic and family risk factors, personality factors, and pre-morbid psychiatric problems (Yehuda & McFarlane, 1995). Furthermore, the high rates of psychiatric illnesses comorbid with PTSD have been argued to reflect shared biological vulnerability to

mental illness, implying that PTSD is in fact secondary to preexisting psychiatric conditions (e.g. Koenen et al., 2003a).

Research on the precursors to PTSD has focused on factors related to the traumatic event, known as *exposure variables*, and factors related to the individual, known as *vulnerability factors.* Usually, these factors are assessed in relation to dichotomous PTSD outcomes including onset of PTSD (e.g. did the individual ever meet criteria for war-related PTSD?) or the persistence of PTSD after some distinct period of time (e.g. does the individual continue to meet criteria for war-related PTSD now?). Since PTSD usually persists for longer than one year (Breslau & Davis, 1992; Kessler et al., 1995), and since research in the general population suggests more than one third of individuals with PTSD do not fully remit after many years (Kessler et al., 1995), studies that examine dichotomous outcomes based on diagnostic criteria for the presence or absence of the disorder at a given point misses the opportunity to consider the course of the disorder. Despite that epidemiological studies have demonstrated that PTSD is a disorder with which individuals must continue to live over months, years, and even decades, very few studies have focused on how the disorder develops and changes over time. A comprehensive review of the literature on war-related PTSD found no studies assessing the disorder's course continuously over time.

Overview of the Study

In this dissertation I will examine the course of war-related PTSD. The participants in this study are from a sample of Vietnam War veterans to whom doctoral level clinicians administered the Structured Clinical Interview for DSM-III Disorders-

Non Patient Version (Spitzer, Williams, & Gibbon, 1987) almost 2 decades after they returned from service. Veterans were asked to describe their symptoms at the worst period of the disorder as well as at the time of the interview, and clinicians rated the severity of symptoms (low, moderate, severe) at both points, thereby assessing whether they met criteria for war-related PTSD at any time since their service (PTSD onset) and within 6 months of the time of the interview (current PTSD). All participants in this sample met diagnostic criteria for war-related PTSD at some point between their service and the clinical interview (PTSD onset). Additionally, veterans were asked to describe how their PTSD symptoms developed and changed over time, and the clinicians produced graphical drawings of the course of PTSD, as per the veterans' descriptions.

Central to this dissertation is the belief that it would be helpful to understand *more* about how PTSD symptoms change over time, in order to examine whether symptom change can be classified into different subtypes of PTSD. This dissertation seeks a) to provide a descriptive analysis of the continuous course of PTSD in Vietnam veterans as they described it retrospectively over a decade after the war, and b) to inquire whether there are different, identifiable subtypes of PTSD course that can be reliably classified, c) to examine whether different courses are associated with specific PTSD outcomes, d) to test whether known risk factors for PTSD outcomes are also risk factors for subtypes of disorder course, and e) examine whether course is related to aspects of the individual's life that coincide with the disorder's progression, such as comorbid disorder, post-war stressful events, and mental health treatment.

I propose that variation in subtypes of PTSD may relate to differences in the function of symptoms, such that in some cases PTSD symptoms progress to relative

health whereas in other cases PTSD symptoms progress to PTSD persistence. Although this data set does not allow for examinations of what functions symptoms serve, I will examine graphical models of the disorder's course in Vietnam veterans and ask whether some kinds of disorder course represent relatively good adaptation to the war zone experiences, learning to regulate their physiological and emotional responses and integrating their memories, whereas other kinds of disorder course signal ongoing, unpredictable, maladaptive symptoms that leave the individual prone to either persistent internal distress, or unpredictable fluctuations in symptom level.

Significance of the Study

PTSD can persist for decades, often with crippling effects (Dirkzwager et al., 2001; Kluznik et al., 1986; Speed et al., 1989; Zeiss & Dickman, 1989), including work impairment, impaired social relations, marital instability, and increased use of outpatient care (Davidson, Stein, Shalev, & Yehuda, 2004). Perhaps partially because of these primary impairments, people with PTSD, especially chronic PTSD, are at significantly greater risk of developing substance use disorders and Major Depressive Disorder compared to the general population (Breslau, 2002; Davidson et al., 2004) as well as compared to trauma survivors who do not develop PTSD (Breslau, 2002).

The high rates of chronic PTSD in military samples are concerning for a variety of additional reasons. In military populations, PTSD is particularly likely to be associated with comorbid disorders including depression, substance abuse, and other anxiety disorders (Blank, 1993; Bremner, Southwick, Darnell, &Charney, 1996; Davidson et al., 2004; Dirkzwager et al., 2001; Kulka et al., 1990); veterans with PTSD are heavy service

users (Beckham et al., 1996); and treatment-seeking veterans with PTSD have been found to have mortality rates that are significantly higher than those for the general population (Johnson, Fontana, Lubin, Corn, & Rosenheck, 2004). Veterans with PTSD also experience a variety of chronic impairments in functioning such as unemployment, income disparities, inability to maintain stable marriages, and lower levels of college education after service amongst those veterans who had not graduated college prior to their service (Dohrenwend, Turner, Turse, Adams, Koenen, & Marshall, 2007). For all of these reasons, it is essential to understand what factors lead to better and worse PTSD outcomes, and to shed light on the varieties of disorder course that may be related to these outcomes and may be elucidate more clearly what it is like for individuals who struggle with this disorder.

This study is also important because it involves a kind of information about PTSD course that has not been previously examined, namely clinician-drawn graphs of the phenomenology of posttraumatic stress disorder course described by veterans. A descriptive analysis of various aspects of disorder course (including time between exposure and onset of symptoms, time between onset and the worst period, length of worst period, frequency of increasing, decreasing, and stable periods of symptoms, etc.) will demonstrate which features of PTSD course are most common and which are rarities. As of yet, these aspects of disorder course have not been described in a comprehensive way with empirical evidence as to their incidence. Beyond demonstrating *what* level of symptoms the disorder had progressed to by the time of interview, the graphs display *how* the symptoms progressed to that point by depicting shifts in symptoms over periods not assessed by conventional two-point data including the period between wartime service

and the worst period of symptoms, and the period between the worst period of symptoms and the interview, where these two periods did not coincide. Furthermore, the only longitudinal studies on disorder course have focused on periods of 1 to 3 years, whereas this data provides information on changes in symptoms over a much longer period.

Probably the major reason that very little research has focused on patterns of PTSD course is due to issues of assessment. Many of the early studies on PTSD course have had retrospective, cross-sectional designs. Veterans' symptoms were assessed at one point, and conclusions were made about the period since exposure to wartime trauma. Other studies involved multiple (usually two) assessments of veterans with PTSD. When comparing ratings of PTSD at various points, it is tempting to make assumptions a) about the course of the disorder between assessments, and b) about whether the change observed between data points is representative of the trend of the disorder course as a whole. Yet data collected at one or two points of time provide no information to substantiate either of these assumptions. It is possible that any number of disorder trajectories unfolded between the time at which the veteran was exposed to wartime trauma and the time of assessment, or between two points of assessment.

Furthermore, almost all of the studies on PTSD course consist of assessments of diagnostic status at different points in time. Very few studies inquire about changes in symptoms in the intervening periods, and most rely on self-reported checklist cut-off scores to indicate that the participant *likely* meets criteria for PTSD diagnosis. These kinds of assessments are less reliable than diagnostic interviews made by experienced clinicians. This study is important because all participants were assessed for a range of diagnoses in an extensive clinical interview with trained providers.

Although they are retrospective in nature and based on self-reports, the graphical representations of over ten years of disorder course allow for a comparison of types of disorder course and the relationship between these subtypes and outcomes, as well as assessments of the association between course subtypes and vulnerability and exposure factors. In a review of the literature on war-related PTSD course, no other study of war-related PTSD was found in which symptoms were either assessed continuously over time. Many of the longitudinal studies reviewed involved multiple assessments over a relatively brief period, usually about one year after traumatic exposure. Given that over half of the people that develop PTSD continue to have symptoms after one year, and the median duration of PTSD in the general population varies from 36 to 64 months (Kessler et al., 1995) but is often longer for veterans, there is great need for a thorough study of the disorder's course over multiple years following traumatic exposure.

I expect that a descriptive analysis of common and uncommon features of war-related PTSD course will illuminate the *process* of posttraumatic stress disorder by informing about the subjective experience of symptoms for those who suffer with and improve from PTSD in contrast to those who suffer without improvements. Since course is inherently related to outcome, it is likely that particular aspects of the disorder course (e.g. delayed onset of symptoms relative to traumatic exposure; early, severe worst period or late, extended worst periods; frequent improvements followed by symptom exacerbation) coincide with particular outcomes over time (e.g. more severe symptoms at the time of clinical interview), or it may be that there are certain, recognizable stages in the disorder's course that indicate a gradual progression towards health and will generate insight into the underlying mechanisms of prolonged responses to traumatic stimuli. Such

information will hopefully shift the focus of research and clinical work from questions of whether individuals continue to meet diagnostic criteria, to a focus on the specifics of individuals' experience of the disorder. It will also aid in the scientific understanding of the disorder as it unfolds and will help clinicians identify signs of PTSD improvement and stagnation, and in tailoring appropriate interventions for those suffering with PTSD and for their related friends and family members.

CHAPTER 2: Literature Review

Sacrificing a portion of your
consciousness so you won't have
to deal with
Being there
and
building mental blocks
so you won't have to deal with
having been there.[1]

Risk Factors Associated with War-Related Post-traumatic Stress Disorder

The majority of research on PTSD has focused on risk factors for developing the disorder (for an extensive discussion on risk factors see Dietrich, 2001). Risk and protective factors can be categorized into pre-trauma factors (e.g. age at the time of exposure, education, ethnicity, etc.); factors specific to the traumatic event (e.g. the kind of trauma experienced, the duration of the traumatic experience, threat to life, or specific aspects such as grotesque death or the individual's involvement in harming others, etc.); or post-trauma factors (e.g. social support in the aftermath of the trauma, PTSD treatment, etc). In this section I will review current knowledge on factors shown to influence PTSD onset, chronicity, and severity, with particular attention to studies on war-related PTSD. I will begin with a section on the National Vietnam Veterans

[1] Al Hubbard as cited in Haley, 1974; cited in Kulka, et al., 1990, p. 279.

Readjustment Survey (NVVRS) since many of the publications on risk factors for war-related PTSD comes out of this research, and since this dissertation focuses on a sub-sample from the NVVRS data.

There has recently emerged a burgeoning field of research exploring biological substrates of PTSD, with particular attention to biological vulnerabilities to the disorder and physiological changes at or immediately after traumatic exposure that have been shown in some studies to predict onset of PTSD (e.g. low cortisol levels, low GABA levels, higher heart rate, increased startle response, and specific skin conductance response, amongst others; see for example Davidson et al., 2004; Hepp et al., 2008; Pitman et al., 2006; Shalev et al., 1998b; Shalev & Freedman, 2005; Vaiva et al., 2006; Yehuda, 2006). Since no physiological assessments were made for the current study, a discussion of this research goes beyond the scope of the current literature review. However, it is important to keep in mind that there may be either pre-existing biological vulnerabilities to PTSD or physiological responses that may be acquired and manifest at, or immediately after, traumatic exposure. These vulnerabilities that may place individuals at greater risk of PTSD onset and/or chronicity are not yet entirely understood or consistently evidenced in empirical studies at this point.

It is also important to note that the influence of risk factors on PTSD outcomes is likely to be affected by the processes of primary and secondary appraisals, through which individuals in a given situation assess threat (primary appraisal) and the resources they have to contend with it (secondary appraisal) (Lazarus & Folkman, 1984). Especially at lower levels of trauma severity, appraisal may well mediate between traumatic event and outcome (Harvey & Yehuda, 1999): For example, if one appraises oneself as helpless in a

given situation, it will likely be perceived as more threatening and more traumatic than if one appraises oneself as less helpless. Furthermore, the ongoing post-traumatic process of evaluating the impact of a traumatic event has been labeled tertiary appraisal and has been studied in relation to adaptive responses to trauma (Dohrenwend et al., 2004). Although a thorough discussion of appraisal is beyond the scope of this literature review, it is mentioned here along with the biological substrates of PTSD to underscore the complexity of the underpinnings of this disorder, which cannot possibly be addressed in any single study.

National Vietnam Veterans' Readjustment Study. The most well known and most frequently referenced study of Vietnam veterans is the National Vietnam Veterans' Readjustment Study (NVVRS) from which the data for this dissertation are taken. Mandated by congress, the NVVRS examined a representative national sample of Vietnam veterans, which makes the findings from this data essential to any discussion of war-related PTSD. Kulka and his colleagues (1990) published the first findings from the NVVRS; however, the NVVRS data continue to be analyzed and re-analyzed, and important new findings have been published as recently as 2008 (Dohrenwend, Turner, Turse, Lewis-Fernandez & Yager, 2008; Lewis-Fernandez et al., 2008).

Background of the NVVRS.[2] In 1979, six years after the evacuation of Saigon, Congress passed Public Law 96-22, directing the Veterans Administration (VA) to establish a readjustment counseling program to provide services to facilitate the transition from military to civilian life for the 8,238,000 men and women who had served in the

[2] This section is based on the NVVRS Public Use Analysis File Documentation: Analysis Variables from the National Vietnam Veterans Readjustment Study, (Hunt et al, 1994).

U.S. Armed Forces during the Vietnam era upon their return from service. 3.14 million of these veterans had served in the Vietnam theater of war. The "Vet Center" program, as it came to be called, was separate from the VA medical center system, and despite expectations that it would meet a short-term need, service utilization exceeded expectations. Congress consequently renewed the program in 1981 and again in 1983 (Hunt et al., 1994).

The National Vietnam Veterans Readjustment Study was mandated with the 1983 renewal of PL96-22 in order to address the needs of those Vietnam veterans readjusting to civilian life who had *not* yet sought help. It was to be of sufficient size and scope to provide national estimates of Vietnam veterans' mental and other health needs and readjustment difficulties. The Research Triangle Institute (RTI) was contracted to carry out this study in 1984 (Hunt et al., 1994). Broadly defined, the three major goals of the study were the following, as stated in the Contractual Report of Findings from the NVVRS (Kulka et al., 1988):

- To obtain information on the incidence, prevalence, and effects of PTSD and related post-war psychological problems among Vietnam veterans.
- To examine the *total* life adjustment of Vietnam theater veterans and to compare it to that of era veterans who did not serve in the Vietnam War theater but who did serve during that period, as well as to nonveterans**.**
- To provide detailed scientific information about post-war PTSD, its antecedents, its course, its consequences, and its relationship to other physical and emotional disorders.

Original findings from the NVVRS. The original analyses of the NVVRS data were published by Kulka and colleagues in 1990. They found that an estimated 15.2% of male Vietnam theater veterans met criteria for current PTSD, measured over a decade after their wartime service, and 30.9% of male Vietnam theater veterans met criteria for PTSD onset at some time in their lifetimes. These prevalence rates contrasted with the much lower PTSD rates published by the Centers for Disease Control in 1988 of 14.7% lifetime PTSD and 2.2% current PTSD, 11 to 12 years after the Vietnam war ended (Centers for Disease Control, 1988, as cited in Dohrenwend et al., 2006). The original NVVRS data evidenced a strong dose-response relationship: veterans' reports of combat exposure were directly related to rates of PTSD (Kulka et al., 1990; 1991). Kulka and colleagues (1990) reported higher rates of lifetime PTSD and current PTSD in Black Vietnam veterans relative to White Vietnam veterans, and even higher rates in Hispanic Vietnam veterans.

Critics underscored the discrepancies between the CDC's prevalence rates and those of the NVVRS and argued that the latter were inflated due to a reliance on self-reports of their postwar symptoms, and a reliance on the DSM-III-R (APA, 1987) criteria for PTSD diagnosis, which did not require impairment of functioning, unlike the criteria of the DSM-IV (American Psychiatric Association, 1994; Schlenger et al., 2007). Lastly, critics argued that the dose-response relationship was exaggerated due to participants' retrospective self-reports of their exposure to war-zone stressors (Schlenger et al., 2007).

Subsequent NVVRS analyses. The NVVRS data were recently subjected to a thorough reanalysis by Dohrenwend and colleagues (2006). These researchers used data from military personnel files (201 files) together with data from military archival sources

and historical accounts to develop an objective measure of probable severity of war-zone stressors, so as not to rely on veterans' retrospective self-reports. They called this objective measure of severity of war-zone stress the Military Historical Measure (MHM), and looked at the relationship between it and rates of PTSD in the subsample of veterans whose PTSD was diagnosed in a Structured Clinical Interview for DSM-III-R by doctoral-level clinicians and confirmed as uniquely war-related. Using veterans' scores on the Global Assessment of Functioning as a proxy for impairment of functioning, Dohrenwend and colleagues adjusted the rates of war-related PTSD to the standards of the DSM-IV criteria (Dohrenwend et al., 2006).

After these adjustments, they found 18.7% of veterans had a lifetime onset of war-related PTSD and 9.1% of veterans had current war-related PTSD (Dohrenwend et al., 2006). They found little evidence of falsification of reports of combat exposure, a strong relationship between objective measures of severity of the war trauma and onset of PTSD (dose-response relationship) and an even stronger relationship between severity of war trauma and chronicity of PTSD. The authors emphasized that even these, most conservative prevalence estimates reveal that near 10% of Vietnam veterans continue to suffer with PTSD over a decade after their service. They also found that the multi-item self-report measure of war-zone stress developed by Kulka and colleagues (1990) showed a strong positive relationship with the record-based MHM of severity of exposure to war-zone stressors (Dohrenwend et al., 2006).

Dohrenwend and colleagues (2008) thoroughly reviewed the NVVRS data to examine whether differences in PTSD prevalence rates across racial/ ethnic groups could be accounted for by factors other than race/ethnicity. After adjusting for war-zone

exposure, they found that the differences in current rates of PTSD between black and white veterans disappeared. In the case of Hispanics veterans, elevated rates of PTSD relative to whites were accounted for by differences in exposure, age, education, and scores on the Armed Forces Qualification Test. Relative only to blacks, higher rates in Hispanics could be accounted for by younger age of entry to the war (Dohrenwend et al., 2008).

A number of researchers have analyzed data from the NVVRS in efforts to identify variables that place individuals at greater or lesser risk for PTSD. These examinations have further emphasized the dose-response relationship, albeit relying on self-reports of combat war-zone stress: Traditional combat was found to be the war-zone stressor with greatest power in predicting PTSD onset (King, King, Foy, & Gudanowksi, 1996); war-zone stress was found to be strongly related to *severity* of PTSD (King et al., 1995); and particularly severe war-zone stress, namely exposure to atrocities and/ or abusive violence, was found to be strongly related to onset of PTSD (King et al., 1999) and symptom severity (King et al., 1995).

Later analyses of the NVVRS data have parsed out the influences of pre-war, war-related, and post-war factors. Focusing on prewar and war-related factors, King and colleagues (1996) found that previous trauma history directly predicted onset of PTSD and younger age at entry to war predicted increased number of PTSD symptoms. Family instability, childhood antisocial behavior, and a younger age at exposure to war also had indirect effects on PTSD. Lastly, they found that history of prior trauma interacted with the level of war-zone stress to exacerbate PTSD symptoms in veterans with high levels of combat exposure. Turning their attention to postwar factors, King and colleagues (King,

King, Fairbank, Keane, & Adams, 1998) found perceived social support, hardiness, and additional negative life events each affected resilience and recovery, with social support playing the largest role as a protective factor against the development of PTSD.

Schnurr and colleagues (Schnurr, Lunney, & Segupta, 2004) conducted a reanalysis of the clinical sample from the NVVRS and combined it with data from the Hawaiian Vietnam Veterans Project, which was modeled on the NVVRS but studied Native Hawaiians and Americans of Japanese Ancestry. Their reanalysis examined pre-war, war-zone, and post-war factors predicting the onset and the persistence of PTSD. Consistent with other studies, they found Hispanic ethnicity, family instability, severe punishment during childhood, childhood antisocial behavior, and pre-war depression to be pre-war variables that increased the risk of PTSD onset. Of all the prewar variables studied, only severe punishment during childhood was related to risk for ongoing PTSD several years after the war. Consistent with the dose-response theory, these researchers found war-zone exposure was a risk factor for both PTSD onset and PTSD chronicity. They also found that peritraumatic dissociation (measured retrospectively) posed a risk for both onset of PTSD and chronicity of PTSD. Serious injury predicted PTSD chronicity, but not onset, whereas depression before, during, or after the war predicted PTSD onset but not chronicity. Similar to findings from other studies, current social support was inversely related to onset of PTSD as well as PTSD chronicity (Schnurr et al., 2004).

Findings from other military studies on risk factors for PTSD. A number of studies, most of convenience samples and less representative populations than was

studied in the NVVRS, have revealed important risk factors for PTSD onset, chronicity, and severity in military populations. Some of these findings are worth mentioning, though they may not be generalized to all or most military populations. These findings will be organized into pre-war, post-war, and peritraumatic, or war-related, variables.

Pre-war variables. Pre-war variables are consistently found to have some effects on PTSD outcomes, though these may be relatively small. History of prior traumatic experiences (i.e. childhood abuse or childhood trauma) has been found to be associated with onset of combat-related PTSD (Bremner, Southwick, Johnson, Yehuda, & Charney, 1993; Donovan, Padin-Rivera, Down, & Blake, 1996), in keeping with the findings of King et al., (1996) and Schnurr et al., (2004) from the NVVRS and Hawaiian Vietnam Veterans Project. Similarly, previous psychiatric history and family history of psychiatric illness are each associated with increased risk for onset of PTSD (Brewin et al., 2000a; Ozer, Best, Lipsey, & Weiss, 2003).

The effects that some pre-war variables have on influencing PTSD onset may be explained by group differences in exposure to trauma. For example, in a study of a convenience sample of Vietnam veterans, by Green and colleagues (1990b), veterans' age and education levels both predicted the severity of combat exposure, and age alone predicted exposure to the grotesque: "younger and less educated soldiers were exposed to the more severe combat experiences" (p. 732). They also found that soldiers with prewar axis I diagnoses were more likely to take part in special assignments and be exposed to grotesque experiences (Green et al., 1990b). Certain pre-war factors also predicted post war life events known to influence PTSD. Better-educated soldiers, for example, reported more social support after the war (Green et al., 1990b).

In a study of a large cohort ($N = 2949$) of Gulf War veterans, Wolfe and colleagues found that being young, single, and having previous combat experience increased veterans' vulnerability to onset of PTSD, though combat exposure was the most significant predictor of onset (Wolfe, Erickson, Sharkansky, King, & King, 1999). The same authors also found that non-officer rank and reserve or National Guard status increased the risk of PTSD onset. In another recent study, LeardMann and colleagues (2009) examined a large military cohort ($N = 5410$) who deployed between their baseline and follow-up surveys, and who reported combat exposure. They found that individuals with low pre-deployment functional health status were two to three times more likely to develop PTSD symptoms or meet criteria for diagnosis by follow up, two to three years later, compared to those individuals with high functional health pre-deployment (LeardMann et al., 2009).

Post-War Variables. Perceived social support is the post war variable that has been repeatedly found to have an inverse effect on the persistence of war-related PTSD (King et al., 1998; Koenen, Stellman, Stellman, & Sommer, 2003b; Port, Engdahl, Frazier, & Eberly, 2002). This is the only post-war variable that was studied in either of the meta-analyses below, although a number of other post-war variables have also been studied. Most notable of these is individuals' reliance on avoidant coping strategies, which has been found to be associated with war-related PTSD.

Hyer and colleagues (Hyer, McCranie, Boudewyns, & Sperr,1996) found, in a sample of treatment-seeking Vietnam veterans with PTSD, that veterans mostly relied upon escape-avoidance coping and distancing to cope with memories of past experiences. Furthermore, increases in self-reported symptom intensity were significantly correlated

with greater relative use of escape-avoidance. Benotsch and colleagues (Benotsch et al., 2000) also found in their study of Gulf War veterans that increases in avoidant coping were related to increases in PTSD symptomatology. They found that avoidant coping one year after the official end of hostilities was predictive of PTSD thirteen months later, even after controlling for emotional distress at the time of the first assessment. Similar findings have corroborated the association between PTSD and avoidant coping (Johnsen, Eid, Laberg, & Thayer, 2002; Stein et al., 2005; Sutker, Davis, Uddo, & Ditta, 1995). An important question to consider for each of these findings is how avoidant coping styles were distinguished from PTSD cluster C symptoms of avoidance.

War-Related/ Peritraumatic Variables. The variables that are most strongly correlated with onset and chronicity of PTSD are those related to the traumatic experience. These can be divided broadly into situational variables specific to the context of war—the intensity of combat, experiences the individual witnessed or in which the individual participated, injuries sustained– and personal variables specific to how the individual responded in the context of war including emotional reactions and dissociative experiences.

Situational variables can be objectively measured, since these aspects of the combat experience can be researched without having to rely on the veteran's retrospective report, which may be affected by his symptomatology at the time of the interview (see for example, Dohrenwend, et al., 2006). Most studies, however, rely on veteran accounts of combat exposure and specific incidents in combat. As reported earlier, the NVVRS self-report measure (Kulka et al., 1990) was found to be strongly related to the record-based measure of combat severity (MHM) developed by

Dohrenwend and colleagues (2006). This may also be the case with other self-report measures of exposure to war-zone stress.

Even before the NVVRS findings confirmed the dose-response relationship with a nationally representative sample of Vietnam veterans, a number of early studies had shown that risk for PTSD onset or PTSD symptoms increases with the severity of combat experience reported by veterans, and with exposure to especially extreme violence or grotesque situations (Breslau & Davis, 1987a; Foy, Sipprelle, Rueger, & Carroll, 1984; Laufer, Gallops, & Frey-Wouters, 1984). Green and colleagues (1990b) also found that onset of PTSD was explained primarily by reported wartime stressors. They delineated each of the following as having significant predictive influences on PTSD onset: threat to life, injury, loss of a close friend in combat, and witnessing or participating in hurting or killing an enemy and/ or civilian.

Since the publication of the original NVVRS findings (Kulka et al., 1990), other studies have confirmed the relationship that the reported intensity of combat exposure has with the onset and chronicity of PTSD (Wolfe et al., 1999; Stein et al., 2005). Roy-Byrne and colleagues (Roy-Byrne et al., 2004), set out to examine changes in the combat exposure-PTSD relationship over the decade between 1987 and 1997 in male-male twin pairs discordant for service in Southeast Asia. While reported combat exposure was strongly associated with PTSD in both 1987 and 1997, and highly significant at each time, the effect diminished over the decade. The authors emphasized the continuing role of combat exposure on the persistence and chronicity of PTSD, since symptoms continued to be elevated in those who reported being exposed to the highest levels of combat, almost 25 years after the end of hostilities (Roy Byrne et al., 2004).

Another aspect of war trauma that has been examined as a risk factor for PTSD is combat injury. In the NVVRS, veterans who reported combat-related injuries had higher rates of PTSD (Kulka et al., 1990). Green and colleagues (1990b) also found injury predicted PTSD onset. Ikin and colleagues (2007) reported that Korean War veterans who endorsed having been wounded in combat were twice as likely to have PTSD at the time of assessment, five decades after the war, compared to those who did not sustain an injury. More recent studies have examined the effects of combat injuries on PTSD onset longitudinally and closer to the time of injury. Between 1998 and 2000, Koren and colleagues (Koren, Norman, Cohen, Berman, & Klein, 2005) studied 60 Israeli soldiers admitted to three major hospitals with combat-related injuries and 40 comparisons matched by rank, military role, and length of service, who took part in the same combat situations but were not injured. They found bodily injury significantly increased the likelihood of PTSD onset after combat, though severity of the injury was not related to PTSD onset. In contrast, Grieger and colleagues (2006) found that, in veterans of wars in Iraq and Afghanistan, severity of physical injury in soldiers hospitalized for combat-related injury was strongly associated with PTSD onset, PTSD severity at 7 months, and later onset PTSD (i.e. after four months).

Another situational aspect of war trauma has to do with the nature of the traumatic event to which the veteran was exposed. In their convenience sample of treatment-seeking and non-treatment seeking Vietnam veterans, (N = 200) Green and colleagues (1990a) found the strongest predictor of PTSD onset to be "exposure to grotesque death" (p. 731), a variable that included "activities subsumed under abusive violence or atrocities" (p. 732). The best predictor of "grotesque experience" was being in a highly

life-threatening combat experience, a factor that made a significant contribution to predicting PTSD above and beyond that made by exposure to grotesque death. These authors also found that special assignment and exposure to grotesque death best predicted persistence of PTSD approximately 15 years after service in the Vietnam War (Green et a., 1990b). These data support that the dose-response relationship applies to PTSD rates over a decade after wartime service.

Another of the risk factors shown to predict PTSD onset in Green and colleagues' study (1990b) was witnessing or participating in hurting or killing an enemy or civilian. This risk factor deserves attention, since it suggests that perpetration of violence may carry unique risks for the development of PTSD. Exposure to atrocities has been shown to contribute to PTSD onset, independent of combat exposure (Kulka et al., 1990; Laufer et al., 1985; Breslau & Davis, 1987a). Most of the studies focusing exclusively on the relationship between perpetrating violent acts and onset or chronicity of PTSD have examined clinical populations, often inpatient samples, of combat veterans who endorse high rates of participation in or witnessing of warzone violence. Breslau and Davis (1987a) examined a clinical sample of Vietnam veterans and found that experience with warzone atrocities correlated with severe PTSD. Participation in said atrocities was a significantly stronger predictor of PTSD than witnessing warzone atrocities.

In a study of 40 male Vietnam combat veterans with PTSD seeking inpatient or outpatient treatment, Yehuda and colleagues (1992) found that veteran-reported experience with atrocities was related to increased severity of current, veteran-reported PTSD symptoms. Experience with atrocities was also related to higher scores for depression and to greater impact of PTSD symptoms on overall functioning. These

authors conclude that both severity and type of stressor may contribute to the course of psychopathology, with experience with atrocities having a role in both severity of symptoms and impairment of functioning.

A study of a sample of inpatients who were also Vietnam veterans (Hiley-Young, Blake, Abueg, Rozynko, & Guzman, 1995) found 92% of the sample endorsed either witnessing and/or participating in three kinds of warzone violence: hurting Vietnamese, killing Vietnamese, and mutilating bodies of Vietnamese. These authors did not specify differences between victims who were Vietnamese civilians and those who were Vietnamese combatants. Their study suggests that specific kinds of participation in violence may be me more pernicious than others. Of the various kinds of violence listed, mutilation of bodies was significantly related to PTSD severity. Furthermore, veterans who witnessed and participated in the three kinds of warzone violence assessed had the highest rates of combat exposure and the most severe PTSD. These authors conclude that treatment-seeking veterans who engaged in violent acts during war often present with extreme PTSD symptoms, including profound guilt (Hiley-Young et al., 1995).

Extending this focus to a nationally representative population of veterans, Dohrenwend and colleagues have recently examined data from the NVVRS for evidence of associations between war-zone violence and PTSD in a nationally representative population of veterans (Dohrenwend et al., in preparation). Amongst veterans who reported having killed enemies at war, those who additionally endorsed having caused harm to civilians or prisoners demonstrated remarkable increases in the odds of developing PTSD. That acts that violate the rules of wartime conduct present increased risk of PTSD beyond killing the enemy, suggests that social codes may be important

filters in individual's appraisals of and reactions to traumatic events (see also Shay, 1994).

Personal factors during war trauma, including psychological responses to combat, are inherently subjective. The risk of such measures is that the individual's psychopathology or psychological symptoms at the point of assessment may influence his recall of his experience during the traumatic event. Despite these inherent measurement problems, dissociative experiences during trauma (peritraumatic dissociation) have been increasingly studied in the past decade as researchers attempt to determine their relationship with PTSD onset and development. In one of the early studies to link dissociation and PTSD, Bremner and colleagues (Bremner et al., 1992) compared rates of current dissociative symptoms in Vietnam veterans with and without PTSD. They found veterans with PTSD had higher levels of dissociative symptoms, even when differences in level of combat exposure were controlled. Veterans with PTSD also reported retrospectively that they had more peri-traumatic dissociative symptoms than veterans who did not have PTSD (Bremner et al., 1992).

In a later study of Vietnam veterans with and without PTSD, Bremner and Brett (1997) found that PTSD patients reported higher levels of peritraumatic dissociative states than patients without PTSD. These persisted in the form of dissociative states in response to postmilitary traumatic events, such that dissociative responses to combat trauma were associated with higher long-term rates of general dissociative symptomatology and increases in number of flashbacks since the war. The authors conclude that peri-traumatic dissociation is a marker of long-term psychopathology (Bremner & Brett, 1997).

In one of the only longitudinal studies on peritraumatic dissociation, Shalev and Freedman (2005) studied victims of motor vehicle accidents and terrorist attacks, beginning 1 week after the traumatic event and over the course of the next 4 months. They found PTSD that persisted at 4 months was predicted by emergency room measures of heartrate, peritraumatic dissociation, and early PTSD symptoms, regardless of the kind of trauma experienced. These data suggest that individual's propensity to dissociate under stress may increase the chance of subsequent onset of PTSD. Linking peritraumatic dissociation with another risk factor for PTSD, Schapiro and colleagues (Schapiro, Glynn, Foy, & Yavorsky, 2002) examined 42 Vietnam veterans with combat-related PTSD and found that participation in war-zone atrocities was associated with trait dissociation, or a general tendency towards dissociating. Veterans who participated in war-zone atrocities were more likely to report long-standing dissociative symptoms. Research has yet to delineate the direction of relationships between trauma, dissociative processes, and PTSD, in order to clarify the nature of causal processes.

Relative Contributions of Pre-war, War-Related, and Post-war Variables to PTSD onset. Green and colleagues (1990a; 1990b) compared the relative contributions of prewar, wartime, and post-war variables to the prediction of current PTSD diagnosis over a decade after wartime service. They found that 19% of the variance in the current rate of PTSD was explained by wartime (peri-traumatic) stressors including threat to life, injury, loss of a close friend in combat, and witnessing or participating in hurting or killing an enemy and/ or civilian. Post-military stressors (social support and post-service adjustment) had a slightly smaller relative contribution to predicting current PTSD, explaining 12% of the variance in prevalence, and pre-trauma stressors including

previous diagnosis and level of pre-service education explained only 9% of the variance in prevalence of current PTSD. These findings are corroborated by the meta-analyses described below.

Meta-analyses on risk factors for PTSD. Two relatively recent publications summarize many of the findings on risk factors for the prevalence of PTSD, though they do not discuss risk factors for chronicity of PTSD. In 2000, Brewin and colleagues published a meta-analysis of risk factors for PTSD based on their review of 77 studies of 14 risk factors for PTSD, and the moderating effects of various sample and study characteristics (Brewin et al., 2000a). This meta-analysis resulted in two major findings. The first was that certain risk factors had uniform, though modest, predictive effects across studies: family history of psychiatric disorder, personal history of psychiatric disorder, and history of childhood trauma. Each of these factors predisposes traumatized individuals to an increased risk of developing PTSD regardless of the kind of trauma to which the individual is exposed. That all three of these factors are pre-trauma characteristics may be one reason the effect size of their influence is relatively small. Secondly, Brewin and colleagues identified a series of other risk factors whose effects on predicting PTSD were often stronger than the three aforementioned, uniform, pre-trauma factors, but whose predictive effects varied with the specific study or the class of study. This led them to argue against a single vulnerability model for PTSD in favor of investigating "more proximal links in the causal chain between pretrauma risk factors and immediate trauma responses" (p. 756; Brewin et al., 2000a).

Brewin and colleagues (2000a) emphasize the distinction between military and civilian samples as the "single largest and most important division of the literature on the prediction of PTSD" (p.752). Younger age at trauma was a risk factor unique to military samples, for example. Furthermore, lower levels of education, minority status, trauma severity, and lack of social support were each found to increase the likelihood of PTSD onset in both civilian and military samples, but they had significantly more effect in predicting PTSD in military samples. Of these, trauma severity (peri-traumatic risk factor) and lack of social support (post-traumatic risk factor), had notably stronger effect sizes for predicting PTSD than the three uniform risk factors consistent across all studies, civilian and military. The finding that minority status predisposes veterans to PTSD should be interpreted with some caution since, as previously noted, Dohrenwend and colleagues (2008) examined the elevated rates of PTSD onset in black and Hispanic Vietnam veterans in the NVVRS and were able to account entirely for the group differences with factors including different rates of combat exposure, younger age of entry, and lower pre-service education levels.

In 2003, Ozer and colleagues published another meta-analysis summarizing the findings of 68 studies of seven predictors of PTSD (Ozer et al., 2003). It included 21 studies that had not been included in the meta-analysis by Brewin et al., (2000a) and included studies on both military and nonmilitary populations. Arguing that Brewin et al.'s review revealed that the effects of demographic factors on predicting PTSD were relatively small, they elected to omit consideration of gender, education, and ethnicity, and focus on personal characteristics "salient for psychological processing and functioning" and aspects of the traumatic event or its sequelae (p. 55). Ozer and

colleagues added two novel peritraumatic risk factors to their analyses: peritraumatic emotionality and peritraumatic dissociation. The other five were: prior trauma, prior psychological adjustment, family history of psychopathology, perceived life threat during the trauma, and posttrauma social support.

Ozer and colleagues found that all seven of these predictors yielded significant effect sizes in predicting PTSD diagnostic status and symptom severity (Ozer et al., 2003). They did not distinguish military from non-military samples, as Brewin and colleagues had (2000a). They examined the effect size of relationships between specific variables and PTSD symptoms, obtained from *t* tests and chi-square analyses as appropriate for each study, where raw data were available. Where raw data were not available, the authors estimated the effect sizes from the degrees of freedom and significance levels provided for each study. Where analyses were not statistically significant and no significance level or data were provided, the authors imputed the effect size of zero (p. 56). Effect sizes were averaged by weighting for the degrees of freedom in each study, so that they could be compared. The predictor variables were divided into two classes based on the weighted effect size of each variable's relationship to PTSD symptoms. Like Brewin et al., (2000a), they found that pretrauma characteristics (i.e. prior adjustment, prior history of trauma, and family history of psychopathology), despite being distal to the traumatic event, produced uniform, small effects on PTSD, with coefficients smaller than .20. A second class of predictive factors yielded coefficients greater than .20, and these were more proximal to the traumatic event, including perceived life threat, perceived social support, peritraumatic emotionality, and

peritraumatic dissociation, which they found had the largest effect size of all (Ozer et al., 2003).

The only finding of Ozer and colleagues that seemed to contradict those of Brewin and colleagues was that of perceived social support (Ozer et al., 2003). Ozer and colleages found perceived social support had an effect size significantly smaller than that of Brewin et al., and they understood this as having to do with the differences in inclusion criteria for the studies in each analysis. They also pointed out that in studies in which the traumatic event had occurred more than three years prior, social support had larger predictive effects than in studies where the traumatic event had occurred more recently (Ozer et al., 2003). This suggests that the protective effects of social support may either have some a cumulative effect over time or may be more salient for chronic rather than for acute PTSD.

Theories on the Course of PTSD

Models of PTSD course. There are four models that attempt to make sense of PTSD onset and course, with varying emphases on the role of pre-existing (i.e. genetic, biological, and/or historical) factors and external, trauma-related (i.e. peri-traumatic situational) factors.

The *stress evaporation model* posits that traumatic stress triggers a reaction that steadily diminishes (or evaporates) over time, after which persisting symptoms are postulated to reflect personal risk factors (described in King et al., 1996). The value of this model has diminished in significance as evidence as accumulated supporting the primacy of war-zone stress (King et al., 1996; Dohrenwend et al., 2008).

Grinker and Spiegel (1945) (cited in Engdahl and Eberly, 1997) posited the *residual stress model* in which symptoms are directly proportional to the severity of the stressors to which the individuals were exposed, and in which symptom severity gradually diminishes over time. The residual stress model contrasts with the stress evaporation model in that it minimizes the influence of preexisting conditions and conceptualizes posttrauma dysfunction as a consequence of the traumatic event itself (King et al., 1996).

The *stress vulnerability model* integrates aspects of the aforementioned models by holding that pretrauma characteristics create increased susceptibility to the deleterious effects of traumatic stress (King et al., 1996). Development of PTSD arises from the interaction of pre-existing vulnerability and traumatic stress, with lower levels of stress necessary to precipitate the disorder in individuals with greater vulnerability, and vice versa.

A fourth *model of additive burden* (Dohrenwend and Dohrenwend, 1981, cited in King et al., 1996) conceives of vulnerability as a continuum between susceptibility and resilience, and takes into account that pre-existing burdens may have main effects on PTSD onset, independent of the traumatic experience.

Stage theories of PTSD course. Only three theories of PTSD course were encountered in this literature review that detailed the progression of symptoms over time, and all of these described these changes as a progression of stages in the disorder.

Horowitz (1978). The first stage theory on PTSD course was proposed by Horowitz, (1978), who suggested there are five phases of adjustment to stressful experiences, each of which varies in duration: 1) outcry, 2) denial, 3) oscillation between

denial and numbing, and intrusive memories, 4) working through, 5) and completion of the response. Horowitz describes the disorder as progressing from one stage to the next, in the stated order, though symptoms may wax and wane over time in relation to individual and external factors. In Horowitz's theory, the individual's completion of all stages culminates in the resolution of the disorder. If progression through the stages is incomplete, however, the theorized outcome is persistent pathology. Other theorists have detailed other pathological outcomes stemming from partial progression through Horowitz's stages. Epstein (1989; cited in Wang and Wilson, 1996), for example, describes a maladaptive outcome of PTSD in which the individual never completes the numbing/ intrusive "oscillation" phase. Epstein suggests that individuals who get stuck in stage 3 ultimately experience one of five outcomes: withdrawal, dissociation, fear, anger, or embracing the trauma.

Frederick (1985, cited in Wang & Wilson, 1996). Frederick described a five-phase model of PTSD in combat veterans that is similar to Horowitz's in that completion of all five stages is ultimately an adaptive process. It differs, however, in that Frederick includes a stage of decompensation as a necessary step towards recovery. The five stages in Frederick's model are: initial impact; resistance/denial; acceptance/repression; decompensation; and trauma mastery and recovery.

Wang and Wilson (1996). Wang and Wilson expand on the idea that decompensation is central to the disease trajectory of many people with severe, chronic PTSD. By decompensation, they intend to mean a specific stage of the disorder during which all aspects of the individual's functioning are severely impaired, and which may also be identified by specific physiological changes including hormonal fluctuations.

Wang and Wilson's model is based on long-term observations and assessments of inpatient and outpatient Vienam veterans with chronic, severe, combat-related PTSD at two national PTSD centers. They point out that, contrary to Frederick's model, for many Vietnam combat veterans decompensation is not a step towards recovery. According to their observations, veterans experience many cycles of decompensation that are extremely disruptive to their lives and efforts at rehabilitation, and that may not lead to any kind of resolution. In fact, individuals with severe PTSD may be unable to emerge from cycles of repeated decompensations.

These authors also emphasize the tremendous variability in veterans' experience of symptoms and their level of functioning over time. They outline notable shifts in reactivity to stimuli, cognitive and problem-solving abilities, coping style, interactions with the environment, capacity for attachment, capacity to articulate emotions and needs (alexithymia), affect regulation, impulsivity, defensiveness, capacity to experience positive emotions, and suicidality. Their model sought not only to delineate cyclic periods of decompensations, but also periods of relatively higher functioning that the veterans they observed experienced, albeit transiently. They suggest that shifts from one stage to another may be accompanied by biological changes and shifts in hormonal patterns.

Wang and Wilson's model (1996) consists of four stages: 1) Most adaptive functioning during which patients demonstrate flexible, appropriate interactions with the environment, have optimal problem solving and goal-oriented behaviors, can experience positive affect, can reflect on emotions and articulate these, and during which PTSD symptoms, if any, are at a minimum. Patients in this stage are able to obtain work and

form meaningful relationships. 2) The *survival stage* is when the individual experiences fair to poor functioning, his orientation becomes defensive, emotions become constricted, as do his physical reactions. PTSD symptoms increase, and the veteran focuses on his survival. His perceptions are restricted to information about potential danger, his affect regulation is notably reduced, as is interest in activities and his capacity to concentrate and remember. Negative affect increases, as does suicidality and homicidal ideation. If the veteran's symptoms worsen beyond a certain threshold, Wang and Wilson describe a pull towards the next stage. 3) *Decompensation* involves very poor functioning characterized by a loss of meaningful contact with reality, lack of relatedness to others, dysregulated affect and severe PTSD symptoms. The veteran typically copes through either sensation seeking or shutting down. In this state the veteran may behave impulsively, even recklessly, or he may completely withdraw and isolate. 4) *Depression/Hopelessness* is another stage of poor to very poor functioning marked by a relative calm after the storm of decompensation (stage 3). The veteran in stage 4 is exhausted and overwhelmed with negative affect, feelings of helplessness and hopelessness, despair, defeat, and at times humiliation. At this point reactivity and startle are at their lowest point, and depression is at its highest level. According to Wang and Wilson, veterans in Stage 4 may follow one of two courses: they may regroup and begin to accept their limitations and available channels of support, and seek reasons for living; or, they may give up, retreat from treatment and support, remain hopeless and despairing, and either attempt suicide directly or indirectly, or cycle back and forth between decompensation Stage 3 and returns to Stage 4.

Unlike the previous stage models, in this model, veterans may not go through the stages in the same order or they may skip a stage altogether. Wang and Wilson (1996) suggest that shifts from one stage to another can be the result of internal stimuli (i.e. memories) or external stimuli (i.e. current events, anniversaries, etc.) that result in increased anxiety and sense of threat that result in increased symptoms. They cite reduced sleep and reduced connections with other people to be critical factors in inducing further decompensation, and insuring that sleep cycles are restored and optimal social support is provided can guard against such devolutions. They suggest that many veterans with chronic, combat-related PTSD spend much of the time vacillating between the first and second stages, and that a goal of treatment may be to maintain the equilibrium between these two stages while preventing decompensation to the third and fourth stages. The authors underscore the importance of state dependent learning in PTSD to their model: Shifts between one stage and another not only represent changes in the severity and quality of symptoms, but represent changes in the "whole reinforcement system affecting the veteran" (p. 243). The larger goal of clinical interventions beyond assessing the stage of the disorder and reducing symptoms and problematic behaviors that are emblematic of that stage, is to equip the veteran with the means to cope with changes in functional capacity, affect regulation, and behavioral tendencies in each of the stages he may come to experience (Wang & Wilson, 1996).

Theories of adaptation to stress. It is beyond the scope of this literature review to detail the intricacies of contemporary psychological theories of PTSD, especially given that this study will not assess the validity of one theory's merits compared with the

validity of those of another. It is relevant, however, to mention briefly three theories on human adaptation to stressful events, since these each present specific ways to think about how individuals reel from and adapt to trauma and adversity.

General Adaptation Syndrome. Hans Selye described a seminal theory on stress response, general adaptation syndrome, from his work through the 1900s on the effects of stress in mice. In this model, there are three stages of stress response: alarm state, resistance, and exhaustion. Selye described the body's initial response to acute stress as the alarm state, during which the hypothalamic-pituitary-adrenal axis is activated in response to acute stressors, and a series of physiological changes takes place to equip the organism to deal with the stressor. Selye also described two later stages—resistance, in which the body's resources become gradually depleted if the stressor persists in its acuteness, and exhaustion, in which resources are exhausted and normal functioning can no longer be maintained. Exhaustion may result in mental illnesses or physical illnesses such as cardiovascular or systemic problems. According to this theory, the body responds to external stressors with an acute response that is helpful in the short term, but becomes damaging to the body if the system does not respond to homeostasis after a short period (Selye, 1956).

Allostatic load. In 1993, McEwen coined the term *allostasis* to describe the body's response to stress, and allostatic load to describe the point at which this response becomes destructive rather than adaptive (McEwen & Norton Lasley, 1994). McEwen's conceptualization departs from the GAS model in that he understands the various stress mechanisms to be constantly engaged in adaptation to stress, and thereby to be constantly maintaining stasis through change and variability. In McEwen's model, the body does not

seek to return to homeostasis, a level of some absence of stress, but rather, it is always adapting to levels of stress by changing its set-point. Allostatic load occurs when allostatis malfunctions—a phenomena that can result if the stress response does not shut off after the acute stress has disappeared, when the stress response does not adapt to a repeated event, or when the stress response chronically, repeatedly, or inappropriately activates the body's systems. These malfunctions may be related to liftestyle and biological/ genetic factors.

Salutary outcomes of traumatic exposure: Resilience & posttraumatic Growth. Whereas *resilience* refers to the ability to sustain trauma without developing PTSD or impaired functioning. As such, resilience is definitionally related to the absence of negative consequences of trauma (Bonanno, 2008; Lepore & Revenson, 2006). *Posttraumatic growth* (PTG) refers to positive changes that occur as a result of a traumatic experience, which may coexist with negative outcomes that also result from trauma. In contrast to resilience, PTG is not mutually exclusive with negative outcomes from trauma (Lindstrom & Triplett, 2010; Joseph, & Linley, 2008). PTG is directly related to trauma whereas resilience is inversely related to trauma, and research has shown that the two are inversely related to one another (Levine, Laufer, Stein, Hamama-Raz, & Solomon, 2009). At least one study has demonstrated that compared to veterans exposed to war trauma who were not POWs, ex-POWs show higher rates of PTG as well as PTSD (Solomon and Dekel, 2007), with the implication being that more severe trauma may be associated with greater amounts of PTG, despite comorbid psychopathology.

PTG comprises the following five domains (Tedeschi & Calhoun, 1996): Personal strength (feeling better able to handle stressful events); new possibilities (interests and

activities); changes in relationships with others (greater compassion, more connection); change in appreciation of life (change in values and priorities); and spiritual change, including increased connection with religion or increased sense of a larger meaning in life (Calhoun & Tedeschi, 2006).

Calhoun and Tedeschi (2006) have presented a complicated general model of PTG that attempts to capture the process of growth by including pre-trauma personal characteristics, the traumatic event, mediating and moderating contextual, cognitive, and interpersonal factors. At its core, this model attempts to capture how positive changes come about in the process of integrating a new, often negative experience into prior beliefs about and ways of seeing the world, relationships, and the self (e.g. Janoff-Bulman, 1999). Elaborating on this concept, Joseph and Linley (2008) emphasize that PTG is about change in psychological well-being, by which they mean "issues of meaning, personality schemas, and relationships rather than positive and negative affect or life satisfaction, which make up subjective well-being" (p. 33).

Criticism of PTG as a concept centers on its overlap with personality traits (i.e. optimism) that existed prior to the traumatic exposure (Feder et al., 2008; Ford, Tennen, & Albert, 2008) or cognitive attribution processes that lead traumatized individuals to believe they have grown from the trauma (Ford et al., 2008). Furthermore, most of the research on PTG relies on the PTGI (Tedeschi & Calhoun, 1996) a self-report inventory of change, which has been criticized for measuring perceived rather than actual growth (e.g. Frazier et al., 2009).

Empirical Findings on the Course of War-Related PTSD

By *course* of the disorder, I mean the changes in symptoms that individuals experience through the duration of the disorder. These changes involve the onset of PTSD symptoms relative to the traumatic event, the chronicity (or duration) of the disorder, as well as the changes in these symptoms over time, including patterns of worsening, amelioration, and/or remission, and patterns of specific types of symptoms (i.e. avoidant, reexperiencing, and hyperarousal clusters). Research has yielded a variety of findings about onset and progression of symptoms and diagnostic status in war-related PTSD. The diversity in findings is related to variations in populations under study, study design (retrospective vs. prospective) and variations in the amount of time between the trauma and the PTSD assessment(s) administered.

Since PTSD was only established as an official diagnostic classification in 1980 (APA, 1980), the study of veterans from wars leading up to and including the Vietnam War, such as WWII and the Korean War, often involved recruiting participants who had been home for decades since their service. The historical distance of these wars has allowed for studies of PTSD many years past veterans' dates of service, and as a consequence, the majority of this research is retrospective in design.

Investigations of veterans of military operations since 1980 have examined veterans longitudinally beginning soon after their return from deployment. Compared to veterans from earlier wars, veterans of the Gulf War, the Iraq War, and the war in Afghanistan have returned from service relatively recently, and those that have developed PTSD have done so more recently than veterans from Vietnam and earlier wars. As a result, PTSD research on these veterans generally spans much shorter periods of time

since the traumatic exposure than research on veterans from earlier wars. This more recent set of research consists of detailed studies of the course of the disorder studied longitudinally over the first year, or the initial few years, after wartime service, including changes in symptoms, symptom severity, and/ or diagnostic status tracked across months or years.

Studies of PTSD course in veterans of wars prior to 1980. Ten publications describing cross-sectional, retrospective studies of PTSD in veterans of wars before 1980 were reviewed in which veterans were assessed at one point, 1 to 5 decades after the beginning of the wars in which they served (with the exception of Port, Engdahl, & Frazier, 2001, who also included a longitudinal and a second assessment point in their study). Four studies focused on former Prisoners of War (POWs) from WWII (Kluznik et al., 1986; Port et al., 2001; Speed et al., 1989; Zeiss and Dickman, 1989), another publication summarized four studies on Dutch resistance fighters from WWII (Op den Velde et al., 1996), three studies focused on veterans of Vietnam (Bremner et al., 1996; Dohrenwend et al., 2006; Schnurr, Lunney, Sengupta, & Waelde, 2003), and one study examined Israeli veterans of the 1973 Yom Kippur War (Solomon and Kleinhauz, 1996). A final publication reports on a case study of a WWII veteran who developed PTSD 30 years after his service (Van Dyke, Zilberg, & McKinnon, 1985). Two of these studies included efforts to chronicle changes in symptoms over time (Bremner et al., 1996; Port et al., 2001). Bremner and colleagues (1996) assessed changes in symptoms since the war retrospectively in an interview in which they went over a timeline, whereas Port and colleagues (2001) who also examined PTSD retrospectively at the first time point,

assessed symptom change longitudinally by measuring diagnostic status and symptom levels with a follow-up assessment four years later.

Two additional studies were longitudinal in design and examined the course of the disorder in veterans of early wars beginning many years *after* the wartime experience. These studies shed light on the development of the disorder over periods well beyond the antecedent trauma (Dirkzwager et al., 2001; Johnson et al., 2004). In 1992 and 1998, Dirkzwager and colleagues (2001) examined 674 Dutch military veterans who had fought in WWII, the former Dutch East Indies, and Korea. The majority of these were on military disability pension, while 198 of them were from the community and not on pension. Johnson and colleagues (2004) examined a smaller sample of Vietnam veterans ($N = 51$) with severe, chronic combat related PTSD decades after the war, who were admitted to a 15-week inpatient treatment for the disorder. Veterans were assessed on admission and six years later, and the most salient finding was the extremely high mortality rate (17%) relative to men of the same age in the general population.

For brevity, all 12 of the aforementioned publications are summarized in Table 1, with specifics on the veterans studied, kind of sample and sample size, number of assessment points, PTSD measures employed in person or by mail, and the stated goal of each study.

Studies of PTSD course in veterans of wars since 1980. Seven longitudinal studies on the course of PTSD in veterans of wars since 1980 were reviewed and summarized in Table 2 (Grieger et al., 2006; McCarroll, Ursano, & Fullerton, 1995; Orcutt, Erickson, & Wolfe, 2004; Solomon & Mikulincer, 2006; Southwick et al., 1995; Thompson et al., 2004; Wolfe et al., 1999). None of those reviewed here were of full

probability samples. Wars in which the veterans studied had fought included the 1982 Israeli war with Lebanon, the Gulf War, and the U.S. invasions of Iraq and Afghanistan. Some of the studies reviewed in this section are included because the investigators obtained information about the course of the disorder, though studying disorder course was not one of their stated goals (e.g. McCarroll et al., 1995; Grieger et al., 2006). Those studies that examined the prevalence of PTSD at different time points (Southwick et al., 1995; Wolfe et al., 1999; Grieger et al., 2006; Solomon and Mikulincer, 2006) all relied on self-report checklist measures to assess for *probable* diagnostic status. Four of the studies reviewed here examined posttraumatic symptom levels in veterans over time *without* assessing whether study participants met criteria for PTSD (McCarroll et al., 1995; Orcutt et al., 2004; Solomon & Mikulincer, 2006; Thompson et al., 2004). It is important to be cautious in generalizing the findings from the latter-mentioned studies since individuals with and without PTSD are grouped together.

Findings on onset of PTSD. The diagnosis of PTSD cannot be made unless the requisite number of re-experiencing (Criterion B), avoidance (Criterion C), and hyperarousal (Criterion D) symptoms have persisted for at least one month (APA, 2000). This is essential because *most* survivors of traumatic events endorse some posttraumatic symptoms in the weeks after being exposed to an acute stressor (Davidson et al., 2004; Foa, Rothbaum, Riggs, & Murdock, 1991; Shalev, Freedman, Peri, Brandes, & Sahar, 1997; Shalev, 2002a). Foa and colleagues (1991) found that whereas 94% of rape survivors had "all the clinical symptoms of PTSD" one week after the event, at three months and nine months, only 15 to 25% had all the symptoms of PTSD. Another study showed that, a week after traumatic exposure, the distribution of PTSD symptoms in

survivors of traumatic events follows a "normal" Gaussian distribution (Shalev et al., 1998b), though most of these people never met diagnostic criteria for PTSD. In the initial days and weeks after a traumatic event, PTSD symptoms do *not* predict later development of PTSD, although the *absence* of symptoms in the initial weeks after a traumatic event is a strong predictor of who will *not* develop PTSD (Shalev, 2002a).

Over the course of the first few weeks after the traumatic event, the individual may be able to regulate his biological and psychological responses to the event, to re-establish homeostasis, and integrate the emotional memory with other memories. Together, these adaptations will result in the reduction of symptoms and failure to meet diagnostic criteria for the disorder at one month (McFarlane, 2000). Research shows that in traumatized individuals who never meet criteria for PTSD, posttraumatic symptoms have both onset and a peak in severity of symptoms soon after the traumatic event, and that symptoms steadily decrease over the subsequent weeks (Shalev, 2002a), whereas those who go on to develop PTSD experience continued, and even progressive, dysregulation over the course of the next month (Davidson et al., 2004; McFarlane, 2000). For this reason, the development of PTSD can be understood as a failure to stabilize after having a normative, acute response to stress (Davidson et al., 2004).

The consensus from this research is that PTSD symptoms generally begin within days or weeks of traumatic exposure and then *persist* until a diagnosis is made. This assertion contradicts reports of the existence of PTSD with delayed onset, defined by the DSM-IV as that in which symptoms do not occur until at least six months *after* the traumatic exposure (APA, 2000). In fact, since the delayed-onset specifier was introduced in DSM-III (APA, 1980), it has been surrounded by controversy, at the heart of which, is

a question of interpretation. As Andrews and colleagues (Andrews, Brewin, Philpott, & Stewart, 2007) point out in their review of studies on delayed onset, there is no "clarification of whether the delayed 'onset of symptoms' refers to delayed onset of *any* symptoms that might eventually lead to the disorder or only to [the delayed onset of] full-blown PTSD itself" (p.1319). This lack of clarity may lead an individual with initial PTSD symptoms that gradually worsen into full PTSD over a period greater than 6-months to be regarded by some researchers as having delayed-onset PTSD, but not by others. Indeed, in their review of 10 case studies and 19 group studies on this issue, Andrews and colleagues concluded "the discrepant findings in the literature concerning prevalence can be largely, but not completely, explained by definitional issues" (p. 1319). They found that "delayed onset PTSD in the absence of any prior symptoms was rare, whereas delayed onsets that represented exacerbations or reactivations of prior symptoms accounted on average for 38.2% and 15.3%, respectively, of military and civilian cases of PTSD" (p.1319).

Different interpretations of what defines delayed-onset PTSD are reflected in the contrasting ways that studies of veterans before and after 1980 define delayed onset. Retrospective studies of veterans from early wars often record time of onset of *any* PTSD symptoms. Since these studies have generally occurred decades after wartime service, questions about the timing of onset have been asked in terms of which *year* veterans began to experience symptoms. The imprecision of this data precludes obtaining prevalence rates of delayed onset of symptoms beginning six months after war trauma, just as it precludes determining when most individuals developed full-blown PTSD relative to the traumatic exposure. These studies can, however, provide rates of long-

delayed onset, i.e. where individuals who were ultimately diagnosed with the disorder reported not experiencing *any* symptoms until one or more years following the traumatic event.

Publications on war-related PTSD in veterans from wars before 1980 demonstrate inconsistent findings on long-delayed onset of PTSD symptoms. Four studies of former POWs from WWII conducted over 40 years after the war, led to conclusions that long-delayed onset of PTSD symptoms is rare (Kluznik et al., 1986; Port et al., 2001; Speed et al., 1989; Zeiss & Dickman, 1989). Kluznik and colleagues (1986) reported that most ex-POWs in their sample described onset of symptoms at repatriation, and reported only one case of "doubtful" delayed onset whose time of onset was not reported. Speed and colleagues (1989) did not report any cases of delayed PTSD in their sample. All of the veterans whose symptoms were deemed consistent with PTSD in Zeiss and Dickman's sample reported symptoms in the first year they returned from war (Zeiss and Dickman, 1989). Lastly Port and colleagues (2001) found the majority of their participants who endorsed PTSD symptoms experienced them early on, usually in the first year after discharge, and only 2% of their total sample reported "long-delayed onset" which they defined as being without symptoms until the 1970s.

Bremner and colleagues (1996) studied Vietnam veterans in an inpatient treatment program for severe, chronic PTSD comorbid with substance use disorder. These were the only authors who used a timeline to go over events since each veteran's military service, inquiring about symptoms every two years between service and the interview, in an effort to ascertain when veterans first met diagnostic criteria for PTSD. The majority of veterans (77%) reported onset of the disorder within two years of the end of their service.

Furthermore, of the13% of veterans who did not meet full criteria for the disorder until 10 years after service, *all* had begun to experience symptoms within 2 years of repatriation. The authors concluded that symptom onset later than 2 years was rare in their sample.

In contrast to the findings that long delayed onset of symptoms is rare in individuals diagnosed with war-related PTSD, three studies of individuals from early wars found evidence of the phenomenon, of which one was a case study. Van Dyke and colleagues (1985) presented a case study of a WWII veteran whose first symptoms of PTSD reportedly occurred more than 30 years after combat during which he had been able to work productively. His symptoms emerged for the first time when his health deteriorated, forcing his retirement. There are some indicators in this study that the veteran may have been minimizing symptoms. For example, his wife reported that he had always attributed loud noises to gunshot, and avoided watching any movies that were war-related. His presentation 30 years from the war, however, was marked by severe impairments in functioning due to changes in mood, repetitive nightmares, and strong emotional reactions to memories of the war, amongst other symptoms.

Evidence of long-delayed onset of PTSD has also been provided by some group studies. Op den Velde and colleagues (1996), reported that there had often been a delay of several decades between the end of the war and reoccurrence or first onset of posttraumatic symptoms in the WWII Dutch resistance fighters they examined. In one study the same authors found 34% of veterans reported experiencing symptoms for the first time after 1970 (1993, cited in Op den Velde et al., 1996.). Similarly, in their publication on findings from two large, congressionally-mandated studies of PTSD in Vietnam veterans, one of which was the NVVRS clinical subsample, Schnurr and

colleagues (2003) found delayed onset relatively common in the 530 veterans: Whereas 31.4% reported symptoms beginning the same year they entered Vietnam, and 30.5% reported onset in the year after their service, 32% reported symptom onset between two and five years after service in Vietnam and 6.4% reported onset between six and 22 years later. Combining the last two groups, close to 40% of this diverse sample report symptom onset two years or more after repatriation. The findings of these two studies are difficult to reconcile with the numerous aforementioned findings that delayed onset of symptoms is rare.

Studies of veterans from more recent wars record onset of full-blown PTSD as per DSM-III or DSM-IV criteria, but generally do *not* chronicle the onset of PTSD symptoms prior to presumed diagnosis. These studies all report rates of PTSD in veterans at a given time, as measured by a cut-off score on a self-report scale purported to indicate probable diagnosis. The risk in this approach to symptoms and diagnosis is that researchers may overlook the presence of earlier, significant, even pervasive symptoms in individuals whose symptoms do not meet the cut-off score at the time the screen scales were administered. It may be that were the participants administered a structured interview with a trained clinician that investigated time of onset, they would in fact be diagnosed with full-blown PTSD at an earlier time. It is important to recognize that just as there may be onset of symptoms well before an individual meets criteria for diagnosis, when relying on self-report scales, there may be onset of symptoms and even onset of diagnosis before the veteran meets the cut-off score. In this situation, the point at which the individual newly meets the cut-off score may be more accurately termed an *exacerbation* of pre-existing symptoms rather than *onset* of symptoms.

Of the four studies of wars since 1980 that tracked the onset of PTSD longitudinally (Southwick et al., 1995; Wolfe et al., 1999; Grieger et al., 2006; Solomon and Mikulincer, 2006), all demonstrate evidence of small, yet considerable percentages of new "cases" of the disorder over the year, two years, three years, or even twenty years under study, as measured by various diagnostic checklists including the Mississippi Scale for Combat-Related PTSD, the PTSD Checklist, and the PTSD Inventory. None of the studies provide rates of symptoms in veterans prior to their meeting the cut-off score.

In Southwick and colleagues' (1995) 2-year study of a small convenience sample of National Guard reservists who had returned from the first Gulf War one month prior to the baseline assessment, a score of 89 on the Mississippi Scale for Combat-Related PTSD was used to indicate likely PTSD diagnosis, a lower score than some of the other studies. These authors found constant increases of 3.2% new PTSD onset at each of the three points of assessment (1 month, 6 months, and 2 years), such that in total, 9.7% of the sample developed PTSD over the two years. Using a scale based on the DSMI-III-R criteria for comparison, they found 13% of the sample developed PTSD by this cut-off criteria over the two years studied. In a much larger study of Gulf War veterans, Wolfe and colleagues, (1999), used a score of 94 or higher on the Mississippi Scale for Combat Related PTSD, to indicate probable diagnosis five days following veterans' return from the war and again two years later. They found 3% of the sample reached the cut-off score upon return from the war and an additional 6% met the cut-off score two years later. In total, near 9% of the sample developed PTSD over the two years studied, similar to Southwick and colleagues (1995). Unfortunately, the authors made no reference to any assessment in the intervening period, which makes it impossible to know whether the

later onset in cases occurred before or after six months post repatriation, the specified time for delayed onset by DSM-IV standards (APA, 2000). The authors consider possible explanations for the doubled rate of onset over the course of the study, including that the increase is artifactual— i.e. that the disorder was present at the initial assessment but not detected due to its being masked (perhaps by positive mood on return, reluctance to endorse symptoms, or misinterpretations of symptoms); that there were unmeasured events such as history effects that may have heightened sensitivity to symptoms at the later assessment; or that the increase represents true delayed onset, which is the explanation they find most likely. Another possibility is that the veterans with later onset according to cut-off score were in fact symptomatic at earlier assessments, but not sufficiently so to reach the threshold for probable diagnosis.

Grieger and colleagues (2006) studied veterans from wars in Iraq and Afghanistan who were hospitalized with severe combat-related injuries. Using the PTSD Checklist, these authors found rates of new onset increased over the course of the study, with 3.3% new onset at one month, and close to 7% new onset at four months and close to 4% new onset at seven months. Although these percentages are relatively low proportions of the sample at large, they evidence an increasing flow of new PTSD cases over the seven months studied with a total post-injury prevalence close to 15% of the sample (Grieger et al., 2006).

Solomon and Mikulincer (2006) studied 214 veterans of the (1982) Israeli Lebanon War who had fought in frontline battles, of whom 131 had been diagnosed with Combat Stress Reaction (CSR) during the war, and 83 were matched controls who had fought in the same units and battles but had not received such diagnoses. Combat stress

reaction consists of "various polymorphic and labile psychiatric and somatic symptoms and is diagnosed based on impaired functioning by trained clinicians… Symptoms may [include] paralyzing fear of death, emotional and physical numbness, withdrawal, severe depression, and impaired combat functioning" (p. 659). These researchers assessed PTSD symptoms and diagnostic status each year for the first three years following wartime service and again 20 years following wartime service with the PTSD Inventory, a self-report instrument based on DSM-III. Additionally, as a proxy for DSM-IV's Criterion F (evidence of clinical impairment) they used the global severity index from the SCL-90 as a measure of clinical distress and disability. The addition of this latter criterion reduced incidence rates by making it more difficult to fulfill diagnostic criteria. Due to the significantly different findings of PTSD incidence produced by using and not using Criterion F in the diagnosis, the authors published incidence rates according to each set of diagnostic criteria, though data on new onset were only published for the DSM-III criteria after the first assessment point.

Although the new onsets of PTSD over the period studied was not the focus of this study, the publication reveals that at the first point of assessment (one year) 44.9% of the sample met DSM-III diagnosis for PTSD (Solomon & Mikulincer, 2006). At year two, an additional 11.2% of the sample newly met diagnostic criteria, and at year three, 1.9% of the sample newly met diagnostic criteria. Lastly, the authors presented new onset rates for the same sample 20 years after repatriation, finding 6.1% of the sample met diagnostic criteria for the first time in the study. These onset rates add to a total PTSD prevalence rate of 64% PTSD in this sample. New incidence rates are presented by these authors broken down into two groups—veterans who had experienced Combat Stress

Reaction (CSR) during the war and those who had not. The percentage of each group that met criteria for PTSD at one year was considerably higher in the CSR group (64.12%) in contrast to the non-CSR group (14.46%). At subsequent assessment points, however, rates of new onset across the two groups were considerably closer (Solomon & Mikulincer, 2006).

Since this study did not assess veterans for PTSD prior to one year after repatriation, it is impossible to tell which of the 44.9% of the sample that met criteria at year one were technically delayed onset cases (i.e. onset at least six months after the trauma) and which were technically acute onset cases, as per DSM-III criteria. This study does provide evidence for considerable rates of new onset at two years (11.2%) as well as at 20 years (6.1%) after repatriation. This finding should be taken with caution, however, given that using scales to diagnose PTSD may lack precision, since slight fluctuations in symptoms close to the cut-off point for PTSD caseness may lead to some individuals with the disorder not meeting the cut-off score, or vice versa.

In conclusion, as Andrews and colleagues (2007) suggest, discrepancies around the issue of delayed onset in veterans center on whether it is considered to mean a six month delay between traumatic exposure and *any PTSD symptoms*, or a six month delay between traumatic exposure and *full blown diagnosis.* They conclude that delayed onset of diagnosis was more common than delayed onset of symptoms, which was quite rare (Andrews et al., 2007). The present review of publications on war-related PTSD is consistent with that finding. More recent studies on veterans of wars since 1980 all provide evidence of small percentages of new cases of PTSD beyond six months after repatriation, as indicated by cut off scores on checklist measures. These findings should

be interpreted with caution given their reliance on self-report screening measures and cut-off scores. It is possible that these individuals had significant prior levels of symptoms in the intervals before they met the cut-off score and went undiagnosed. If this were true, then the "new" diagnoses would in fact be more like exacerbations of symptoms. Diagnostic assessments, including history, need to be done in person by trained clinicians in order to obviate this lack of precision and to confirm whether similar rates of new clinician-made diagnoses are observed years after the traumatic event.

In sum, there has been very little evidence provided in any of the studies here of long delays between traumatic exposure and the onset of any symptoms. With the exception of one case study (Van Dyke et al., 1985), the retrospective studies of veterans from earlier wars have been collectively better at learning about the timing of symptom onset, though not the timing of onset of diagnosis. These studies all report that the majority of veterans reported onset of symptoms during wartime service or in the one to two years after repatriatriation (Bremner et al., 1996; Kluznik et al., 1986; Op den Velde et al., 1996; Port et al., 2001; 1996; Schnurr et al., 2003; Speed et al., 1989; Zeiss & Dickman, 1989). Although some studies have concluded that long-delayed onset of symptoms up to decades after the war make up a small, but important minority of cases (Op den Velde et al., 1996; Schnurr et al., 2003), others have found rare or dubious evidence or no evidence at all of such delays in their samples (Bremner et al., 1996; Kluznik et al., 1986; Port et al., 2001; Speed et al., 1989; Zeis & Dickman, 1989). The most compelling evidence of delayed onset of symptoms, demonstrated by Schnurr and colleagues, relies on comparing age at onset to age military service, which is an inherently less precise manner to track the timing of events. Furthermore, the reported

rate (34%) is still a minority of the total diagnosed sample and the "delay" reported describes a fairly large period (2-5 years after military service).

Whether delayed onset PTSD is associated with greater severity or greater chronicity of the disorder has also been debated: One study found no measurable differences in PTSD severity found between veterans with delayed onset and those with non-delayed PTSD onset (Watson, Kucala, Manifold, Vassar, & Juba, 1988). Andrews and colleagues' (2007) literature review of studies on delayed onset PTSD concluded that differences in PTSD outcomes between groups of delayed-onset and acute-onset PTSD could not be meaningfully described given the small numbers of study participants with delayed onset that made interpretations of non-significant findings questionable. It remains to be clearly established whether delayed onset PTSD has replicable, negative effects on severity and chronicity of PTSD.

Duration/ chronicity of PTSD. By chronicity of the disorder, I refer to the duration of the period during which an individual continues to meet diagnostic criteria for PTSD. The most accurate findings on PTSD chronicity involve regularly scheduled assessments of whether individuals continue to meet diagnostic criteria at short intervals tracked over long periods time. However, the most common means of approaching the subject is by assessing the prevalence of the disorder in a sample of individuals who have been exposed to war trauma some years after the war, and assuming that individuals meeting criteria for diagnosis at the time of assessment met criteria during the intervening period. It is far from certain, however, that prevalence rates at a current time point are accurate proxies for rates of ongoing, persistent disorder in the intervening period. Often current rates are compared to lifetime rates of war-related PTSD, in an effort to see in

what percentage of persons the disorder has "persisted" compared to the total number of individuals who have ever had PTSD. The term persisted is in quotations here, since there has been no intervening assessment to examine whether the disorder did indeed persist throughout the period.

Four of the publications reviewed on retrospective studies of veterans from WWII provided rates of current PTSD several decades after the war, but did not assess lifetime rates of the disorder (Dirkzwager et al., 2001; Op Den Velde et al., 1996; Port et al., 2001; Zeiss & Dickman, 1989). Dirkzwager and colleagues examined a sample of Dutch veterans from the community and found a 9% rate of current PTSD, which the authors compared to a 27% current rate in a sample of Dutch WWII veterans receiving military disability pension. Similarly, Port and colleagues, who studied ex-POWs of WWII and the Korean War, found that 27% met criteria for current PTSD about 4 to 5 decades later, though this number increased to 34% just four years later, and they estimated both of these rates to be substantially lower than the prevalence rates in the first six years after repatriation. Zeiss and Dickman (1989) examined ex-POWs from WWII and found the current rates to be higher than the other studies—55.7%. Op den Velde and colleagues' (1996) concluded that between 25 and 50% of the 1,098 veterans from the Dutch Resistance during WWII studied continued to suffer with PTSD between four and five decades after the end of the war, and they estimated the lifetime prevalence to be substantially higher. The measures used in each study to confirm or indicate presumed diagnosis are listed in Table 1.

Five retrospective studies of early wars published lifetime prevalence rates in addition to current prevalence rates (Dohrenwend et al., 2006; Kluznik et al., 1986; Speed

et al., 1989; Schnurr et al., 2003, Solomon & Kleinhauz, 1996). Two studies examined ex-POWs from WWII in the 1980s and both found particularly high rates of current disorder (Kluznik et al., 1986; Speed et al., 1989). Kluznik and colleagues (1986) found that 67% of their sample had lifetime PTSD, and 47% of the total sample met diagnosis at the time of the study, while Speed and colleagues (1989) retrospectively assessed prevalence of the disorder at one year after repatriation, finding a prevalence rate of 50%, and a current rate of 29% of the total sample. In two other studies, lifetime rates were twice as large as current rates (Dohrenwend et al., 2006; Schnurr et al., 2003). Schnurr and colleagues (2003) found 30.6% of the Vietnam veterans in their study had met diagnostic criteria for full DSM-III-R PTSD at some point, and 14.7% met diagnostic criteria for full PTSD at the time of the interview; Dohrenwend and colleagues (2006), who re-analyzed the NVVRS clinical subsample that was included in the Schnurr study (2003), adjusting for documented exposure to trauma and the DSM-IV criterion F, functional impairment, found a lifetime PTSD rate of 18.7% and a current PTSD rate of 9.1%. Lastly, Solomon and Kleinhauz (1996) found that 30% of their entire sample of Israeli veterans of the Yom Kippur war had lifetime war-related PTSD and 7% met diagnostic criteria at the time of the study.

The main objective of the study by Solomon and Kleinhauz (1996) was to compare PTSD prevalence and symptomatology in veterans who experienced Combat Stress Reaction during combat with matched veterans who had not experienced Combat Stress Reaction. Consequently, these researchers divided the sample into groups with and without CSR according to CSR diagnoses made during war according to the military record. They found a lifetime PTSD prevalence of 38.4% in those veterans who had been

treated for CSR, and a current prevalence of 13.4%; in those who had never been treated for CSR they found lifetime prevalence of 14.3% and current prevalence of 3.2%. The authors conclude that time had a significant impact on recovery from PTSD in both groups, though veterans who had experienced CSR were at greater risk for both onset and chronicity of PTSD.

In sum, retrospective studies of war-related PTSD on veterans from early wars demonstrate abundant evidence of the disorder's persistence over decades. The four longitudinal studies of veterans from more recent wars that tracked diagnostic status over time corroborate this finding, albeit over shorter periods of time (Grieger et al., 2006; Solomon and Mikulincer, 2006; Southwick et al., 1995; Wolfe et al., 1999). Since these studies have the advantage of assessing probable diagnostic status at different intervals, they also provide the changing rates of recovery at multiple points along the disorder's course.

Southwick and colleagues' (1995) study of Gulf War veterans at three time points over two years after service found no evidence of the disorder remitting. In the much larger study by Wolfe and colleagues (1999), 3% of the sample met the cut-off score for presumed diagnosis at 5 days after repatriation, and one third of these (i.e. 1% of the sample) no longer met the cut-off score at two years after repatriation. At two years, 8% of the sample met cut-off for probable diagnosis, of which one quarter had met the cut-off score at baseline, and the rest (6%) were new cases. Since the strongest predictors of PTSD at the second assessment were presumed PTSD diagnostic status at the first assessment and the level of combat exposure, these authors concluded that PTSD is a "relatively stable, chronic condition." Grieger and colleagues (2006) found that just over

half of seriously injured veterans who met cut-off for probable PTSD at one month continued to do so at four months. Likewise, close to half of the veterans who met PTSD criteria at four months met criteria at seven months. As a consequence of these trends and factoring in new cases of presumed PTSD at 4 months and 7 months, most veterans with PTSD at seven months did not meet criteria had not met the cut-off score for presumed diagnosis at one month.

Solomon and Mikulincer's (2006) study found that 44.9% of their total sample met the cut-off score for presumed PTSD diagnosis in the first year. Three fifths of these veterans no longer met the cut-off score by year 3, whereas the disorder persisted in the other two fifths. By 20 years after the war, close to one third (30.2%) of those with PTSD at the first assessment continued to meet the cut-off score for presumed diagnosis. Of those who did not experience PTSD onset until the second year of the study, a similar percentage (29.2%) continued to meet diagnosis at year 3 and at year 20. Lastly, of those who did not experience PTSD onset until the third year of the study, 50% met criteria the cut-off score again and 50% no longer met diagnostic criteria at year 20. In this study later onset of presumed diagnosis appears to have been related to greater rates of the disorder's persistence.

In sum, the four studies reviewed that tracked individuals' war-related PTSD diagnostic status longitudinally over time all demonstrated the disorder's chronicity, with rates of persistence amongst those who meet cut-offs for presumed diagnosis early on ranging from 30% over 20 years (Solomon & Mikulincer, 2006), to 41% over three years (Solomon & Mikulincer, 2006), to 66.6% (Wolfe et al., 1999) or 100% (Southwick et al., 1995) over two years. Evidencing the disorder's stability, these studies show that one of

the most reliable predictors of current war-related PTSD is having been diagnosed with the disorder at some point in the past.

Re-exacerbation of PTSD. In this literature review I have defined as re-exacerbation the phenomenon whereby PTSD is diagnosed in an individual who subsequently fails to meet diagnostic criteria, and proceeds to once more meet diagnostic criteria at a third assessment point. It differs from the term *exacerbation*, which I used in the section on onset, because exacerbation refers to the worsening of pre-existing symptoms that results in the veteran's newly meeting diagnostic criteria or the cut-off score for presumed diagnosis. Preliminary evidence of the phenomenon of re-exacerbation has been presented in retrospective studies of veterans from wars before 1980. Zeiss and Dickman (1989) reported that 62% of their sample was "intermittently troubled" by symptoms over 40 years since their wartime service, while Port and colleagues (2001) reported that 11% of their sample had been seriously troubled by symptoms during one period after the war, had *not* been troubled in a subsequent period, and returned to being seriously troubled again in a later period.

Dirkzwager and colleagues (2001) provide further evidence of the fluctuating course of PTSD. Dutch WWII veterans were assessed in 1992 and 1998, and the authors found the prevalence of presumed PTSD increased slightly but not significantly, from 27% to 29%. Despite this seeming stability, 9% of the sample met the cut-off score for probable diagnosis in 1992 alone, and 10% met the cut-off score in 1998 alone. The authors emphasize that caseness at each time point was not only significantly associated with caseness at the other time point, it was also significantly associated with the severity of symptoms at the other time point. In other words, the 19% of veterans who

only met the cut-off score at one of the two time points were at much greater odds of having severe PTSD symptoms at the other time point, even though they scored below the level of presumed diagnosis. These findings are important not only because they evidence the persistence of PTSD symptoms, but also because they evidence the fluctuations in diagnostic status in a sizeable portion of traumatized individuals who hover just below or just above meeting diagnostic criteria. Lastly, this study found no significant relationships between post-war stressful life events, measured by a checklist, and changes in PTSD rates or severity.

Further evidence of re-exacerbated PTSD was presented in one of the longitudinal studies of veterans of later wars. Solomon and Mikulincer (2006) tracked PTSD status at yearly intervals for three years, and again at 20 years after service, showing evidence of onset, remittance, and subsequent re-exacerbation of the full-blown disorder. In their 20-year study, over one half of those individuals whose probable disorder remitted after having met cut-off score in the first two years after repatriation ultimately met cut-off scores for presumed diagnosis again in subsequent years.

Although there have been attempts to understand reexacerbation (and indeed increases in prevalence and long-delayed onset) as a function of post-war life stressors (Bonwick and Morris, 1996; Dirkzwager et al., 2001; Morgan, Hill, Fox, Kingham, & Southwick, 1999; Van Dyke et al., 1985), there has been little evidence to corroborate such a relationship. The only study on the course of war-related PTSD reviewed here that examined the influence of subsequent stressful life events on changes in PTSD prevalence or symptom severity found no associations (Dirkzwager et al., 2001). In their study of a very small sample of Vietnam veterans who resided in the U.S. during the

Persian Gulf War, Leetz, Martino-Saltzman, & Dean (1993) found no evidence of worsening PTSD symptomatology during the onset of air and ground combat in the Persian Gulf. In an overview of the current knowledge on PTSD in elderly war veterans, Bonwick and Morris (1996) reported that "the reemergence of reexperiencing and arousal symptomatology, especially intrusive recollections, nightmares, insomnia, and hypervigilance in response to a stressor is a common feature…" (p. 1074), but they cite only one study that has reported such a phenomenon. These authors discuss stressors associated with later life (i.e. retirement, physical illness, loneliness, bereavement, cognitive impairment, and diminution of family responsibilities), as being potential triggers for re-exacerbation, but the few publications that have examined the influence of post-war life stressors on reexacerbation of symptoms have failed to substantiate this relationship (Dirkzwager et al., 2001; Leetz et al., 1993; Sungur and Kaya, 2001).

There are a couple of exceptions to this: Moyers (2004) outlines theoretical processes through which re-exacerbation may occur, and presents anecdotal cases demonstrating exacerbated PTSD symptoms in veterans of World War II, the Korean War, and Vietnam in the context of the 1995 Oklahoma City bombing. Furthermore, Morgan and colleagues (1999) found evidence of "anniversary reactions" in Gulf War veterans six years after the conclusion of the war, and McCarroll and colleagues (1997) found evidence of re-exacerbation of symptoms with the second deployment of veterans who had met criteria for PTSD diagnosis after their first deployment. The relationship between life events and the exacerbation of PTSD symptoms remains to be clearly elucidated in the literature.

Change in symptoms over the course of war-related PTSD. Central to any discussion of disorder course is the question of how symptoms progress over time in individuals diagnosed with PTSD. It is not uncommon for researchers to take prevalence data at two points in time and assume that symptoms and/or diagnostic status have remained static in the intervening period. For example, Speed and colleagues (1989) describe the 29% of POWs in their sample who met diagnostic status forty years after repatriation as having "severe persistent" PTSD, implying a consistent level of severe symptoms. This description stems in part from their finding that the best predictor of diagnostic status at the time of the study was the severity of PTSD that POWs described having one year after repatriation (Speed et al., 1989). These authors did not inquire, however, as to whether the veterans experienced periods of remission or relapse over the four intervening decades, a fact that leaves open the possibility that the course of each POW's PTSD may in fact have been something other than persistently severe.

It is also common to extrapolate information about the changes in symptoms over time from data on the prevalence of certain symptoms in the population at various time points. Such approaches provide *population averages* that are informative as to population trends, but should *not* be confused with the course of the disorder in individuals. Kluznik and colleagues (1986), for example, surmise that PTSD symptoms in former POWs of WWII initially persist over months or years and then undergo "a gradual diminution" (p.4) with complete remission in some. This statement is apparently drawn from their finding that while 67% of their sample experienced PTSD at some point since the war, this reduced to a current incidence of 40% at the time of the study, of which the

majority continued to experience mild, rather than moderate or marked, symptoms (Kluznik et al., 1986).

Although the conclusion drawn by the authors may well be true, there are two possible errors in their logic. First, as in the case of Speed and colleagues (1989), the assumption is that a reduction in prevalence from 67% lifetime to 40% at the time of the study implies a straight line from time A to time B which would demonstrate that prevalence of PTSD in this population steadily reduces over time. Since these authors have not taken assessments over time longitudinally, it cannot be confirmed that the prevalence curve for the population is a straight, negatively sloped line. Secondly, though the overall trend for the population may indeed be a diminution in symptoms, that does not necessarily mean that change in symptoms for each individual follows the average course for the population. Such a conclusion would only be valid if symptoms were measured at multiple time points over the course of the disorder for each of the individuals. Data about average symptom change across the population do not inform us about the course of the disorder in individuals. Six publications on studies on war-related PTSD in veterans from early wars provide information about changes in individuals' PTSD symptoms over time (Bremner et al., 1996; Dirkzwager et al., 2001; Johnson et al., 2004; Op den velde et al., 1996; Port et al., 2001; Zeiss & Dickman, 1989), while four studies of war-related PTSD in more recent wars are also relevant to this discussion. In a number of these, changes in PTSD symptoms are tabulated for the entire sample without distinguishing between those who do or do not meet criteria for PTSD diagnosis.

In studies by Zeiss and Dickman (1989) and Port and colleagues (2001) ex-POWs of WWII were asked, *regardless of their diagnostic status*, if they were "seriously

troubled" by PTSD symptoms across all time intervals including one year after repatriation, five years after repatriation, and in the several subsequent decades. In the earlier study, 60% of POWs reported having been seriously troubled by symptoms in the first six years after repatriation, whereas this percentage dropped to 48% in three decades prior to the study (Zeiss & Dickman, 1989). Port and colleagues (2001) found that similarly high percentages of their sample reported being seriously troubled by symptoms in the first six years after repatriation (close to 70% in the first year and close to 60% in the next five years), and this percentage dropped precipitously each decade thereafter, bottoming out at close to 20% in the 1970s.

Port and colleagues (2001) additionally reported that, after bottoming out in the 1970s, the percentage of seriously troubled POWs increased significantly in the 1980s and 1990s, though not to the earlier percentages. Furthermore, the increase in numbers of veterans who reported being seriously troubled coincided with an increase in disorder prevalence over the same period. The authors suggest that their findings "contradict the notion that PTSD is stable overall in its chronic form or that it uniformly decreases in severity over time" (p. 1477; Port et al., 2001).

Lastly, Port and colleagues (2001) provided percentages of participants who reported not being seriously troubled in any time period (16%), those who reported being seriously troubled in the first six years after repatriation only (29%-- the largest group), those who reported being seriously troubled through the first 26 years but not thereafter (17%), and those who were seriously troubled five of the seven periods (18%). Other categories included individuals who reported being seriously troubled for the first time after 1970 (i.e. over 25 years after the war) (2%), and those who reported being seriously

troubled in the first time intervals, then not being troubled for at least three periods, and then being troubled again (11%).

The distribution of veterans endorsing such varied categories provides a window into the diversity of veterans' experience of PTSD symptom course, a finding that was also demonstrated in two other studies (Op den Velde et al., 1996; Zeiss and Dickman, 1989). Op den velde et al. (1996) noted that veterans of the Dutch Resistance described a variety of disorder trajectories including acute onset, delayed onset, persistence of symptoms, as well as amelioration and reoccurrence of symptoms across the groups of veterans examined, though these authors did not provide information on the percentage of veterans endorsing each trajectory. Zeiss and Dickman (1989) inquired if veterans were never troubled, were continuously troubled, or were intermittently troubled over the 40 years since their service. Almost one quarter of the sample (23.5%) reported having been continuously troubled by symptoms over four decades, 13.8% reported never having been troubled, and by far the largest group (62.2%) reported being intermittently troubled, though no clear patterns in this changing course were evident (Zeiss & Dickman, 1989).

Although veterans who did not meet criteria for diagnosis were included in each of these samples, the conclusion here is clear: the number of people seriously distressed by PTSD symptoms after war decreases after the first six years. Although individuals may experience persistent, high distress after the first six years, such a group, if it exists, seems to comprise a minority of cases. In each of these samples of traumatized veterans, progressively fewer veterans over the first few decades report severe symptoms. There may, however, be increases in severe symptoms amongst some veterans four to five decades after the war, and there may be individuals who newly meet diagnostic criteria

this many years later, having not met diagnostic criteria for at least some of the intervening period. Lastly, the specific course of symptoms may vary from one individual to the next. Such variability has been corroborated in a number of longitudinal non-military studies of PTSD as well (e.g. Hepp et al., 2008; McFarlane, 1988; Perkonigg et al., 2005; Sungur and Kaya, 2001).

Two studies of veterans with severe, chronic PTSD admitted to inpatient treatment facilities reveal further information about changes in symptoms (Bremner et al., 1996; Johnson et al., 2004). In the study by Johnson and colleagues (2004), veterans were assessed on admission and six years later, and demonstrated increases in hyperarousal symptoms and social isolation, though none in reexperiencing or avoidance symptoms, and no significant change in total PTSD symptom scores over six years. This finding contrasts with that of Bremner and colleagues (1996) who examined a sample of veterans with severe, chronic PTSD and comorbid substance use disorder. They tabulated the average number of PTSD symptoms each veteran experienced during service and then two years, four years, and so on, for over two decades after veterans' service in Vietnam. The average number of symptoms reported increased from five symptoms during service to eleven two years later, and to an average of fifteen symptoms by the time of the interview. The authors conclude that symptoms typically increase after the war and then plateau, becoming chronic and unremitting. This statement is likely true for much of the sample; however, since it is based on population averages, it should not be assumed to describe each individual's disorder course. The averages may mask individuals who had periods during which they experienced markedly higher or markedly lower numbers of symptoms. Furthermore, there may be subgroups of veterans with persisting, high

numbers of symptoms that mask symptom patterns of others that fluctuate over time. These two studies provide evidence that veterans with the most severe, chronic form of PTSD experience persistence of symptoms and may experience increases in symptoms, even decades after their service.

Amongst the longitudinal PTSD studies carried out with veterans from more recent wars reviewed here, five sets of researchers focused on changes in PTSD symptomatology over time, either with (e.g. Southwick et al., 1995) or without (e.g. McCarroll et al., 1995; Orcutt, Erickson, & Wolfe, 2004; Solomon & Mikulciner, 2006; Thompson et al., 2004) including information on proportions of participants meeting diagnostic cut-offs for PTSD.

In an early study of veterans returning from the Persian Gulf War of 1990-1991, McCarroll and colleagues (1995) sought to test the hypothesis that male and female veterans who had handled human remains during their time at war would develop higher levels of PTSD symptoms compared to veterans who had not handled human remains. None of the veterans in this sample had experienced combat directly. This investigation provides an early window into the changes in posttraumatic symptoms between an assessment at 3 months and one at 15 months after veterans' return to the U.S. The authors did not report whether veterans met diagnostic criteria for PTSD. They found that the severity of symptoms, including avoidance, intrusion, and a total symptom scale, *decreased* for both groups studied over the year, though individuals who had handled human remains during the war had higher levels of symptoms at all time points than those who had not. In contrast, Southwick and colleagues (1995) found that from 1 month

to 6 months, and from 6 months to two years, PTSD symptoms *increased* over time, with the largest increases in the first six months.

In the 20 year study of Israeli combat veterans by Solomon and Mikulincer (2006), researchers tracked changes in veterans' PTSD symptoms, regardless of diagnostic status, at four assessment points. This approach is similar to that taken by Thompson and colleagues (2004) and Orcutt and colleagues (2004), who measured PTSD symptoms in veterans at two and three assessment points respectively, without regard to diagnostic status. Similar to McCarroll's findings on the impact of handling human remains, Solomon and Mikulincer (2006) found that a history of CSR in war had a significant main effect on the number of symptoms experienced after the war at all time points. Additionally, for both veterans with and veterans without histories of CSR, the number of total PTSD symptoms decreased very slightly in the first two years after wartime service, then decreased significantly between year two and year three, and increased significantly at year 20, approaching the mean number of symptoms endorsed during the second year after service (Solomon & Mikulincer, 2006).

Solomon and Mikulincer (2006) present graphical profiles of PTSD symptoms at each assessment point for veterans with a history of CSR, and separately for veterans with no history of CSR. Since veterans with CSR were 6.6 times more likely to meet criteria for PTSD at all four time points and had incidence rates of PTSD as high as 64%, compared to 6% of those without histories of CSR, this review will focus on symptom profiles for veterans with histories of CSR. For every year assessed, the authors graphed the percentage of veterans endorsing each PTSD symptom. At all four time points (years 1, 2, 3, and 20), the proportion of the various PTSD symptoms to one another remained

the same, whether they were endorsed at high or low levels: The following symptoms—recurrent images and thoughts about the war, hyperalertnesss, and intensification of symptoms following experiences reminiscent of the war— were endorsed by most veterans; in contrast, survivor guilt, constricted affect, and guilt about functioning, were endorsed by few veterans at all time points. The only year that proportions of symptoms endorsed changed slightly was year 20, when veterans endorsed less avoidance of activities recalling the war than they had done in prior years, and when sleep difficulties and memory difficulties were endorsed in slightly higher proportions than other symptoms, perhaps due to the aging process (Solomon & Mikulincer, 2006).

Thompson and colleagues (2004) used the PTSD Checklist to assess specific symptom clusters at two time points: 14 and 27 months after veterans returned from deployment in the Gulf War. Although these authors did not assess diagnostic criteria for PTSD, they found that overall PTSD symptomatology increased over time. This was due to increases in the emotional numbing and hyperarousal symptom clusters, a finding that echoes that of Johnson and colleagues (2004) who saw increases in social isolation and hyperarousal over six years in a severe, chronic PTSD population. In contrast, levels of avoidance and re-experiencing symptoms remained static. These authors also found that levels of emotional numbing and hyperarousal symptoms at 1 year post deployment predicted generalized distress, depression, anxiety, hostility, and somatic symptoms a year later, whereas other PTSD symptoms did not (Thompson et al., 2004).

Orcutt and colleagues (2004) presented a 6-year study of PTSD course that examines the Millenium Cohort, a sample of 2,949 veterans of the Gulf War (see also Wolfe et al., 1999). Regardless of PTSD diagnostic status, the authors assessed PTSD

symptoms of all veterans at three intervals: five days, 18 months, and 72 months after returning from deployment, finding further evidence of increased PTSD symptomatology over time. The authors applied growth mixture modeling, a specialized structural equation modeling approach, in order to examine whether the data consisted of different groups of symptom trajectories. They found that the data best fit a two-group model of disorder course represented by two distinct growth curves—one that began with low levels of PTSD symptoms and demonstrated little increase over time, and the other with higher levels of initial symptoms that increased significantly over time.

In conclusion, four of the five prospective studies reviewed here that tracked changes in PTSD symptoms over time (McCarroll et al., 1995; Orcutt et al., 2004; Solomon and Mikulincer, 2006; Thompson et al., 2004) did *not* distinguish PTSD cases from noncases. Two of the studies demonstrated decreases in total numbers of PTSD symptoms over the first two or three years after wartime service (McCarroll et al., 1995; Solomon and Mikulincer, 2006), whereas two other studies found evidence of increases in PTSD symptoms over the first two years after repatriation (Southwick et al., 1995; Thompson et al., 2004). Two other longer studies found evidence of increasing symptomatology in veterans followed for six years (Orcutt et al., 2004), and 20 years (Solomon and Mikulincer (2006). Studies that have examined specific symptom clusters reveal that increases in overall symptoms are due primarily to increases in hyperarousal symptoms (Johnson et al., 2004; Southwick et al., 1995; Thompson et al., 2004) and emotional numbing symptoms (Thompson et al., 2004).

The only longitudinal study found for this literature review that examines and graphically charts groups of similar PTSD trajectories, while relying exclusively upon

data from diagnosed participants, is a study of non-war related PTSD (Hepp et al., 2008). These authors assessed 90 severely injured accident victims in a hospital ICU within one month of the accidental trauma, at 6 months, 12 months, and 36 months after the trauma. To more closely examine different courses of PTSD, the authors began by limiting their data to "cases" that met diagnosis at some point over the 3-year study, defined by scoring above the cut off of 30 on the CAPS (Clinician-Administered PTSD Scale).

Hepp and colleagues (2008) found three separate groups of diagnosed cases, categorized according to common changes in course. Cases in the "decreasing" group all began the four-point assessments with a caseness score. It should be noted that this assessment occurred only 2 weeks after the trauma, so these participants could not, by definition, meet criteria according to DSM-IV. By the second assessment at 6 months, these participants' scores on the CAPS decreased to a sub-threshold level, and over the last two assessments they continued to remain at this low level. It may be that the trajectory graphed for members of this group describes that of a "normal" response to trauma, and that many of the participants in this group never met DSM-IV criteria for PTSD. The second common PTSD trajectory was that of the "increasing group." These individuals had some symptoms of PTSD at two weeks, though the majority did not score above 30 on the CAPS. They all showed steady increases in PTSD symptoms, to the extent that each of them ultimately met the cut-off score for PTSD. 70% of them met the cut-off score at 6 months, and of these, half reached plateaus at high levels of symptoms over the next 2.5 years, while the other half decreased over the same period. The other 30% of this group had symptoms that increased steadily over all four assessments, ultimately meeting the cut-off by one year, and continuing to increase gradually

thereafter. Lastly, the "delayed increasing group" consisted of individuals who generally had severe symptoms at the first assessment, had significantly decreased symptoms at 6 months, with almost half of them not meeting criteria at that assessment, and then demonstrated significant increases in symptom level at one year or three years, at which points those who had fallen below threshold reached, or superceded, threshold for caseness. Both the increasing group and the delayed increase group reported significantly more stress due to life events at each time point, and described higher rates of somatic complaints, litigation, and compensation claims (Hepp et al., 2008).

Study Goals and Hypotheses

A number of conclusions can be drawn from this review of the literature. Although the prevalence of war-related PTSD in veterans generally appears to diminish over many years or decades, the disorder persists in significant numbers of individuals, particularly those who have experienced the most severe traumas. Furthermore, there is evidence for a variety of disorder course trajectories, with symptom improvement in some individuals, persistence or worsening of symptoms in others, and intermittent re-exacerbations of symptoms in others. Stage theories of PTSD course generally describe a worsening of symptoms, which leads to opportunities for working through and processing the traumatic experience with subsequent progression towards health, though this progression does not always take place (e.g. Horowitz, 1978; Frederick, 1985; Wang & Wilson, 1996). Individuals may get stuck in one stage (Horowitz, 1978) or cycling through stages without long-lasting improvements in symptoms (Wang & Wilson, 1996). No study was found that clearly delineated different kinds of course profiles, their

associations with other PTSD outcomes, and their relationships to pre-, peri- and post-war risk factors for PTSD.

This study will provide the first comprehensive description of graphs of the course of war-related PTSD and will examine the relationships between profiles of disorder course and PTSD outcomes. It will also attempt to shed light on the factors related to different kinds of course graphs including pre-war vulnerabilities, war-zone stressors, and post-war variables. By examining these relationships, the graphs will provide a window into the processes that may underlie different courses of PTSD and in so doing, may further our understanding of how these processes differ across veterans afflicted with war-related PTSD.

The central hypotheses of this study are that the shapes of graphs of PTSD symptom change indicate subtypes of PTSD course, and that different subtypes will be related to better or worse PTSD outcomes. It is presumed that course subtypes are a function of underlying processes of PTSD. These processes may be affected by preexisting vulnerabilities, peritraumatic factors related to the situation of the trauma, and/ or post-war factors. This study has the following goals: To thoroughly describe the features of each of the course subtypes identified; to examine the relationship between course subtypes and demographic characteristics; to establish an association between course subtypes and PTSD outcomes; to examine the relationships between course subtypes and pre-war vulnerability factors, peritraumatic exposure factors, and post-war factors known to affect PTSD outcomes; and to investigate whether the subtype of PTSD course that a veteran experiences moderates the dose-response relationship.

Part I: The development of course profiles

The first part of this study will answer the following question: *On the basis of veterans' responses in the clinical interviews, how do clinicians portray the continuous change of veterans' symptoms over the course of their war-related PTSD?* This will involve an examination of 84 clinician-drawn graphs of PTSD course, drawn with the intention of capturing visually the changes in veterans' PTSD symptoms over nearly two decades after their military tours.

A preliminary analysis of the graphs in this study revealed three subtypes or profiles of curve depicting a) single-peak course: featuring acute onset followed by gradual improvement over time; b) flat course: featuring persistent symptoms with little change over time, and c) multi-peak course: in which symptoms fluctuate over time. The goals for the first part of this dissertation are to develop codes that describe the course profiles that have been identified based on common graphical features; to subject these codes to a test of inter-rater reliability; to provide a descriptive analysis of qualitative differences between and within each of the subtypes identified; and to identify common aspects of PTSD course from the quantitative data that co-vary with the identified sub-types.

Part II: The association of course profiles with course outcomes

Hypothesis 1. *Profiles of PTSD course will differ systematically on demographic characteristics of race/ ethnicity, level of pre-military education, level of cognitive ability, and age.* The association between demographic factors and PTSD course profiles will be examined to determine whether any of these factors should be controlled for in later analyses. It has been demonstrated in prior studies that these four demographic

characteristics are related to PTSD outcomes, especially for military populations (Brewin et al., 2000). Their effects may be small relative to peritraumatic and posttraumatic factors (Green et al., 1990b; Ozer et al., 2003). The smallest of these is expected to be race/ ethnicity, whose effects on PTSD outcomes have been explained by other demographic factors and severity of combat exposure (Dohrenwend et al., 2008).

Hypothesis 2. *Profiles of PTSD course will predict PTSD outcomes, with single-peak graphs being associated with better outcomes and flat graphs and multi-peak graphs being associated with worse outcomes.* Four PTSD outcomes will be measured: PTSD diagnostic status at the time of the interview over a decade after the war; rates of symptoms in each of the three symptom clusters; rates of improvement in symptoms at the clinical interview relative to the worst period of symptoms, controlled for severity at the worst period; and membership into one of five ranked ordinal categories of symptom change over time.

It is expected that veterans with single-peak symptom course will have better PTSD outcomes at the time of the interview than veterans with other subtypes of PTSD course. This is in part inherent to the definition of the single-peak course code. The graphical courses of PTSD in individuals in this subtype all have a unique period of exacerbated symptoms (single peak), and in most cases, this peak is followed by reductions in symptoms over time. This expectation is also based on the presumption that the course of PTSD in individuals with single-peak disorder course takes the form of a protracted version of PTSD symptoms in traumatized individuals that never meet criteria for PTSD diagnosis. In healthy individuals posttraumatic symptoms emerge, escalate, and subside over the first month after the traumatic event, and it is expected that in veterans

with single-peak courses of PTSD, the disorder will emerge and peak over several months or years, and then subsequently subside with the passage of time.

Secondly, I expect to find that individuals with flat or multi-peak PTSD course subtypes will have worse PTSD outcomes at the time of the interview. Again, this expectation is in part based on the overlap between course and outcome: By definition, flat graphs depict symptoms that persist at the same level throughout the disorder's course and multi-peak graphs depict symptoms that are prone to exacerbations secondary to temporary improvements. This expectation is also based on the literature: Two studies reviewed here of veterans with severe, chronic war-related PTSD over a decade after the war presented longitudinal evidence of persistent or worsening symptoms over time (Bremner et al., 1996; Johnson et al., 2004), and a stage theory based on clinical observations of veterans with severe, chronic war-related PTSD depicted repeated cycles through stages of relatively better and extremely impaired functioning (Wang & Wilson, 1996). It is presumed that flat and multi-peak course subtypes indicate the failure or absence of an underlying adaptive mechanism that regulates the emotional and physiological dysregulation precipitated by the traumatic experience. This failure may be related to pre-war vulnerability factors, peritraumatic exposure factors, or postwar factors.

Part III: The association of course profiles with pre-war vulnerability, war-zone exposure, and post-war factors.

Hypothesis 3. *Subtypes of PTSD course are expected to differ on vulnerability factors that predispose individuals to worse outcomes of PTSD including pre-military psychiatric disorder, pre-military conduct disorder, history of physical abuse as a child,*

and family history of substance abuse. The rationale for this hypothesis is that since PTSD outcomes are known to be associated with these four vulnerability factors (Bremner et al., 1993; Donovan et al., 1996; King et al., 1996; Schnurr et al., 2004), albeit to a lesser degree than the more proximal, peritraumatic exposure variables (Brewin et al., 2000; Green et al., 1990a; Green et al., 1990b; Ozer et al., 2003), and since course subtype is believed to be related to better (in the case of single-peak graphs) or worse (in the case of flat or multi-peak graphs) outcomes, then course subtype is expected to be related to vulnerability factors.

Hypothesis 4. *Subtypes of PTSD course will differ systematically on exposure variables established as being risk factors for PTSD, including combat exposure, high life threat during service, obtaining a purple heart, seeing a friend in the unit killed, being betrayed in a life-threatening situation, killing an enemy, or doing harm to civilians and/ or prisoners.* This hypothesis is based on prior findings that exposure variables have the most robust association with persistence of PTSD (Dohrenwend et al., 2006; Green et al., 1990b; Grieger et al., 2006; Kulka et al., 1990; Roy-Byrne et al., 2004; Stein et al., 2005; Wolfe et al., 1999). Since PTSD course profiles are expected to be associated with outcomes, and outcomes have been shown to have associations with exposure variables, I reason that course profiles will be related to exposure variables.

Hypothesis 5. *Subtypes of PTSD course will differ systematically on the following post-war variables: lifetime onset of psychiatric disorders during or after Vietnam, and mental health treatment history during or after Vietnam. Subtypes of PTSD course will not differ systematically on post-war stressful life events.* I expect to find that PTSD course profiles will be most significantly related to comorbid psychiatric disorders, since

previous findings have established a relationship between these disorders and severe, chronic PTSD (Blank, 1993; Bremner et al., 1996; Davidson et al., 2004; Dirkzwager et al., 2001; Kulka et al., 1990), and since I expect to find course profiles are related PTSD outcomes. I expect that PTSD course profiles will be related to treatment history since treatment history is likely to be a marker of more severe, persistent PTSD. This expectation is based on the consistent findings that military populations are especially vulnerable to chronic, severe PTSD, irrespective of treatment (Blank, 1993; Kessler et al., 1995), and the expectation that outcomes will be related to course subtypes. I expect to find no association between PTSD course profiles and stressful life events, based on the absence of consistent findings of such an association in the literature (Dirkzwager et al., 2001; Leetz et al., 1993; Sungur & Kaya, 2001).

Hypothesis 6. *It is hypothesized that PTSD course subtype will moderate the established association between level of combat severity and the PTSD outcome, failure to remit (i.e. current diagnosis).* In the event that PTSD course profile is found not to be related to the strongest exposure variable, level of combat severity, in Hypothesis 4, this will provide support for the theory that course subtype is distinct from course outcomes, in being less clearly associated with exposure variables. I expect to find that for some course subtypes, the impact of exposure variables on outcomes is stronger than for other course subtypes, for which the impact of personal factors may be stronger. I will test the relationship between level of combat severity and disorder persistence (i.e. current diagnosis) separately across different course profiles to see if this relationship is moderated by PTSD course subtype.

CHAPTER III: Methods

Participants

The National Vietnam Veterans Readjustment Study (NVVRS) involved a representative sample of 1200 veterans who were administered the National Survey of the Vietnam Generation (NSVG; Kulka et al. 1988). Since there was no master list of the more than 8 million veterans who served in the military during the Vietnam era, a preliminary goal was to create one from which veterans could be selected. This sampling frame was created from the following military records: The National Personnel Records Center, the Defense Manpower Data Center, and a Veterans Administration list purported to comprise all female theater veterans. Since participants were dispersed throughout the nation and sometimes abroad, researchers relied on an interagency agreement with the IRS to acquire current addresses for all participants. Furthermore, other tracing procedures were employed to find individuals whose addresses were not supplied, and for those whose information was inaccurate.

A core feature of the NVVRS was its intention to study nationally representative samples of Vietnam theater veterans, era veterans, and non-veterans or civilian counterparts matched in age, sex, race/ethnicity, and (for women) occupation. Although the current study will address only male theatre veterans, the original NVVRS studied as contrasting groups male and female veterans, African-American, White, and Hispanic male veterans, nurses and non-nurse female veterans, and theater veterans with service-connected disabilities as well as those without. A full, national probability sample was recruited with women, African-American and Hispanic males, and disabled veterans oversampled.

Of the original 1200 veterans, a subsample of 260 theatre and era veterans was administered a Structured Clinical Interview for DSM-III Disorders (SCID; Spitzer et al. 1987) by doctoral-level clinicians. The 260 participants in this clinical sample were drawn from veterans who lived in or near one of 28 metropolitan regions. Individuals who appeared from the preliminary survey to have PTSD based on screening scales and those who reported high exposure to war-zone stressors were over-sampled. Veterans who were diagnosed with PTSD prior to the war, or those diagnosed with non-war related PTSD at any point were omitted from the sample. Of the 260 veterans administered diagnostic interviews, 88 (33.8%) male veterans were diagnosed with first onsets of war-related PTSD at some point between their wartime service and the interview. Individuals who had PTSD related to other traumas were excluded. Four of the 88 participants (4.5%) were excluded due to missing data on course.

Participants in this study were White (25%), African American non-Hispanic (32.1%), or Hispanic (42.9%) veterans. Individuals from the latter two ethnic groups were over-sampled in order to allow for statistical analyses of cross-racial differences in study outcomes. Since members of the heterogeneous "minor" minorities, including Pacific Islander, Asian American, and Native American ethnic groups, were not over-sampled, there were only 17 of them in the original pool of 1200, and only 5 in the weighted sub-sample (n = 260) who received SCIDs. This made it impossible for their data to be included in examinations of racial and ethnic differences across variables. Consequently, they were excluded from the current study.

The 84 participants in the current sample ranged in age from 36 to 54 at interview, and the average was 40 years old. A majority of veterans (68, or 81%) had served one

military tour. Of the remaining 16 veterans in this sample, 13 (15.5%) served two tours, 2 veterans (2.4%) served three tours, and 1 veteran served four tours. At the time of the interviews, 27 veterans (32.1% of the 84 with war-related PTSD onset) no longer met the threshold criteria for PTSD, whereas 57 (67.9% of the 84 with war-related PTSD onset) met diagnosis for PTSD within the 6 months prior to the interview date, and were consequently referred to as having current, war-related PTSD.

Design

This study was a secondary analysis of cross-sectional data. Although the NVVRS was originally intended to have multiple time points of data collection, only the first round of interviews were ever funded. Male theater veterans in this sample were interviewed once to administer the National Survey of the Vietnam Generation (Kulka et al., 1987), and a second time to administer the Structured Clinical Interview for DSM-IIIR- Non-Patient Version (Spitzer et al., 1987). Veterans were diagnosed with PTSD based on retrospective reports of their symptoms since wartime service. Just as they were assessed for current PTSD at the time of the interview and lifetime PTSD prior to the interview, they were also assessed for other current and past (i.e. lifetime) Axis I disorders. The change in veterans' PTSD symptoms over time was evaluated retrospectively. Additional data about veterans' dates and experiences in the military, medals received, and scores from military tests scores were taken from military records.

Instruments and Measures

The following instruments were administered to participants of the NVVRS subsample included in this study:

Structured Clinical Interview for DSM-IIIR- Non Patient Version (SCID-DSM-III-R-NP; Spitzer et al., 1987). The SCID-DSM-III-R is a semi-structured interview designed to be administered by a trained clinician. It uses a decision tree approach to guide the clinician in making major Axis I diagnoses from DSM-III-R. The SCID confirms the presence or absence of each disorder within the 6 months prior to the interview and over the course of the respondent's lifetime (Spitzer, Williams, Gibbon, & First, 1992).

Items from the SCID-DSM-III-R were used to screen for psychotic disorders and to assess the following psychiatric diagnoses: substance use disorders, mood disorders, somatoform disorders, adjustment disorders, antisocial personality disorder, conduct disorder, and other DSM-III-R Axis I anxiety disorders. Items from the PTSD Module included age at onset of symptoms, start and end dates of reported worst period of symptoms, specific PTSD symptoms from each of the three clusters (re-experiencing, avoidance/numbing, and hyperarousal) endorsed since wartime service and within the 6 months prior to the interview, clinician ratings of PTSD symptom severity in the past and at the time of the interview. The PTSD module also contained Cartesian graphs of PTSD course. These graphs consisted of a horizontal axis representing time between wartime trauma and the interview. The vertical axis of each graph represented severity of PTSD symptoms, and was divided into thirds labeled "low," "moderate," and "high." Veterans were asked to describe the changes in severity over time since their wartime service, and

clinicians used their responses to roughly map out the disorder course for each veteran on the graph.

The SCID for DSM-III-R was used in over 100 psychiatric studies even before information on test-retest reliability was published (Williams et al., 1992). An independent reliability check of current and lifetime PTSD diagnoses resulted in kappa (κ) values of 0.87 and 0.94 respectively (Weiss et al., 1992).

National Survey of the Vietnam Generation (NSVG; Kulka et al., 1988). This survey consisted of an average of three to five hours of highly sensitive questioning. For the current dissertation the demographics form was used to obtain veterans' dates of birth, dates of wartime service, number of tours served, education completed prior to wartime service, and pre-war psychiatric history. All of this information was checked against the Official Military Personnel Files, or *201 files*, kept by the government for each veteran. Where there were discrepancies, the information provided by the 201 files was privileged in the interests of ensuring objective, verifiable data.

Military Historical Measure (MHM; Dohrenwend et al, 2006). This is the most objective measure of combat exposure in Vietnam to date. Created for a prior study by Dohrenwend and colleagues (2006), the MHM is a composite score of war-zone stress calculated for each veteran from objective sources of information including historical records, military occupational specialties (MOS), monthly killed-in-action rate during Vietnam service, and the killed-in-action rate of the veteran's particular military unit given the specific place and time of service (Dohrenwend et al, 2006). Since this measure is entirely based on objective indicators of severity of combat exposure, it has notable

advantages over veteran self-reports of combat exposure, which may be biased by PTSD status and psychological wellbeing at the time of the interview.

Armed Forces Qualification Test (AFQT; Uhlaner & Bolanovich, 1952). AFQT scores from the military record were used as an indicator of general cognitive ability. The AFQT is a 100-item paper-and-pencil test assessing knowledge of vocabulary, arithmetic word problems, knowledge and reasoning about tools and mechanical relations, and visual-spatial organization. It is highly correlated with measures of general cognitive ability (Uhlaner & Bolanovich, 1952) and specifically with scores on the Wechsler Adult Intelligence Scale (Lyons et al., 2009; McGrevy, Knouse, & Thompson, 1974; Wechsler, 1955).

Procedures[3]

Training. All 140 interviewers who administered the NVVRS questionnaire, the National Survey of the Vietnam Generation (Kulka et al., 1988), underwent a 10-day training and certification procedure to prepare for the survey administration. The clinical component of the interview was administered by 28 doctoral-level clinicians experienced in diagnosing and treating stress disorders.

Implementation. NSVG interviews were administered in a variety of settings, depending on where participants were located. The NVVRS located over 95% of veterans sampled and administered 3,016 total interviews. Interviewers traveled an average of 200 miles and 7 hours per case for theater veterans, and interviews were conducted in all 50 states and Puerto Rico. Over 83% of the theatre veterans sampled and eligible were

[3] This section is based on the Contractual Report of Findings from The NVVRS, Kulka et al., 1988.

interviewed, ranging from 81% of Hispanic males to 86% of females. Of the 403 Vietnam theater veterans selected for the Clinical Examination Component, 85% were successfully interviewed. This data set was collected between 1986 and 1988, and has been the subject of numerous studies and publications (e.g. Dohrenwend et al. 2006; Kulka et al. 1990; Marmar et al. 1994; Marshall et al. 2006;).

Inter-rater reliability. Three graphical subtypes of disorder course were identified in a qualitative analysis of the 84 graphs. The author of this dissertation developed codes, or qualitative descriptions of each subtype (see Appendix). These codes were subjected to a test of interrater reliability: 6 doctoral level clinicians were shown the 84 graphs of PTSD course, and asked to classify them into graph subtypes according to the codes. Interrater reliability was assessed by determining Cohen's Kappa coefficient for each rater, relative to each of the other raters, and by calculating the mean coefficient for all 15 possible combinations.

Data

Veteran responses to the NSVG, data from the SCID, and information from the Official Military Personnel Files, or 201 files, have been compiled in an SPSS database that has been the subject of numerous research studies since the late 1980s. Each veteran has a unique study ID number and there is no other identifying information associated with each veteran's data. Hard copies of veterans' SCIDs have been stripped of any identifying information and have been kept on file at the Social Psychiatry Research Unit, in the Department of Epidemiology at Columbia University. The current investigation utilized quantitative data from the SPSS database and qualitative data from the hard

copies of Cartesian graphs drawn by clinicians from veteran reports of the course of their PTSD in the SCID (Spitzer et al., 1987).

Part I. Graphs of PTSD course were copied and a qualitative analysis of each of the three course subtypes was conducted. Pearsons Chi-Square analyses were performed to detect whether there were significant differences across graph subtypes on disorder severity at the worst period. One-way analyses of variance were conducted to detect whether there were significant differences across subtypes in other aspects of disorder course including the timing of symptom onset and the worst period of symptoms relative to wartime service.

Part II. Before the relationship between course profile and PTSD outcomes were examined, analyses were conducted to assess whether course subtype varied systematically with demographic characteristics. Next, analyses assessed the relationship between course profile and four measures of PTSD outcomes. One-way analyses of variance and independent samples t-tests were conducted to examine differences in the means of continuous variables across course subtypes. Pearsons Chi-Square analyses were performed to examine the differences across subtypes in percentages of categorical variables.

Part III. Differences across PTSD course subtypes on pre-war variables, war-zone stressors, and post-war factors were assessed. One-way analyses of variance and independent samples t-tests were conducted to examine differences in the means of continuous variables across course subtypes. Pearsons Chi-Square analyses were performed to examine the differences across subtypes in percentages of categorical variables. Where necessary, due to low cell count, low expected frequencies, and/or high

risk of Type II error, course subtype was condensed into a two-category variable of typical (i.e. single-peak) and less-typical (i.e. flat and multi-peak) disorder courses. Since the sixth hypothesis examined whether two-category course subtype moderated the relationship between combat exposure and PTSD outcomes, and since each of the latter variables were also dichotomous and categorical, a three-way contingency table was utilized to perform layered chi square analyses.

CHAPTER 4: Results

Demographics

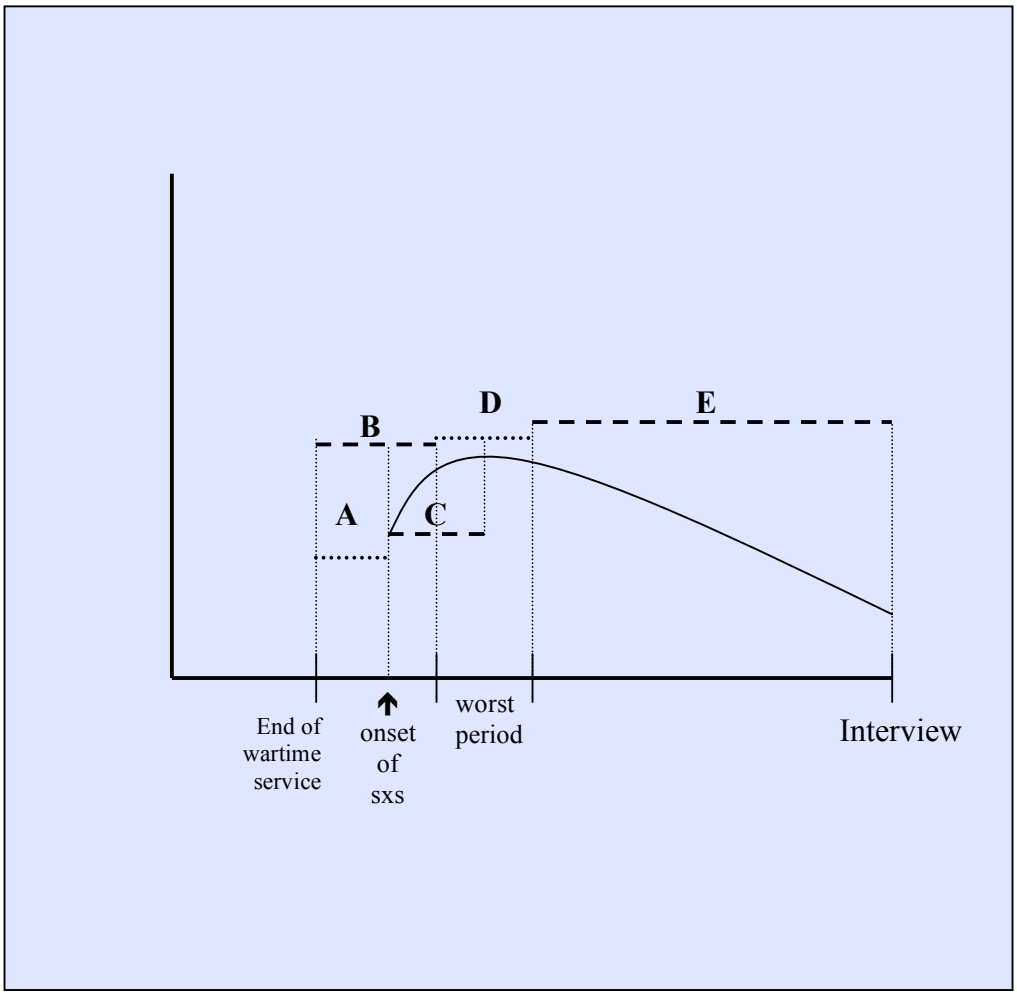
This sample consisted of 84 veterans of the Vietnam War born between 1933 and 1951, who began their service in Vietnam between 1963 and 1972. The average age at the beginning of the first military tours was 20.5 years (*SD* = 2.62 years) with a range of 17 to 34 years. Since the average age at the interview was 40 years old, the average time from the beginning of military service to the interview was two decades. The average duration of service in this sample was just over 15 months, (*SD* = 8.51 months), though the median was 12 months, with a range of 6 months to 4.5 years. Over half of the veterans in this sample (51.2%) had attained a high school diploma but no more at the time they entered the military. Close to one third (31.0%) had not graduated from high school upon entering the military. The remaining 17.9% had received some college education, though only one veteran (1.2% of the sample) had actually graduated from college at the time of entry to the military.

Timeline of the Course of PTSD

All of the individuals in this sample developed war-related PTSD as a consequence of their service in Vietnam. To obtain a sense of the average timeline of PTSD-related events, mean values were calculated for the length of time between the events listed in Figure 1.

Figure 1.

Timeline of PTSD course: events from war exit to clinical interview.



A: End of wartime service to the onset of PTSD symptoms (sxs)

B: End of wartime service to the beginning of the worst period

C: Onset of symptoms to the midpoint of the worst period

D: Start to the end of the worst period of symptoms

E: End of the worst period of symptoms to the clinical interview

Means and standard deviations for each of these periods are presented for the total sample in the first three columns of Table 3. Six of the veterans in this sample had missing information for the worst period, either because they denied having experienced a discrete worst period of symptoms, or because they could not remember the dates of the worst period of symptoms. One veteran recalled the beginning of the worst period, but provided no information about the end of it. A number of individuals cited an age of onset that *preceded* the age at which they began their first tour, as per the military record. These discrepancies were never more than one year off and were likely due memory error or confusion between age at signing up for the military, age at the beginning of the first military tour, and age at onset of symptoms.

In this data set the timing of some events was marked by age whereas that of other events was marked by dates. For example, veterans provided the age at which they recalled their symptoms beginning, but were asked the precise dates of the beginning and end of the worst period of symptoms. Relying on differences between age at two events to measure the time between them is inherently imprecise by virtue of the range of dates possible within any given age. For example, a veteran who left Vietnam at age 18 on the eve of his 19th birthday may have developed PTSD symptoms just one week after his return from the war. If his birthday occurred in the interim period, this data set would reflect his age as 19 for symptom onset and 18 for end of military tour, indicating a one-year interval, despite that the events were only one week apart. As a consequence, for periods that utilize age difference as a marker of time (items A and C in Figure 1), any one-year difference in age should be interpreted to mean a *maximum* of one year between

the event and the time of symptom onset, with the understanding that the events may have occurred *within* one year of each other.

Descriptive analysis of sub-types of PTSD course

Graphs of veterans' descriptions of the course of their PTSD were sorted into three distinct categories of curve, based on the existence of zero, one, or more than one high points, or peaks: Subtypes were labeled flat courses, single-peak courses, and multi-peak courses. The following sections will provide detailed, qualitative descriptions of the course sub-types, as well as a summary of differences in quantitative aspects of PTSD course for each subtype, including the timing between events and severity of symptoms at the worst period.

It should be noted that the qualitative information afforded by the graphs sometimes contradicts the quantitative information provided by veterans on other sections of the interview. For example, veterans with flat graphs that appear not to have any worst period of symptoms may have reported dates for the worst period of their symptoms; and individuals who reported one or two years between the end of military service and the onset of symptoms may have graphs that depict immediate onset of symptoms at the time of the trauma. These discrepancies are not understood here as detracting from the validity of the graphs, since the importance of the graphs in this study is to provide an overall impression of each veteran's subjective experience of the change in severity of symptoms over time rather than to provide detailed specifics on information about the timing of events in the disorder's trajectory.

Reliability. Three sub-types of PTSD course were identified and inductive codes were developed to describe each (see Appendix). These codes were subjected to a test of interrater reliability. Six doctoral-level psychologists rated each of the 84 graphs according to the codes. To assess reliability, Cohen's kappa coefficient was calculated for each of the six raters relative to each of the other raters yielding 15 possible combinations, each with a kappa coefficient. Kappa coefficients ranged from $\kappa = 0.645$ to $\kappa = 1.0$. The mean kappa coefficient was $\kappa = .872$, demonstrative of substantial to near-perfect interrater agreement (Landis & Koch, 1977).

Single-peak graphs: A qualitative description. Sixty-four (76.2%) of the 84 veterans in this study had single-peak graphs. The graphs varied according to the following four aspects:

1. *Time between the traumatic event and the onset of symptoms.* In 60 of the 64 single-peak graphs, (and 80 of the 84 total sample), PTSD symptoms were depicted as having begun at the time of the traumatic event. Two graphs, however clearly depicted a delay between the event and the onset of symptoms and these are presented in Figures 2 and 3. A third graph depicted symptoms that began just slightly after the traumatic event. Depending on the precision of the drawing, this may be a third example of a slightly delayed onset of symptoms (Figure 4). A fourth graph denoted points that were not connected by a line, making it impossible to clearly interpret when symptom onset occurred relative to the traumatic event. In sum, 95% of the total sample depicted onset of symptoms at the time of the traumatic event.

Figure 2

Graphical depiction of delayed onset of symptoms I.

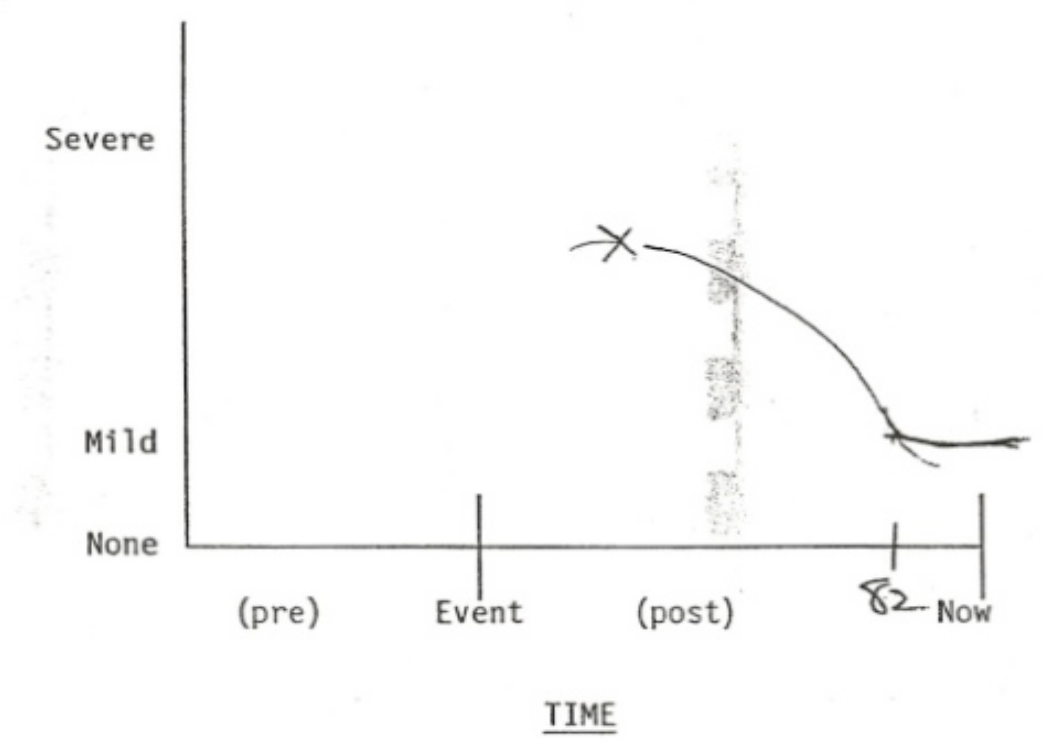


Figure 3

Graphical depiction of delayed onset of symptoms II.

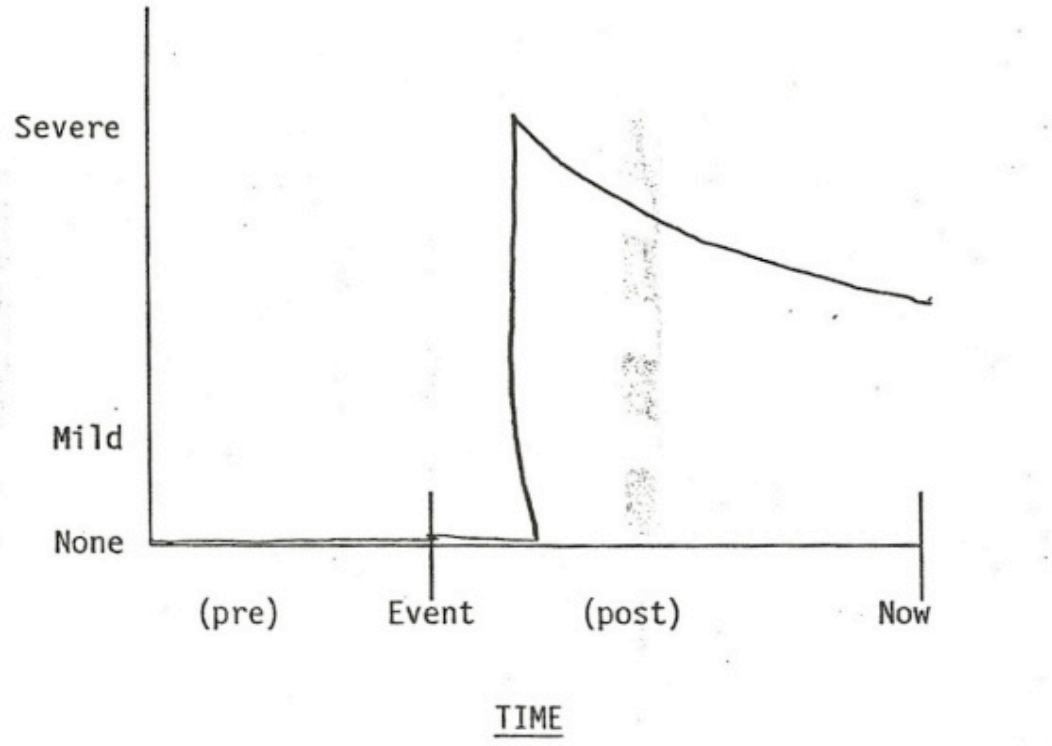
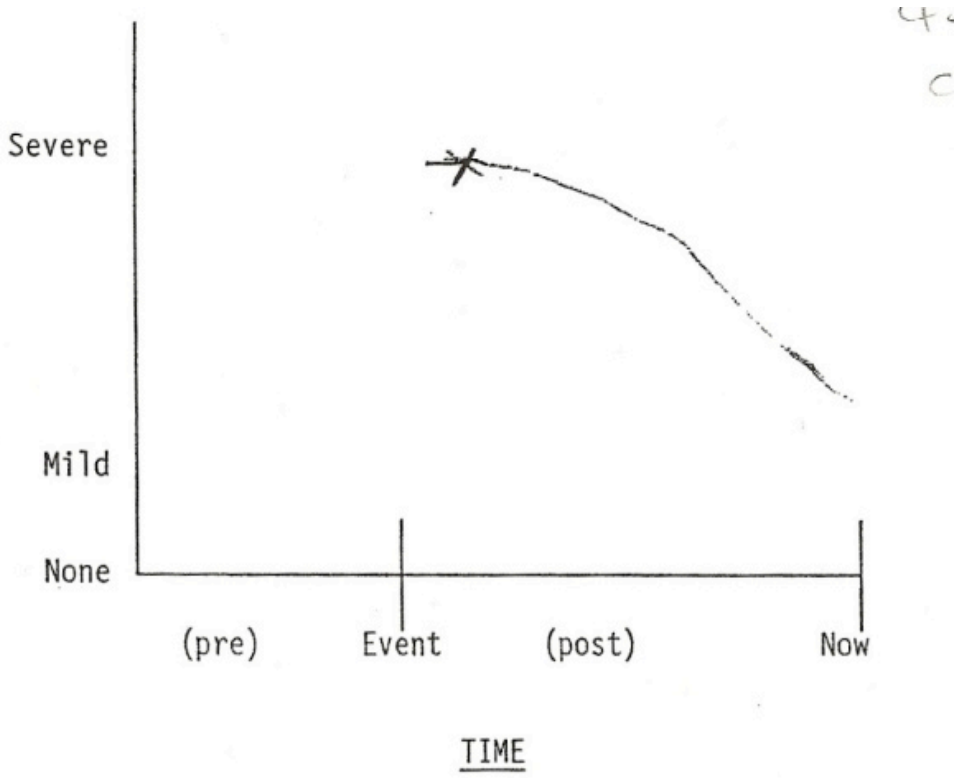


Figure 4

Possible depiction of delayed onset of symptoms.



2. *Time between the traumatic event and the highest peak in symptoms (peak at onset versus peak after onset).* In about half of the single-peak graphs, onset of symptoms began at the highest level of severity, which occurred at the traumatic event (e.g. Figure 5). These graphs display the worst symptoms at the time of the event, usually followed by a reduction of symptoms (i.e. a negative slope). In other graphs, however, the highest point occurred *after* the traumatic event, with a period of escalation preceding the peak, (Figure 6). The majority of single-peak graphs depicted the most acute symptoms, i.e. the peak, in the first half of the course. There were a small number of graphs, however, in which there was an escalation of symptoms over several years, as in Figure 7. In one of the 64 graphs, symptoms escalated over the entire course until the interview (Figure 8).

3. *Slope of the line following the peak (steady negative slope vs. negative slope followed by plateau).* In the 63 single-peak graphs that peaked prior to the interview, there was considerable variation in the slope of the line following the peak. Contrary to expectations, not all single-peak graphs featured simple diminution of symptoms until the interview. In the graphs that did depict steady reductions in symptoms over time, symptoms had rarely ceased entirely by the time of the interview (Figure 9). Only 2 of the 64 single-peak graphs (and only 2 of the entire sample of 84) depicted declines followed by complete cessation of symptoms (Figure 10). Other graphs depicted a negative slope after the peak that gradually became less negative and flattened into a plateau. In 5 graphs this plateau occurred at the lowest possible level of symptom severity above the x-axis (Figure 11). In contrast, many of the other single-peak graphs depicted a period of reducing symptoms (i.e. negative slope) and subsequent symptomatic plateaus at moderate or severe levels of severity (e.g. Figures 12 and 13).

Figure 5

Single-peak graph depicting peak at the onset of symptoms.

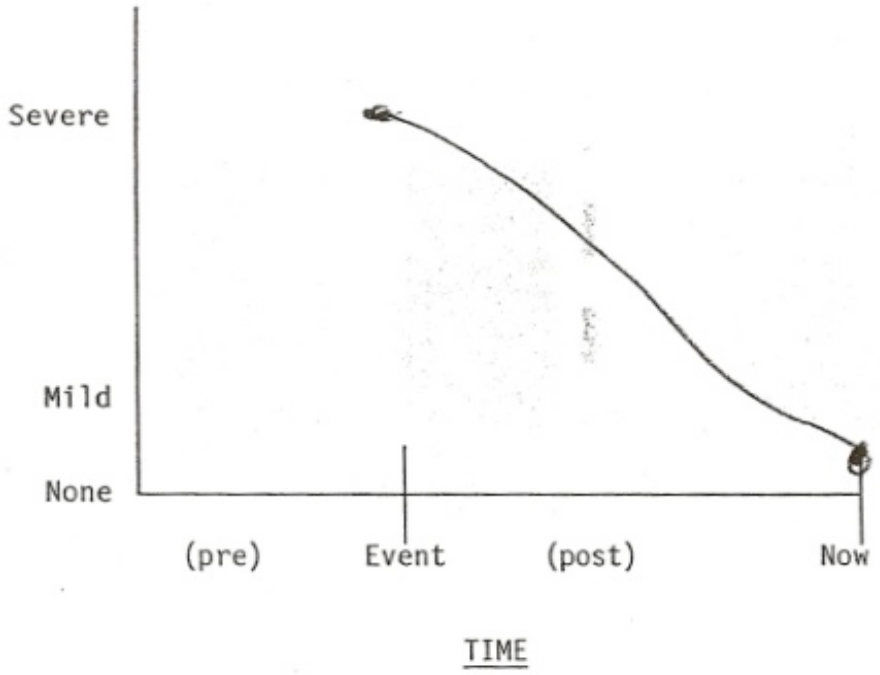


Figure 6

Single-peak graph with escalation of symptoms between onset and peak.

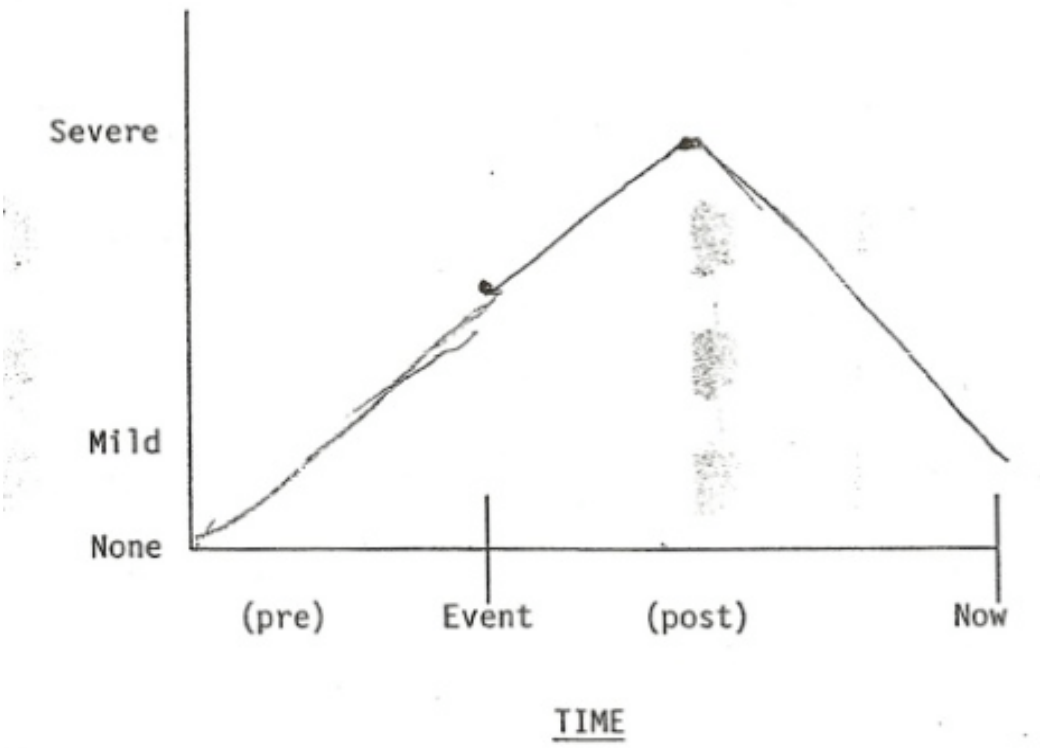


Figure 7

Single-peak graph with extended escalation of symptoms from onset to peak.

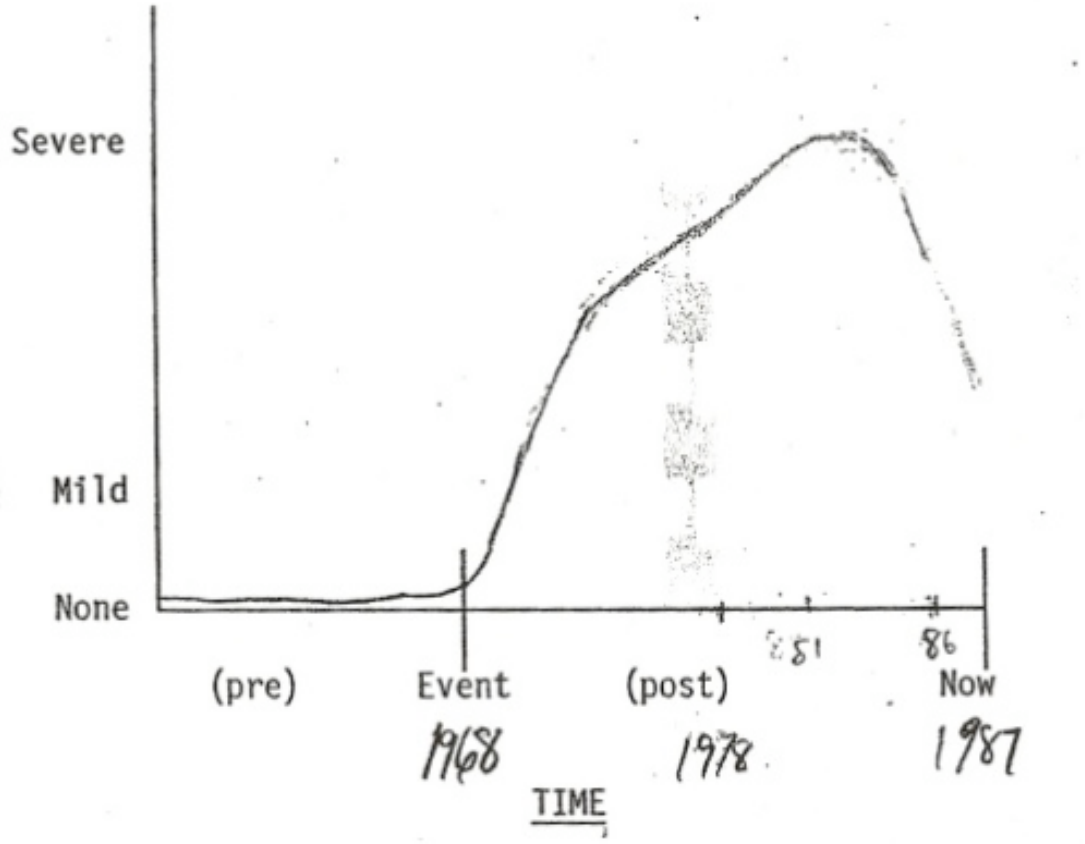


Figure 8

Single-peak graph with escalation of symptoms over the entire course.

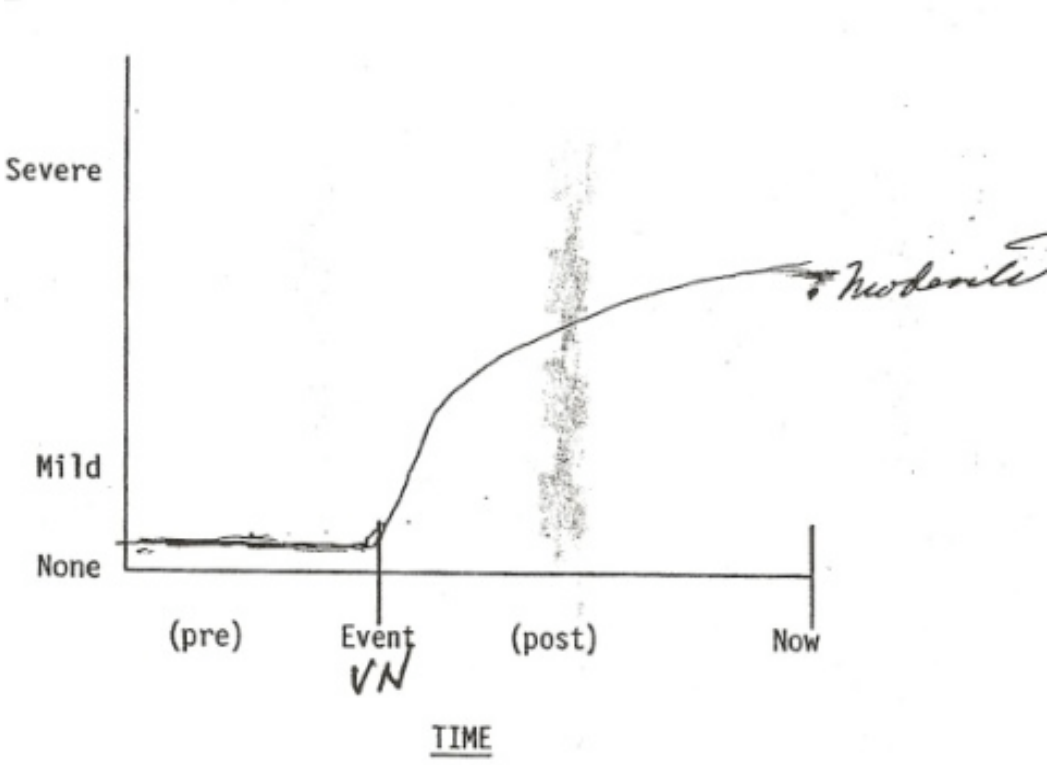


Figure 9

Single-peak graph with steady decline in symptoms but not full remission.

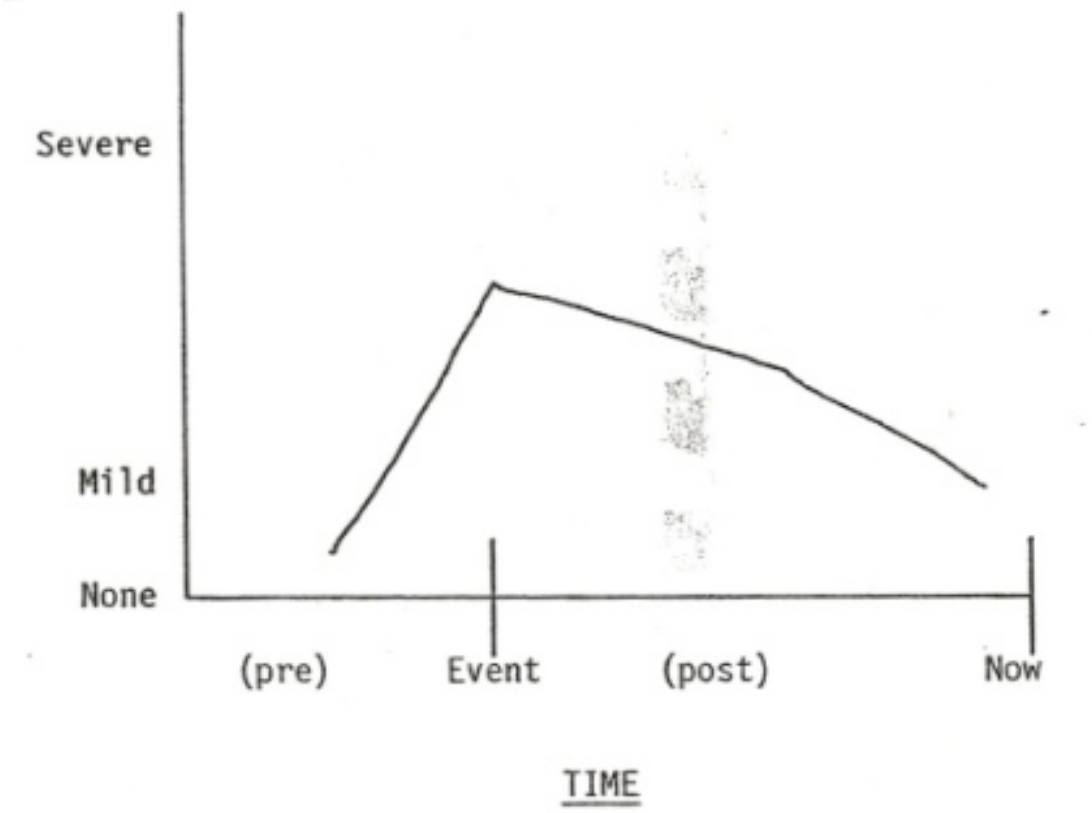


Figure 10

Single-peak graph with decline in symptoms to cessation of symptoms.

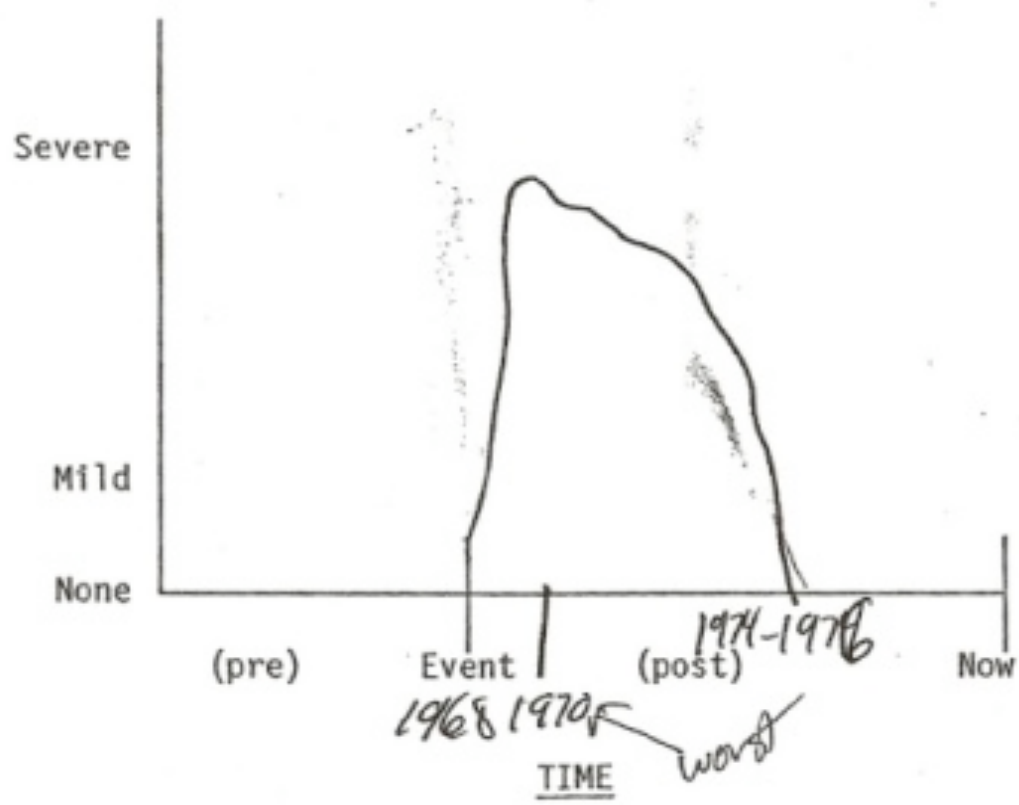


Figure 11

Single-peak graph with decline in symptoms to plateau at lowest level of symptoms.

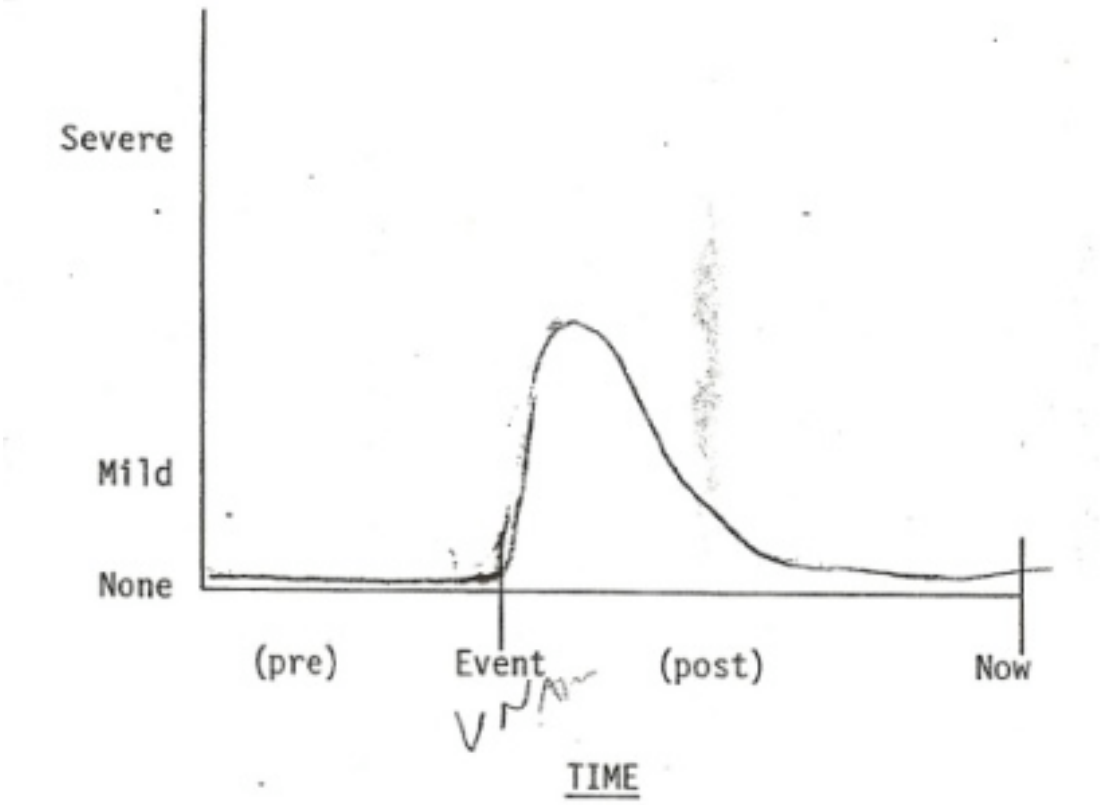


Figure 12

Single-peak graph with plateau at severe/ moderate level I.

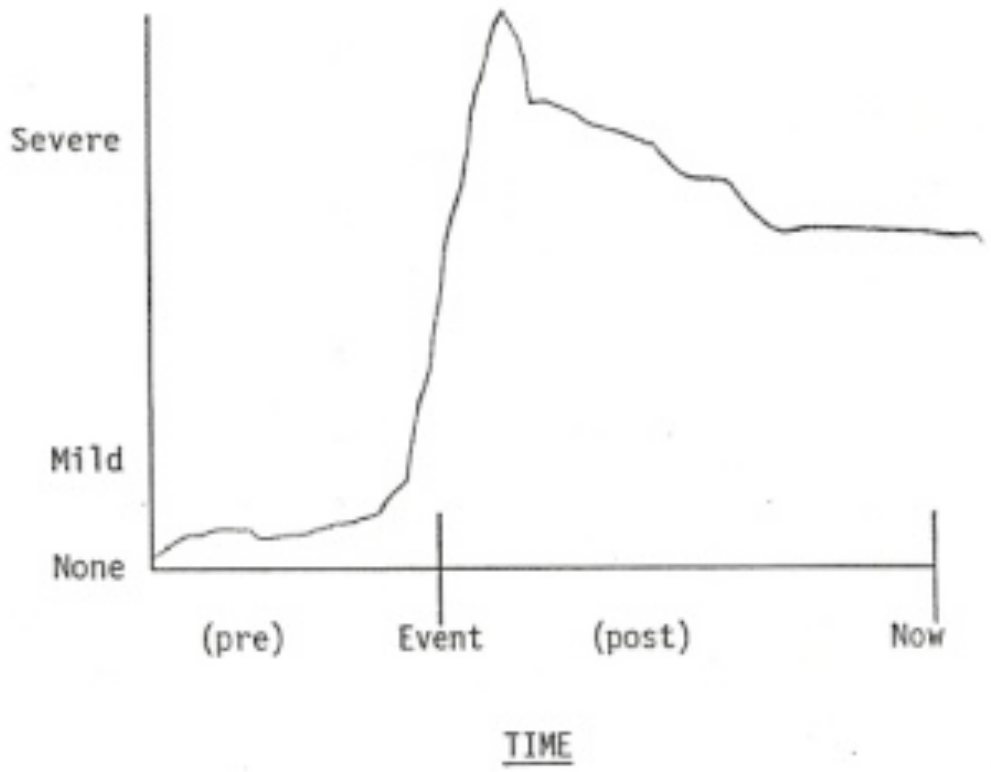
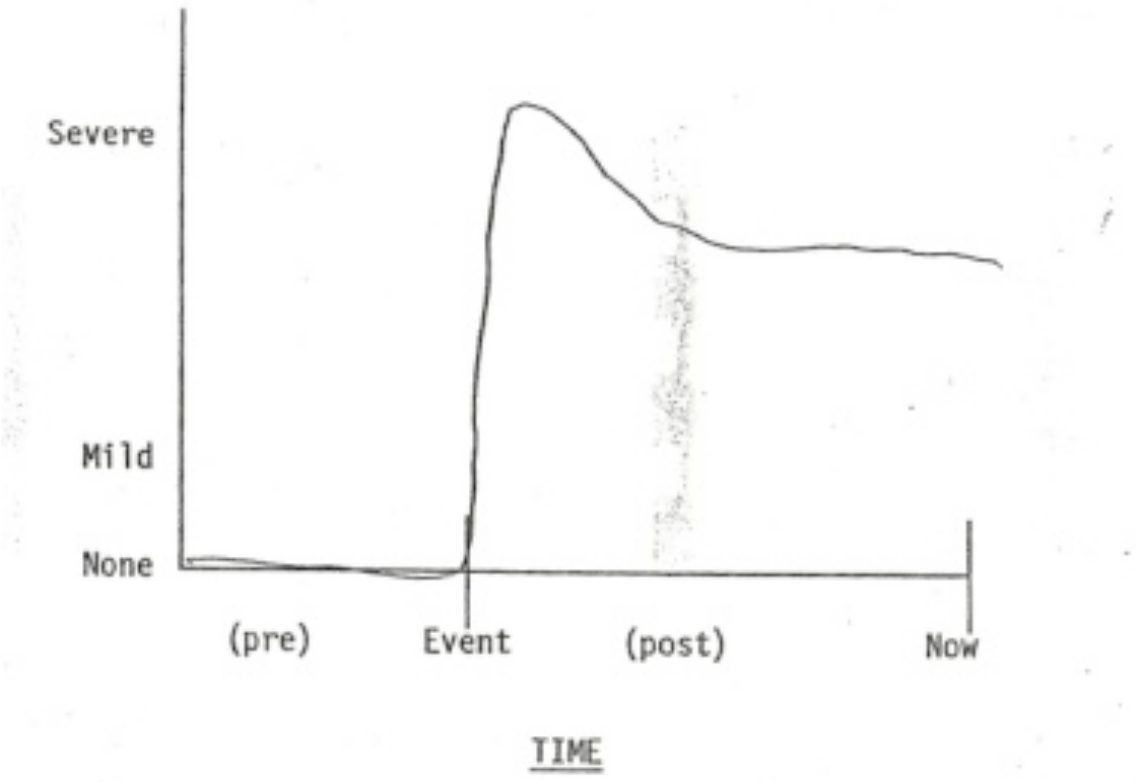


Figure 13

Single-peak graph with plateau at severe/ moderate level II.



4. *Lastly, single-peak graphs varied as to whether the peak consisted of a point or a set of points, even a plateau.* Sharper peaks, (e.g. Figure 14), or those that consisted of a point, or an acutely curved peak, were notably more common than extended peaks consisting of plateaus (e.g. Figure 15).

Flat graphs: A qualitative description. Graphs with slopes that neither increased nor decreased over the period between the traumatic event and the interview were considered flat graphs. Raters were instructed to group graphs that were "flat or almost flat" into this group (see Appendix), to allow for drawings that may have been imprecise but that appeared to depict PTSD symptoms with an unchanging course from the time of the traumatic event until the interview. This subtype made up the smallest group, with only 7 veterans, or 8.3% of the total sample. Flat graphs were most likely to depict persistence of severe symptoms: Five of the seven flat graphs depicted persistent symptoms in the severe range (Figure 16), whereas the other two flat graphs demonstrated persistent symptoms in the mild or moderate range (Figure 17). Despite the impression that these veterans experienced no change in symptoms over time, five of the seven veterans with flat graphs provided start and end dates of worst periods of symptoms.

Multi-peak graphs: A qualitative description. Fourteen (15.5%) of the 84 veterans in this sample featured graphs of PTSD course with more than one peak, or multiple, distinct periods of increasing (i.e. exacerbating) symptoms separated by periods of decreasing (i.e. improving) symptoms. There was such heterogeneity amongst multi-peak graphs that each would have to be described individually to report the variations within this group. Figures 18, 19, 20, and 21 fell into this category.

Figure 14

Single-peak graph with acute peak.

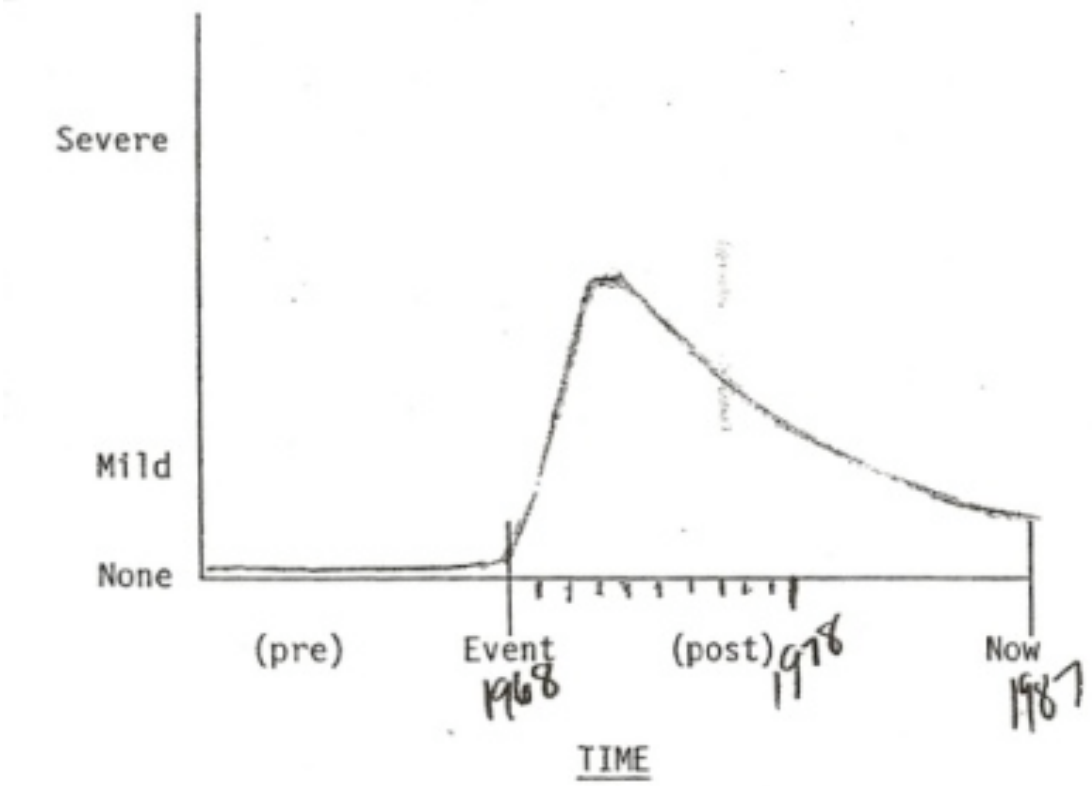


Figure 15

Single-peak graph with plateau at peak.

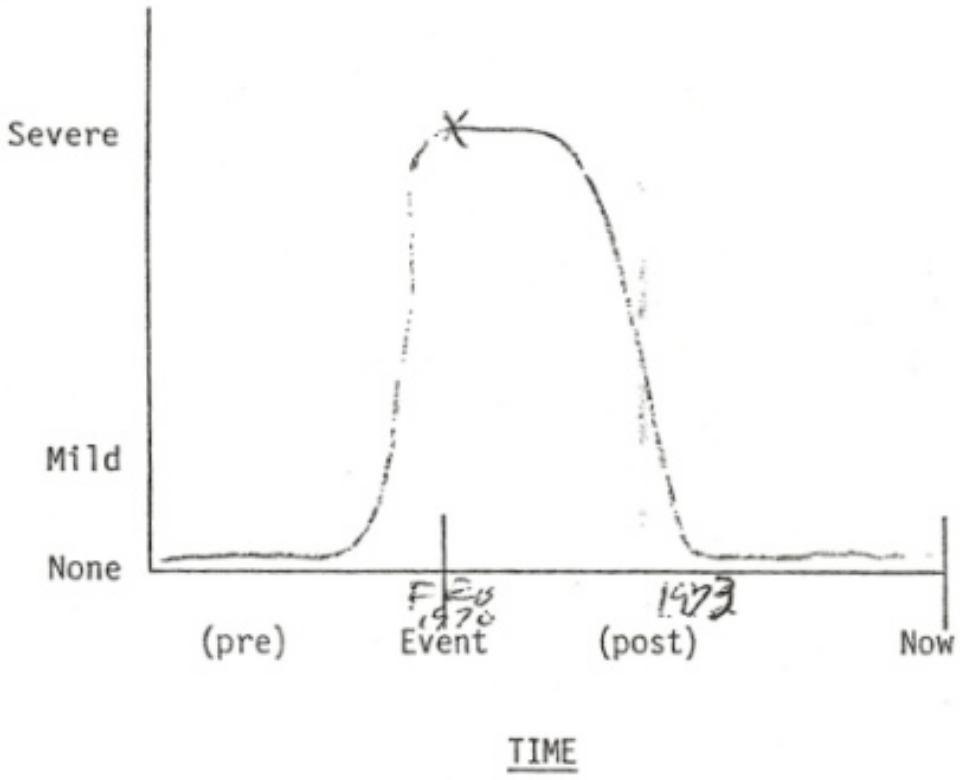


Figure 16

Flat course subtype with persistent symptoms in the severe range.

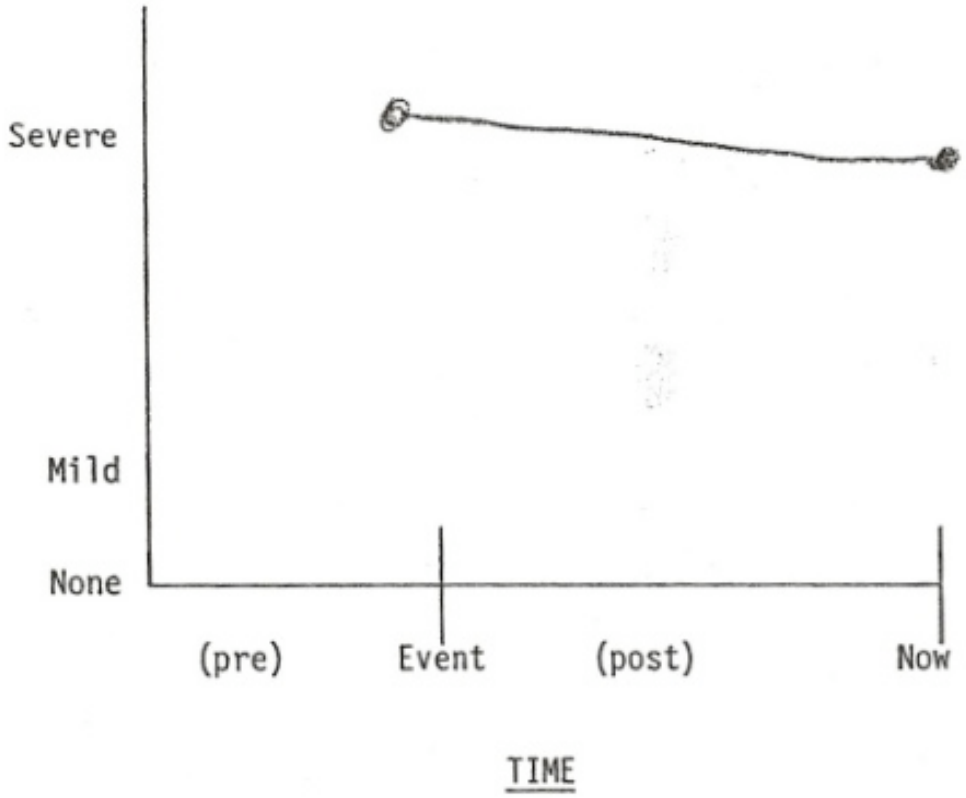


Figure 17

Flat course graph with persistent symptoms in the mild range.

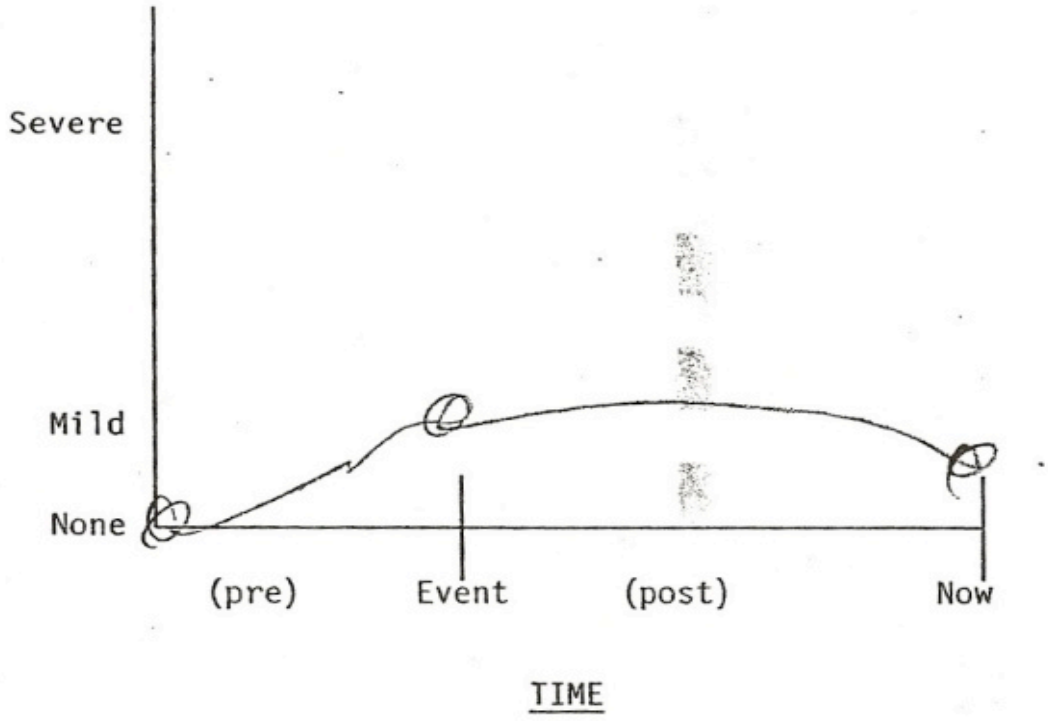


Figure 18

Multi-peak graph with two peaks.

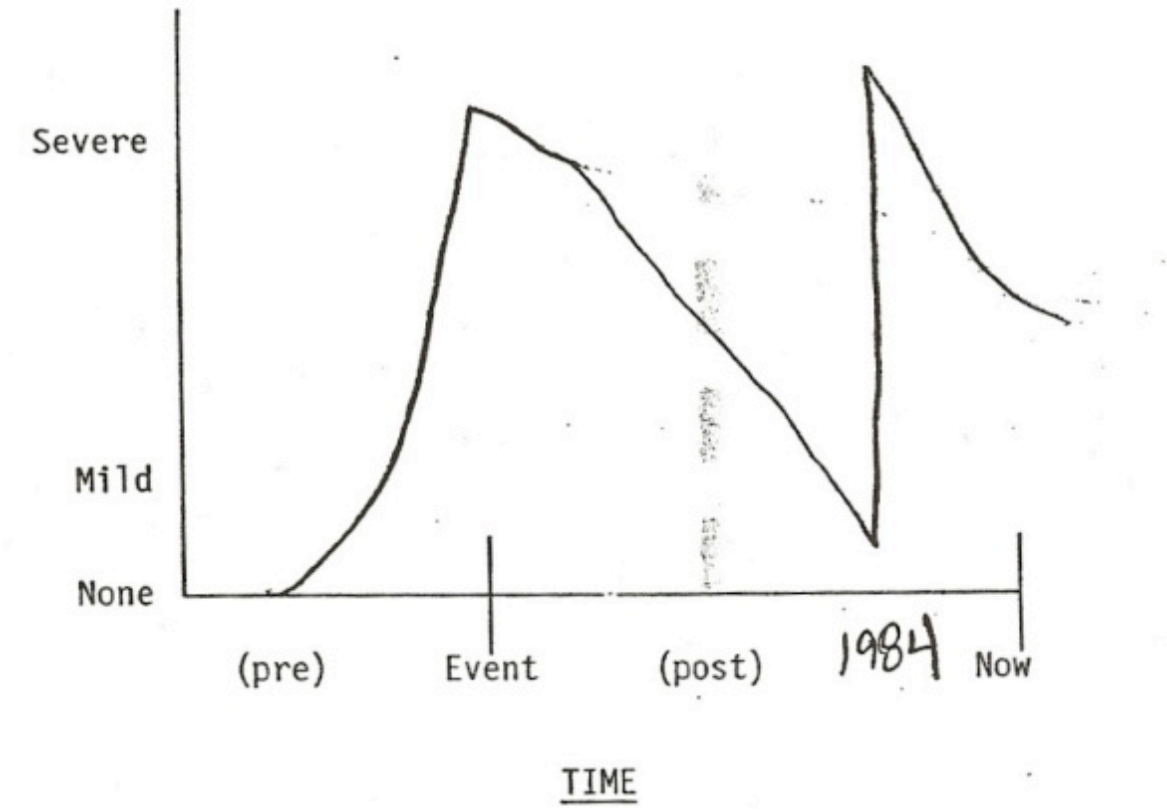


Figure 19

Multi-peak graph with exacerbations and ameliorations of symptoms within the severe—moderate range.

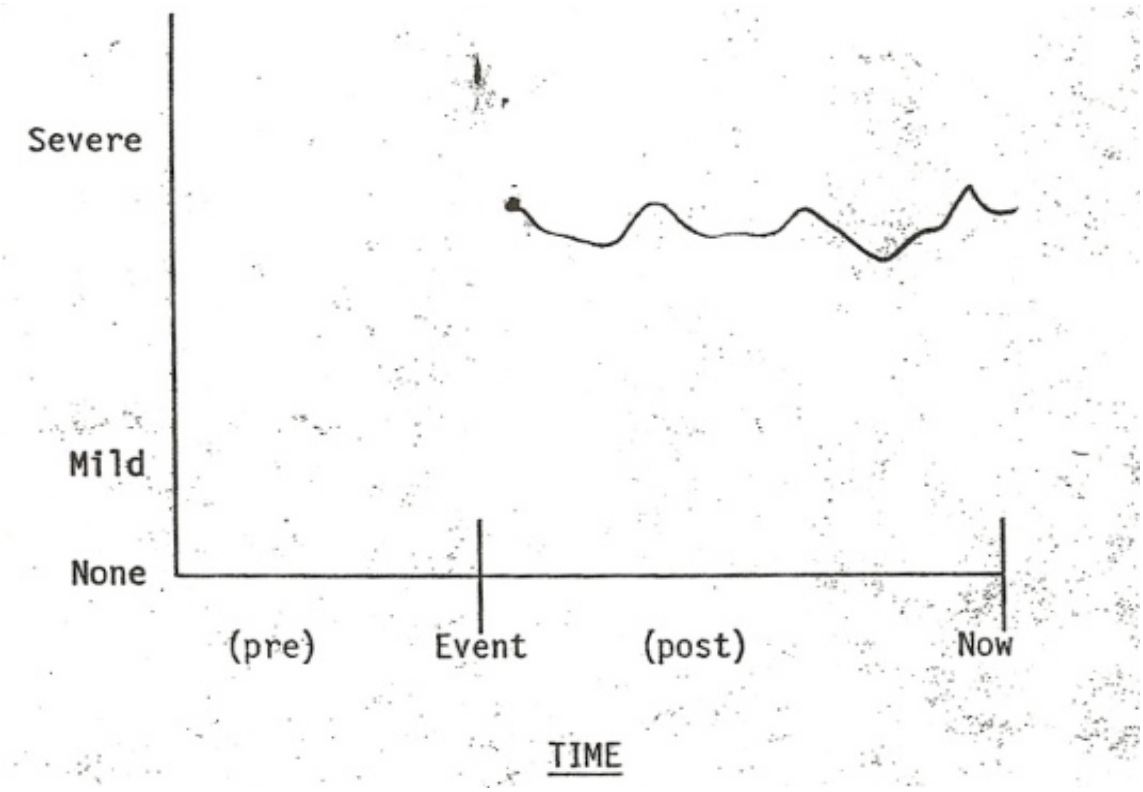


Figure 20

Multi-peak graph with two symptomatic plateaus.

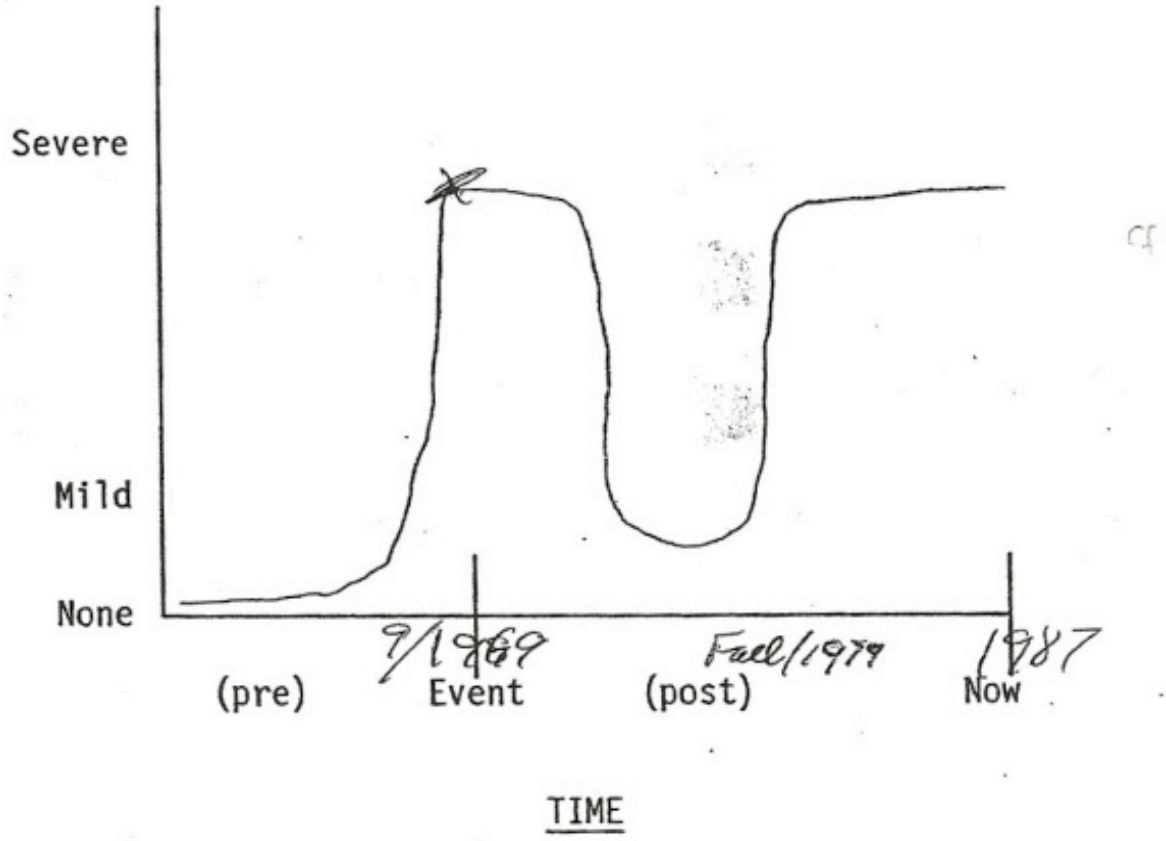
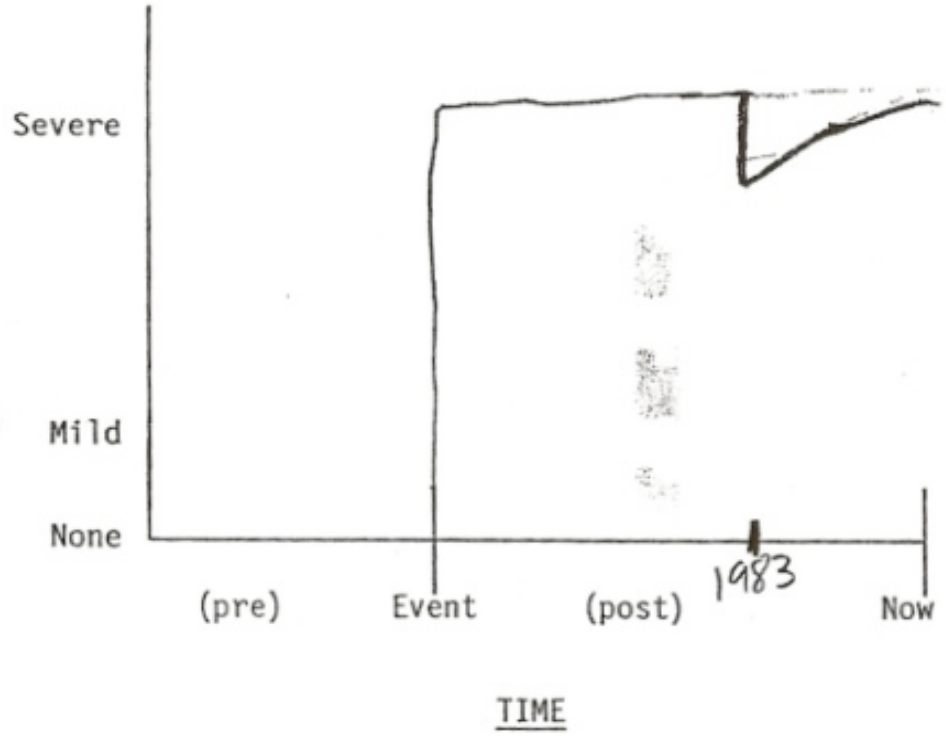


Figure 21

Multi-peak graph with plateau at first peak, symptomatic improvement, and re-exacerbation.



Differences across course sub-types on other quantitative aspects of PTSD course

One possible explanation for different subtypes of disorder course is that they correspond to different levels of PTSD severity at the disorder's worst period. Pearson's Chi-Square analyses were conducted to assess whether course subtype (flat, single-peak, or multi-peak) was associated with severity of symptoms at the worst period (Table 4: χ^2 (4, N = 83) = 8.02, p = 0.091). One veteran was missing a rating for symptom severity at the worst period. Since six cells had low expected frequencies, the same analysis was conducted for a two-category course subtype variable that combined flat and multi-peak courses to make one subtype of less common disorder courses and another of the more common, single-peak subtype (Table 5: χ^2 (2, N = 83) = 5.56, p = 0.062). Though neither set of analyses were significant, each set demonstrated a trend towards significance, with veterans with multi-peak and flat course subtypes more likely to have had severe PTSD at the disorder's worst period relative to those with single-peak disorder course. The analysis had a medium effect size, (Φ = 0.311), for the 3-category course subtype analysis (Table 4), and approached a medium effect size, (Φ = 0.259), for the 2-category analysis (Table 5).

One-way analyses of variance were conducted to test whether there were significant differences between course subtypes on the average times between the five events on the PTSD timeline (Figure 1). Findings for differences between the three course subtypes on each of the following periods are presented in Table 6: A) End of wartime service to the onset of PTSD symptoms; B) End of wartime service to the beginning of the worst period; C) Onset of symptoms to the midpoint of the worst period;

D) Start to the end of the worst period of symptoms; and E) End of the worst period of symptoms to the clinical interview.

No significant differences were found between course subtypes on the mean duration of time from age at the last military tour to age at the onset of symptoms (Time Period A, Table 6: F (2, 81) = 0.51, $p = 0.60$). The average age of the onset of symptoms that veterans recalled was 21.7 years old ($SD = 3.1$ years), though this figure ranged from 18 to 36. The majority of veterans in this sample (70.2%) reported onset of symptoms at the same age or earlier than their age at the end of their military service, and almost 90% of the sample (88.1%) reported having experienced onset of PTSD symptoms by the time they were one year older than they were at the end of their last military tour. Furthermore, the maximum delay between age at the end of service and age at onset of symptoms was two years, though this should be interpreted to mean between one and two years since this is based on a comparison of ages and not dates.

Forty one percent of the veterans in this sample experienced the worst period of symptoms at or before the end of their military service, and another 30% experienced the worst period of symptoms within one year of the end of their last tour. The maximum delay between service and the worst period of symptoms was close to 18 years (215 months). Such a delay in worst period was rare: Only two of the 84 individuals (2.4%) in this sample reported experiencing their worst period of symptoms at the time of the interview, close to two decades after the military service of many of the veterans.

One-way analyses of variance revealed a significant main effect for course subtypes on the average time from the end of military service to the beginning of the worst period of symptoms (Period B, Table 6: F (2, 75) = 5.37, p = .007). Post hoc

Tukey's test confirmed that with a mean period of 5.19 years, (*SD* = 5.21 years), veterans with multi-peak courses of PTSD had significantly greater delays between the end of military service and the beginning of the worst period of symptoms than veterans with flat graphs (p = .048) and veterans with single-peak courses (p = .007). The latter two subtypes were not significantly different from one another.

One-way analyses of variance also revealed significant differences across course subtypes on the average time from the onset of symptoms to the midpoints of their worst period of symptoms (Time Period C, Table 6: F (2, 74) = 5.72, p = .005). Whereas veterans with multi-peak courses of PTSD averaged seven years from onset to the mid point of their worst period of symptoms (*SD* = 5.5 years), veterans with single-peak PTSD courses had an average of 2.8 years (*SD* = 3.73 years) and veterans with flat courses had an average of 1.8 years (*SD* = 1.3 years) from symptom onset to the midpoint of their worst periods of symptoms. Post hoc Tukey's test confirmed that veterans with multi-peak courses of PTSD were significantly different from both those with single-peak courses (p = .005) and those with flat courses of PTSD (p = .043), though these latter subtypes were not significantly different from one another.

Analyses of variance also revealed a main effect for course subtype on the duration of the worst period (Time Period D, Table 6: F (2, 74) = 5.61, p = .005). Post hoc Tukey's test confirmed that veterans who experience multiple peaks in the course of their PTSD recall significantly longer worst periods of symptoms than those with single-peak graphs (p = .004). Veterans with flat graphs did not differ significantly on duration of the worst period from either veterans with single-peak PTSD course or from veterans with multiply peaked courses.

The majority of veterans (57.1%) recalled that the end of the worst period of symptoms had occurred more than 14 years before the clinical interview. One-way analyses of variance showed a main effect for course subtype on the time from the end of the worst period to the date of the clinical interview (Time Period E, Table 6: F (2, 74) = 6.61, p = .002). Consistent with the findings that veterans with multi-peak graphs generally had later and more protracted worst periods of symptoms, post hoc Tukey's test demonstrated that these veterans had significantly less time between the worst period and the clinical interview compared to veterans with flat graphs (p = .01) and compared to veterans with single-peak graphs (p = .004).

In sum, course subtype was not associated with time to onset of PTSD symptoms, but it was associated with the timing of the worst period of symptoms, with veterans who reported multi-peak courses having later worst periods that lasted longer than those of veterans who had either single-peak or flat courses of PTSD.

Part II: The association of course profiles with course outcomes

Hypothesis 1. *Profiles of PTSD course will differ systematically on demographic characteristics of race/ ethnicity, level of pre-military education, level of cognitive ability, and age.* Pearson's Chi-Square analyses were conducted to test for significant differences between the three course sub-types (flat course, single-peak course, and multi-peak course) on the following categorical demographic variables: race and level of pre-military education. Main effects were not found for race, (Table 7: χ^2 (4, N = 84) = 2.79, p = 0.59), nor for education by 3-category course subtype (Table 8: χ^2 (6, N = 84) = 2.28, p = 0.89). Due to low cell counts and expected frequencies, Chi square tests using

the three-category sub-type variable had increased risk of Type II error. Consequently, the flat and multi-peak courses were combined into one group (flat/multi-peaked course) to form a two-category variable. Chi-Square analyses were conducted to test for significant differences between the two groups on race and education and no significant differences were found between course subtypes on either of these variables (2-category course subtype by race: χ^2 (2, N = 84) = 1.58, p = 0.45; 2-category course subtype by pre-military education: χ^2 (3, N = 84) = 0.52, p = 0.92).

One-way analyses of variance were conducted to test for main effect of 3-category course sub-type on the following continuous demographic variables: scores on the Armed Forces Qualification Test of general cognitive ability and age at the first military tour (see Tables 9 and 10). No main effects were found (for AFQT, Table 9: F (2, 67) = 0.10, p = 0.91; for age of entry, Table 10: F (2, 81) = 0.50, p = 0.61). Independent t-tests were performed combining flat and multi-peaked graphs into one group of less typical PTSD course to be compared to the more typical, single-peak course in a 2-category variable for course subtype. No significant differences across the two subtypes were found (Table 11: for AFQT: t (70) = 0.43, p = 0.67; for age at entry: t (84) = -1.0, p = 0.32). It was not necessary to control for any of these factors in later analyses.

Hypothesis 2. *Profiles of PTSD course will predict PTSD outcomes, with single-peak graphs being associated with better outcomes and flat graphs and multi-peak graphs being associated with worse outcomes.*

The first outcome assessed was whether or not veterans met criteria for current diagnosis of PTSD (i.e. at the time of the interview). Chi-Square analyses were performed to measure the relationship between three-category course subtype and PTSD

caseness at interview with significant results (Table 12: χ^2 (2, N = 84) = 12.43, p = .002). A second analysis condensed the less common course subtypes (flat course and multi-peak course) into one category, and tested for a relationship between two-category course subtype and PTSD caseness at interview, finding significant results as well (Table 13: χ^2 (1, N = 84) = 12.43, p < .001). Each of these analyses had a medium effect size (Φ = 0.39).

A second outcome assessed current scores on specific PTSD symptom clusters (intrusive, avoidance, and arousal symptoms) at the time of the clinical interview. One-way analyses of variance were conducted to test for significant differences in the mean number of symptoms within each cluster endorsed by veterans across each of the three course subtypes. Results are displayed in Table 14. A main effect for course subtype was found on all three symptoms clusters: (F (2, 81) = 4.17, p = .019) for differences in intrusive symptoms; (F (2, 77) = 5.77, p = .005) for differences in avoidance symptoms; (F (2, 79) = 7.25, p = .001) for differences in arousal symptoms.

Post-hoc Tukey tests revealed that mean scores for veterans with flat courses and veterans with multi-peak courses were closer to each other than they were to scores for veterans with single-peak courses on all three clusters. Significant differences were found between veterans with multi-peak courses and single-peak courses for symptoms of avoidance and arousal (p = .007 and p = .001, respectively), though the differences between veterans with flat-courses and those with single-peak courses on these two clusters were not significant. For intrusive symptoms, differences between mean scores for veterans with flat courses compared to those for veterans with single-peak courses

approached significance (p = .061), though the mean differences between veterans with multiple-peak courses and those with single-peak courses were not significantly different.

Consistent with these findings, when veterans with flat courses and those with multi-peak courses were combined into a 2-category course subtype variable and independent samples t-tests were conducted, a significant main effect was found for course subtype on each of the three symptom clusters (Table 15: Intrusive Cluster: t (57.48) = -3.73, $p < 0.001$; Avoidance Cluster: t (78) = -3.35, $p = 0.001$; Arousal Cluster: t (80) = *-3.93*, $p = 0.001$).

The third PTSD outcome assessed in this study was change in severity of PTSD symptoms from the worst period to the interview. This change was assessed in two different ways. Severity at the worst period was rated as either severe, moderate, or mild, and severity at the interview included all three of those ratings as well as a category called "remitted," for individuals who no longer met criteria for PTSD diagnosis. Since severity ratings were considered ordinal categories, they were each assigned a value (0-3) according to their ranking from the least (remit = 0) to the most (severe = 3) severe. A score of improvement in symptom severity was created through calculating the difference in severity scores at the two time points, (see Table 16). By definition, veterans could not have a severity rating that was worse at interview than at the worst period. One veteran, however, had been rated as having moderate symptoms during the worst period and severe symptoms at interview. A close examination of the recency and frequency of PTSD symptoms this veteran had in each of the three clusters led his severity rating at interview to be confirmed as severe. The rating of symptoms as moderate at the worst period was presumed to be in error and was changed to a severe rating.

Chi-Square analyses were performed to test for a main effect of course subtype on number of steps of improvement in severity of symptoms from worst period to interview, a variable that ranged from zero, or no change, to the maximum of three steps of improvement from severe at worst period to remitted at interview. Improvement scores were treated as an ordinal but not continuous category, since it was not possible to assume that each step was equidistant from the next. Since Chi-Square analyses of three subtypes by four steps of improvement produced 12 potential cells with low expected frequencies (seven cells had expected counts lower than five), the analysis was conducted using a two-category course subtype variable that combined flat courses and multi-peaked courses. Significant differences on number of steps of improvement across course subtypes were found for both three-category and two-category analyses, with flat and multi-peak disorder courses being associated with less improvement, and single-peak disorder course associated with greater improvement (Table 17: χ^2 (6, N = 84) = 22.62, p = .001 for three-category subtypes; Table 18: χ^2 (3, N = 84) = 16.40, p = .001 for two-category subtypes).

One potential criticism of examining steps of improvement in symptom severity between the worst period and the clinical interview is that each step is weighted equally in the prior Chi-Square analyses: It may be argued, however, that a one-step improvement from mild PTSD to remitted PTSD is qualitatively different from a one-step improvement from severe PTSD to moderate PTSD. For this reason, an assessment of the relation between course subtypes and a more nuanced indicator of improvement in PTSD symptoms over time was also performed. Chi-Square analyses were employed to examine differences between course subtypes and veterans' membership in one of five different

categories of improvement. These categories were derived from the nine potential levels of improvement displayed in Table 16.

A preliminary cross tabulation analysis of veterans' PTSD symptom severity at the worst period by the symptom severity rating at the interview was conducted to examine frequency distribution in each of the nine cells (see Table 19). Cells were then collapsed due to low cell counts and with the intention of sharpening the contrasts between degrees of improvement (see Table 20). The five-category variable that was created in this process consisted of the following ordinal levels: 1) severe-severe; 2) mild or moderate- mild or moderate; 3) severe-mild or moderate; 4) mild or moderate- remit; 5) severe- remit. The most nebulous category of these five is 2) mild/moderate-mild/moderate which blends a mixture of three cells with low cell counts from Table 19, including veterans who were rated moderate at both worst period and interview, veterans whose symptoms were rated mild at both time points, and those whose symptoms were rated at moderate at the worst period and mild at the interview.

Chi-Square analyses were performed to test for significant differences between three-category and two-category course subtypes on membership in each of the five improvement categories. Results are displayed in Tables 21 and 22. Consistent with the prior findings, both sets of analyses found significant differences in membership in the improvement categories across graph subtypes, with flat course and multi-peak course being associated with no improvement or little-improvement groups, and single-peak course being associated with higher rates of membership in more-improvement groups (Table 21: χ^2 (8, N = 83) = 30.92, p < .001 for three-category subtypes; Table 22: χ^2 (4, N = 83) = 25.14, p = .005 for two-category subtypes).

In conclusion, as predicted in hypothesis 1, course subtype was related to all six PTSD outcome measures. Findings demonstrated that veterans whose PTSD has developed a multi-peak or a flat course were significantly more likely to have diagnosable PTSD at the clinical interview, nearly two decades after veterans' wartime service, to have higher rates of symptoms in all three symptom clusters at the time of the clinical interview, and to experience less improvement in PTSD symptoms compared with veterans with single-peak courses. That multiple-peak courses and flat courses were repeatedly related to poorer PTSD outcomes justified grouping them together in some future analyses where the number of cells and low sample size contributed to high risk of Type II error. It should be noted, however, that the similarity between flat and multi-peak graphs in relation to PTSD outcomes contrasts with the *differences* between these two groups found in Part I of this study, where an analysis of course timeline revealed that veterans with flat courses of PTSD are more similar to veterans with single-peak disorder courses in the timing and duration of the worst period of symptoms.

Part III: The association of course profiles with pre-war vulnerability, war-zone exposure, and post-war factors.

Hypothesis 3. *Subtypes of PTSD course are expected to differ on vulnerability factors that predispose individuals to PTSD including pre-military psychiatric disorder, pre-military conduct disorder, history of physical abuse as a child, and family history of substance abuse.* Since PTSD outcomes have been shown to be related to veterans' pre-existing stress mechanisms, it was suspected that there would be some overlap between different course subtypes and the following pre-war vulnerability factors: history of pre-

military psychiatric disorders (substance abuse disorder, major depressive disorder, or other anxiety disorders), pre-military childhood conduct disorder, history of physical abuse as a child, and family history of substance abuse.

Chi-Square analyses revealed no differences in premorbid vulnerability factors across the three course subtypes (see Table 23). Since all four analyses had at least 2 cells with low expected counts, the same analyses were conducted with a 2-category course subtype variable. Contrary to expectations, none of the vulnerability factors were significantly related to 2-category course subtype. For Axis I disorders, χ^2 (1, N = 84) = 0.52, p = 0.47); for childhood conduct disorder, χ^2 (1, N = 84) = 0.52, p = 0.47); for childhood physical abuse, χ^2 (1, N = 83) = 0.17, p = 0.68; for family history of substance abuse χ^2 (1, N = 84) = 0.64, p = 0.42).

Hypothesis 4. *Subtypes of PTSD course will differ systematically on exposure variables including combat exposure, high life threat during service, obtaining a purple heart, seeing a friend in the unit killed, being betrayed in a life-threatening situation, killing an enemy, or doing harm to civilians and/ or prisoners.*

One-way analyses of variance were performed on the only continuous exposure variable, total months service, to test for significant differences in the mean number of months served by veterans across each course subtype. None were found on months served by 3-category course subtypes (Table 24: F (2, 81) = 0.22, p = 0.80). An independent samples t-test was conducted on the 2-category course subtype by total months service and again no main effect was found (Table 25: t (84) = 1.44 p = 0.51).

The remaining exposure variables were all categorical. Chi-Square analyses were conducted to test for significant differences across course subtypes. All of the exposure

variables were dichotomous with the exception of the Military Historical Measure of level of combat exposure and the scale of life threatening situations, which each had four ordinal levels. To increase cell counts, these four-category variables were condensed into two-category variables consisting of the two lower levels of combat exposure or life threat that were compared to the two highest levels of combat exposure or life threat. Due to low cell counts and expected frequencies with the 3-category course subtype variable, it was condensed into a 2-category variable by combining flat courses and multi-peaked courses into one group, and Chi-Square analyses were conducted again. Two-category course subtype cross tabulations had acceptable cell counts and expected frequencies.

A significant main effect was found for only one exposure variable, having been awarded a purple heart, on both the 3-category course subtype variable (Table 26: χ^2 (1, N = 84) = 8.39, p = 0.02) and the 2-category course subtype variable (Table 27, purple heart: χ^2 (1, N = 84) = 7.47, p = 0.01). Furthermore, analyses with the 2-category course subtype variable demonstrated trends toward significance for differences on having seen a friend in the unit killed, (Table 27, friend killed: χ^2 (1, N = 83) = 2.86, p = 0.09), and having killed an enemy (Table 27, killed enemy: χ^2 (1, N = 84) = 2.69, p = 0.10). Tables 26 and 27 display all results for Chi-Square Analyses on course subtype by exposure variables. Close examination of Table 26 reveals an important pattern across four exposure variables: life threat, receipt of a purple heart, friend killed, and killed enemy. For each, the percentage of veterans in the single-peak group who endorse the item is slightly lower than that of the entire sample, the percentage of veterans in the flat-peak group who endorse the item is slightly higher than that of the entire sample, and by far the highest percentages of veterans endorsing the item are in the multi-peak group. This

pattern of rates on these four variables suggests that some effect of exposure cannot be discounted in contributing to the distribution of veterans across different disorder course profiles. Of note, life threat, having a friend killed, and killing an enemy are each self-report items, in contrast to the objective rating of combat severity, the Military Historical Measure, which did not demonstrate any association with graph profiles. Also notable is the absence of association between course subtype and harmed civilians and/or prisoners, a variable that has been found to be strongly associated with *onset* of PTSD (Dohrenwend et al., in preparation).

Hypothesis 5. *Subtypes of PTSD course will differ systematically on the following post-war variables: lifetime onset of psychiatric disorders during or after Vietnam, and mental health treatment history during or after Vietnam. Subtypes of PTSD course will not differ systematically on post-war stressful life events.*

Chi-Square analyses were conducted for 3-category course subtype by the following five lifetime psychiatric diagnoses with onset during or after Vietnam: alcohol abuse or dependence; other drug abuse or dependence; Major Depressive Disorder; Panic Disorder, Simple Phobia, Social Phobia, Agoraphobia, or Obsessive Compulsive Disorder; and any of these four diagnostic categories. No main effects were found (Table 28, Alcohol: χ^2 (2, N = 84) = 4.65, p = 0.098; Other drugs: χ^2 (2, N = 84) = 1.01, p = 0.60; MDD: χ^2 (2, N = 84) = 1.81, p = 0.40; Panic/Phobia/OCD: χ^2 (2, N = 84) = 4.22, p = 0.12; Any of these: χ^2 (2, N = 84) = 2.95, p = 0.23). An interesting trend was revealed: With the exception of Major Depressive Disorder, the percentages of veterans in the multi-peak group who received a non-PTSD Axis 1 diagnosis was *less* than or approximated that of the total sample, and consequently the multi-peak group was essentially equivalent to the

single-peak group for comorbid diagnoses. The flat-peak group, however, demonstrated very much higher rates of diagnosis in all five categories. As a result, running the same analyses for 2-category course subtype in which flat-peak graphs and multi-peak graphs are combined resulted in greater *p* values and masked the main effects of course subtype on comorbid disorders.

Chi-Square analyses were conducted for 3 category course subtype by five categories of mental health treatment that were not mutually exclusive: The use of Veterans Administration mental health services; the use of a hospital emergency room for mental health services; the use of non-VA mental health services for substance abuse treatment; use of any other non-VA mental health services; and an inclusive variable that combined *any* use of professional mental health services within or outside of the VA (Table 29).

A main effect was demonstrated for the use of any VA mental health service, with veterans with multi-peak graphs being considerably more likely to endorse having had mental health treatment compared with veterans of the other two subtypes, and those with flat graphs were more likely to endorse having had VA treatment than those with single-peak disorder courses (Table 29, Any VA MH Service: χ^2 (2, N = 84) = 6.03, p = 0.05). Although significant main effects were not found for the other four categories of treatment, veterans with multi-peak graphs had the highest rates of all five categories of mental health services. Interestingly, veterans with flat graphs, who proved to have the highest rates of comorbid disorders (Table 28), had the *lowest* rates of the combined variable measuring utilization of any mental health service. These veterans had the lowest rates of all non-VA mental health services besides use of a hospital ER for mental health

services. They had 0% usage of non-VA substance use services (Table 29), a finding that is striking, given that their usage (28.6%) of VA services—which may have been substance use-related— was only slightly above the average for the total sample, and 100% of veterans in this group suffered with comorbid alcoholism (Table 28). Since veterans with flat graphs had the second highest rates of VA mental health service use and the ER for mental health services, Chi-Square analyses with a 2-category course subtype variable that combined flat graphs and multi-peak graphs resulted in main effects for both kinds of treatment (2-category course subtype x Hospital ER: χ^2 (2, N = 84) = 6.78, p = 0.034; 2-category course subtype x VA treatment history: χ^2 (2, N = 84) = 7.47, p = 0.006).

Lastly, Chi-Square analyses were conducted for course subtype on the following post-war life events: Divorce or separation; periods of unemployment longer than six months; and negative fateful life events (illness and non-illness), defined as adverse events over which the individual had no control (Shrout et al., 1989). As expected, no significant associations were found between either 3-category or 2-category course subtypes and any of these post-war life events (Table 30). Likely related to their higher rates of symptoms and PTSD persistence, veterans with flat and multi-peak courses were found to have higher, though not significantly higher, rates of the negative, non-fateful life events of marital divorce/ separation and long-term unemployment.

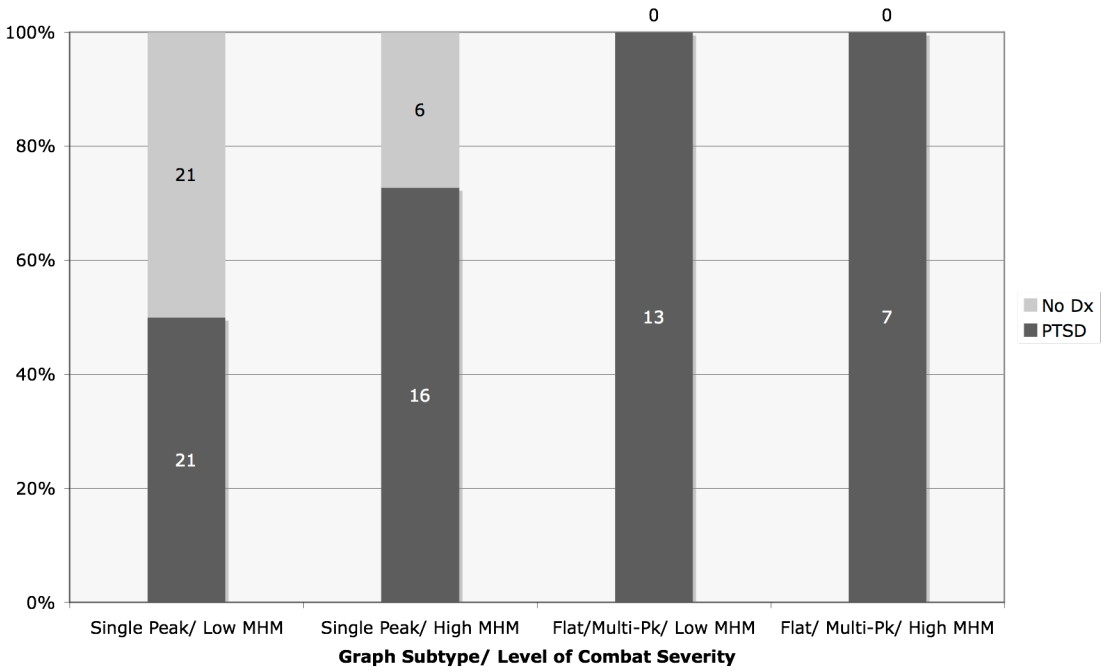
Hypothesis 6. *It is hypothesized that PTSD course subtype will moderate the established association between level of combat severity and the PTSD outcome, failure to remit (i.e. current diagnosis).* It was established in the analyses for Hypothesis 4 that course subtype was not associated with the objective measure of combat severity

(Military Historical Measure). For Hypothesis 6, a three-way contingency table (2x2x2) was utilized to assess how the relationship between current PTSD diagnosis at interview and course subtype differed at different levels of combat exposure (Current PTSD Diagnosis x Course Subtype x Combat Severity). At low levels of combat severity, there was a significant relationship between current PTSD diagnosis and course subtype (χ^2 (1, N = 84) = 10.52, p = .001), with veterans who had single-peak course subtypes having significantly lower rates of current PTSD (50%) than veterans with multi-peak or flat course subtypes, of whom 100% had current PTSD at the time of the interview. At high levels of combat severity, however, the relationship between course subtype and PTSD caseness was no longer significant (χ^2 (1, N = 84) = 2.41, p = 0.121). This was because a much greater percentage of veterans with single-peak disorder course (73%) met criteria for current diagnosis, and this percentage was not significantly different from the 100% of the seven veterans with flat or multi-peak graphs who were exposed to high levels of combat severity (see Figure 22).

This finding demonstrates the interaction of the severity of combat trauma with specific subtypes of disorder course in determining PTSD outcomes. Most veterans with PTSD in this sample developed the more common, single-peak disorder course, and for these veterans, more severe combat trauma was significantly associated with a failure to remit nearly two decades after the war. Veterans who developed the less common, more complicated course subtypes, however, *all* met criteria for PTSD diagnosis at the clinic interview, regardless of the level of combat severity to which they were exposed. This finding underscores the preeminence of disorder course in predicting outcomes over the dose response relationship. The implication is that the well-documented dose-response

relationship already confirmed to exist in this population (Dohrenwend et al., 2006) pertains to most veterans with war-related PTSD (i.e. those with the most common, single-peak course subtype), but it does not pertain to the small subset of veterans for whom exposure to even low levels of trauma precipitates flat or multi-peak disorder course, since *all* of these individuals develop severe PTSD that endures for over a decade. The preeminence of the course subtype over levels of combat severity can be seen clearly in Figure 22, in which the two bars on the right side of the graph demonstrate 100% rates of PTSD diagnosis at both levels of combat severity compared to the two bars on the left that demonstrate the shifting rates of current diagnosis according to level of combat severity.

Figure 22: *Percentage of veterans in each course subtype diagnosed with PTSD at interview across levels of combat severity*



Chapter Five: Discussion

This dissertation has focused on the course of war-related posttraumatic stress disorder in Vietnam Veterans, a disorder that is frequently chronic, lasting for years or even decades, and the rates of which have been shown to be related to the severity of the combat trauma to which veterans have been exposed (Dohrenwend et al., 2006; King et al., 1995; King et al., 1999; Kulka et al., 1990; Green et al., 1990a; Green et al., 1990b; Hoge et al., 2004; Smith et al., 2008; Yehuda, 1992). A comprehensive review of publications on war-related PTSD suggests that, depending on the time period studied, there may be a variety of trajectories of disorder course. These include a gradual diminution of symptoms over decades (Kluznik et al., 1986; Op den Velde et al., 1996) gradual increases in both frequencies of symptoms and severity of symptoms over anywhere from 2 years to 20 years (Bremner et al.,1996; Johnson et al., 2004; Orcutt et al., 2004; Solomon & Mikulincer, 2006; Southwick, et al., 1995; Thompson et al., 2004), and intermittent exacerbation of symptoms punctuated by temporary symptomatic abatements (Op den velde et al., 1996; Port et al., 2001; Solomon & Mikulincer, 2006; Zeiss & Dickman, 1989).

Given the absence of any comprehensive examination of the disorder's trajectory over time, the goals of this dissertation were to provide a detailed account of qualitative and quantitative aspects of the course of war-related PTSD in 84 Vietnam veterans, to investigate the relationship between subtypes of disorder course and other PTSD outcomes, and to examine whether the pre-war, post-war, and war-related factors known to be related to PTSD outcomes are also correlates of the disorder's course. A number of the findings from this study are notable and will be briefly summarized.

Findings

Features of the three course subtypes of PTSD. Three subtypes of course were identified and codes for each were developed. Raters demonstrated strong reliability using the codes to classify graphs of PTSD course into these subtypes. The majority of veterans in this sample (76.2%) experienced a course of PTSD with exactly one period of exacerbated symptoms that was followed by some symptomatic improvement (i.e. single-peak course subtype). A smaller percentage of veterans in this sample (15.5%) experienced exacerbations of symptoms followed by some improvement in symptoms and then at least one re-exacerbation in symptoms (multi-peak course), and an even smaller group (8.3%) experienced consistent, unrelenting symptoms at the same level throughout the 10 to 20 years assessed.

The distribution of veterans into these three course subtypes corroborates reports in the literature of a variety of war-related PTSD trajectories. Furthermore, this finding is consistent with the repeated findings in the literature of reduced disorder prevalence over time (e.g. Dohrenwend et al., 2006; Kessler et al., 1995; Kulka et al., 1990) and of diminutions in rates of veterans who report being seriously troubled by symptoms after the first 6 years post repatriation (Kluznik et al., 1986; Port et al., 2001; Zeiss & Dickman, 1989).

Across *all* course subtypes, veterans reported similar patterns of PTSD onset. Close to 90% of the entire sample experienced symptoms either before leaving their last tour or within the first year of repatriation. A minority of veterans, 11.9%, reported onset of symptoms one to two years after the end of the last military tour, but there was no evidence of onset of symptoms delayed more than two years from military service, in

contrast to reports by a number of authors (Van Dyke et al., 1985; Op den velde et al., 1996; Schnurr et al., 2003). The findings of this study corroborate conclusions from a review of 29 studies of delayed onset PTSD, that long-delayed onset of PTSD in the absence of *any* prior symptoms is rare (Andrews et al., 2007), since symptoms (though not necessarily the full-blown disorder) generally appear fairly soon after the traumatic event.

On average, veterans reported the worst period of symptoms beginning between one and two years after repatriation. The timing and duration of the worst period varied by disorder course subtype. Those with multi-peak disorder courses experienced the worst period of symptoms over 4.5 times later than those with single-peak courses whose average delay between service and the worst period was just over one year. Veterans with flat graphs experienced the beginning of the worst period of symptoms before the end of service, or some months thereafter. Furthermore, veterans with multi-peak courses had much longer worst periods of symptoms, lasting an average of nearly six years compared to 2.5 years for those with single peak and flat courses. Of note, veterans with flat courses were more similar to those with single peak graphs than those with multi-peak graphs when comparing the average times between service and the beginning and end of the worst period of symptoms.

One possible explanation for different types of PTSD course is that course subtype is related to the severity level of the disorder at its worst. Simply stated, people afflicted with more severe PTSD may suffer with a more complicated disorder course. The results showed this to be only partially true. All (100%) of the veterans with multiple-peak courses were rated as having PTSD that was severe at its worst period,

compared to 71% of veterans with flat-course and 64% of veterans with single-peak disorder course. The differences in severity across these groups produced a statistical trend, though no main effect of severity on disorder course was found. It is important to note that the multi-peak course subtype was the only one lacking variance in severity level at the worst period of symptoms. Both flat-course and single-peak subtypes included veterans who were rated as having had mild, moderate, and severe PTSD at the disorder's worst period. It appears that the subtype of PTSD that features multiple exacerbations over time *only* occurs in the most severe of cases, whereas the other course subtypes may develop in severe, mild, or moderate PTSD.

Associations between PTSD course profiles and PTSD outcomes. The second part of this study tested the associations between PTSD course profile and six PTSD outcomes. First, the relationship between course profiles and demographic characteristics were examined to determine whether any of these needed to be controlled for. Course profile was not related to the demographic characteristics of race, pre-military education level, AFQT scores, or age at entry to service. As predicted, subtypes of PTSD course were significantly related to all six PTSD outcomes at the time of the clinical interview, between one and two decades after veterans served in the war. A startling 100% of veterans with flat and multi-peak disorder courses had current PTSD at the time of the interview, whereas just under 58% of the veterans with single-peak courses met criteria for diagnosis at the interview. Furthermore, veterans with multi-peak and flat courses of PTSD were more symptomatic on all three PTSD symptom clusters at the time of the interview than those who reported single-peak disorder courses. The multi-peak disorder course was associated with the highest rates of symptoms on the avoidance and arousal

clusters, whereas the flat disorder course was associated with the highest rates of intrusive symptoms. Since previous publications have linked symptoms of avoidance and arousal with both chronicity and severity of PTSD (Breslau and Davis, 1992; Marshall et al., 2006), this finding may indicate more severe disorder in the multi-peak group relative to the flat course group, though both groups are clearly at equally high risk for very chronic PTSD.

Analyses of the differences between course subtypes and change in symptom severity from the worst period to the interview provided a more nuanced view of the relationship between course and outcome than did the dichotomous outcome of current PTSD diagnosis at interview. Veterans with multi-peak disorder course *do* in fact experience some improvement in PTSD over time, despite that they all continued to meet criteria for diagnosis at the interview. Nearly 62% of veterans with multi-peak disorder course improved from severe PTSD at the worst period to moderate PTSD at the interview, whereas the other 38% were rated as having severe symptoms at both times. In contrast, consistent with the depiction of an unchanging course, six of the seven veterans with flat courses (85.7%) remained at the original level of severity by the time of the clinical interview (either severe or moderate), and only one demonstrated improvement from severe at the worst period to moderate symptoms at the interview. Veterans with single-peak courses showed a much greater range of improvement, with a fairly even distribution across levels of improvement: Whereas two fifths (41%) of them improved just one level, the other three fifths of this group were distributed evenly between no improvement, two levels of improvement, or three levels of improvement. Of note, veterans with single-peak disorder course whose PTSD was rated as severe at the worst

period were much more likely to improve than those rated as severe who had either of the other two course subtypes.

The third part of this study involved an investigation of the relationships between PTSD course subtype and pre-war vulnerabilities, war-zone exposure variables, and post-war factors.

PTSD course profiles and pre-war vulnerability factors: Contrary to expectations, pre-military psychiatric problems, abuse history, or family substance abuse history did *not* impact the kind of disorder course veterans experienced. This may be because course subtype is more directly related to the more proximal factors related to the trauma (i.e. war-zone stressors) rather than distal vulnerability factors. It may also be that there is not enough power in these analyses to reveal a relationship with a small effect size, as pre-war vulnerability factors have been shown to have (Brewin et al., 2000a). Another possibility is that course subtype is in fact related to individual factors that are not measured here or that do not correlate with the individual factors measured here. For example, course subtype may be related to unique neuro-physiological characteristics that do not correlate with the vulnerability factors examined for this study. Also, course subtype may be related to personality factors which went undetected in this study: Veterans were not assessed for the full range of Axis II disorders (APA, 1994) beyond antisocial personality disorder and childhood conduct disorder.

Associations between PTSD course profiles and war-zone exposure variables. Seven of the eight exposure variables examined for this study were not significantly related to course subtype, including the objective measure of combat severity, the Military Historical Measure (MHM), that has been shown to be most strongly related to

PTSD outcomes in this sample (Dohrewnend et al., 2006). These findings support the theory that course subtype is a feature of the disorder with an association to outcomes that is separate from the relationship that has been shown to exist between PTSD outcomes and exposure variables. A closer examination, however, revealed that in addition to the significant relationship between course profile and being the recipient of a purple heart, there was a strong trend of association between course subtype and the self-report exposure variables of life threat, having seen a friend killed, and having killed an enemy. Greater percentages of veterans with flat course profiles endorsed each of these exposure variables compared to the single-peak group, and even greater percentages of veterans with multi-peak course profiles endorsed each variable compared with both flat and single-peak groups. The direction of this pattern of association between self-report exposure variables and course profile was the same for the one variable that *was* found to be significantly associated with course profile, receipt of a purple heart, which was verified by the military record and did not rely on self-report.

Given that there was a strong relationship between some exposure variables and PTSD course subtype, an interesting question that comes of these findings is why the most established exposure variable, level of combat severity, was found *not* to be related to PTSD course subtype. Importantly, the relationship between MHM and PTSD persistence and onset has been found amongst a sample of traumatized veterans of whom some did and others did not develop subsequent onset of war-related PTSD (Dohrenwend et al., 2006). In contrast, the question asked in the current study is: Amongst a sample of traumatized veterans *with war-related PTSD*, which exposure variable best predicts course profile, i.e. one marker of the poorest PTSD outcomes? Whereas the objective

measure of war-zone stress (MHM) has predictive value in parsing out the resilient (i.e. those who will not develop PTSD) from the less resilient, it is not helpful in parsing out different course trajectories amongst those who develop PTSD. It appears that the variables best at the latter task are those that capture the subjective experience of vulnerability amidst war such as the self-report items of degree of life threat, witnessing a close friend killed, and killing an enemy.

Another very personal experience of vulnerability in war is the experience of combat-related injury, for which veterans receive a purple heart. Regardless of the objective level of combat severity to which a veteran is exposed, combat-related injury indicates traumatic exposure severe enough to cause harm, and potentially be experienced as life threatening; it also indicates physical involvement of the veteran's body as a recipient of the traumatic force; and the psychological correlates of having received physically injurious, potentially life-threatening bodily damage in the context of war. The psychological ramifications of being injured physically may precipitate a particularly pernicious disorder course by way of a number of possible channels. Physical injury in combat may simply have overwhelming effects on the stress response mechanism. It may be that when one is physically injured in combat the trauma's magnitude is more intensely and personally felt compared to the stress of combat measured objectively, since the effects of the war are physically imposed upon the body, thereby penetrating any psychological distancing from the experience and forcing themselves to be felt subjectively. This experience may overload the veteran's capacity to adapt to the trauma and precipitate a particularly dire PTSD course. It may also be that bodily injury during combat-related trauma may cause physiological changes that short-circuit the body's

more adaptive stress-response mechanism and precipitate less common disorder course. Indeed, a number of studies have shown combat-related injury to be significantly associated with either onset (e.g. Green et al., 1990b) or persistence of PTSD (Grieger et al., 2006; Kulka et al., 1990). It is also possible that an enduring disability may serves as a continuous reminder of the traumatic event which could impact the disorder's course.

Associations between PTSD course profiles and post-war variables. No statistically significant main effects were found for the relationship between course subtype and Axis I disorders diagnosed during or after military service in Vietnam. However, a fascinating trend was revealed. With the exception of the diagnosis of non-alcohol substance use disorders, veterans with flat courses of PTSD had the highest percentages of each category of clinician-made non-PTSD diagnoses compared to veterans with both multi-peak and single-peak disorder courses. Furthermore, veterans with multi-peak graphs who had the highest rates (100%) of severe PTSD at the worst period, and the highest rates (100%) of current PTSD at the time of the interview, had the lowest rates of comorbid substance abuse disorders of any of the three groups of course profiles, and they had rates of comorbid Panic, OCD, and other non-PTSD anxiety disorders similar to the group with single-peak courses. Veterans with multi-peak course profiles had rates of Major Depressive Disorder that were greater than those of the group with single-peak graphs but still lower than rates of the group with flat disorder courses.

Although they should be interpreted with caution, since only 7 veterans in the sample had flat courses of PTSD and only 13 had multi-peak courses, these findings are notable for a number of reasons. First, It is often reported that as a group, veterans with severe, chronic PTSD have high rates of comorbid disorders. This data suggests that there

may be a distinction between those with severe, persistent PTSD who have higher rates of comorbid disorders from those with severe, re-exacerbating PTSD whose rates of comorbid disorders are notably lower. Secondly, amongst those with flat graphs, it may be that veterans' perceptions of PTSD as unchanging are heavily influenced by the complicating effects of comorbid disorders. Of note, 6 out of 7 veterans (85.7%) with flat graphs were diagnosed with Vietnam onset or later alcohol abuse or dependency—a rate that near doubled that of the multi-peak and single-peak groups. There may be complex relationships between PTSD, comorbid disorders, alcoholism, and perceptions of the disorder's course.

The high percentage of comorbid disorders in veterans with flat graphs was also striking given the relationship between PTSD course profile and self-reports of treatment history: As was expected, the individuals shown to have the most severe PTSD symptoms (see Table 4), veterans with multi-peak disorder courses, demonstrated the highest rates of mental-health treatment of all kinds during or after their military service—ER mental health services, VA mental health services, and the combined VA and non-VA mental health services. With the exception of Major Depressive Disorder, veterans with multi-peak graphs generally did *not* demonstrate rates of comorbid disorders higher than those of veterans in the single-peak group. It seems that veterans with multi-peak disorder courses may have suffered with and sought treatment directly for PTSD-related symptoms more so than the other groups, and related to this, they may have been particularly attuned to temporary ameliorations and exacerbations of their PTSD symptoms. In contrast, all of the veterans with flat disorder courses suffered with both PTSD and other Axis I disorders, and this combination may have reduced their

attunement to the disorder's ups and downs. It is notable that, despite the presence of comorbid disorders in *all* veterans with flat disorder courses, only 2 of this group (28.6%) reported having received mental health services, and none of them reported having received specific substance abuse treatment outside of the VA.

In sum, veterans with flat graphs stood out as being a subgroup of veterans with war-related PTSD who are afflicted by multiple psychiatric disorders and under-treated with mental health services. Veterans with multi-peak disorder courses stood out as the most severely symptomatic individuals at the time of the clinical interviews, the most attuned to ameliorations and exacerbations in symptoms, and most likely to have received VA or non-VA mental health services.

Course as a moderator of the dose-response relationship. Course subtype was found to mediate the relationship between level of severity of combat trauma and current PTSD diagnosis. It had previously been established amongst veterans from the NVVRS sample with and without lifetime diagnoses of PTSD, that combat severity is strongly related to rates of current PTSD (Dohrenwend et al., 2006). This study reduced the sample to veterans with lifetime diagnoses of war-related PTSD, and asked whether the relationship between combat severity and PTSD persistence changes when veterans with different PTSD course subtypes are separated into groups. As predicted, for veterans with single-peak disorder course, (i.e. the majority of veterans), the dose-response relationship held: severity of combat trauma measured by the MHM was directly related to PTSD diagnosis at the time of the clinical interview, with those exposed to higher levels of combat severity being significantly more likely to meet criteria for current diagnosis. For veterans who experienced flat or multi-peak disorder courses, however, the dose-response

relationship did not hold. All of these individuals met criteria for current PTSD regardless of whether they were exposed to high or low levels of combat severity. This finding supports the hypothesis that the dose-response relationship is contingent on PTSD course subtype. The dose response relationship appears to hold true for some, indeed a majority, of the veterans in this sample, but it holds no relevance for the minority of individuals afflicted with multi-peak or flat courses of PTSD.

The Etiology of PTSD Course Subtype: The Role of Future Research

This study underscores the importance of examining PTSD course as an additional source of information about war-related PTSD, and as a predictor of PTSD outcomes. It also demonstrates that course subtype acts as a condition under which the dose-response relationship operates, a finding that is particularly relevant in the ongoing struggle to parse out the various etiological threads of PTSD. It remains unclear, however, what causes different veterans to develop different PTSD course subtypes.

On one hand, course subtype may be a function of the individual's pre-existing capacity to adapt to and recoil from traumatic stress though there are no findings here to support this theory. Before this possibility can be conclusively ruled out, future research must examine with greater detail, and in prospectively designed investigations, the roles of pre-morbid Axis II disorders (other than antisocial personality disorder and conduct disorder) and other possible premorbid correlates of poor stress response mechanisms that were not examined in this study.

On the other hand, it appears from this study course subtype is significantly related to one exposure variable (i.e. purple hearts), and there are clear trends reflecting

the association between course profile and other exposure variables. These findings present a conceptual challenge because they seems to suggest that course subtype *both* moderates the relationship between trauma severity and PTSD outcomes, *and* is to some degree a function of trauma severity.

One way to understand this finding is to consider course subtype as a marker of extremely poor PTSD outcomes predicted by very severe traumatic events that go undetected using the Military Historical Measure to assess combat severity. In this interpretation, the course is in fact not moderating the relationship between dose and response, as it appears, but is actually an artifact of the poor outcome. Why, then, in this scenario, would the pre-established measure of the dose-response relationship not apply to individuals with flat and multi-peak course subtypes? Perhaps because there is a hierarchy of traumatic events, some of which go undetected by the objective Military Historical Measure. For example, a soldier stationed in a unit with a comparatively low risk of combat and a low rate of combatants killed in action at the time of service, (i.e. low MHM combat severity), may nonetheless experience a severe trauma if he gets shot by a stray bullet while engaging in support activities. If this high dose of trauma results in a PTSD course with ameliorations and re-exacerbations, the veteran will be an example of someone who displays the relationship between combat-related injury and course profile. This high dose of trauma he received will go undetected by the MHM in our study, and the multi-peak course subtype, which is in fact an artifact of the traumatic exposure, will appear to mediate the dose-response relationship since this veteran's trauma does not meet criteria for the MHM rating of high combat severity.

Another way to understand how course subtype may mediate the dose-response relationship and also be a function of trauma severity involves conceptualizing the worst PTSD outcomes as occurring after some threshold is reached in the individual's stress response mechanism. This explanation blends individual vulnerability with factors related to traumatic exposure. If an individual's pre-set threshold for traumatic stress is met or exceeded, PTSD symptoms either persist relentlessly (flat course) or seem to produce no traction in the progression towards relative health as is indicated by multiple exacerbations over time (multi-peak course). According to this theory, course subtype may be related to the individual's internal threshold for adapting to stress, which may be different for different veterans. Certain traumatic events (i.e. physical injury in combat or traumatic brain injury) may cause the mechanism to reach or surpass its internal threshold in all or most individuals, precipitating greater rates of flat and multi-peak disorder courses and chronic, recalcitrant, PTSD outcomes, but provided the threshold has not been exceeded, the dose-response relationship as measured by objective measures of combat severity continues to hold. Future research must examine prospectively the role of physical injury and combat-related changes in the brain's physiology on PTSD course and later outcomes. It is also important to develop ways of testing support for the theory that specific, extremely poor outcomes of PTSD related to flat and multi-peak disorder course subtypes are related to exceeding an internal threshold that other veterans *who also develop PTSD* have not exceeded.

Other findings that call for further study include the differences in the timing of the worst period of symptoms and the vastly different experiences of symptom change over time across PTSD course subtype. That the worst PTSD outcomes were uniformly

associated with the multi-peak disorder course and that this group had the most delayed symptomatic crescendo to the worst period, informs us that there is something adaptive about experiencing the worst period closer to the traumatic event, and suggests there may indeed be something adaptive in the experience of PTSD symptoms for those with single-peak disorder course that is not adaptive for those with multi-peak courses. This idea should be pursued in greater detail in studies that frequently, even continuously, track the change in specific types of symptoms as well as the severity of symptoms over time, to delve into the mechanisms of PTSD and to understand better the functions of symptoms in healthy people exposed to trauma compared to traumatized people diagnosed with PTSD and those diagnosed with chronic PTSD and its variety of disorder course subtypes.

Clinical Implications

The clinical implications of this study are multifold. The confirmation of specific subtypes of disorder course is important information for clinicians working with veterans with PTSD, especially given that flat courses of PTSD and multi-peak courses of PTSD are invariably linked with chronic, and usually severe, outcomes. It may be important to develop specific interventions to work with these recalcitrant subtypes of disorder course, since these individuals are most likely to have to adapt to living *with* their disorder over time, and should therefore be provided specific kinds of support that prepare them to deal optimally with chronic forms of the disorder. It may also be that treatment goals for individuals with flat and multi-peak disorder subtypes should differ than for those with single-peak disorder course. For example, the current findings suggest that veterans with

the highest rates of comorbid disorders, (i.e. those with flat graphs of disorder course) must be treated for the co-existing disorders and receive ongoing assessments of the course of their PTSD, since they may experience changes in the course of their PTSD once the comorid problems are effectively treated.

Further research is needed to see if these different groups respond differently to psychopharmacological interventions, exposure therapies, group interventions, acceptance-based treatments, and treatments directed at integrating memories of traumatic events. Whereas such treatments may be mutative for veterans with certain kinds of disorder subtype, they may be less effective, and even frustratingly ineffective and lead to poor treatment adherence for veterans with other kinds of disorder subtype.

If further research confirms the existence of different PTSD course subtypes, as well as the correlates, likely outcomes, and specific responses to treatment of these outcomes, it will be important to integrate this information in psychoeducational components of treatment interventions. This information will increase veterans' and providers' understanding of the disorder, facilitate communication with treatment providers about their symptoms and the course of the disorder, and better equip veterans to cope with their PTSD and make informed choices about treatment.

Limitations

This study was limited by the small number of veterans in the diagnosed subsample with lifetime war-related PTSD, and the even smaller proportions of these that had the less typical flat and multi-peak disorder courses. Frequently, analyses were hampered by low cell counts, low expected frequencies, and higher than average risks of

Type II error. Another potential issue with so many analyses is the increased risk of Type I error that develops due to multiple comparisons. The data were also limited by their dependence on veterans' self-reports of the disorder's course over the nearly two decades prior to the interview. Although subjective reports of disorder symptoms are essential, valid subjects of inquiry, these are also difficult to compare across participants since there is no objective standard of reference. It should be noted, however, that in a longitudinal investigation of the agreement between Vietnam veterans' reports of PTSD symptoms and clinicians' reports of veterans' PTSD symptoms across two separate randomized clinical trials, there were significant associations between patient and clinician ratings of total symptoms and PTSD symptom clusters, and no differences in between clinician and patient ratings of improvement and exacerbations (Monson, C.M., Gradus, J.L., Young-Xu, Y., Schnurr, P.P., Price, J.L., & Schumm, J.A., 2008).

The retrospective recollection of changes in disorder course over time may have been limited, in part because it is a challenge to remember symptom changes of any disorder over years, let alone over a decade or more, and in part because memories of the disorder may have been influenced by the mental status and the PTSD-related status of the veteran at the time of recall, even despite that these recollections were assessed by a trained clinician during the administration of the SCID. The visual data on the Cartesian graphs of changes in symptom severity over time were hindered by the absence of detailed time markers. The precise collection of this continuous data could have been greatly improved were the graphs to have been drawn with reference to specific years and major life events, and such prompts would have likely aided veterans' recollections since these markers would have served to provide a context for past symptoms and have

prompted veterans' recall of memorable time period. Nevertheless, this is the only study of representative samples of Vietnam veterans or veterans of Iraq and Afghanistan in which rigorous diagnoses were conducted by experienced clinicians—diagnoses that give information about factors in both onset and course of war-related PTSD—by modern diagnostic criteria. These findings, at the very least, should serve as strong hypotheses for further research on the course of war-related PTSD.

Table 1. Studies on veterans of wars before 1980 with findings on the course of war-related PTSD

Authors	**Veterans**	**Sample**	**N**	**Time points**	**PTSD Measure(s)**	**Study Goal**
Kluznik et al., 1986	Ex-POWs from WWII (males)	Convenience sample of volunteers recruited from VA office (Minneapolis).	188	T1: 40 yrs after war	Lifetime PTSD and Current PTSD with severity (mild, medium, or marked) assessed with: -Structured psychiatric interviews with psychiatrist-administered DSM III symptom checklist	To examine rates of lifetime and current PTSD in WWII ex-POWs 40 yrs after the war. Also looked at rates of recovery and delayed onset.
Port et al., 2001	Ex- POWs from WWII and Korean War (males)	Convenience sample of volunteers contacted through the VA Medical Center (Minneapolis).	244 (retro-spective) 177 (long-itudinal)	T1 (Retro-spective): over 40 yrs after war T2 (long-itudinal): four yrs later than T1	Prevalence rates and symptom levels assessed with: -T1: In person interview with administration of Mississippi Scale for Combat-Related PTSD (self-report) -T2: Mississippi Scale for Combat-Related PTSD (self-report) sent in by mail.	To examine PTSD prevalence, symptom levels, and long-delayed onset retrospectively at T1, and longitudinally between T1 and T2
Speed et al., 1989	Ex-POWs from WWII (males)	Convenience sample of volunteers recruited from VA office (Minneapolis), randomly scheduled with and examined by first author.	62	T1: 40 yrs after war	Lifetime PTSD and Current PTSD with severity (mild, medium, or marked) assessed with: -Structured psychiatric interviews with psychiatrist-administered DSM III symptom checklist	To estimate relative contributions of trauma and premorbid disposition in onset and persistence of PTSD 40 years after the war.
Zeiss & Dickman, 1989	Ex- POWs from WWII (males)	Convenience sample of volunteers mailed questionnaires through VA in Virginia.	442	T1: 3-4 decades after war	PTSD symptoms assessed with: -Questionnaire (designed by authors) that inquired about specific symptoms from diagnostic criteria for PTSD (DSM-III).	To examine current and past difficulties with PTSD-related symptoms.

Table 1. Studies on veterans of wars before 1980 with findings on the course of war-related PTSD

Authors	Veterans	Sample	N	Time points	PTSD Measure(s)	Study Goal
Dirkzwager et al., 2001	Dutch veterans of WWII, Dutch East Indies, and Korea (males)	198 volunteer Dutch military veterans who had participated in previous study of randomly selected community sample of 1,461 war survivors (born 1920-1929) plus 576 veterans receiving Dutch military disability pension.	674	T1: 1992 T2: 1998	-Self-Rating Inventory for PTSD, mailed to veterans at each assessment.	To examine longitudinally the course of PTSD over six years.
Op den Velde et al., 1996	WWII Dutch resistance fighters (males)	4 separate studies of convenience samples	1098	T1: decades after war	PTSD prevalence assessed with: -PTSD questionnaire.	To examine current rates of PTSD and estimate lifetime prevalence.
Van Dyke et al., 1985	WWII Veteran (males)	Case study	1	T1: over 3 decades after war	Clinician-made diagnosis of DSM-III-R PTSD.	To describe case of WWII veteran with 30-yr delayed PTSD onset.

Table 1. Studies on veterans of wars before 1980 with findings on the course of war-related PTSD

Authors	**Veterans**	**Sample**	**N**	**Time points**	**PTSD Measure(s)**	**Study Goal**
Bremner et al., 1996	Vietnam (males)	Convenience sample from combat veterans admitted voluntarily for inpatient treatment for chronic PTSD and co-morbid Substance Use Disorder	61	1: 2-3 decades after service	PTSD diagnosis and assessment of symptoms made with: -Mississippi Scale for Combat-Related PTSD (self report) (cut-off score greater than 108) -verification of diagnosis from 2 psychiatrists. -SCID-DSM-III-R, where available Past course of PTSD assessed in interview with timeline and checklist of symptoms.	To measure the longitudinal course of specific PTSD symptoms and related symptoms of substance abuse.
Dohrenwend et al., 2006	Vietnam (males)	NVVRS clinical subsample	260	1: 1-2 decades after service	-SCID-DSM-III-R, adjusted for functional impairment as per DSM-IV criteria using the Global Assessment of Functioning.	To cross-check the original findings of the NVVRS of lifetime and current rates of PTSD and measures of combat exposure in Vietnam.
Johnson et al., 2004	Vietnam (males)	Convenience sample of veterans in 15-week inpatient treatment for PTSD at VAMC (CT).	51	T1: At intake 2-3 decades after service. T2: 6 years later	-Mississippi PTSD Scale (Cut-off of 107 for indicated diagnosis) self report administered at treatment center at T1, and administered by research assistants in person at T2 where possible, or filled out by veterans and returned by mail.	To examine longitudinally PTSD course and functional outcomes six years after inpatient treatment program for severe, chronic PTSD.

Table 1. Studies on veterans of wars before 1980 with findings on the course of war-related PTSD

Authors	**Veterans**	**Sample**	**N**	**Time points**	**PTSD Measure(s)**	**Study Goal**
Schnurr et al., 2003	Vietnam (males and females)	Clinical samples from both the NVVRS and Hawaiian Vietnam Veterans Project (included veterans identifying as Native Hawaiian and Americans of Japanese Ancestry)	530	1: 1-2 decades after service	-SCID for DSM-III-R.	To examine onset and chronicity of PTSD in 2 large, ethnically diverse samples.
Solomon & Kleinhaz, 1996	Israeli veterans of Yom Kippur War (males)	Combat Stress Reaction casualties were identified and recruited through medical records; controls were identified through Israeli Defense Forces	301	1: 18 yrs after war.	-PTSD Inventory, a self-report questionnaire based on DSM-III-R to assess lifetime and current diagnosis and symptom levels.	To examine retrospectively rates of current and lifetime PTSD in veterans who had been diagnosed with combat stress reaction with comparable controls.

Table 2. Studies on veterans of wars since 1980 with findings on the course of war-related PTSD

Authors	**Veterans**	**Sample**	**N**	**Time points**	**PTSD Measure(s)**	**Study Goal**
Grieger et al., 2006	Iraq & Afghan-istan	Convenience sample of hospitalized patients following serious combat injury at the Walter Reed Army Medical Center	243	T1: 1 mo. T2: 4 mos. T3: 7 mos. after injury	-PTSD Checklist- self-report	To examine rates, predictors, and course of probable PTSD and depression among seriously injured soldiers during and following hospitalization.
McCarroll et al.,1995	Non-combat veterans from Gulf War (males and females)	Volunteer veterans who had served in Army mortuary affairs companies and who had handled at least one body were selected and matched with veterans who had participated in the war doing logistical functions other than handling human remains.	111	T1: 3-5 T2: 13-15 months after service	Authors did *not* measure diagnostic status. PTSD and other psychiatric symptoms measured using -Impact of Event Scale -SCL-90R	To examine whether individuals who participated in the recovery of war dead were more likely to experience later symptoms of PTSD than those who did not handle human remains.
Orcutt et al., 2004	Gulf War (males and females)	Millenium Cohort Study of Gulf War veterans who volunteer to participate.	2949	T1: 5 days T2: 18 mos. T3: 72 mos. after service	Authors did *not* assess PTSD diagnostic status. PTSD symptoms were examined using: -PTSD Checklist- Military version-self-report -Mississippi Scale for Desert Storm War Zone Personnel adapted from Mississippi Scale for Combat-Related PTSD.	To use structural equation modeling approach to examine the trajectory/ies of PTSD symptoms across three time points.

Table 2. Studies on veterans of wars since 1980 with findings on the course of war-related PTSD

Authors	**Veterans**	**Sample**	**N**	**Time points**	**PTSD Measure(s)**	**Study Goal**
Solomon & Mikulincer, 2006	Lebanon War (1982) (males)	Veterans identified by military mental health providers as having had combat stress reaction diagnosed on the battlefield and matched controls from same units who were not identified as having had CSR.	214	T1: 1 yr T2: 2yrs T3: 3 yrs T4: 20 yrs after war	-PTSD Inventory: A self-report instrument based on DSM-III -supplemented by measures of distress and disability to assess DSM-IV criteria of functional impairment	To assess psychopathological effects of combat in veterans with and without combat stress reaction.
Southwick et al., 1995	Gulf War (males and females)	Convenience sample of National Guard reservists who returned from Gulf War one month prior to T1.	62	T1: 1 mo. T2: 6 mos. T3: 2 yrs after service	-Self-report Mississippi PTSD Scale (cut-off of 89) -DSM-III-R PTSD scale.	Prospective examination of development of trauma-related symptoms over two years after service in Operation Desert Storm.
Thompson et al., 2004	Gulf War (males and females)	Veterans who had received psychological testing at New Orleans VAMC PTSD Evaluation, Debriefing, and Treatment Team volunteered to be re-tested at T2 and be part of study.	348	T1: 1 yr T2: 2 yrs after service	Authors did *not* assess PTSD diagnostic status. PTSD symptoms were examined using: -PTSD Checklist- Military version-self-report -Mississippi Scale for Desert Storm War Zone Personnel adapted from Mississippi Scale for Combat-Related PTSD.	To examine course of psychological symptoms over 2 years after service in the Gulf War
Wolfe et al., 1999	Gulf War (males and females)	Millenium Cohort Study of Gulf War veterans who volunteer to participate.	2949	T1: 5 days T2: 2 yrs after service	-Self-report Mississippi PTSD Scale (cut-off 94).	To examine rates and predictors of PTSD immediately following war and 2 years later.

Table 3

Means and standard deviations of the total sample for periods on the PTSD timeline.

Time Period		Total Sample Means (years)		
		N	***M***	***(SD)***
A	End of service to symptom onset	84	-0.17	(1.54)
B	End of service to worst period start	78	1.70	(4.31)
C	Symptom onset to mid point worst period	77	3.33	(4.17)
D	Length of worst period	77	3.03	(3.05)
E	End of worst period to interview	77	13.71	(5.19)

Table 4

Chi-Square analyses of 3-category course subtype by PTSD symptom severity at the worst period.

	PSTD Symptom Severity at Worst Period								
	Mild		**Moderate**		**Severe**		**Tests of Significance**		
Course Subtype	% of row	(*n*)	% of row	(*n*)	% of row	(*n*)	χ^2	*p*	*Φ*
Single-peak (*n* = 63)	6.3%	(4)	30.2%	(19)	63.5%	(40)			
Multi-peak (*n* = 13)	0%	(0)	0%	(0)	100%	(13)			
Flat (*n* = 7)	14.3%	(1)	14.3%	(1)	71.4%	(5)			
Total Sample (*N* = 83)	6.0%	(5)	24.1%	(20)	69.9%	(58)	8.02	0.09*	0.311

*Trend toward significance (where significance is considered $p < 0.05$)

Table 5

Chi-Square analyses of 2-category course subtype by PTSD symptom severity at the worst period.

	PSTD Symptom Severity at Worst Period								
	Mild		**Moderate**		**Severe**		**Tests of Significance**		
Course Subtype	% of row	(*n*)	% of row	(*n*)	% of row	(*n*)	χ^2	*p*	***Φ***
Single-peak (*n* = 63)	6.3%	(4)	30.2%	(19)	63.5%	(40)			
Flat/Multi-peak (*n*= 20)	5%	(1)	5%	(1)	90%	(18)			
Total Sample (*N* = 83)	6.0%	(5)	24.1%	(20)	69.9%	(58)	5.56	0.06*	0.26

*Trend toward significance (where $p < 0.05$).

Table 6

One-way analyses of variance for 3-category course subtype by time periods on the PTSD timeline.

Time Period (years)	PTSD Course Subtype									Significance Tests		
	Single-Peak			Flat			Multi-Peak					
	n	***M***	***(SD)***	***n***	***M***	***(SD)***	***n***	***M***	***(SD)***	***F***	***Df, df***	***p***
A	64	-0.23	(1.47)	7	-0.29	(0.76)	13	0.23	(2.13)	0.51	2, 81	0.60
B	61	1.15	(3.99)	5	-0.05	(0.42)	12	5.19	(5.21)	5.37	2, 75	0.007*
C	61	2.80	(3.73)	5	1.80	(1.3)	11	7.0	(5.50)	5.72	2, 74	0.005*
D	61	2.58	(2.34)	5	2.60	(1.67)	11	5.73	(5.26)	5.61	2, 74	0.005*
E	61	14.29	(4.69)	5	16.85	(2.65)	11	9.03	(6.24)	6.61	2, 74	0.002*

Time periods measured: A) End of service to symptom onset; B) End of service to worst period start; C) Symptom onset to mid point worst period; D) Length of worst period; E) End of worst period to interview. (See Figure 1 for graphical depiction of time periods).

* $p < 0.05$.

Table 7

Chi-Square analyses of 3-category course subtype by race.

	PTSD Course Subtype							
	Single-Peak (*n* = 64)		**Flat** (*n* = 7)		**Multi-Peak** (*n* = 13)		**Total Sample** (*N* = 84)	
Race	% of column	*n*	% of column	*n*	% of column	*n*	% of column	*n*
White	26.6%	17	14.3%	1	23.1%	3	25%	21
Black	34.4%	22	14.3%	1	30.8%	4	32.1%	27
Hispanic	39.1%	25	71.4%	5	46.2%	6	42.9%	36
Tests of Significance	χ^2	2.79						
	p	0.59						
	Φ	0.18						

Table 8

Chi-Square analyses of 3-category course subtype by pre-military education.

	PTSD Course Subtype							
	Single-Peak (*n* = 64)		**Flat** (*n* = 7)		**Multi-Peak** (*n* = 13)		**Total Sample** (*N* = 84)	
Education	% of column	*n*	% of column	*n*	% of column	*n*	% of column	*n*
Some HS	29.7%	19	42.9%	3	30.8%	4	30.1%	26
HS Grad	51.6%	33	57.1%	4	46.2%	6	51.2%	43
Some College	17.2%	11	0%	0	23.1%	3	16.7%	14
College Grad	1.6%	1	0%	0	0%	0	1.2%	1
Tests of Significance	χ^2	2.28						
	p	0.89						
	Φ	0.17						

Table 9

One-way analyses of variance of 3-category PTSD course subtype by AFQT scores.

	PTSD Course Subtype											
	Single-Peak			**Flat**			**Multi-Peak**			**Total Sample**		
	n	*M*	*(SD)*	*n*	*M*	*(SD)*	*n*	*M*	*(SD)*	*N*	*M*	*(SD)*
AFQT Scores	55	39.8	(23.7)	6	41.8	(8.4)	9	43.3	(29.5)	70	40.4	(23.4)
Tests of significance	*F*	0.10										
	Df, df	2, 67										
	P	0.91										

Table 10

One-way analyses of variance of 3-category PTSD course subtype by age at entry to military service in the Vietnam War.

	PTSD Course Subtype											
	Single-Peak			**Flat**			**Multi-Peak**			**Total Sample**		
	n	*M*	*(SD)*	*n*	*M*	*(SD)*	*n*	*M*	*(SD)*	*N*	*M*	*(SD)*
Age (years)	64	20.6	(2.8)	7	20.0	(1.3)	13	19.9	(2.3)	84	20.5	(2.6)
Tests of significance	*F*	0.50										
	Df, df	2, 81										
	P	0.61										

Table 11

Independent samples t-test for 2-category PTSD course subtype by AFQT scores and age at entry to military service in the Vietnam War.

	PTSD Course Subtype						**Tests of significance**		
	Single-Peak			**Flat/ Multi-Peak**					
	n	*M*	*(SD)*	*n*	*M*	*(SD)*	*T*	*df*	*p*
AFQT	55	39.8	(23.7)	15	42.7	22.9	0.43	68	0.67
Age (years)	64	20.6	(2.8)	20	20.0	(2.0)	-1.0	82	0.32

Table 12

Chi-Square analyses of 3-category course subtype by current PTSD diagnosis at interview.

	Diagnostic status at interview				**Tests of significance**		
	PTSD		No PTSD		3-Category Subtype		
Course Subtype	% row	(*n*)	% row	(*n*)	χ^2	*p*	Φ
Single-Peak (*n* = 64)	57.8%	(37)	42.2%	(27)			
Flat Course (*n* = 7)	100%	(7)	0%	(0)			
Multi-Peak (*n* = 13)	100%	(13)	0%	(0)			
Total Sample (*N* = 84)	67.9%	(57)	32.1%	(27)	12.43	0.002*	0.39

* $p < 0.05$.

Table 13

Chi-Square analyses of 2-category course subtype by current PTSD diagnosis at interview.

	Diagnostic status at interview				Tests of significance		
	PTSD		No PTSD		3-Category Subtype		
Course Subtype	% row	(*n*)	% row	(*n*)	χ^2	*p*	***Φ***
Single-Peak (*n* = 64)	57.8%	(37)	42.2%	(27)			
Flat/ Multi-Peak Course (*n* = 20)	100%	(20)	0%	(0)			
Total Sample (*N* = 84)	67.9%	(57)	32.1%	(27)	12.43	0.00*	-0.39

* $p < 0.05$.

Table 14

One-way analyses of variance for 3-category course subtypes by mean scores on 3 PTSD symptom clusters.

		Mean scores on PTSD symptom clusters								
		Intrusive			Avoidant			Arousal		
Course Subtype		***n***	***M***	***(SD)***	***n***	***M***	***(SD)***	***n***	***M***	***(SD)***
Single-Peak (*n* = 64)		64	1.83	(1.39)	61	3.28	(2.02)	63	2.95	(1.75)
Flat (*n* = 7)		7	3.00	(0.82)	7	4.57	(1.72)	6	3.5	(1.64)
Multi-Peak (*n* = 13)		13	2.62	(0.77)	12	5.17	(1.27)	13	4.85	(0.90)
Total Sample (*N* = 84)		84	2.05	(1.33)	80	3.68	(2.02)	82	3.29	(1.76)
Tests of significance	***F***			4.17			5.77			7.25
	Df, df			2, 81			2, 77			2, 79
	p			0.019*			0.005*			0.001*

* $p < 0.05$.

Table 15

Independent samples t-test for 2-category course subtypes by mean scores on 3 PTSD symptom clusters.

		Mean scores on PTSD symptom clusters								
		Intrusive			Avoidant			Arousal		
Course Subtype		***n***	***M***	***(SD)***	***n***	***M***	***(SD)***	***N***	***M***	***(SD)***
Single-Peak										
(*n* = 64)		64	1.83	(1.39)	61	3.28	(2.02)	63	2.95	(1.75)
Flat/ Multi-Peak										
(*n* = 20)		20	3.00	(0.82)	19	4.57	(1.72)	19	4.42	(1.30)
Total Sample										
(*N* = 84)		84	2.05	(1.33)	80	3.68	(2.02)	82	3.29	(1.76)
	t			-3.73*			-3.35			-3.39
Tests of	***df***			57.48*			78			80
significance	***p***			0.00**			0.001*			0.001*

*Equal variances not assumed since significance for Levene's test for equality of variances was below .05.

** $p < 0.05$.

Table 16

Steps of improvement in PTSD symptom severity between worst period and interview.

Severity at the clinical Interview:		**Severe**	**Moderate**	**Mild**	**Remitted**
Severity at the worst period of symptoms	**Severe**	No change: Severe-severe	1-step improvement: severe-mod	2-steps improvement: severe-mild	3-steps improvement: severe-remit
	Moderate		No change: Mod-moderate	1-step improvement: Mod-mild	2-steps improvement: mod-remit
	Mild			No change: Mild-mild	1-step improvement: mild-remit

Table 17

Chi-Square analyses of 3-category course subtype by improvement scores in PTSD symptom severity from worst period to interview.

	Improvement in PTSD symptom severity from worst period to interview											
	0		**1**		**2**		**3**		**Tests of significance**			
Course Subtype	% row	(*n*)	% row	(*n*)	% row	(*n*)	% row	(*n*)	χ^2	*p*	***Φ***	
Single-Peak (*n* = 63)	17.5%	(11)	41.3%	(26)	22.2%	(14)	19%	(12)				
Flat (*n* = 7)	85.7%	(6)	14.3%	(1)	0%	(0)	0%	(0)				
Multi-Peak (*n* = 13)	38.5%	(5)	61.5%	(8)	0%	(0)	0%	(0)				
Total Sample (*N* = 84)	26.5%	(22)	42.2%	(35)	16.9%	(14)	14.5%	(12)	22.62	.001*	.52	

* $p < 0.05$

Table 18

Chi-Square analyses of 2-category course subtype by improvement scores in PTSD symptom severity from worst period to interview.

	Improvement in PTSD symptom severity from worst period to interview										
	0		**1**		**2**		**3**		**Tests of significance**		
Course Subtype	% row	(*n*)	% row	(*n*)	% row	(*n*)	% row	(*n*)	χ^2	*p*	*Φ*
Single-Peak (*n* = 63)	17.5%	(11)	41.3%	(26)	22.2%	(14)	19%	(12)			
Flat/Multi-Peak (*n* = 20)	55.0%	(11)	45.0%	(9)	0%	(0)	0%	(0)			
Total Sample (*N* = 84)	26.5%	(22)	42.2%	(35)	16.9%	(14)	14.5%	(12)	16.4	.001*	.44

* $p < 0.05$

Table 19

Steps of improvement in PTSD symptom severity with frequency counts.

Severity at the clinical Interview:		**Severe**	**Moderate**	**Mild**	**Remitted**
Severity at the worst period of symptoms	**Severe**	No change: SS Severe-severe *n* = 12	1-step: SM severe-mod *n* = 28	2-steps: SD severe-mild *n* = 6	3-steps: SR severe-remit *n* = 12
	Moderate		No change MM Mod- Mod *n* = 8	1-step: MD Mod-mild *n* = 4	2-steps: MR mod-remit *n* = 8
	Mild			No change: DD Mild-mild *n* = 2	1-step: DR mild-remit *n* = 3

Table 20

Symptom improvement collapsed into five categories.

Severity at the clinical Interview:		**Severe**	**Moderate**	**Mild**	**Remitted**
Severity at the worst period of symptoms	**Severe**	No change: SS Severe-severe *n* = 12	Severe- mild/moderate *n* = 34		severe-remit *n* = 12
	Moderate		Mild/moderate-mild/moderate *n* = 14		Mild/mod-remit *n* = 11
	Mild				

Table 21

Chi-Square analyses of 3-category course subtype by membership in PTSD symptom improvement categories.

	Improvement in PTSD symptom severity from worst period to interview									
	Severe-Severe		**Mild/Mod-Mild/Mod**		**Severe-Mild/Mod**		**Mod/Mild-Remit**		**Severe-Remit**	
Course Subtype	% row	(*n*)	% row	(*n*)	% row	(*n*)	% row	(*n*)	% row	(*n*)
Single-Peak (*n* = 63)	4.8%	(3)	19.0%	(12)	39.7%	(25)	17.5%	(11)	19%	(12)
Flat (*n* = 7)	57.1%	(4)	28.6%	(2)	14.3%	(1)	0%	(0)	0%	(0)
Multi-Peak (*n* = 13)	38.5%	(5)	0%	(0)	61.5%	(8)	0%	(0)	0%	(0)
Total Sample (*N* = 83)	14.5%	(12)	16.9%	(14)	41.0%	(34)	13.3%	(11)	14.5%	(12)
Tests of Significance	χ^2	30.92								
	p	0.00*								
	Φ	0.61								

*Statistically significant where $p < 0.05$

Table 22

Chi-Square analyses of 2-category course subtype by membership in PTSD symptom improvement categories.

	Improvement in PTSD symptom severity from worst period to interview									
	Severe-Severe		**Mild/Mod-Mild/Mod**		**Severe-Mild/Mod**		**Mod/Mild-Remit**		**Severe-Remit**	
Course Subtype	% row	(*n*)	% row	(*n*)	% row	(*n*)	% row	(*n*)	% row	(*n*)
Single-Peak (*n* = 63)	4.8%	(3)	19.0%	(12)	39.7%	(25)	17.5%	(11)	19%	(12)
Flat/ Multi-Peak (*n* = 20)	45.0%	(9)	10.0%	(2)	45.0%	(9)	0%	(0)	0%	(0)
Total Sample (*n* = 83)	14.5%	(12)	16.9%	(14)	41.0%	(34)	13.3%	(11)	14.5%	(12)
Tests of Significance	χ^2	25.14								
	p	<0.005*								
	Φ	0.55								

* $p < 0.05$

Table 23

Chi-Square analyses of 3-category PTSD course subtype by pre-war vulnerability factors.

	PTSD Course Subtype										
	Single-peak (*n* = 64)*		**Flat** (*n* = 7)		**Multi-Peak** (*n*=13)		**Total Sample** (*N* = 84)*		**Tests of significance**		
Pre-war Vulnerability	% group	(*n*)	% group	(*n*)	% group	(*n*)	% group	(*n*)	χ^2	*p*	*Φ*
Axis I Dx	28.1	(18)	14.3	(1)	23.1	(3)	26.2	(22)	0.70	0.70	0.09
No history	71.9	(46)	85.7	(6)	76.9	(10)	73.8	(62)			
Conduct Disorder	28.1	(18)	28.6	(2)	15.4	(2)	26.2	(22)	0.93	0.63	0.11
No history	71.9	(46)	71.4	(5)	84.6	(11)	73.8	(62)			
Physical abuse	30.2	(19)	42.9	(3)	30.8	(4)	31.3	(26)	0.47	0.79	0.08
No history	69.8	(44)	57.1	(4)	69.2	(9)	68.7	(57)			
Family subst. abuse	23.4	(15)	14.3	(1)	15.4	(2)	21.4	(18)	0.65	0.72	0.09
No history	76.6	(49)	85.7	(6)	84.6	(11)	78.6	(66)			

* *N* (single-peak course subtype) = 64 and *N* (total sample) = 84 for all variables except childhood physical abuse for which only 63 veterans in the single-peak subtype provided information. Consequently, *N* (total sample) = 83 for that variable.

Table 24

One-way analyses of variance of 3-category PTSD course subtype by mean total months of military service in the Vietnam War.

	PTSD Course Subtype											
	Single-Peak			**Flat**			**Multi-Peak**			**Total Sample**		
	n	*M*	*(SD)*	*n*	*M*	*(SD)*	*n*	*M*	*(SD)*	*N*	*M*	*(SD)*
Total Months Service	64	14.9	(8.1)	7	16.0	(8.7)	13	16.5	(10.9)	84	15.3	(8.5)
Tests of significance	*F*	0.22										
	Df, df	2, 81										
	P	0.80										

Table 25

Independent samples t-test for 2-category PTSD course subtype by mean total months of military service in the Vietnam War.

	PTSD Course Subtype						
	Single-Peak			**Flat/Multi-Peak**			**Tests of significance**
	n	*M*	*(SD)*	*n*	*M*	*(SD)*	*t* *df* *p*
Total Months Service	64	14.9	(8.1)	20	16.4	(10.0)	1.44 82 0.51

Table 26

Chi-Square analyses of 3-category PTSD course subtype by exposure factors.

		PTSD Course Subtype											
		Single-peak (n = 64)**		**Flat** (n = 7)		**Multi-Peak** (n=13)		**Total Sample** (N = 84)**		**Tests of significance**		
Exposure Variables		% group	(n)	% group	(n)	% group	(n)	% group	(n)	χ^2	p	Φ
MHM High		34.4	(22)	28.6	(2)	38.5	(5)	34.5	(29)	0.20	0.91	0.05
MHM Low		65.6	(42)	71.4	(5)	61.5	(8)	65.5	(55)			
Threat high		67.2	(43)	71.4	(5)	84.6	(11)	70.2	(59)	1.58	0.46	0.14
Threat low		32.8	(21)	28.6	(2)	15.4	(2)	29.8	(25)			
Purple heart	Yes	12.5	(8)	28.6	(2)	46.2	(6)	19.0	(16)	8.39	0.02*	0.32
	No	87.5	(56)	71.4	(5)	53.8	(7)	81.0	(68)			
Friend killed	Yes	71.4	(45)	85.7	(6)	92.3	(12)	75.9	(63)	2.97	0.23	0.19
	No	28.6	(18)	14.3	(1)	7.7	(1)	24.1	(20)			
Betrayed in threat	Yes	10.9	(7)	0.0	(0)	23.1	(3)	11.9	(10)	2.55	0.28	0.17
	No	89.1	(57)	100	(7)	76.9	(10)	88.1	(74)			
Killed enemy	Yes	59.6	(34)	71.4	(5)	84.6	(11)	64.9	(50)	3.04	0.22	0.20
	No	40.4	(23)	28.6	(2)	15.4	(2)	35.1	(27)			
Harmed civs/pris	Yes	40.3	(25)	28.6	(2)	41.7	(5)	39.5	(32)	0.39	0.82	0.07
	No	59.7	(37)	71.4	(5)	58.3	(7)	60.5	(49)			

* Statistically significant where p <0.05

** N (single-peak course subtype) = 64 and N (total sample) = 84 for all variables except saw friend killed for which only 63 veterans in the single-peak subtype provided information. Consequently, N (total sample) = 83 for that variable.

Table 27

Chi-Square analyses of 2-category PTSD course subtype by exposure factors.

		PTSD Course Subtype								
		Single-peak (*n* = 64)**		**Flat/ Multi-Peak** (*n* = 20)		**Total Sample** (*N* = 84)**		**Tests of significance**		
Exposure Variables		% group	(*n*)	% group	(*n*)	% group	(*n*)	χ^2	*p*	*Φ*
MHM High		34.4	(22)	35.0	(7)	34.5	(29)	0.00	0.96	-0.01
MHM Low		65.6	(42)	65.0	(13)	65.5	(55)			
Threat high		67.2	(43)	80.0	(16)	70.2	(59)	1.20	0.27	-0.12
Threat low		32.8	(21)	20.0	(4)	29.8	(25)			
Purple heart	Yes	12.5	(8)	40.0	(8)	19.0	(16)	7.47	0.01*	-0.30
	No	87.5	(56)	60.0	(12)	81.0	(68)			
Friend killed	Yes	71.4	(45)	90.0	(18)	75.9	(63)	2.86	0.09	-0.19
	No	28.6	(18)	10.0	(2)	24.1	(20)			
Betrayed in threat	Yes	10.9	(7)	15.0	(3)	11.9	(10)	0.24	0.62	-0.05
	No	89.1	(57)	85.0	(17)	88.1	(74)			
Killed enemy	Yes	59.6	(34)	80.0	(16)	64.9	(50)	2.69	0.10	-0.19
	No	40.4	(23)	20.0	(4)	35.1	(27)			
Harmed civs/pris	Yes	40.3	(25)	36.8	(7)	39.5	(32)	0.07	0.79	0.03
	No	59.7	(37)	63.2	(12)	60.5	(49)			

* Statistically significant where $p < 0.05$

** *N* (single-peak course subtype) = 64 and *N* (total sample) = 84 for all variables except saw friend killed for which only 63 veterans in the single-peak subtype provided information. Consequently, *N* (total sample) = 83 for that variable.

Table 28

Chi-Square analyses of 3-category PTSD course subtype by psychiatric disorders with onset at or after Vietnam.

		PTSD Course Subtype										
		Single-peak (*n* = 64)		**Flat** (*n* = 7)		**Multi-Peak** (*n*=13)		**Total Sample** (*N* = 84)		**Tests of Significance**		
Co-morbid Diagnoses		% group	(*n*)	% group	(*n*)	% group	(*n*)	% group	(*n*)	χ^2	*p*	*Φ*
Alcohol abuse/ depend-ence	Yes	45.3	(29)	85.7	(6)	38.5	(5)	47.6	(40)	4.65	0.098	0.24
	No	54.7	(35)	14.3	(1)	61.5	(8)	52.4	(44)			
Other drug abuse/ depend-ence	Yes	32.8	(21)	14.3	(1)	30.8	(4)	31.0	(26)	1.01	0.60	0.11
	No	67.2	(43)	85.7	(6)	69.2	(9)	69.0	(58)			
Major Depress-ive Disorder	Yes	34.4	(22)	57.1	(4)	46.2	(6)	38.1	(32)	1.81	0.40	0.15
	No	65.6	(42)	42.9	(3)	53.8	(7)	61.9	(52)			
Panic/ phobia/ OCD	Yes	21.9	(14)	57.1	(4)	23.1	(3)	25.0	(21)	4.22	0.12	0.22
	No	78.1	(50)	42.9	(3)	76.9	(10)	75.0	(63)			
Any of the above	Yes	70.3	(45)	100.0	(7)	76.9	(10)	73.8	(62)	2.95	0.23	-.13
	No	29.7	(19)	0.0	(0)	23.1	(3)	26.2	(22)			

Table 29

Chi-Square analyses of 3-category PTSD course subtype by treatment history.

		PTSD Course Subtype										
		Single-peak** (*n* = 63)		**Flat** (*n* = 7)		**Multi-Peak** (*n*=13)		**Total Sample**** (*N* = 83)		**Tests of significance**		
Self-Reported Treatment		% group	(*n*)	% group	(*n*)	% group	(*n*)	% group	(*n*)	χ^2	*p*	*Φ*
VA mental health services	Yes	15.9	(10)	28.6	(2)	46.2	(6)	21.7	(18)	6.03	0.05*	0.27
	No	84.1	(53)	71.4	(5)	53.8	(7)	78.3	(65)			
ER for mental health services	Yes	3.2	(2)	14.3	(1)	23.1	(3)	7.2	(6)	7.17	0.13	0.29
	No	95.2	(60)	85.7	(6)	76.9	(10)	91.6	(76)			
Non-VA subst. abuse services	Yes	11.1	(7)	0.0	(0)	15.4	(2)	10.8	(9)	1.46	0.83	0.13
	No	87.3	(55)	100.0	(7)	84.6	(11)	88.0	(73)			
Non-VA other mental health services	Yes	39.7	(25)	28.6	(2)	61.5	(8)	42.2	(35)	2.69	0.26	0.18
	No	60.3	(38)	71.4	(5)	38.5	(5)	57.8	(48)			
Any of the above	Yes	42.9	(27)	28.6	(2)	69.2	(9)	45.8	(38)	3.93	0.14	0.22
	No	57.1	(36)	71.4	(5)	30.8	(4)	54.2	(45)			

N = 83, since one of the participants was not incorporated in this analysis since the mental health treatment he received was from before his military service.

* Statistically significant where $p < 0.05$

** For use of hospital ER for mental health services and for any substance abuse treatment, one veteran in the single-peak group provided no responses, making for an *n* of 62 in the single-peak group and an *N* of 82 in the total sample for that variable alone.

Table 30

Chi Square analyses of 3-category course subtype by fateful and non-fateful life events during or after military service in Vietnam.

		PTSD Course Subtype											
		Single-peak (*n* = 64)		**Flat** (*n* = 7)		**Multi-Peak** (*n*=13)		**Total Sample*** (*N* = 84)		**Tests of significance**		
Post-Military Life Events		% group	(*n*)	% group	(*n*)	% group	(*n*)	% group	(*n*)	χ^2	*p*	*Φ*
Fateful Illness	Yes	12.5	(8)	0.0	(0)	15.4	(2)	11.9	(10)	1.12	0.57	0.12
	No	87.5	(56)	100.0	(7)	84.6	(11)	88.1	(74)			
Fateful event: Non-Illness	Yes	68.8	(44)	71.4	(5)	38.5	(5)	67.9	(57)	0.30	0.86	0.06
	No	31.3	(20)	28.6	(2)	61.5	(8)	32.1	(27)			
Any marital divorce or separation	Yes	43.8	(28)	57.1	(4)	61.5	(8)	47.6	(40)	1.65	0.44	0.14
	No	56.3	(36)	42.9	(3)	38.5	(5)	52.4	(44)			
Un-employed 6 months or more	Yes	61.9	(39)	57.1	(4)	83.3	(10)	64.6	(53)	2.21	0.33	0.16
	No	38.1	(24)	42.9	(3)	16.7	(2)	35.4	(29)			

** For unemployed for 6 months or more, two veterans were missing data, making for an *n* of 63 in the single-peak group, an *n* of 12 in the multi-peak group, and an *n* of 82 in the total sample for that variable alone.

Appendix

Directions for Raters:

Thank you for volunteering. This study examines the course of PTSD in veterans of the Vietnam War. Over a decade after their wartime service, veterans with PTSD were asked to describe how their symptoms had changed over time since the traumatic event they experienced in combat. As each veteran described the ups and downs in the severity of his symptoms over the past decade, a clinician sketched the disorder's course as a graph. In a preliminary review of the graphs, 3 major categories of graphs have been identified. The justification for these categories is provided in the codes below. The goal of this part of the study is to assess whether you and other raters can use these codes to replicate the same categories identified in the preliminary review.

Directions:

- Read the codes for the three kinds of graphs: Flat Graphs, Single-peak Graphs, and Multi-Peaked Graphs.
- Use the codes to classify each of the 84 graphs into one of the three categories.
- **Important:** Your classification of the graphs should be based on the part of the slope between "EVENT" and "NOW." **IGNORE ANY PARTS OF THE GRAPH DRAWN PRIOR TO THE "EVENT" MARK.**
- **Remember:** These graphs are not drawn to scale. They are not drawn with great precision. Rather, they provide an overall impression of how the disorder felt to the veteran. As you decide into which category to classify each graph, you should ask yourself "What is the overall impression this veteran was demonstrating by drawing this graph?"

CODES

Flat Graphs: These are graphs with slopes that are completely level or almost completely level from the "event" to the point "now" on the graph. *Flat: The graph's slope neither increases nor decreases over the course. The overall impression is that there has been no change in symptoms over time.*

Single Peak Graphs: These are graphs with exactly one peak between the "event" and the point "now" on the graph. *Peak: the high point or set of high points after a discrete period of increasing (i.e. positive), or preceding a discrete period of decreasing (i.e. negative) slope.* The overall impression is that symptoms exacerbated once over the course. They may have remained exacerbated (plateau), improved (downwards slope), or had some combination of these, but there is NO re-exacerbation after the initial peak in symptoms.

Multi Peak Graphs: These are graphs with more than one peak between the "event" and the point "now" on the graph. The overall impression is that there was an exacerbation of symptoms that improved (downward slope), and then worsened again (increased slope leading to at least one other peak).

Thanks for your time!

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