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Trauma, PTSD, and Self-Reported Physical Health Prior to and Following Counselling: A Longitudinal Study

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March 2006

A thesis submitted for the degree of Doctor of Psychology (Clinical) of the Australian National University
I hereby certify that the work embodied in this thesis is the result of original research and contains acknowledgement of all non-original work.

[Signature]

Jo Houston
ACKNOWLEDGEMENTS

I would like to thank the following individuals, without whom this work would not have been possible:

- Professor Don Byrne. Many thanks for your ongoing guidance, support, and encouragement over the lengthy gestation of this work.
- My partner, Stuart, for his support during the period of this research, and dedication in meticulously proof-reading various drafts (we'll have to agree to disagree over the use of commas, though, I'm afraid)
- My young daughter Sarah, born during a critical period of this research: thank you for sharing Mummy with the thesis, darling
- The staff at the Victims' Services Scheme and Victims of Crime Assistance League for their invaluable assistance in distributing questionnaires or facilitating their distribution
- Last, but not least: many, many thanks to the individuals who took the time to participate in this study, in the process sharing some harrowing experiences via the questionnaires
ABSTRACT

Posttraumatic stress disorder (PTSD) is an often chronic condition affecting a substantial minority of individuals exposed to traumatic events, particularly when multiple or complex trauma histories are present (e.g., repeated violence, child sexual abuse). Research conducted primarily with Vietnam veterans and sexual assault victims has supported an association between a diagnosis of PTSD and increased self-reported—and in some cases medically diagnosed—health problems.

This dissertation reports two pieces of research—together forming one longitudinal study—that examine the self-reported physical health of victims of crime in the Canberra area prior to and following receipt of counseling in the community. Participants were predominantly female (n = 88, or 88.9% of the sample), treatment-seeking, and in many cases victims of domestic violence and/or child sexual abuse. The mean age was 35.9 years (sd = 11.14). 99 participants completed three questionnaires in each study: the Posttraumatic Diagnostic Scale, the BDI-II, and a health questionnaire specifically designed for this research (a slightly different version of this latter questionnaire was used in the second study).

The initial study found a PTSD prevalence rate of 44.3%, with over 30% of participants reporting that they had experienced four or more traumatic events. Hierarchical multiple regression analyses confirmed that PTSD was a significant predictor of both specific self-reported health symptoms and a poorer perception of health following exposure to the trauma(s), over and above the contribution of depression and other variables associated with poor health. However, contrary to expectations PTSD was not a significant predictor of self-reported medically diagnosed conditions (possibly attributable to both the relative youth of this sample and recency of the traumatic event). Similarly, the PTSD avoidance/numbing cluster was the only significant predictor of self-reported health problems, rather than the expected hyperarousal symptom cluster.
Participants were followed up five to seven months after the initial study, with 59 returning the second set of questionnaires. The aim of the second study was to evaluate whether the provision of counselling between the two studies led to a reduction in both PTSD symptoms and self-reported health problems. The results were complicated, with initial repeated measures analyses of covariance (ANCOVA) showing a reduction in PTSD symptoms over time, but little change in health symptom reporting between the two studies in either the counselling or no counselling group, raising questions about the effectiveness of the treatment being provided to this sample. Further analyses of variance were undertaken depending on whether participants had received no or up to seven sessions of counselling, or more than seven sessions. These subsequent analyses revealed a significant reduction in PTSD symptoms in the shorter-term (or no) counselling group, as well as a non-significant trend towards reduced health symptom reporting for those who met criteria for PTSD. Contrary to expectations, provision of more counselling not only did not improve PTSD or health symptoms, but actually appeared to result in greater endorsement of self-reported health symptoms. Reasons for these results canvassed in this dissertation relate to the effectiveness of the counselling being provided in the community for this client group, in addition to certain complex characteristics of this sample (including exposure to repeated trauma, and the nature of the traumatic events experienced). The need to stipulate the nature of therapy provided to victims of crime is highlighted as one area for further research in the PTSD – health relationship.
# TABLE OF CONTENTS

ACKNOWLEDGEMENTS ......................................................................................................................... iii
ABSTRACT .............................................................................................................................................. iv

Chapter One: Trauma and Posttraumatic Stress Disorder - Background ............. 1

1.1 Introduction ................................................................................................................................. 1
1.2 Range of responses to trauma ..................................................................................................... 1
1.3 PTSD: historical background ...................................................................................................... 3
1.4 Current definition of PTSD ......................................................................................................... 5
1.5 Other traumatic reactions .......................................................................................................... 7
1.6 Prevalence of PTSD .................................................................................................................. 8
  1.6.1 General community studies of PTSD prevalence ................................................................. 9
  1.6.2 PTSD prevalence in at-risk populations ............................................................................. 12
  1.6.3 Prevalence: summary .......................................................................................................... 15
1.7 Chronicity ..................................................................................................................................... 16
1.8 Risk factors .................................................................................................................................. 18
  1.8.1 Gender .................................................................................................................................. 18
  1.8.2 Age ....................................................................................................................................... 19
  1.8.3 Ethnicity .............................................................................................................................. 20
  1.8.4 Pre-trauma experiences ....................................................................................................... 20
      1.8.4.1 Personality variables ................................................................................................. 20
      1.8.4.2 Exposure to previous traumatic events ...................................................................... 21
  1.8.5 Mental health problems and genetic variables .................................................................. 22
  1.8.6 Peri- and post-traumatic environment ............................................................................... 22
1.9 Comorbidity with other disorders ......................................................................................... 23
1.10 ‘Complex’ PTSD .................................................................................................................... 25
1.11 Psychobiology of PTSD .......................................................................................................... 27
  1.11.1 Normal stress response .................................................................................................... 29
  1.11.2 Harmful effects of prolonged stress .................................................................................. 30
  1.11.3 PTSD: a different category of stress response ................................................................. 30
1.12 Summary .................................................................................................................................... 33
Chapter Two: Trauma, PTSD, and Physical Health

2.1 Introduction

2.2 War trauma and physical health

2.3 Health outcomes after war trauma: PTSD as a critical variable

2.3.1 Vietnam veterans

2.3.2 Somatisation or ‘real’ medical conditions?

2.3.3 Veterans of other wars and conflicts

2.3.4 Summary: Trauma, PTSD, and health outcomes in veterans

2.4 Health outcomes following non-combat trauma

2.4.1 Health following physical or sexual assault

2.4.1.1 Health outcomes for assault victims with PTSD

2.4.1.2 Summary: PTSD and health outcomes following physical or sexual assault

2.4.2 Other traumatic events and health outcomes

2.4.2.1 Natural disasters and other violent events

2.4.2.2 Motor vehicle accidents

2.4.2.3 Torture

2.4.3 Community samples: non-specific trauma

2.5 Summary: PTSD, trauma, and health outcomes in various populations

2.6 Hypotheses

Chapter Three: Methodology

3.1 Introduction

3.2 Participants

3.3 Measures

3.3.1 Background

3.3.2 Description of Measures

3.3.2.1 Trauma and PTSD Measures

3.3.2.2 Beck Depression Inventory, 2nd edition

3.3.2.3 Health Questionnaire Parts 1 and 2

3.4 Procedure

3.4.1 Study One
3.4.1.1 Victims' Services Scheme (initial clients) ............................................ 71
3.4.1.2 VOCAL ............................................................................................. 74
3.4.1.3 VSS – past clients ............................................................................. 75
3.4.1.4 University students at ANU ............................................................... 75
3.4.2 Study Two ............................................................................................. 75

Chapter Four: A Study Examining the Self-Reported Health of Victims of
Crime ............................................................................................................. 77

4.1 Results ............................................................................................................. 77
4.1.1 Overview ................................................................................................. 77
4.1.2 Outline of Statistical Analysis .................................................................. 78
4.1.3 Preliminary Data Screening ....................................................................... 78
4.1.4 Descriptive statistics ................................................................................ 79
4.1.4.1 Comorbidity and prevalence issues ................................................... 80
4.1.5 Multivariate analyses - multiple regression ............................................. 84
4.1.5.1 Multivariate data screening: Health Score regressed on PTSD and
other predictors ............................................................................................. 84
4.1.5.2 Correlation matrix interpretation and health score regressed on PTSD
and other predictors ...................................................................................... 86
4.1.5.3 Multivariate data screening: Health Score regressed on PTSD
symptom clusters and antidepressant medication ......................................... 88
4.1.5.4 Correlation matrix interpretation: health score regressed on PTSD
symptom clusters .......................................................................................... 89
4.1.6 Multivariate analyses: multiple regression - difference score
regressed on five independent variables ......................................................... 90
4.1.6.1 Correlation matrix interpretation and multiple regression: difference
score regressed on PTSD and other variables ............................................. 92
4.1.7 Multivariate Analyses: logistic regression – medical problems
endorsed ............................................................................................................. 93
4.2 Discussion ........................................................................................................ 94
4.2.1 Sample characteristics ............................................................................ 95
4.2.2 Multiple Regression Analyses with (a) PTSD and (b) PTSD symptom clusters as predictors of Health Score ................................................. 97
4.2.3 Logistic regression analysis: PTSD as a predictor of self-reported medically-diagnosed conditions .......................................................... 103
4.2.4 Limitations ........................................................................................... 104
4.2.5 Summary .............................................................................................. 104
4.2.6 Conclusion ............................................................................................ 105

Chapter Five: An Examination of Self-Reported Health and PTSD Severity in Victims of Crime Following Counselling ........................................ 106
5.1 Results ........................................................................................................... 106
5.1.1 Objective ............................................................................................... 106
5.1.2 Overview ............................................................................................... 106
5.1.3 Outline of statistical analysis .................................................................. 107
5.1.4 Preliminary Data Screening .................................................................. 108
5.1.5 Descriptive Statistics ............................................................................ 110
5.1.6 Comparison of responders with non-responders .............................. 112
5.1.7 Bivariate and Multivariate Analyses ............................................... 112
  5.1.7.1 Bivariate relationships ....................................................................... 112
  5.1.7.2 Repeated Measures Analysis of Covariance: Health Score 2 ........ 114
  5.1.7.3 Repeated Measures ANCOVA: PTSD severity score ...................... 117
  5.1.7.4 Repeated measures ANOVAs examining health scores by PTSD diagnosis and number of counselling sessions .......................... 119
  5.1.7.5 Paired Samples t-tests for PTSD severity score by counselling duration ........................................................... 122
5.2 Discussion ..................................................................................................... 123
  5.2.1 Initial hypotheses and summary of results ......................................... 123
  5.2.2 Sample characteristics ......................................................................... 124
  5.2.3 Interpretation of ANCOVA/ANOVA results ....................................... 126
  5.2.4 Summary and conclusions .................................................................. 131

Chapter Six: General Discussion ...................................................................... 133
References ........................................................................................................ 145
Appendices ................................................................................................................................................. 160
Copies of the following forms used in these studies are attached: ..................................................... 160
1. Health Questionnaire Part 1 ................................................................................................................. 161
2. Posttraumatic Diagnostic Scale (PDS) .............................................................................................. 165
3. Beck Depression Inventory, 2nd Edition (BDI-II) ............................................................................. 168
4. Health Questionnaire Part 2 ................................................................................................................. 170
### LIST OF TABLES AND FIGURES

Table 1.1: DSM-IV Diagnostic criteria for 309.81 Post-traumatic stress disorder .......... 5
Table 1.2: Selected Studies of PTSD Prevalence in the General (US) Community .......... 9
Table 1.3: Biological Abnormalities in PTSD ............................................................... 28
Table 4.1: Characteristics of PTSD positive and PTSD negative groups ...................... 80
Table 4.2: Mean health scores for each of the PTSD groups ......................................... 81
Table 4.3: PTSD diagnosis and different traumatic events ......................................... 82
Table 4.4: Self-reported health problems endorsed by participants ......................... 84
Table 4.5: Pearson r correlations for PTSD, health score and associated variables ..... 85
Table 4.6: Hierarchical multiple regression for prediction of frequency of self-reported physical health symptoms, PTSD entered in final step ......................... 87
Table 4.7: Pearson r correlations for health score, PTSD symptom clusters, and antidepressant medication use ............................................................... 88
Table 4.8: Hierarchical multiple regression with PTSD symptom clusters predicting health symptoms ............................................................... 89
Table 4.9: Pearson r correlations for difference score (change in health perception), PTSD, and associated variables ............................................................... 91
Table 4.10: Hierarchical multiple regression for prediction of change in health perception (difference score), PTSD entered in final step ......................... 93
Table 4.11: Logistic regression for medical problems endorsed, with age, depression, and finally PTSD entered as independent variables ........................................ 94
Table 5.1: Summary of skewness and kurtosis for PTSD severity score, BDI score, age, and health score ............................................................... 108
Table 5.2: Characteristics of PTSD positive and PTSD negative groups at the time of this study ............................................................... 110
Table 5.3: Self-reported health problems endorsed by participants in studies 1 and 2 111
Table 5.4: Independent samples t-tests comparing responders to this study (n = 59) with non-responders (n = 40) on initial mean PTSD severity score, BDI score, health score, and age ............................................................... 112
Table 5.5: Pearson \( r \) Correlations between the main outcome measures and independent variables of interest.................................................................113

Figure 5.1: Graphic representation of mean health scores for the PTSD and non-PTSD groups, by counselling status...............................................................115

Table 5.6: Results of repeated measures (Split-Plot) ANCOVA on health score, with PTSD, time, and counselling status as factors, and depression (study 2) as covariate ..........................................................116

Table 5.7: Simple effects analyses (t-tests) on health score 2 by groups......................116

Figure 5.2: PTSD severity scores for each study by counselling status.......................118

Table 5.8: Results of repeated measures (Split-Plot) ANCOVA on PTSD severity score, with counselling status as a factor, and depression (time2) as covariate ....119

Table 5.9: Mean health scores by PTSD diagnosis and counselling duration..........120

Table 5.10: Results of repeated measures ANOVA on health score for participants who received 0 – 7 sessions of counselling, with PTSD as a factor ..........121

Table 5.11: Results of repeated measures ANOVA on health score for participants who received 8 or more sessions of counselling, with PTSD as a factor ..........121

Table 5.12: Paired samples t-tests examining mean PTSD severity scores for the 0 – 7 and 8+ sessions of counselling groups ..................................................122
Chapter One: Trauma and Posttraumatic Stress Disorder -
Background

1.1 Introduction

The impact of psychological trauma has attracted increasing attention in the past two
decades. Such interest can be largely attributed to the aftermath of the Vietnam war and
concurrent heightened awareness or acknowledgement of the extent and impact of
sexual abuse and domestic violence in the community. This introductory chapter
examines some of the typical emotional responses to trauma, particularly where this
leads to psychopathological consequences such as post-traumatic stress disorder
(PTSD). The background to PTSD, its diagnostic criteria, epidemiological factors such
as the condition's prevalence and chronicity, risk factors for its development, other co-
morbid psychiatric conditions, and an outline of the underlying neurobiology of stress
and PTSD are all discussed. A discussion of evidence associating PTSD with physical
health problems follows in Chapter Two, after which two studies (comprising one
longitudinal health study) are described in detail. This research focuses on the issue
central to this dissertation: is PTSD associated with more medical problems, self-
reported or otherwise?

1.2 Range of responses to trauma

At some point in their lifetime, the majority of individuals will experience a traumatic
event that may be life-threatening (to the person or others close to them), or threatening
to the individual's sense of self in some indelible way. A number of studies demonstrate
that over 50% of the general population will report experiencing a traumatic incident
such as physical or sexual assault, armed robbery, sexual abuse, domestic violence,
natural disaster, a serious accident, torture, or armed combat (eg Kessler, Sonnega,
Bromet, Hughes and Nelson, 1995). Given the prevalence of traumatic events in the
community, it is not surprising that interest has focussed on the potential sequelae to
such trauma, particularly in terms of the psychological impact on the individual and any resultant impairment in functioning.

While some individuals report little or no adverse reactions in the aftermath of trauma, these appear to be in the minority. Traumatic, stressful events often occur suddenly, particularly in the case of accidents, disasters and criminal events such as assault, and with little or no warning. However, some traumatic incidents are not so unexpected, as may be the case in situations involving domestic violence, torture, combat, or sexual abuse. The victim may be left with an impression of impending death. It is common for traumatised survivors of assaults, disasters, and so forth to make statements such as “I thought I was going to die” or “He was going to kill me”. In the first few weeks following a traumatic event (regardless of whether the individual experienced it personally or witnessed another person being subjected to such trauma) many traumatised people find themselves feeling anxious, being hypervigilant, ruminating on the traumatic event, and having disturbed sleep and/or nightmares. These are not uncommon reactions; in fact some studies have shown that the majority of trauma victims will initially meet the criteria laid down in the American Psychiatric Association’s (1994) Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV) for post-traumatic stress disorder (PTSD) or acute stress disorder (ASD). Rothbaum, Foa, Riggs, Murdock and Walsh (1992) reported that 94% of female rape victims met DSM-IV (1994) criteria for PTSD one week after their rape; however this dropped to 47% three months after the event. The majority of trauma victims will continue to recover and no longer meet diagnostic criteria for either PTSD or ASD. However, only relatively recently has PTSD been recognised as a distinct psychiatric disorder. An overview of the historical context leading to its inclusion in the DSM follows.
1.3 PTSD: historical background

The term ‘post-traumatic stress disorder’ was first introduced in DSM-III in 1980; however, reactions to traumatic stress had been periodically discussed and described for many years, if not centuries. Terms such as “battle fatigue”, “combat neurosis”, “traumatic neurosis” and others were used to describe what are now recognised as post-traumatic reactions in World War I and II soldiers. A British psychologist, Charles Myers, examined some of the casualties of the First World War who exhibited symptoms such as mutism, hysteria, weeping, amnesia, and emotional numbing and attributed these to the concussive effects of exploding shells. He called the condition “shell shock” (Myers, 1940). Once it became clear that the symptoms were psychological in nature, early medical and psychiatric interest focussed on the “constitution” and “moral character” of the soldier, viewing severe emotional reactions to combat as cowardice, or “moral invalidity”. One prominent psychiatrist treating World War I soldiers, Lewis Yealland, advocated treating traumatised soldiers with electric shocks, threats of court martial, and humiliating comments (Herman, 1997). However, more progressive psychiatrists recognised the emotional basis of this disorder and proposed treating the symptoms using psychoanalytical principles. One early pioneer of this approach, Kardiner (1941), referred to the trauma-related disorder that he observed as a “physioneurosis” and identified five clinical features which are recognisable in the current DSM-IV (1994) definition of PTSD (see Table 1.1 in Section 1.4):

- Irritability
- Startle pattern
- Fixation on the trauma
- An atypical dream life
- A propensity to experience an explosive aggressive reaction

Early 20th century psychologists such as Pierre Janet had also remarked on the symptoms observed in traumatised individuals, particularly as they impacted on memory and the tendency in some people to dissociate as a means of escaping the intense distress
evoked by the trauma (van der Kolk and van der Hart, 1991). Controversially, Sigmund Freud had also initially noted the extent of sexual abuse reported by his “hysterical” female patients during psychoanalysis, but later repudiated his hypothesis that “premature sexual experience” lay behind every case of hysteria. Instead, he claimed that such reports were “fantasies” created by his female patients. Herman (1997) noted that Freud could not come to terms with what appeared to be endemic sexual abuse in so-called respectable society.

Despite much of this early interest in trauma and its impact, only relatively recently has PTSD been categorised and recognised as a distinct psychiatric condition resulting from an overwhelming traumatic stressor. This may be attributable partly to cultural and political factors, as well as the impact of both the Vietnam war and the women’s movement in the 1960s and 1970s. Returned servicemen and women from the Vietnam war were vocal in speaking out about their symptoms and experiences, partly fostered by negative community attitudes towards the war and in some cases the veterans themselves. Shatan (1973) noted individual cases where veterans reported suffering nightmares, flashbacks, somatic problems, insomnia, depression, and ‘survivor guilt’, while also commenting (somewhat sardonically) on the US Veteran Administration’s claim that “the Vietnam war has resulted in fewer psychiatric casualties than has any other in which the US has fought” (p644).

Women in the 1970s also began to raise issues that had previously been relegated to the “private” or domestic front, including child sexual abuse, physical abuse, incest, rape, and domestic violence. Syndromes that appeared very similar to those reported in returning Vietnam veterans were described in some women, with names such as “rape trauma syndrome” and “battered women’s syndrome” (Resick, 2001). Attention focussed once more on trauma-related symptoms, leading to the genesis of post-traumatic stress disorder in DSM-III (1980).
1.4 Current definition of PTSD

The original DSM-III (1980) definition of what constituted a stressor specified that the traumatic event must be “outside the normal range of events”, which does not necessarily reflect the reality that the majority of people will experience some form of traumatic incident in their life. DSM-IV (1994) was revised to recognise this fact. However, DSM-III (1980) (and later DSM-III-R, 1987) essentially described the form of PTSD that is now reflected in the current version of the DSM, which lists symptoms in clusters as outlined in Table 1.1 below.

| Table 1.1: DSM-IV Diagnostic criteria for 309.81 Post-traumatic stress disorder |

**A.** The person has been exposed to a traumatic event in which both of the following were present:

(1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.

(2) the person’s response involved intense fear, helplessness, or horror. Note: in children, this may be expressed instead by disorganised or agitated behaviour.

**B.** The traumatic event is persistently re-experienced in one (or more) of the following ways:

(1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. Note: in young children, repetitive play may occur in which themes or aspects of the trauma are expressed.

(2) recurrent distressing dreams of the event. Note: in children, there may be frightening dreams without recognizable content.

(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). Note: in young children, trauma-specific re-enactment may occur.
(4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

(5) physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

(1) efforts to avoid thoughts, feelings, or conversations associated with the trauma
(2) efforts to avoid activities, places, or people that arouse recollections of the trauma
(3) inability to recall an important aspect of the trauma
(4) markedly diminished interest or participation in significant activities
(5) feeling of detachment or estrangement from others
(6) restricted range of affect (eg unable to have loving feelings)
(7) sense of a foreshortened future (eg does not expect to have a career, marriage, children, or a normal life span)

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

(1) difficulty falling or staying asleep
(2) irritability or outbursts of anger
(3) difficulty concentrating
(4) hypervigilance
(5) exaggerated startle response

E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
Specify if:
  Acute: if duration of symptoms is less than 3 months
  Chronic: if duration of symptoms is 3 months or more
Specify if:
  With delayed onset: if onset of symptoms is at least 6 months after the stressor

The emphasis in Criterion A on the threat to self or others, accompanied by fear, helplessness, or horror is important, as it deals with an individual’s perception of how the traumatic event has impacted upon them. There is sometimes a misconception in the community that a person must be badly injured or directly exposed to trauma in order for PTSD to develop. This is not necessarily the case; the critical variable is whether the individual concerned perceived themselves (or someone close to them, physically or emotionally) to be at risk of death, serious injury, or loss of integrity. For instance, one study of earthquake survivors in Turkey found that the strongest predictor of PTSD symptoms was experiencing fear during the earthquake (Basoglu, Kilic, Salcioglu and Livanou, 2004). The focus on “threat to integrity” is important when one considers childhood sexual abuse in particular; the child concerned may not necessarily have feared that they would die or suffer serious injury, however their sense of self has been extensively damaged (see van der Kolk, 1998, for a discussion on this topic).

1.5 Other traumatic reactions

A symptom cluster similar to PTSD (but with a greater focus on dissociation) that occurs only in the first four weeks after the traumatic incident was somewhat controversially included as a new psychiatric condition in DSM-IV (1994). Acute stress disorder (ASD) was introduced to the DSM after some research identified a subset of people who dissociate more during or immediately after a traumatic incident. These individuals are more likely to develop chronic PTSD (Spiegel, Koopman, Cardeña, and Classen, 1996). In addition to experiencing one or more of the symptoms from each of the PTSD symptom clusters, people with ASD experience reduced emotional responsiveness, a
lack of pleasure in previously enjoyable activities, and concentration difficulties. They may also report dissociative amnesia, depersonalisation, and derealisation. By definition, ASD can only be diagnosed for the first four weeks after a traumatic stressor: after this, the individual either meets the criteria for PTSD, another DSM-IV (1994) disorder, or recovers to a level where they no longer meet criteria for any psychiatric condition. The inclusion of ASD as a separate diagnostic entity in DSM-IV (1994) attracted some criticism as it was based on fairly limited research (Resick, 2001).

Victims of traumatic events who do not meet the criteria for either ASD or PTSD may sometimes be diagnosed with other DSM-IV (1994) disorders, including an adjustment disorder, panic disorder, a specific phobia (not uncommon after motor vehicle accidents, in particular), generalized anxiety disorder, major depressive disorder, or substance abuse. One study that examined a community sample of patients suffering from panic disorder found that 24.2% of females and 5% of males with this condition reported a past history of sexual abuse or assault, even after excluding comorbid PTSD symptoms (Leskin and Sheikh, 2002). More commonly, however, other psychiatric disorders appear comorbidly with PTSD (see Section 1.9 for a discussion on this topic).

1.6 Prevalence of PTSD

The prevalence of PTSD has been studied in a number of populations, including the general community (national samples in particular countries), particular geographic areas, and samples drawn from specific populations where trauma exposure is highly likely (such as Vietnam veterans, torture and trauma survivors, people exposed to natural disasters or criminal events, and victims of crime). Prevalence of this often chronic disorder may be measured in terms of lifetime prevalence (the percentage of individuals who meet the diagnostic criteria for PTSD at some time in their lifetime) and current prevalence (the percentage who meet the diagnostic criteria for PTSD at the time of assessment). Typically, estimates of the prevalence of disorders such as PTSD are gathered by way of surveys (questionnaires or phone-based, more rarely interview-based).
Studies conducted since 1980, when PTSD was formally recognised as a psychiatric disorder in the DSM, have tended to find a high rate of exposure to trauma in the general community. However, it should be noted that the majority of research has been conducted in the USA.

1.6.1 General community studies of PTSD prevalence

Results from a number of large-scale community studies investigating the prevalence of trauma and PTSD in adults are summarised in Table 1.2. These studies have been selected on the basis of their large sample size, frequency of citation in the PTSD literature, and rigorous methodology.

Table 1.2: Selected Studies of PTSD Prevalence in the General (US) Community

<table>
<thead>
<tr>
<th>Authors</th>
<th>Population</th>
<th>Exposure to Trauma (%)</th>
<th>PTSD prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Helzer, Robins &amp; McEvoy (1987)</td>
<td>General adult population, US metro area (St Louis)</td>
<td>-</td>
<td>1.0%</td>
</tr>
<tr>
<td>Breslau, Davis, Andreski &amp; Peterson (1991)</td>
<td>Young adults from a health maintenance organisation in Detroit, USA</td>
<td>39%</td>
<td>9.2%</td>
</tr>
<tr>
<td>Norris (1992)</td>
<td>Adults in four southern US cities</td>
<td>-</td>
<td>5.1% - 6.2%</td>
</tr>
<tr>
<td>Resnick, Kilpatrick, Dansky et al (1993)</td>
<td>General population of adult women: USA</td>
<td>-</td>
<td>12.3% 4.6%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60.7% (men)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>60.7% (men)</td>
<td></td>
</tr>
<tr>
<td>Breslau, Kessler, Chiloact et al (1998)</td>
<td>US population of persons aged 18 – 45 (Detroit)</td>
<td>89.6%*</td>
<td>9.2%</td>
</tr>
</tbody>
</table>

* includes sudden unexpected death of a close friend or relative as a traumatic event
As shown in Table 1.2, the lifetime prevalence of PTSD in these American studies has been variously estimated at 1.0% of the general adult population through to 12.3%. This is obviously a wide variation, although the extent of the variation is diminished if one excludes the Helzer et al (1987) study. The majority of studies conducted since Helzer et al (1987) published their research generally report a consistent lifetime prevalence rate for PTSD of around 8% - 10%. However, it would appear that Helzer et al (1987) underestimated the prevalence of PTSD in the community as they used a measure for assessing PTSD based on the more restrictive DSM-III (1980) criteria. Although men may report exposure to traumatic incidents more than women (see Kessler et al, 1995), it appears that women are more likely to meet the criteria for PTSD (Kessler et al, 1995; Breslau et al, 1991) due in all likelihood to the nature of the trauma they experienced (eg sexual assault, although see Section 1.8.1 for further discussion of gender as a potential risk factor for PTSD).

More recently, studies of PTSD prevalence have also been undertaken in European, Australian, non-western, and developing countries, providing important data about the universality of this disorder. Despite the difficulties associated with using PTSD measures often translated from English and developed in western countries, useful data has been obtained from studies conducted in Afghanistan, Mexico, South Africa, and Bosnia. Scholte, Olff, Ventevogel, et al (2004) interviewed 1011 adults in eastern Afghanistan and found that 20.4% met DSM-IV (1994) criteria for PTSD, with over 50% of their total sample experiencing more than eight traumatic events in the past 10 years (including bombardments, fleeing war, being close to death, and experiencing interrogation or harassment). A study of 2,509 adults in four Mexican cities revealed a PTSD rate of 11.2% (Norris, Murphy, Baker, Perilla, Rodriguez, and Rodriguez, 2003), while a high rate of exposure to trauma (94%) and development of PTSD symptoms was also found in a sample of 201 adults who were patients of a South African township health clinic (Carey, Stein, Zungu-Dirwayi, and Seedat, 2003). Finally, Rosner, Powell, and Butollo (2003) undertook research into the levels of PTSD in a sample of civilians (n = 311) who had been residing in Sarajevo during the siege of the city by Bosnian Serb
forces in the mid-1990s. 18.6% of the non treatment-seeking group met DSM-IV (1994) criteria for PTSD, while 32.7% of those in medical treatment and 38.6% of those receiving psychological treatment also met current criteria for this disorder.

Australian data on the prevalence of PTSD in the general community is also available. The National Survey of Mental Health and Wellbeing was conducted throughout Australia in 1997 (Australian Bureau of Statistics, 1998). The sample consisted of 10,641 adults, with 57.4% reporting lifetime experience of at least one of the specified traumatic events. Men were significantly more likely than women to report a traumatic incident (64.5% vs 49.5%). The most common types of trauma reported were witnessing someone being badly injured or killed (37.8% of men and 16.1% of women), being involved in a life-threatening accident (28.3% of men and 13.6% of women), and being involved in a natural disaster (19.9% of men and 12.7% of women). Rosenman (2002) further interrogated this data to estimate the prevalence of PTSD according to both DSM-IV (1994) and ICD-10 criteria in the twelve months preceding the survey. The twelve-month prevalence of PTSD in the population sample as a whole was 1.5% (DSM-IV criteria) and 3.6% (ICD-10 criteria). Analysis of the traumatised sample (n = 6104) revealed a PTSD prevalence of 2.8% (DSM-IV) and 6.8% (ICD-10). Rosenman (2002) attributed the significant difference in diagnosis between DSM-IV and ICD-10 to classification and threshold differences. He noted that women who had experienced trauma were significantly more likely than men to develop PTSD, especially if one uses the ICD-10 diagnosis. Another interrogation of this data by Creamer, Burgess and McFarlane (2001), sampling 10,641 participants, found a 12 month prevalence rate for PTSD of 1.33%, using a modified version of the Composite International Diagnostic Interview. The prevalence rates for PTSD are low when compared with overseas studies, a fact noted by Creamer et al (2001). Even when 12 month prevalence data from the Kessler et al (1994) study was obtained, the rate of PTSD in that American study was still three times the rate of PTSD in the Australian study. Creamer et al (2001) considered a number of explanations, including higher rates of pre-existing psychiatric conditions in the US sample, differences in measurement instruments, and a cross-cultural difference in resilience to stress. Contrary to Rosenman’s findings
(Rosenman, 2002), Creamer et al (2001) did not find any evidence of significantly higher PTSD prevalence among women: PTSD prevalence was 1.2% for males and 1.4% for females, using DSM-IV criteria.

1.6.2 PTSD prevalence in at-risk populations

The prevalence of PTSD and other psychiatric disorders has also been studied extensively in specific at-risk populations – in other words, those individuals who have experienced some sort of traumatic event. The most commonly studied groups are war veterans, women who have been sexually assaulted, and victims of natural disasters or terrorist attacks. Vietnam veterans have been one of the most widely studied populations. A comprehensive examination of symptoms present in this latter group comes from the National Vietnam Veterans Readjustment Study (NVVRS, Kulka, Schlenger, Fairbank, et al, 1990). This study of 1632 Vietnam veterans, other veterans of the same era (n = 716) and nonveterans (n = 668) found that 15.2% of men and 8.5% of women who served in Vietnam met the DSM-III (1980) criteria for PTSD in the 6 months prior to their interview. This compared with a current prevalence of PTSD among other veterans of 2.5% for men and 1.1% for women, with nonveterans having a PTSD prevalence of 1.2% and 0.3% for men and women, respectively. Lifetime prevalence of PTSD was reported to be 30.9% for men and 26.9% for women. A diagnosis of PTSD was made based on a multimodal assessment consisting of self-report scales and structured clinical interviews. Other studies of Vietnam veterans conducted in the USA and New Zealand (MacDonald, Chamberlain, and Long, 1997) have tended to find a lifetime prevalence of PTSD of 15% - 20%, and current prevalence of 11% - 18%.

The traumatic effects of other wars and military conflicts have also been studied to establish the proportion of the population adversely affected by such trauma. One study of 125 World War II and Korean War veterans found that 39% met the criteria for PTSD (Hyer, Summers, Boyd, Litaker, and Boudewyns, 1996). An Australian study of 1991 Gulf War veterans reported that 5.4% of their sample met DSM-IV (1994) criteria for
PTSD, assessed via the Composite International Diagnostic Interview (Ikin, Sim, Creamer et al, 2004). The rigour of an interview-based measure might explain the relatively low prevalence of PTSD in this population, compared with other studies of war veterans.

Civilian populations traumatised by war, particularly in South-East Asia, have also been studied in recent times. One such study reported that 70% of Indochinese refugees in a clinic setting met the criteria for PTSD, although the rates varied according to nationality (eg 54% of Vietnamese patients had PTSD, compared with over 90% of the Cambodians). Many of these individuals had been interred in refugee or concentration camps for a substantial number of years (Kinzie, Boehnlein, Leung, Moore, Riley, and Smith, 1990).

Other studies have examined the prevalence of PTSD in torture and trauma survivors, sexual assault victims, victims of crime other than sexual assault, natural disaster or terrorist attack survivors, and motor vehicle accident victims. There are some major differences in the prevalence of PTSD between these groups, with torture and trauma survivors amongst the worst affected. Van Velsen, Gorst-Unsworth and Turner (1996) assessed 60 asylum seekers in Britain from Turkey and other middle-eastern countries. 85% of these individuals reported physical torture, and 52% of the total group met DSM-III-R (1987) criteria for current PTSD. Similarly, the rates of PTSD resulting from sexual assault (particularly rape) are disturbingly high: Kessler et al (1995) reported that 65% of men and 45.9% of women who reported being raped (as their “most upsetting” trauma) met the criteria for PTSD at some point in their life, while Breslau et al (1991) found a much higher rate of PTSD (80%). Resnick et al (1993) reported that 32% of rape victims in their study met criteria for lifetime PTSD, with 12.4% meeting criteria for current PTSD. However, PTSD rates for physical assault were even higher in this latter study (38.5% lifetime prevalence and 17.8% current prevalence), which may suggest that both physical and sexual assault pose a greater threat to life, and are more personal (or targeted) in their nature. Norris (1992) also found that sexual and physical assault led to similar rates of PTSD, although the
prevalence in her study for these traumas was more conservative (13.6% and 13.3%, respectively). This sense of personal targeting and threat to life may also be felt in domestic violence situations, where abuse is ongoing: studies of domestic violence victims (primarily women seeking assistance through shelters, refuges, or clinics) also show a high rate of current and lifetime PTSD: Gleason (1993) reported a lifetime prevalence of 31% while Kemp, Rawlings, and Green (1991) found 84% of domestic violent victims assessed a few days after arrival at a refuge met the criteria for PTSD. A recent Israeli study reported that 51.6% of “battered women” met DSM-IV (1994) criteria for current PTSD (Sharhabani-Arzy, Amir, Kotler and Liran, 2003). Similarly, a study of 100 female victims of domestic violence residing in women’s shelters in Adelaide, South Australia, found that 45% met all diagnostic criteria for PTSD (Mertin and Mohr, 2000). The authors noted that those women who met criteria for PTSD reported experiencing higher levels of violence, including threats of death from their spouse or partner, as opposed to those women without PTSD.

Survivors of terrorist attacks (which by their nature are impersonal, often unexpected, and designed to cause maximum injury and devastation) also appear vulnerable to PTSD: 31% of a sample of 228 victims directly exposed to a wave of bombings in France during the mid-1990s were diagnosed with PTSD (Verger, Dab, Lamping, et al, 2004), while 20% of individuals living close to the World Trade Centre who were surveyed five to eight weeks after the destruction of the twin towers on September 11 2001 met criteria for PTSD (Galea, Ahern, Resnick, et al, 2002).

Other violent and/or traumatic crimes tend to result in lower rates of PTSD than torture, sexual and physical assault, or abuse. Norris (1992) reported that 6.0% of robbery victims met criteria for PTSD, while Resnick et al (1993) found that 9.7% of women in their sample who had experienced “any crime victimisation” met criteria for current PTSD. Kessler et al (1995) reported an interesting finding that only 1.9% of men were diagnosed with lifetime PTSD after being “threat[ened] with a weapon”, compared with 32.6% of women in relation to this trauma. This suggests a greater sense of
vulnerability and fear experienced by women when confronted with a potentially deadly weapon.

Mayou, Tyndel, and Bryant (1997) studied a sample of motor vehicle accident (MVA) victims (n = 111) five years after the accident in question, and found that 8% met the full criteria for PTSD. Similarly, Norris (1992) found a current PTSD rate of 11.5% in her sample of MVA victims. Natural disasters also appear less likely than sexual assault, terrorist attacks, torture, and possibly combat to lead to a diagnosis of PTSD: McFarlane and Papay (1992) found that 13% of firefighters met the criteria for PTSD 42 months after bushfires in Australia, while Lai, Chang, Connor, Lee, and Davidson (2003) reported that 10.3% of earthquake survivors in Taiwan met full diagnostic criteria for PTSD ten months after the disaster.

While the majority of studies investigating the prevalence of PTSD are limited in one respect by their retrospective nature (relying on the recall and possibly fallible memories of participants), the few prospective studies in this area have tended to support the rates of PTSD reported (see for instance Rothbaum, Foa, Riggs, Murdock, and Walsh, 1992).

1.6.3 Prevalence: summary

The majority of large community studies have established a lifetime prevalence of PTSD of between 5% and 10%, and a current prevalence of between 2% and 4% in their samples. The variation of prevalence estimates can be attributed primarily to differences in diagnostic measures and criterion thresholds between DSM-III (1980) and DSM-IV (1994).

The prevalence of PTSD in traumatised populations is, unsurprisingly, substantially higher than in the general community, with around 15% of Vietnam veterans, 30% of rape victims, 8% of motor vehicle accident survivors, and 10 – 15% of disaster survivors having met criteria for PTSD at some point in their lives.
While men report exposure to a greater number of traumatic events, women appear more likely than men to meet diagnostic criteria for PTSD, primarily (it would appear) due to the nature of the trauma most commonly experienced (sexual assault, and domestic violence) rather than any intrinsic gender differences (see Section 1.8.1 on gender for further information).

1.7 Chronicity

PTSD was once considered to be a short-term, 'adjustment' reaction to an extreme stressor. However, evidence gathered since the Vietnam war in particular has demonstrated that, for a minority of sufferers, PTSD can be a chronic condition that may wax and wane over time (Kessler et al, 1995, note that the DSM-IV sub-work group on PTSD suggested that this condition should be considered chronic if it endures for more than three months; consequently DSM-IV (1994) now considers a duration of greater than three months to be a chronic condition). As outlined in the 'Prevalence' section, 11% - 18% of Vietnam veterans studied in the 1990s and more recently still meet the DSM-IV (1994) criteria for PTSD several decades after their return from that particular conflict.

There is also evidence that a proportion of veterans have 'partial' PTSD, meeting some but not all of the criteria (Kulka et al, 1990; Weiss, Marmar, Schlenger, et al, 1992). This also appears to apply to other populations with chronic PTSD (eg sexual assault or child abuse victims, see Davidson and Foa, 1991), suggesting that a substantial number of trauma victims have clinical or sub-clinical symptoms of PTSD at any point in time. Some experts have criticised this focus on partial or sub-threshold PTSD (see for instance McNally, 2003), believing that it blurs the boundaries between what is a clinical disorder and a more normal stress reaction. There is also disagreement over what definition, if any, should be used to describe partial PTSD (for instance, should one include individuals who have previously met the full criteria for PTSD, but who are now in remission?). Breslau, Lucia, and Davis (2004) studied the level of impairment (including work loss days and social impairment) in a sample of 2181 adults in Detroit.
who met the full DSM-IV criteria for PTSD (n=152), or were considered to have partial PTSD (n=444), or who were exposed to a trauma but did not meet the criteria set down for either full or partial PTSD (n=1010). Partial PTSD was defined as meeting at least one of each of the DSM-IV symptoms groups (B, C and D) and having a symptom duration of at least one month. They found that, while individuals with partial PTSD reported greater impairment than those who had been exposed to trauma only, people who met the full DSM-IV criteria for PTSD were significantly more impaired again. In other words, full PTSD leads to substantially greater impairment than partial PTSD; this arguably suggests that the research focus should be on those who meet full criteria for this condition.

Breslau and Davis (1992) reported that 57% of individuals with PTSD in their sample of young adults in an urban location had met the criteria for this disorder for at least one year. Several prospective studies of survivors of a dam collapse in the USA which resulted in major flooding have also demonstrated that, for some sufferers of PTSD, their symptoms remain for many years. Two years after the 1972 Buffalo Creek Dam collapse, 44% of the sample met the criteria for PTSD, with 28% continuing to fulfil the criteria for this disorder 14 years after the event (Green, Grace, Lindy, Gleser, and Kramer, 1990, and Green, Lindy, Grace, Gleser, Leonard, and Korol et al, 1990).

Typically, PTSD symptoms are most severe very shortly after the traumatic incident in question, with the rates of PTSD decreasing over time. A small percentage of individuals (usually 3 – 5%) experience a delayed onset of symptoms: in these cases, some symptoms of PTSD are generally present from the time of the trauma, with these worsening over time until the individual meets the full criteria for this disorder (Resick, 2001).

Generally, however, studies demonstrate that the majority of individuals who initially meet the criteria for PTSD continue to improve until they no longer have a diagnosis of this disorder. This improvement typically occurs up until 12 or 18 months post-trauma. Much less improvement is evident in trauma samples after this time. Kessler et al
(1995) suggest that if PTSD has not resolved within 3 years, it has a substantial probability of remaining chronic. Breslau et al (1998) also found that PTSD tends to persist longer in women than men (mean duration of 48.1 months and 12 months, respectively).

1.8 Risk factors

The etiology for PTSD has been widely studied, with theories for the origin of this disorder encompassing psychosocial, demographic, biological, genetic, and other risk factors, including prior exposure to trauma and proximity to the traumatic event. Obviously not everyone who is exposed to a traumatic event develops PTSD, hence research on the variables that might predispose an individual to this disorder is valuable.

1.8.1 Gender

Gender has received considerable attention as a potential risk factor for developing PTSD after trauma. Breslau et al (1998) reported that this emerged as a significant risk factor for PTSD when other sociodemographic factors were controlled in their community-based study, with the women meeting criteria for this disorder twice as much as men. This appears consistent with the data reported earlier from the Kessler et al (1995) community study, where women were twice as likely to meet PTSD criteria as men (see Table 1.2). This may be at least partly explained by the fact that women are more likely to experience a trauma such as rape, child sexual abuse, or be exposed to domestic violence; such traumatic events are potentially more devastating in type and severity (Solomon and Davidson, 1997) than some other traumas (eg robbery, although see also Kessler et, 1995, for the high PTSD prevalence in women threatened with a weapon).

Interestingly, some studies that have examined the sequelae of a single event such as a natural disaster or motor vehicle accident have failed to replicate the finding that women are at greater risk of developing PTSD (eg Kuo, Tang, Tsay, Lin, Hu, and Chen, 2003;
Mayou, Bryant and Ehlers, 2001). Terrorist attacks appear to be an exception. Research conducted in the five to eight weeks following the September 11 terrorist attacks on the New York World Trade Centre found that women were twice as likely as men to report symptoms consistent with probable PTSD (Pulcino, Galea, Ahern, Resnick, Foley, and Vlahov, 2003). Similarly, Verger et al (2004) reported that women were at greater risk than men of developing PTSD in the aftermath of bombings in France during the mid-1990s. It is therefore open to debate whether being female is in itself a risk factor for PTSD, or whether the higher rates of this disorder reported among women is related to the nature of the trauma they have been exposed to and possibly a heightened sense of vulnerability.

1.8.2 Age

Age has also attracted attention as a potential risk factor in the development of PTSD, although research findings have been mixed. Norris (1992) reported that a group of older adults in a community-based sample appeared to be more resilient in terms of the impact of general stress and PTSD. Similarly, Fontana and Rosenheck (1994) found that the older veterans of three wars (World War II, the Korean War, and the Vietnam War) were less symptomatic than the younger men, although some of the more traumatised individuals in the older population may have already died, leaving a relatively healthy cohort to be studied. However, a number of other studies have not shown any association between age and PTSD (eg Mayou, Bryant, and Ehlers, 2001; Allden, Poole, Chantavanich, Ohmar, Aung and Mollica, 1996), while others have actually reported that older individuals are at greater risk of developing PTSD (for instance survivors of terrorist bombings in France, Verger et al, 2004). Clearly further research is required to establish under what circumstances age might present as a risk factor for the development of PTSD.
1.8.3 Ethnicity

Some American studies have reported that people of Hispanic background are at greater risk of developing PTSD than white Americans, particularly after a natural disaster (eg Perilla, Norris, and Lavizzo, 2002) or terrorist attack (Galea et al, 2002). The NVVRS (Kulka et al, 1990) found a higher incidence of PTSD among Hispanic veterans (28%), followed by African-Americans (21%) and then whites/others (14%). However, these findings are countered by others that have not reported any relationship between PTSD and ethnicity (eg Kessler et al, 1995). Non-white soldiers have often been placed in the 'front-line' in combat situations, increasing the likelihood that they will be exposed to traumatic events. However, this does not explain the greater rates of PTSD amongst Hispanic people caught up in disasters and attacks; cultural influences may potentially play a role here.

1.8.4 Pre-trauma experiences

Personality variables, early life experiences, and prior trauma have also emerged as potential risk factors for developing PTSD. Davidson, Hughes, Blazer and George (1991) found that people in the community who met criteria for PTSD were three times more likely to have experienced parental poverty, a history of family psychiatric illness, separation from the parents, and/or child abuse. Breslau et al (1991) also reported that prolonged childhood separation from parents, family history of anxiety and/or antisocial behaviour, and neuroticism (see below) were associated with greater vulnerability to PTSD following exposure to traumatic events (see also Breslau and Davis, 1992).

1.8.4.1 Personality variables

One personality factor that has been consistently associated with the development of PTSD symptoms is neuroticism. Studies have found significantly higher neuroticism scores among individuals with PTSD, compared with control groups who also experienced traumatic events. Specific examples include individuals exposed to combat
(Casella and Motta, 1990), bushfires (McFarlane, 1992), and other traumatic events in a community sample (Breslau et al., 1991). However, these studies were retrospective in nature, leaving open the question of whether neuroticism was also a significant pre-trauma variable. One prospective study of women who had suffered a pregnancy loss collected information on neuroticism and baseline arousal symptoms at various points throughout pregnancy until one month after the due date of birth (Engelhard, van den Hout, and Kindt, 2003). The results showed that pre-trauma neuroticism predicted PTSD symptoms, particularly the arousal symptom cluster. However, neuroticism was also strongly related to pre-trauma arousal symptoms. The authors speculated that there was a significant overlap between PTSD arousal and items commonly measured in neuroticism, which may account for the relationship between these two factors.

1.8.4.2 Exposure to previous traumatic events

One of the risk factors that has been most closely associated with the development of PTSD is exposure to previous trauma, particularly if this occurs during childhood or adolescence (see for example Bremner, Southwick, Johnson, Yehuda and Charney, 1993, and Cimino and Dutton, 1991). Abused or neglected children are more vulnerable to developing personality disorders and other psychiatric conditions, have poorer coping skills, and inadequate models (in terms of their caregivers) upon which to base their own problem-solving and adaptive strategies. There is also emerging evidence of distinct neurobiological alterations in abused children which may render them more vulnerable to future trauma (see Perry, 1999, and Putnam and Trickett, 1997).

Adults who develop PTSD may also be at risk of future episodes of this disorder after the initial symptoms subside. Similarly, individuals exposed to past trauma appear to be at greater risk of both exposure to subsequent traumatic events and development of psychiatric symptoms such as PTSD (see for instance Breslau, Davis, and Andreski, 1995, and Kessler et al., 1995, for a discussion on the subject of frequent exposure to traumatic events amongst certain sectors of the population). Resick (1988) studied rape victims to assess the extent to which prior trauma (including domestic violence, child
abuse, incest, and others) would affect recovery. She found that previous victimization was associated with greater distress one year after the rape.

1.8.5 Mental health problems and genetic variables

Some studies have found that the existence of pre-existing mental health problems in trauma victims impedes recovery. For instance, McFarlane (1989) noted that a prior history of psychiatric disorders was a better predictor of post-traumatic symptoms in firefighters after a bushfire than the degree of exposure to the fire or losses sustained.

Relatively little research exists on whether there is any genetic predisposition to PTSD. However, one study of 2092 male, monozygotic twin pairs (with one of each pair serving in the Vietnam war) found a current PTSD prevalence of 16.8% among the twins who served in Vietnam, compared with 5.0% for those who served elsewhere (Goldberg, True, and Eisen et al, 1990). This suggests a very limited role (if any) for genetic (or at least familial) variables in the development of this condition.

1.8.6 Peri- and post-traumatic environment

Finally, it appears that certain characteristics of the trauma and the postexposure environment are related to post-traumatic symptoms. Several studies have identified social support as an important variable, with limited or dysfunctional social support post-trauma associated with greater likelihood of a diagnosis of PTSD or poor adjustment (Keane, Scott, Chavoya, Lamparski, and Fairbank, 1985; Burgess and Holmstrom, 1979). The type of trauma is also implicated in the development of post-traumatic symptoms. Kessler et al (1995) found that rape was the traumatic event most likely to be associated with PTSD. Similarly, Breslau et al (1998) reported that the conditional risk of PTSD was highest for "being held captive/tortured/kidnapped" (53.8%) or rape (49%). Roth, Newman, Pelcovitz, van der Kolk and Mandel (1997) also reported that sexual abuse history resulted in greater dysfunction than physical abuse, with sexually abused women more likely to meet the criteria set out in this study, a
DSM-IV field trial, for a complex form of PTSD (see later discussion on this issue). Kilpatrick and his colleagues have shown that certain aspects of serious assaults against women, primarily rape, are more likely to be associated with the subsequent development of PTSD. These predisposing factors include perceiving one’s life to be at risk, suffering physical injury during the assault, completion of the rape, and testifying about the rape in court (Epstein, Saunders, and Kilpatrick, 1997; Kilpatrick, Saunders, Amick-McMullan, Best, et al, 1989).

Torture and exposure to terrorist attacks have also been shown to result in high rates of PTSD. For instance, van Velsen et al (1996) found that 52% of their sample of primarily Kurdish asylum-seekers (the majority of whom reported some type of torture) met the DSM-III-R (1987) criteria for PTSD, with sexual torture in particular leading to higher levels of PTSD symptomatology. Motor vehicle accidents, natural disasters, diagnosis with a life-threatening illness, and learning about others’ traumas are all associated with a relatively low risk of developing PTSD (eg Breslau et al, 1998).

Proximity to the trauma (probably best able to be researched in disaster and terrorist attack survivors) also appears to play an important role in whether individuals go on to develop PTSD. Residents who lived close to the World Trade Centre were more likely to report PTSD symptoms that those Manhattan residents living further away (20% vs 7.5%, Galea et al, 2002). Similarly, earthquake survivors who were physically close to the quake epicentre have greater rates of diagnosable PTSD. Basoglu et al (2004) reported PTSD rates of 23% for those at the epicentre of a Turkish earthquake, as opposed to 14% for those further away.

1.9 Comorbidity with other disorders

Both epidemiological studies of community populations and studies of traumatised and/or clinical populations have found a high comorbidity of other psychiatric disorders with PTSD. Some of these disorders (eg substance abuse) may be secondary to the primary diagnosis of PTSD as the traumatised individual attempts to control his or her
symptoms using whatever means are available (including use of illicit substances). The National Comorbidity Study (Kessler et al, 1995) revealed that 88.3% of men with lifetime PTSD and 79% of women with lifetime PTSD had at least one other diagnosable psychiatric disorder. The most commonly diagnosed comorbid disorders were major depression (48% in men and 49% in women), alcohol abuse (52% for men, 28% in women), and drug abuse (35% in men, 27% in women). Phobias, conduct disorder (in men), and dysthymia were also frequently diagnosed in these individuals with PTSD. Other community studies have also reported high rates of psychiatric comorbidity in individuals with PTSD, including Helzer et al (1987): 80%, Breslau et al (1991): 83%, and Davidson et al (1991): 62%. Unsurprisingly, studies of at-risk populations have also identified comorbid psychiatric disorders in individuals with PTSD. For instance, Keane and Wolfe (1990) found high rates of comorbid substance abuse (84%), major depression (68%), dysthymia (34%) and antisocial personality disorder (26%) amongst a sample of treatment seeking veterans. Similarly, Kulka et al (1990) in the NVVRS reported that almost all (98.9%) Vietnam veterans with PTSD met the criteria for one or more other psychiatric disorders, with substance abuse the most commonly occurring comorbid problem (73%). In their study of primarily Kurdish asylum-seekers who reported experiencing torture, Van Velsen et al (1996) reported that, of the 31 individuals meeting DSM-III-R (1987) criteria for PTSD, 15 also met criteria for other psychiatric disorders. Survivors of natural disasters who meet criteria for PTSD also appear to have significant comorbid psychiatric disorders, particularly major depression and generalized anxiety disorder (Green, Lindy, Grace, and Leonard, 1992; McFarlane and Papay, 1992).

Several studies have also identified high rates of comorbid Axis II disorders, specifically personality disorders. Results from the Keane and Wolfe (1990) study, reported above, suggested that 26% of treatment seeking veterans with chronic PTSD also met criteria for antisocial personality disorder. Kulka et al (1990), and Kessler et al (1995) also reported a high comorbidity between these two disorders (31% amongst Vietnam veterans, and 43% (male) and 15% (women) in a community sample). Southwick, Yehuda and Giller (1993) and Cashman, Molnar and Foa (1995) both found high rates of
comorbid paranoid personality disorder in their samples of individuals with PTSD (veterans and sexual assault victims, respectively). Southwick et al (1993) also reported a very high rate of borderline personality disorder (76%) in their sample of treatment seeking male Vietnam veterans with chronic PTSD. One might question whether the personality disorder pre-dated the onset of PTSD symptoms, given that one of the criteria for a personality disorder is that it has been present since early adulthood, or whether it in fact developed as a consequence of dysfunctional belief systems resulting from living with PTSD.

Conjecture relating to the reasons for such a high comorbidity rate centres on the overlap between PTSD and major depression symptoms (eg Keane and Wolfe, 1990), as well as the ‘self-medication’ hypothesis, where PTSD sufferers attempt to alleviate their distressing symptoms (eg high arousal levels, sleep disturbance, intrusive thoughts) with drugs or alcohol. An alternative explanation has been offered by Hyer, Fallon, Harrison and Boudewyns (1987) who found that individuals with PTSD tend to report higher levels of symptoms than those individuals who meet criteria for other psychiatric disorders. Hence, the high level of comorbidity between PTSD and other psychiatric conditions could be a function of symptom overreporting, or demonstrate the level of distress (even despair) felt by the individual with PTSD. There is also evidence that pre-existing psychological problems may render an individual more vulnerable to developing PTSD following exposure to a traumatic event (eg Kulka et al, 1990). Notwithstanding these hypotheses, it is clear that the relationship between PTSD and other psychiatric diagnoses is complicated and interwoven, presenting researchers with complex and challenging issues in studying this group.

1.10 ‘Complex’ PTSD

The preceding sections have presented evidence that PTSD is essentially an atypical reaction to a traumatic event that may become chronic and disabling, and is in many cases accompanied by other psychiatric disorders. Indeed, it appears that only a small minority of traumatised individuals have a sole diagnosis of PTSD. The significant
overlap in diagnosis between PTSD and some personality disorders, in particular, has led a number of researchers to believe that the criteria set out in DSM-III-R (1987) (and to a certain extent, DSM-IV) for a diagnosis of this disorder do not adequately describe its complexities, with potential for a new, related condition to be included in future editions of the DSM. This is particularly the case for survivors of childhood sexual and/or physical abuse, domestic violence victims, and possibly torture and trauma survivors. These types of traumatic events are often repeatedly enacted over weeks, months, or years, usually involve an abuse of power by a trusted figure (for childhood abuse), and include subjugation, boundary violations, terror, and captivity, with victims often feeling a deep sense of shame and helplessness. There is also the knowledge that the abuse will be repeated by the perpetrator, with the victim feeling powerless to prevent this. Herman (1997), one of the strongest advocates for the creation of a new diagnostic concept that would adequately describe victims of repeated trauma, states, “Even the diagnosis of “post-traumatic stress disorder,” as it is presently defined, does not fit accurately enough. The existing diagnostic criteria for this disorder are derived mainly from survivors of circumscribed traumatic events ... In survivors of prolonged, repeated trauma, the symptom picture is often far more complex. Survivors of prolonged abuse develop characteristic personality changes, including deformations of relatedness and identity. Survivors of abuse in childhood develop similar problems with relationships and identity; in addition they are particularly vulnerable to repeated harm, both self-inflicted and at the hands of others. The current formulation of post-traumatic stress disorder fails to capture either the protean symptomatic manifestations of prolonged, repeated trauma or the profound deformations of personality that occur in captivity.” (Herman, 1997, p119).

There is also evidence that victims of the types of trauma described above may be more likely to dissociate in an effort to cope (see Roth, Newman, Pelcovitz, van der Kolk and Mandel, 1997, and Van Velsen, et al, 1996). Discussion prompted by the work of
psychiatrists such as Herman and van der Kolk prompted research in the form of field trials prior to the DSM-IV (1994) revision to examine whether a new diagnosis of complex PTSD (a term coined by Herman, 1997), also known as Disorders of Extreme Stress Not Otherwise Specified (DESNOS) was warranted. Herman (1997) outlined seven diagnostic criteria for complex PTSD, which included being subject to prolonged totalitarian control, and alterations in affect regulation, consciousness, self-perception, relationships with others, systems of meaning, and perception of the perpetrator of the abuse.

The DSM-IV field trial conducted by Roth et al (1997) examined a sample of treatment-seeking childhood physical and sexual abuse survivors. They reported that 50% of participants met criteria for lifetime complex PTSD, with 72% of those who met criteria for PTSD also meeting the criteria set out in the study for complex PTSD. Sexual abuse (particularly in combination with physical abuse) appeared to be the strongest risk factor for developing this latter condition. However, as complex PTSD did not occur in the absence of PTSD, it was decided not to include a separate diagnostic disorder in DSM-IV (1994) incorporating symptoms of extreme stress. In other words, disorders of extreme stress could be seen as a subset of PTSD. Hence, these symptoms have been included in DSM-IV as ‘associated features’ of PTSD.

In summary, there is evidence that victims of ongoing trauma involving shame, violence, possibly captivity, and abuse of power as occurs in childhood sexual and physical abuse and torture meet criteria for PTSD but also suffer a more chronic loss of self-esteem and affect dysregulation. This is an important issue that needs careful consideration in any research involving trauma victims who have been exposed to chronic abuse.

1.11 Psychobiology of PTSD

This section will briefly review some of the extensive research undertaken on the psychobiology of PTSD, particularly as it relates to neuroendocrine alterations. Space does not permit a thorough review of the various neuroanatomical structures involved in
the fear and stress response; Table 1.3 (adapted from van der Kolk, 1997) instead summarises the main findings.

Table 1.3: Biological Abnormalities in PTSD

<table>
<thead>
<tr>
<th>Psychophysiologic</th>
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<tbody>
<tr>
<td>Extreme autonomic responses to stimuli reminiscent of the trauma</td>
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<tr>
<td>Nonhabituation to startle stimuli</td>
</tr>
<tr>
<td>Reduced response to event-related potentials</td>
</tr>
<tr>
<td>Response to sound intensifies below threshold</td>
</tr>
<tr>
<td>Decreased EEG cortico-cortical synchronization in children</td>
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<thead>
<tr>
<th>Neurotransmitter</th>
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<tr>
<td>Noradrenergic</td>
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<tr>
<td>Elevated urinary catecholamines</td>
</tr>
<tr>
<td>Increased MHPG to yohimbine challenge</td>
</tr>
<tr>
<td>Reduced platelet MAO activity</td>
</tr>
<tr>
<td>Down-regulation of adrenergic receptors</td>
</tr>
<tr>
<td>Serotonergic</td>
</tr>
<tr>
<td>Decreased serotonin activity in traumatised animals</td>
</tr>
<tr>
<td>Best pharmacologic responses to serotonin selective uptake inhibitors</td>
</tr>
<tr>
<td>Endogenous opioids – increased opioid response to stimuli reminiscent of trauma</td>
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</tbody>
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<table>
<thead>
<tr>
<th>HPA axis</th>
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<tbody>
<tr>
<td>Decreased resting glucocorticoid levels</td>
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<tr>
<td>Decreased glucocorticoid response to stress</td>
</tr>
<tr>
<td>Down-regulation of glucocorticoid receptors</td>
</tr>
<tr>
<td>Hyperresponsiveness to low-dose dexamethasone</td>
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<tr>
<th>Memory</th>
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<tbody>
<tr>
<td>Amnesias and hyperamnesia</td>
</tr>
<tr>
<td>Traumatic memories precipitated by noradrenergic stimulation, physiologic arousal, reminders</td>
</tr>
<tr>
<td>Memories generally sensorimotor rather than semantic</td>
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<table>
<thead>
<tr>
<th>Neuroanatomical</th>
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<tbody>
<tr>
<td>Decreased hippocampal volume</td>
</tr>
<tr>
<td>Activation of right amygdala and parahippocampal structures during flashbacks</td>
</tr>
<tr>
<td>Activation of right sensory areas during flashbacks</td>
</tr>
<tr>
<td>Decreased activation of Broca's area during traumatic exposure</td>
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<tr>
<td>Marked hemisphere lateralization</td>
</tr>
<tr>
<td>Decreased left hemisphere cortical synchronization in abused children</td>
</tr>
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<table>
<thead>
<tr>
<th>Miscellaneous</th>
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</thead>
<tbody>
<tr>
<td>Traumatic nightmares often not oneiric but exact replicas of visual elements of trauma: may occur in stage II or stage III sleep</td>
</tr>
<tr>
<td>Impaired psychoimmunologic function</td>
</tr>
</tbody>
</table>

adapted from van der Kolk, 1997
1.11.1 Normal stress response

An individual’s response to an acute stressor was conceptualised by Cannon (1914) as the ‘fight or flight’ reaction. Selye (1956/1976) went on to conceptualise a ‘normal’ stress response with three stages: alarm and mobilisation, resistance, and exhaustion. As part of this first stage, catecholamines (adrenaline and noradrenaline, often referred to as epinephrine and norepinephrine) are secreted by the adrenal medulla (part of the adrenal glands) and locus coeruleus (located in the brainstem) in order to mobilise the organism and effect a defence or escape from danger. These hormones cause an increase in skeletal muscle contraction, heart rate, blood pressure, respiration, oxygen consumption, and blood sugar secretion (Lating and Everly Jr, 1995). Noradrenaline in particular plays a critical role in initiating the flight or fight behaviours and mobilising the organism to deal with the stressor. It is also important in memory consolidation (van der Kolk, 1997). Other chemicals also play a role in assisting the organism to cope with an acute stressor (part of the ‘resistance’ stage): serotonin modulates noradrenaline, possibly inhibiting the ‘fight response’ (low levels of serotonin are associated with exaggerated emotional arousal, such as irritability, excitability, and hypersensitivity), and endogenous opiates are released into the bloodstream to numb pain.

A different aspect of the stress response is controlled by the hypothalamic-pituitary-adrenal (HPA) axis, which is regulated by corticotrophin-releasing factor (CRF). The HPA axis serves to stimulate the release of neurohormones in order to help the organism deal with the stressor. The hypothalamus in the brain releases CRF, which stimulates the pituitary gland to secrete adrenocorticotropic-releasing hormone (ACTH) which in turn regulates the release of cortisol from the adrenal cortex (van der Kolk, 1997; Heninger, 1995). Cortisol helps to regulate and reduce the stress response (Yehuda, Southwick, Mason, and Giller, 1990), prompting a release of additional blood sugar while increasing muscle tone in the heart and blood vessels and reducing inflammation. Yehuda has referred to cortisol as the “antistress” hormone which acts as part of a negative feedback loop, signalling the brain to shut off the stress hormones once a threat has passed.
In addition to the structures that form part of the HPA axis, the primary brain structures involved in the stress response are the amygdala and hippocampus, which are part of the relatively primitive limbic system which plays a major role in self-preservation, procreation, parental care, and play (van der Kolk, 1997). The amygdala and hippocampus are critical to the fear response and consolidation of memories associated with this. The locus coerules, the cells of which secrete noradrenaline and opioids, also plays a critical role in the stress response. At the neocortical level, the prefrontal cortex has a role in the conscious elaboration of the trauma, providing it with meaning and context. Other structures implicated in responses to traumatic stress include the anterior cingulate gyrus, Broca’s area (responsible for translating experience into speech), insula, and medial temporal lobe (van der Kolk, 1997).

1.11.2 Harmful effects of prolonged stress

Under normal circumstances levels of stress hormones rapidly return to a state of homeostasis after the immediate threat has passed. However, when an organism is exposed to chronic stress, continued high levels of blood sugar and an elevated metabolism (stimulated by the increased amounts of cortisol) may suppress the immune system (Selye, 1956/1976). In addition, there is evidence that prolonged stress may be associated with a reduced percentage of natural killer cells (important components of the immune response to viral infections, etc) in the blood (see Heninger, 1995, for a review). An immune system which is suppressed may ultimately lead to ill health and disease. It has also been noted that prolonged serotonin depletion may be associated with rage, impulsivity, depression, anxiety, and disrupted slow-wave and rapid eye-movement sleep (Resick, 2001; van der Kolk, 1997).

1.11.3 PTSD: a different category of stress response

PTSD develops in certain individuals after exposure to a traumatic stressor, involving a reaction incorporating fear, helplessness or horror; therefore one might reasonably
expect the typical stress response as mediated by the HPA axis. This was in fact assumed to be the case for around a decade after PTSD was first included under the category of anxiety disorders in DSM-III (1980), particularly given the fact that PTSD often occurs co-morbidly with major depression. However, one important difference between the normal stress response and that which occurs in PTSD is that chronic stress tends to escalate gradually over time (with the individual often feeling exhausted and overwhelmed) while the impact of traumatic stress is generally sudden and dramatic. The neurobiological responses of the two stressful states have been found to differ.

While high levels of cortisol (as occurs in the stress response) have been found in patients suffering from depression (Sacher, Hellman, and Roffwarg, 1973) and chronic stress or anxiety, most recent research has reported that PTSD sufferers in fact have chronic low levels of cortisol compared with normal controls, other psychiatric patients, or trauma survivors without PTSD (Mason, Giller, Kosten, Ostroff and Podd, 1986; Yehuda, Boisoneau, Mason, and Giller, 1993). Yehuda (1997) noted that her studies of urinary cortisol levels in Vietnam veterans and Holocaust survivors with PTSD demonstrated that cortisol was low regardless of whether the participants were inpatients, outpatients, treatment-seeking, community-based, male, female, or had comorbid depression. She suggests that low cortisol levels in PTSD sufferers reflect symptom severity, rather than an adaptation to trauma. Yehuda also speculates that this chronic low level of cortisol may indicate a sensitization of the stress response (mediated by the HPA axis) such that cortisol is quickly metabolised. Yehuda and her colleagues have reported that Vietnam veterans with PTSD have significantly more glucocorticoid receptors compared to normal controls or other psychiatric patients (Yehuda et al, 1993; Yehuda, Boisoneau, Lowy, and Giller, 1995). Given research that glucocorticoid receptors mediate the strength of negative feedback in the HPA axis, this may reflect a system that is maximally responsive to stress. Sufferers of PTSD often appear to be unusually responsive to stress, demonstrating hypervigilance, an exaggerated startle response, and a reduced ability to deal with stressors in the environment; this is consistent with a biological sensitisation of the HPA axis with chronic low levels of cortisol (Yehuda, 1997). Low cortisol levels have been found during psychological
trauma, in the immediate aftermath of a traumatic event such as rape, and in individuals with chronic PTSD (Yehuda, 1997). Hence, low cortisol levels as measured shortly after a traumatic incident tend to predict the development of PTSD several months later, according to some studies (Resnick, Yehuda, Pitman, and Foy, 1995; McFarlane, Atchison, and Yehuda, 1997). The question as to whether individuals with PTSD may have had low cortisol levels prior to developing this psychiatric disorder has not been resolved. Yehuda (1997) speculates that PTSD could reflect a biological sensitization following stress due to preexisting risk factors; she suggests an alternative name: posttraumatic sensitization disorder rather than posttraumatic stress disorder. Van der Kolk (1997) suggests that an inability to mount an adequate cortisol response to acute stress may interfere with the individual’s capacity to integrate the experience, predisposing them to PTSD.

Logically, one might expect low levels of cortisol to be associated with enhanced immune functioning, given the role of cortisol as an immune suppressor. However, there is evidence (reviewed in Chapter 2) that individuals with PTSD report an array of medical problems and symptoms, while generally feeling that their health is poor. This picture is further complicated by recent evidence that the relationship between traumatic stress and cortisol levels may not be as clear-cut as originally assumed. Bonne, Brandes, Segman, Pitman, Yehuda, and Shalev (2003) undertook a prospective study of plasma cortisol levels and PTSD symptoms in a small sample of primarily motor vehicle accident victims who did not receive serious physical injuries. The authors reported that cortisol levels taken one week after the trauma did not predict chronic PTSD, with symptoms assessed again at six months post-trauma. In other words, there was no significant difference in cortisol levels between trauma survivors with and without PTSD. They speculate that HPA dysfunction may develop along with PTSD symptoms, implying a progressive neuronal sensitization.

There is also emerging evidence of damage to brain structures in PTSD. A number of studies have shown that some people with PTSD (Vietnam veterans or women reporting chronic childhood sexual abuse) have reduced hippocampal volume when compared
with controls (eg Bremner, Randall, Scott, et al, 1995; Bremner, Randall, Vermetten, et al, 1997). Van der Kolk (1997) speculates that this decrease in the size of the hippocampus is a result of the long-term effect of intrusive re-living of the trauma. Damage to the hippocampus combined with high levels of emotional arousal may be responsible for the difficulties PTSD sufferers sometimes report in ‘taking in’ and processing information of an arousing nature, leaving them vulnerable to perceiving such stimuli as a threat, and reacting in an aggressive or avoidant manner.

1.12 Summary

This chapter has provided an historical context to the recognition of PTSD as a psychiatric disorder, in addition to providing an overview of epidemiological, etiological, and possible underlying psychobiological factors. Research suggests that while a majority of individuals will be exposed to at least one traumatic event in their lifetime, not everyone who is so exposed will develop PTSD. The lifetime prevalence rate, as estimated by community studies, generally appears to be in the order of 8% - 10% (eg Breslau et al, 1991; Kessler et al, 1995). Once traumatised populations are studied, the prevalence rate unsurprisingly increases to around 30% for rape or domestic violence victims (Resnick et al, 1993; Gleason, 1993), 50% for torture and trauma survivors/refugees (eg Van Velsen et al, 1996; Kinzie et al, 1990), and 15% - 20% for Vietnam veterans (eg Kulka et al, 1990). Studies of this latter group, in particular, have demonstrated that PTSD can be a chronic condition that at times subsides to “sub-clinical” levels. However, the majority of individuals who initially meet criteria for PTSD improve within 12 months of experiencing the traumatic event responsible for the onset of the condition. PTSD also rarely occurs in isolation, with comorbid depression, substance abuse, and personality disorders common features in studies of traumatised populations.

Potential risk factors for developing PTSD include personality variables (primarily neuroticism), exposure to prior trauma, having a limited social support network, and possibly proximity to the trauma. There is also evidence that the type of trauma is an
important variable, with events such as rape, captivity, torture, sexual abuse, and exposure to terrorist attacks (in some instances) leading to higher rates of PTSD. While age, ethnicity, and gender have also received attention as potential risk factors, research has produced mixed results.

Finally, the psychobiology of PTSD is an area currently attracting extensive research. If alterations in certain neurochemicals can be implicated in the development and chronicity of PTSD (such as low cortisol levels), then pharmaceutical companies can tailor specific medications for PTSD (a potentially lucrative area, as no pharmaceutical products are currently available to “treat” PTSD in the same way that antidepressant medications can treat depression).

If PTSD is a chronic, disabling condition for a minority of trauma victims, then the implications for physical health and well-being are worth considering, given evidence that prolonged stress can harm an organism (e.g., Heninger, 1995). The following chapter considers this issue in some detail.
Chapter Two: Trauma, PTSD, and Physical Health

2.1 Introduction

The previous chapter reviewed evidence that a substantial proportion of the population will at some point in their lifetime experience a traumatic event. A minority of these individuals will go on to develop PTSD. This may become a chronic condition, often presenting with comorbid psychiatric conditions such as major depression and substance abuse. While PTSD develops in response to an often acute stressor, the symptoms typical of the disorder, such as re-experiencing, avoidance, and hyperarousal, may constitute an ongoing stressor in and of themselves. Given research linking poor health with certain types of stress (see Rosen and Fields, 1988; Heninger, 1995, and Williams, 1995, for reviews), it is worthwhile examining whether PTSD and/or exposure to trauma might also be related to poorer health outcomes. Individuals with PTSD often report a perception that their health is poor, leading some researchers to suggest that this reflects a degree of somatisation or symptom over-reporting. Van der Kolk (1997) has in fact stated that somatisation should form part of the diagnostic criteria for PTSD in the DSM. This contrasts with the need to consider separately the impact of any injuries sustained during the traumatic event, and the extent to which trauma or PTSD may lead to diagnosable diseases or illness. If evidence suggests that PTSD is associated with poor health, there are a number of possible explanations for this that need not be mutually exclusive:

- The stress associated with PTSD may directly lead to poor health, possibly through a compromised immune system, and/or other factors including increased catecholamines and white blood cell counts;
- Having PTSD may result in an increase in certain health-compromising behaviours (eg smoking, alcohol abuse, substance abuse, poor diet, sleeplessness, etc) or affective states (eg hostility and anger) which in turn may have an adverse impact on health;
• There may be a heightened perception of poor health and an increased preoccupation with somatic complaints in individuals with PTSD;

• Comorbid physical injuries or conditions (e.g., injuries sustained in an attack or accident, or a life-threatening condition that precipitated PTSD) may complicate the picture.

As outlined in the previous chapter, stress has been associated with neurochemical changes and a reduced immunological response; however, PTSD presents somewhat differently from the 'typical' stress response. There is evidence of low cortisol levels in individuals diagnosed with PTSD, and increased white blood cell counts (Boscarino and Chang, 1999a; Ironson, Wynings, Schneiderman, et al., 1997). The latter variable has been associated with coronary heart disease, even after controlling for possible confounding variables such as age, gender, and smoking (Bovill, Bild, Heiss et al., 1996; Danesh, Collins, Appleby, and Peto, 1998), although the mechanisms by which such an association is mediated are unclear. Danesh et al. (1998) note that the immunological response is highly complex, and alterations associated with coronary heart disease may involve increases in some types of white blood cells, and decreases in others.

There is also evidence that PTSD sufferers have lower natural killer cell activity in the aftermath of natural disasters (Inoue-Sakurai, Maruyama, and Morimoto, 2000; Ironson et al., 1997). Natural killer cells present the first line of defense against illnesses such as cancer. Kiecolt-Glaser, McGuire, Robles, and Glaser (2002) provide a comprehensive review of psychoneuroimmunological research that demonstrates correlations between affective states (primarily depression and anxiety), exposure to severe stressors, and altered immunological responses in humans such as decreased CD4+ T-cell counts, increased B-cell counts, and reduced natural killer cell activity. Some of the studies reviewed by Kiecolt-Glaser et al. (2002) also reported evidence of poorer health in their samples, such as greater rates of respiratory infections and an accelerated course of HIV in one prospective study. Importantly, Kiecolt-Glaser et al. (2002) note evidence that both short and long-term stressors may be associated with immune dysregulation and deleterious health effects including chronic infectious diseases, and inflammation linked
to cardiovascular disease, osteoporosis, arthritis, and type 2 diabetes. It is therefore plausible that immunological alterations such as changes in white blood cell counts and activity in individuals with PTSD may render an individual more vulnerable to ill health in both the short and long term.

This chapter will review research examining the relationship between trauma, PTSD, and ill health. It will also consider a number of complex issues associated with any such relationship, specifically:

- whether PTSD (as distinct from exposure to trauma alone) is directly associated with negative health outcomes, and if so whether specific aspects of this condition (eg sleeping problems; any of the specific symptom clusters) are more closely related to poor health
- the degree to which any perception of ill health is attributable to medically diagnosable conditions as opposed to somatisation
- the impact of comorbid conditions such as depression, substance abuse, and smoking on any relationship between PTSD and poorer health
- whether different types of trauma (eg combat as opposed to sexual assault, or one-off versus multiple traumas) impact on health outcomes in different ways.

### 2.2 War trauma and physical health

Combat veterans represent an interesting and unique group in studying any relationship between trauma, PTSD, and health problems. Recruitment into the armed forces is usually conducted in a rigorous fashion to ensure that only individuals meeting strict health and medical standards are employed. This healthy (and overwhelmingly male) cohort is then subjected to combat and the associated physical injuries and trauma that may eventuate. Research on the relationship between exposure to traumatic events and physical health outcomes in this group focussed initially on World War II veterans, including concentration camp survivors and resistance fighters (eg a longitudinal study by Weisaeth and Eitinger, 1993; also Elder, Shanahan, and Clipp, 1997). Research
findings were mixed: the health of veterans/survivors appeared to suffer to some extent (compared with an age and gender-matched control group who were not exposed to such trauma); however major confounding variables – including the failure to account for the physical deprivation present in concentration camps – were present.

In the aftermath of the Vietnam War, amid claims of ill health among veterans attributed to factors including physical combat trauma and herbicide exposure, the health of Vietnam veterans has been examined extensively. Several large scale studies have been undertaken in the USA by the Centers for Disease Control, in addition to the National Vietnam Veterans Readjustment Study (NVVRS). The Centers for Disease Control (1988) found that male veterans reported current and past health problems more frequently than veterans who did not serve in Vietnam. However, medical examinations generally did not find any difference between these two groups in terms of physical health, apart from greater hearing loss in the Vietnam veterans (due presumably to the use of heavy artillery), and to some extent an increased incidence of stool occult blood and left ventricular hypertrophy in this group. Although this study employed a large sample, and was methodologically rigorous, the age of the veterans at the time it was conducted (mid- to late- 30s, on average) may indicate that there had been insufficient time for health problems to manifest following the Vietnam War. Additionally, this study did not consider the differential impact of PTSD on health outcomes. The National Vietnam Veterans Readjustment Study (NVVRS, Kulka, Schlenger, Fairbank, et al, 1990) also considered the physical health of male and female Vietnam veterans a decade after the conflict ceased. While they found no difference in the prevalence of self-reported health problems among Vietnam theatre veterans (who saw combat), veterans who were not in Vietnam (era veterans), and civilian counterparts, a significant relationship between health and degree of combat stress was evident. Vietnam veterans who were exposed to higher levels of war-zone stress tended to report poorer perceptions of their physical health, and a larger number of chronic medical conditions than a variety of comparison groups.
Researchers have also examined the contribution of genetics to reports of ill-health among Vietnam veterans. Eisen, Goldberg, True, and Henderson (1991) examined over 2,000 monozygotic twin pairs, both of whom served in the armed forces during the Vietnam War era. While some served in South-East Asia, others were stationed elsewhere. By examining health records and perceived physical health of these individuals (while looking for health differences among twin pairs), Eisen et al (1991) concluded that military service in South East Asia led to increased reports of hearing difficulties and skin problems. However, it was unclear from this study whether there were occasions when both twins in each pair were exposed to combat. In a later study, Eisen, Neuman, Goldberg et al (1998), again considered the self-reported physical health of Vietnam era veterans and their twins, this time both monozygotic and dyzygotic. They concluded that combat exposure made a small but significant contribution to the propensity to report current health problems, but that genetic effects accounted for the greatest variance in this variable. Again, PTSD was not considered separately as a variable of interest.

In Australia, the recent Vietnam Veterans' Morbidity Study (Commonwealth Department of Veterans' Affairs, 1998) concluded that Vietnam veterans in Australia reported their health to be worse than that of other Australians of a similar age. In particular, a higher incidence of certain types of cancer, motor neurone disease, multiple sclerosis, asthma, diabetes, and ischaemic heart disease was reported in male veterans, although some of these findings were not supported by a later validation study (Australian Institute of Health and Welfare, 1999). The results were limited to some extent by the use of general community health data for comparison purposes. It could be argued that this population is different from that of the armed forces by virtue of the fact that recruits to the defence forces are screened carefully for health problems and initially represent (hypothetically) a healthier population. As with the preceding studies, no consideration was given to the impact of PTSD on health.

Another recent study, this time of a large group of Gulf War veterans, was undertaken in the United Kingdom by Ismail, Blatchley, and Hotopf et al (2000). This cross-sectional
study of 3905 UK Gulf veterans who were surveyed six to seven years after the first Gulf War ended found that the only significant variable related to poor health was rank; privates were most likely to report poor health outcomes. PTSD was only considered in the context of being a ‘health outcome’.

Finally, the Centers for Disease Control (1987) conducted an extensive study into the mortality of Vietnam veterans up until 1983. They reported that mortality was 17% higher in Vietnam veterans compared with a sample of veterans of the same era who had served in Korea, Germany or the USA. The most common causes of death were motor vehicle accidents, suicide, homicide, and accidental poisoning; the majority of deaths occurred in the first five years following discharge from active duty. PTSD was not considered separately as a variable.

2.3 Health outcomes after war trauma: PTSD as a critical variable

2.3.1 Vietnam veterans

Research undertaken initially with Vietnam veterans in the USA has provided evidence that it is the ongoing stress associated with the avoidance, intrusion, numbing, and re-experiencing symptoms of PTSD that is the prime mediating variable in the development and/or reporting of physical health problems following trauma (Friedman and Schnurr, 1995). Several studies of non-treatment seeking female Vietnam veterans undertaken by Wolfe and her colleagues have established that PTSD symptoms account for a significant portion of the variance in health problems. Wolfe, Schnurr, Brown, and Furey (1994) considered the differential effects of the severity of exposure to trauma and PTSD on self-reported physical health complaints in their sample. While both war-zone exposure and PTSD independently predicted health complaints, adjusting the former variable to take PTSD into account led to the effects of exposure diminishing markedly, while the impact of PTSD on reported health remained essentially unchanged. Kimerling, Clum, and Wolfe (2000) also considered whether PTSD symptoms were significant mediating variables in the relationship between exposure to a traumatic event
and subsequent self-reported health complaints. Their sample of 52 female Vietnam veterans included 12 who met full criteria for PTSD, another five who met ‘partial’ criteria (two of the three symptom clusters were met), and another 12 who had previously fulfilled the diagnostic criteria for PTSD. The remainder had never met criteria for PTSD. The group who met full current criteria for PTSD reported significantly more stomach cramps, muscle weakness, and neck pain or stiffness than those who did not. Kimerling et al (2000) found that both trauma exposure and PTSD symptoms significantly predicted both physical symptoms and health perception. According to the authors, the direct effect of trauma exposure on these health outcome variables was accounted for by the indirect effects of trauma exposure through PTSD symptoms. They also reported that the PTSD hyperarousal symptom cluster was associated with higher levels of physical symptoms and poorer health perception.

More extensive studies of Vietnam veterans’ health have also examined the relationship between health outcomes and PTSD. Kulka et al (1990), in the comprehensive National Vietnam Veterans Readjustment Study (NVVRS), noted that Vietnam theatre veterans (i.e., those who saw combat) with a current diagnosis of PTSD and/or a lifetime diagnosis of substance abuse or dependence rated their current physical health as worse than theatre veterans without these disorders. In addition, Zatzick, Marmar, and Weiss et al (1997) undertook archival analysis of data from the NVVRS to assess the extent of PTSD comorbidity with other psychiatric conditions, and the relationship with physical health problems. They reported that individuals with PTSD had a significantly higher risk of poor health, physical limitations, and reduced well-being than veterans without PTSD, even after controlling for comorbid conditions such as Major Depressive Disorder. Similarly, Zatzick, Weiss, Marmar et al (1997) looked at data from the NVVRS relating to female veterans (primarily nurses), and also found a high comorbidity of PTSD with depression, alcohol abuse, and panic disorder. Again, PTSD was associated with poorer functioning and an increased number of medical conditions when these comorbid conditions were controlled for. Taft, Stern, King, and King (1999) also examined data from the NVVRS in considering whether hardiness and degree of
social support mediated a link between PTSD and health problems. No such relationship was found.

Litz, Keane, Fisher, Marx, and Monaco (1992) examined a small number (n = 37) of treatment-seeking Vietnam veterans, with and without PTSD. They reported that the group with PTSD reported more current health problems (mainly of a cardiopulmonary and gastrointestinal nature) than veterans without PTSD. However, the small sample size in this study raises questions about the generalizability of these findings. Interestingly, Litz et al (1992) collected a number of physiological measures, and reported a relationship between higher baseline heart rate prior to veterans being exposed to stressful stimuli, and self-report of heart problems.

Comparatively little research has been undertaken outside the USA into any association between PTSD and physical health problems in Vietnam veterans. However, researchers in Australia reported that a diagnosis of PTSD in a group of 641 Vietnam veterans was associated with significantly higher health care costs (Marshall, Jorm, Grayson, and O'Toole, 1998, 2000).

A recent study took into account sub-clinical or ‘partial’ PTSD in examining health care usage among Vietnam veterans (Deykin, Keane, Kaloupek et al, 2001). This study found that those veterans with the highest health care usage were more likely to meet the criteria for PTSD than low users of health care, with the high usage group having a greater PTSD symptom severity score (as assessed via the Clinician Administered Posttraumatic Stress Scale, CAPS). No differences were found in relation to the number of health visits made by non-PTSD and partial-PTSD groups. Importantly, this survey-based study also found that veterans with PTSD had a greater number of medically diagnosed health complaints. Health care utilisation by male Vietnam veterans with and without PTSD was also considered by Schnurr, Friedman, Sengupta, Jankowski, and Holmes (2000). The authors examined data from the NVVRS and Hawaiian Vietnam Veterans Project to investigate the role of comorbid Axis I disorders on the relationship between PTSD and medical utilisation. They found that PTSD was significantly related
to greater utilisation of medical services; part of this relationship was mediated by comorbid psychiatric conditions (particularly depression).

2.3.2 Somatisation or 'real' medical conditions?

The issue of whether PTSD symptomatology leads to a higher degree of somatisation or does in fact reflect some underlying physical pathology has been difficult to assess adequately in many of the studies reviewed above, with the possible exception of Deykin et al (2001), and Zatzick, Weiss, Marmar et al (1997) in relation to female veterans. Earlier research by White and Faustman (1989) provided some evidence of an association between PTSD and medical conditions by means of a comprehensive review of the medical discharge summaries of 543 Vietnam veterans (mean age 36.9 years) who would have met criteria for PTSD. 60% of the veterans with PTSD had some kind of physical condition listed on Axis III of the DSM, including musculoskeletal problems, combat trauma/injury, and sensory impairment. There was no evidence of co-morbid somatisation disorder. Limitations of this study included its retrospective, 'file-audit' nature, and the fact that a control group of veterans without PTSD was not included in the analysis.

Recently published research undertaken by Boscarino and his colleagues in the mid-1980s has provided further evidence that PTSD can have direct deleterious effects on physical health, over and above somatic preoccupations. In one extensive study of 1399 male Vietnam veterans, Boscarino (1997) explored the relationship between PTSD and disease 20 years after combat exposure, while controlling for hypochondriasis, substance abuse, comorbid psychiatric disorders, and smoking. He found that PTSD was associated with 6 out of the 10 diseases examined (circulatory, digestive, musculoskeletal, nervous system, respiratory, and infectious diseases). Boscarino speculated that chronically increased catecholamines and chronically decreased cortisol secretions could play a role in any relationship between PTSD and disease. Further research on this sample showed that PTSD was also associated with atrioventricular (AV) conduction defects and infarctions, even after controlling for depression, anxiety,
and a variety of other factors associated with negative health outcomes (Boscarino and Chang, 1999b).

Boscarino and Chang (1999a) followed this up with research examining white blood cell counts in Vietnam veterans with 'current partial' PTSD (individuals with 'partial' PTSD who had also met the diagnostic criteria for this condition in the past) and those meeting criteria for anxiety and/or depression. The authors reported that the current partial PTSD group had higher overall mean white blood cell, lymphocyte, T-cell, and CD4 cell counts (all adjusted for the covariates described in the previous paragraph), which was not the case in those suffering from depression. As outlined earlier in this chapter (Section 2.1), elevated white blood cell counts have been linked to coronary heart disease, although the mechanisms behind this association are unclear (Danesh et al, 1998).

Further evidence of a direct relationship between PTSD and medically diagnosed health conditions was reported by Beckham et al (1998), in their examination of both self-reported and physician-rated health in a sample of treatment-seeking Vietnam veterans with (n = 225) and without (n = 51) PTSD. PTSD severity predicted both types of health outcome, as did cigarette smoking (the extent of which was similar across both the PTSD and non-PTSD groups). Hypochondriasis was also strongly related to self-reported health problems in the PTSD group. However, the authors did not control for comorbid psychiatric disorders such as depression, although it may be noted that 83% of the PTSD group recorded high scores on the Beck Depression Inventory (BDI).

Relatively few mortality studies of veterans with PTSD have been carried out, although this may change with an ageing population of veterans and the potential for certain diseases to lead to death. Bullman and Kang (1994) reported that Vietnam veterans with PTSD were more likely to have died from accidental death, accidental poisoning, suicide, and cirrhosis of the liver than veterans without PTSD. This may partly be due to some of the behaviours associated with PTSD, such as increased anger, smoking, alcohol use, and drug abuse.
2.3.3 Veterans of other wars and conflicts

Veterans from other conflicts have also been studied to establish any relationship between trauma, PTSD, and poor health outcomes. Falger, Op den Velde, Hovens, et al (1992), in a study of Dutch Resistance Fighters from World War II, considered a number of factors in the relationship between PTSD and physical health (specifically, cardiovascular disease), including 'vital exhaustion', Type A behaviour, and prevalence of smoking. Veterans were compared with two age and sex matched control groups: surgical patients and people recovering from a recent myocardial infarction. Falger et al (1992) reported that the veterans were more likely to express Type A behaviour, have greater rates of vital exhaustion, and report significantly higher rates of angina (although the level of angina pectoris was equivalent in the PTSD and myocardial infarction groups). A surprising finding was the fact that the veterans were less likely to smoke than either of the control groups.

Hankin, Abueg, Gallagher-Thompson and Laws (1996) examined PTSD arising from any event (as opposed to combat alone) in older treatment seeking male veterans. They reported that one third of their small sample (nine out of 30 individuals) met the criteria for lifetime PTSD, and reported more chest pain and arthritis than the veterans without PTSD. Apart from the small sample size, this study was limited by the fact that the type of trauma was not defined, while the number of individuals who had been exposed to combat was not revealed.

Several studies of Israeli veterans of the 1982 war with Lebanon have also been published. Solomon and Mikulincer (1987) reported that soldiers with PTSD claimed to have more physical health problems than soldiers who had never been diagnosed with this condition, while Shalev, Bleich, and Ursano (1990) considered both self-reported health measures and medically diagnosed conditions. This latter study found that veterans with PTSD reported significantly more health problems than those without PTSD; however medical examinations did not reveal any difference between the two groups, except with respect to smoking (individuals with PTSD smoked at a greater rate). The authors noted that the veterans with PTSD performed poorly on an effort test.
(walking on a treadmill) compared to those without PTSD. An issue with both of these studies was the relative youth of the participants: even in the Shalev et al (1990) study, the mean age of veterans was around 33 years of age. One might expect physical health problems such as cardiovascular disease to manifest at a much later stage in life, or at least after many years of experiencing chronic PTSD symptoms.

In contrast, Schnurr and Spiro (1999) examined an older cohort of male veterans from the Korean and Second World Wars (mean age: 65 years). They found that severity of PTSD symptoms had a direct impact on self-reported physical health. This relationship between PTSD and physical health was not mediated by either smoking or alcohol use. Smoking had a small but significant effect on veterans’ self-reported health. The authors attributed the lack of any apparent relationship between alcohol use and health outcomes to a shortcoming in the instrument they used to measure alcohol consumption (the CAGE, which is a measure of lifetime rather than current alcohol-related problems).

Wagner, Wolfe, Rotnitsky, Proctor, and Erickson (2000) undertook a longitudinal study of male and female Gulf War veterans (n = 2949) to examine the relationships between PTSD, gender, and self-reported health problems over a two year period. They found that the level of PTSD symptomatology as measured immediately after returning from the Gulf War was predictive of self-reported health problems two years later, controlling for demographic variables, degree of combat exposure, and initial levels of health problems. Gender did not have a significant impact on the relationship between PTSD and health outcomes, although the number of women in this sample was relatively small (n = 193).

2.3.4 Summary: Trauma, PTSD, and health outcomes in veterans

The research reviewed above provides support for a direct relationship between PTSD and self-reported health outcomes in veterans, and emerging evidence that PTSD may also have a significant impact on health over and above any somatisation. It appears from research undertaken by Schnurr, Wolfe, Boscarino, and their colleagues that it is
the symptomatology associated with PTSD (particularly the hyperarousal symptom cluster) that plays a mediating role in the relationship between trauma and ill health in veterans from various conflicts.

2.4 Health outcomes following non-combat trauma

Research has also focussed on the health of individuals exposed to trauma unrelated to combat. It is important to consider whether the evidence of poor health reported above in veterans is generalisable to survivors of other types of trauma. The nature of different traumatic events, in addition to whether the trauma is an isolated incident or ongoing (as in domestic violence or torture) may lead to distinctive PTSD symptomatology, and could therefore impact on health differently. It is also the case that veterans prior to recruitment into the military were screened closely for any health conditions, and therefore may not be directly comparable to a community sample. In addition, the vast majority of veterans are male, a factor which again reduces the generalizability of any results to the rest of the community.

2.4.1 Health following physical or sexual assault

Victims of sexual assault (the majority of whom are female) often suffer profound psychological and physical trauma as a result. One study examining marital rape reported that victims suffered injuries such as anal or vaginal injuries, bladder infections, miscarriages or stillbirths, infertility, and sexually transmitted diseases (Campbell and Alford, 1989). The rates of PTSD arising from sexual assault are also high: Resnick et al (1993) reported a lifetime prevalence rate of 32%, while Breslau, Peterson, Poisson, Schultz, and Lucia (2004) found that 49% of rape victims in their community sample met current PTSD criteria. One might expect sexual assault victims to report poorer overall health than comparable community samples; the question that arises is whether those with PTSD report worse health outcomes than traumatised individuals without this disorder.
A number of retrospective studies have examined the impact of physical or sexual assault on the health of victims. Koss, Koss, and Woodruff (1991), in a comprehensive study of 316 women who had been “criminally victimised”, reported that—compared with 74 female non-victims—the victimised women reported greater general distress, made more visits to a doctor in the year following the trauma, and had significantly higher outpatient costs. A study of 234 “battered” women (who had been or still were in violent marital relationships) also found high levels of depression, persistent headaches, back and limb problems, and stomach complaints in this group; additionally the women reported that these health problems were worse during and to some extent following an abusive relationship, compared with health problems beforehand (Follingstad, Brennan, Hause, Polek, and Rutledge, 1991). A major limitation of this study—in addition to the fact that PTSD was not considered as a variable—was that a control group was not used.

Similarly, Leserman, Drossman, Li, Toomey, Nachman, and Glogau (1996) examined the relationship between reported health problems and sexual or physical abuse in a sample of 239 female patients in a gastroenterology clinic. This study considered a number of variables, including the invasiveness of the abuse, the age at which it first occurred, and the differential contributions of sexual and physical abuse. Leserman et al (1996) found that a history of self-reported sexual abuse was associated with greater pain, non-gastrointestinal somatic symptoms, days in bed due to illness, and worse functional status when compared with women with no sexual abuse history. Reports of past physical abuse were also associated with increased physical pain, somatic symptoms, and poorer functional status. Importantly, however, those individuals reporting a history of rape (as opposed to other forms of sexual or physical abuse) appeared to be the most disabled in terms of their physical health. Likewise, Walker, Katon, Neraas, Jemelka, and Massoth (1992) examined 22 women with a history of chronic pelvic pain, in addition to 21 women attending the same health clinic with no such history of pelvic pain. The women with pelvic pain were significantly more likely to report childhood sexual abuse, higher levels of dissociation, and greater somatic distress.
A potential limitation of these studies is their retrospective nature, as the authors asked women presenting to health clinics or refuges if they had ever experienced physical or sexual abuse or pain, potentially introducing some recall bias. In addition, PTSD was not considered independently as a variable. Given the evidence reported earlier documenting a direct relationship between PTSD and health outcomes, this is a significant limitation.

Golding and her colleagues have also explored the relationship between sexual assault and perceptions of poor health. Golding (1994) reported evidence that women who provided a history of sexual assault claimed to have higher rates of diabetes, arthritis, and physical disabilities than non-victims. This issue was addressed more broadly in a review of data from seven population surveys looking at sexual assault and health perceptions in 10,000 adults and adolescents (Golding, Cooper, and George, 1997). While the authors examined the contribution of depression to perceptions of poor health in sexual assault victims (finding that it did not account completely for any association between these variables) they did not consider the impact of PTSD, as insufficient information was available to include this in their meta-analysis. An interesting finding of this study was that individuals reporting more than one sexual assault were significantly more likely to report poorer health than those who had sustained a single assault. However, repeated assault by the same offender was not significantly related to poorer subjective health. Sexual assault that occurred during childhood was also not significantly related to poorer perceptions of health.

A history of sexual assault was also considered in a study examining the health of 219 female veterans (Stein, Lang, Laffaye, et al 2004). The authors found that women with a sexual assault history reported more physical symptoms (including headache, chest pain, and fatigue) than those without such an assault history. The degree of somatization and number of sick days were also higher in the sexual assault group. Unfortunately neither of these studies included PTSD as an independent variable of interest.
2.4.1.1 Health outcomes for assault victims with PTSD

As noted in Section 2.3, there is compelling evidence that PTSD is a critical mediating variable in the relationship between trauma and reports of poor health in veterans (Wolfe et al., 1994; Boscarino, 1997; Schnurr and Spiro, 1999; Wagner et al., 2000). One might therefore expect a similar relationship to exist between PTSD arising from assault and negative health outcomes, notwithstanding the different nature of the trauma (assault as opposed to combat) and some demographic differences between veterans and assault victims. Examining trauma other than combat in a group of female veterans might be a useful initial step in broadening research into the relationship between PTSD and health.

One such study of self-reported health problems in a sample of female veterans (mean age 45.9 years) reported that PTSD arising from various traumatic events was a significant intervening variable in the relationship between trauma and health (Dobie, Kivlahan, Maynard et al., 2004). Although the questionnaire utilised by the authors to assess PTSD – the PTSD Checklist – Civilian Version (PCL-C) – did not identify the nature of the traumatic event, the authors assumed, on the basis of prior evidence, that the most common traumas were physical or sexual assaults. They found that the women with PTSD (as opposed to those without this disorder) reported higher levels of somatic distress, and were more likely to report higher rates of obesity, fibromyalgia, irritable bowel syndrome, emphysema, and sexually transmitted diseases. Limitations of this study included the failure to control for smoking, substance abuse, or comorbid depression. In addition, the disparate list of health problems outlined might suggest the involvement of one or more mediating variables, such as adverse lifestyle and socioeconomic factors.

Resnick, Acierno, and Kilpatrick (1997) reviewed existing data relating to the prevalence of rape, physical assault, and health problems in women. They identified several potential mechanisms that might increase women’s risk of health problems after a violent assault, including injuries suffered at the time, increased stress, and mental health problems. The authors postulated that the latter two variables would lead to impaired immune system functioning, increased health risk behaviours, and
inappropriate health care utilisation, all of which also have a direct impact on the risk of negative health outcomes. They particularly noted the potential impact of PTSD and other mental health problems such as depression and panic on health through mechanisms such as substance abuse, self-neglect of health, risky health behaviours, persistent physiological arousal, and misinterpretation of psychological distress as a sign of physical illness. A number of studies have been undertaken to explore in more detail the links between PTSD, physical or sexual assault, trauma, and ill health, particularly in relation to the physiological arousal hypothesis.

Two recent studies by Clum and her colleagues (Clum, Calhoun, and Kimerling, 2000; Clum, Nishith, and Resick, 2001) examined the self-reported physical health of female sexual assault victims suffering from depression and/or PTSD. The first of these studies concluded that both PTSD and depressive symptoms had a prolonged negative impact on self-reported general health in female assault victims. However, even after controlling for depression, PTSD symptoms were a significant predictor of health symptoms in this study. In the second study, Clum et al (2001) specifically examined the role of trauma-related sleep disturbance in considering health outcomes of female rape victims with PTSD. The authors reviewed evidence suggesting that sleep disturbance is linked to alterations in immune function. Using hierarchical multiple regression, Clum et al (2001) reported that PTSD symptoms accounted for the larger portion of the variance in self-reported health symptoms (headaches, stomach upsets, back pain, etc), followed by depressive symptoms and finally PTSD-related sleep disturbance. The analysis also suggested that the hyperarousal symptoms of PTSD – even with sleep disturbance removed – remained the strongest predictor of any of the PTSD symptom clusters of poor physical health.

PTSD and the role of sleep disturbance in predicting self-reported health were also studied in a sample of 741 police officers who had been exposed to a variety of traumatic incidents, with the majority appearing to be assault-related (Mohr, Vedantham, Neylan et al, 2003). The authors reported that, while PTSD symptoms were
significantly associated with measures of health functioning and somatisation, the relationship was mediated by subjective sleep quality.

Woods and Wineman (2004) examined the impact of PTSD symptom clusters on the self-reported health of 50 women who had been abused in the past by ex-partners or spouses. Those women who met the diagnostic criteria for PTSD reported significantly more muscle weakness and trembling than the sample without PTSD. While a PTSD diagnosis was not associated with a perception of poor physical health (although a trend existed, with p = .055), the hyperarousal and avoidance symptom clusters were significantly associated with physical health symptoms. Similarly, Zoellner, Goodwin, and Foa (2000) examined 76 treatment-seeking female sexual assault victims with a primary diagnosis of PTSD to test a number of hypotheses, one of which predicted that the hyperarousal component of PTSD symptoms (rather than the avoidance and reexperiencing clusters) was related to self-reported physical symptoms beyond the effects of general psychological disturbance, general life stressors, and assault-related injuries. Results of this study indicated that severity of PTSD predicted self-reported physical symptoms above and beyond anger, general life stressors, and depression. Contrary to the findings of Woods and Wineman (2004) and Clum et al (2001), the authors found that severity of re-experiencing symptoms (but not hyperarousal or avoidance) predicted physical symptoms. Zoellner et al (2000) theorised that this may be due to severe re-experiencing symptoms (eg flashbacks, nightmares) prompting strong physiological reactions such as a rapid heart rate, trembling, shaking, and sweating, which may in turn be related to an underlying biological susceptibility to disease or a heightened awareness of physical symptoms.

2.4.1.2 Summary: PTSD and health outcomes following physical or sexual assault

Research into the health of assault victims is attracting increasing attention and interest. Evidence reviewed above points to a demonstrable link between PTSD symptomatology and self-reported health problems, with mixed evidence regarding the impact of particular PTSD symptom clusters on health. Unfortunately there appears to be a dearth
of research investigating the relationship between assault, PTSD, and objectively diagnosed medical problems. However, one would expect the relationship to mirror that of veterans, if Friedman and Schnurr's (1995) view that the stress associated with the symptoms of PTSD is the “prime mediating variable” in the development of health problems following trauma is correct. Further evidence has been obtained through examining victims of other forms of trauma, although this research has been more limited.

2.4.2 Other traumatic events and health outcomes

2.4.2.1 Natural disasters and other violent events

Ironson and her colleagues have undertaken several prospective studies into the health of people exposed to Hurricane Andrew in the USA. Lutgendorf, Antoni and Ironson, et al (1995) reported that patients suffering from Chronic Fatigue Syndrome (CFS) who were exposed to the worst effects of Hurricane Andrew suffered greater physician-reported relapses than those CFS sufferers living in areas less affected by the hurricane. Ironson, Wynings, Schneiderman, et al (1997) extended this research by looking specifically at immune function in hurricane survivors with PTSD. The authors interviewed 180 voluntary participants and drew blood from them one to four months after the hurricane. They reported that a diagnosis of PTSD (using DSM-III-R criteria) was significantly associated with lower natural killer cell cytotoxicity (NKCC) and higher white blood cell counts, particularly if the individuals came from areas that sustained high levels of damage from the hurricane. Ironson et al (1997) also explored whether the PTSD symptom clusters were related to these measures of immune functioning. These results were somewhat complicated: the reexperiencing cluster was significantly associated with lower NKCC, and higher white blood cell counts (high damage group only), while the arousal cluster had a significant association with increased white blood cell counts in the entire sample and the high damage group. Natural killer cell (NKC) activity was also examined in 155 male survivors of a Japanese earthquake: survivors assessed as meeting DSM-III-R criteria for PTSD were found to have reduced NKC activity.
compared to those survivors who did not meet criteria for this disorder (Inoue-Sakurai et al, 2000). “Poor lifestyles” (including behaviours such as smoking, alcohol consumption, lack of exercise, sleeping problems, poor nutrition, subjective mental stress, and not eating breakfast) were also significantly associated with lower NKC activity.

Escobar, Canino, Rubio-Stipec, and Bravo (1992) examined the aftermath of another natural disaster (flash floods and mudslides in Puerto Rico). They found that individuals who were exposed to this disaster reported new gastrointestinal and pseudo-neurological symptoms (such as amnesia, paralysis, fainting, and double-vision) at a greater rate than people who were not exposed to the disaster. However, they did not assess survivors for PTSD and therefore this was not included in their analysis.

Finally, in terms of natural disasters, McFarlane, Atchison, Rafalowicz, and Papay (1994) undertook a longitudinal study of 140 volunteer firefighters who had been exposed to bushfires devastating south-eastern Australia in 1983. 50% met criteria for PTSD. The PTSD group reported statistically higher rates of cardiovascular, respiratory, musculoskeletal, and neurological symptoms than the non-PTSD group. McFarlane et al (1994) established that PTSD sufferers who complained of physical symptoms were more likely to have a comorbid diagnosis of major depression. Examinations of these firefighters by general practitioners failed to find any evidence of more medical problems among the PTSD group as compared with the non-PTSD group. However, the GPs also failed to diagnose PTSD in the majority of firefighters who met the criteria for this disorder. McFarlane et al (1994) attributed the greater self-reporting of physical symptoms in firefighters with PTSD to chronic hyperarousal, misinterpretation of somatic sensations, and the impact of comorbid depression.

Man-made violent events such as terrorist attacks or cataclysmic accidents have received less attention in the PTSD/health literature. However, one recent study followed up survivors of an explosion (attributed to a terrorist attack) in a Lebanese church in 1994 one year after the event, along with family members and neighbours (Farhood and
Noureddine, 2003). Individuals with PTSD reported more doctors’ visits than those without PTSD, although the authors noted that severity of injuries sustained in the explosion was a strong risk factor for the development of PTSD. Hence, the increase in health utilisation among the PTSD group might at least partly be explained by physical injuries related to the church explosion.

2.4.2.2 Motor vehicle accidents

Mayou, Tyndel, and Bryant (1997) undertook research on the physical and psychological health of 111 motor vehicle accident victims (mean age: 31 years) as part of a prospective study five years after their accident. They reported that those individuals with PTSD appeared to have a significantly greater number of physical health problems than those without PTSD, although the authors did not evaluate specific types of health problems, some of which may have been directly related to injuries received in the accidents.

2.4.2.3 Torture

Van Velsen et al (1996), in a study of predominantly Kurdish asylum seekers (the vast majority of whom reported past torture), found a strong relationship between ‘loss of health’ and PTSD symptoms. Interestingly, Van Velsen et al (1996) also reported that different types of torture were associated with different symptoms of PTSD. Sexual torture led primarily to symptoms of avoidance, for instance, while other forms of torture resulted mainly in intrusive thoughts. A potential problem with this study, however, was the fact that the authors were also preparing official medical/psychiatric reports for the participants as part of their application for political asylum, which might give an impression of conflict of interest or experimenter bias. However the authors denied the existence of any such bias. Allden, Poole, Chantavanich, et al (1996), in a study of 104 Burmese political dissidents hiding in Thailand, found that 54% rated their health as fair or poor, while 27% stated they had medical problems diagnosed by a physician. 23% of this group had elevated posttraumatic stress symptoms, with
avoidance and increased arousal most strongly associated with cumulative trauma. The majority of individuals in this group reported experiencing traumatic events such as interrogation (89%), imprisonment (78%), and in some cases torture (38%). Unfortunately the authors did not consider the differential impact of PTSD on self-reported health in this sample.

### 2.4.3 Community samples: non-specific trauma

A number of studies have investigated the link between trauma, PTSD, and health in community samples without necessarily seeking information about the nature of the traumatic event. Such research can be useful in examining the extent to which PTSD arising from a variety of traumatic events contributes to poor health. Breslau and Davis (1992) found that young people who had met the criteria for PTSD for over one year had more self-reported medical conditions (such as arthritis, bronchitis, migraines, and gynaecological complaints) than people with non-chronic PTSD. Participants reported a range of traumatic events; however war-related combat was not identified by any of them as a traumatic stressor that they had experienced. Similarly, Andreski, Chilcoat, and Breslau (1998) undertook a prospective study over five years of 1007 young adults, examining the relationship between PTSD and somatic complaints. They found that individuals diagnosed with PTSD were over three times more likely to have a history of somatisation symptoms than the non-PTSD group. A limitation of this study was the authors’ focus from the outset on ‘somatisation’ (using abridged DSM-III-R criteria for somatisation disorder) rather than a broader consideration of self-reported health problems.

A cross-sectional study examining the health of Canadian bus drivers explored relationships between lifetime PTSD, trauma exposure, and current health problems (Vedantham, Brunet, Boyer, Weiss, Metzler, and Marmar, 2001). The authors reported that participants exposed to a traumatic event who developed PTSD were more likely to report poorer health, a higher number of health complaints, and show increased health utilization than the exposed participants who did not develop PTSD. Interestingly, the
study included a third group (bus drivers who did not report exposure to any traumatic stressor); results indicated that this group had a health profile similar to that of the trauma exposed (non-PTSD) individuals. This provides further support for the theory that it is PTSD, rather than exposure to trauma, that is associated with poorer self-reported health and increased health service utilization.

Ullman and Siegel (1996) also considered a variety of traumatic events in reporting that traumatised adults in a community sample had poorer perceptions of their physical health, while also reporting chronic limitations in their physical functioning compared with a control group who did not report any history of trauma. The traumatised group were also five times more likely to report chronic medical conditions. They did not consider PTSD as a separate variable of interest in the relationship between trauma and poor health.

Finally, Roy-Byrne, Smith, Goldberg, Afari, and Buchwald (2004) explored the relationship between trauma, poor health, and PTSD from a reverse perspective. They examined 571 patients suffering from fibromyalgia and Chronic Fatigue Syndrome (CFS) for symptoms of PTSD and major depression. They found a significant relationship between certain fibromyalgia symptoms (tender points and diffuse pain) and lifetime PTSD; however this association lost significance once symptoms of major depression were removed from the regression equation. The authors' rationale for considering an association between PTSD and some fibromyalgia components stemmed from evidence reviewed in their paper that both regulation of pain and the emotional dysregulation of PTSD may be mediated by areas such as the anterior cingulate cortex. Roy-Byrne et al (2004) postulated that it may follow that a bi-directional relationship might exist between pain and PTSD, with each exacerbating the other.

2.5 Summary: PTSD, trauma, and health outcomes in various populations

In the introduction to this chapter a number of possible theories that might explain any association between PTSD and poor health were outlined. These included a possible
direct pathway from PTSD to poor health; an explanation taking into account certain comorbid conditions (particularly depression) and health-compromising behaviours often associated with PTSD (smoking, substance abuse, alcohol consumption, for instance); the fact that individuals with PTSD might have altered perceptions of their health status in the form of somatic preoccupations or somatisation; and the impact of any injuries sustained or exposure to toxic substances at the time of the traumatic event(s). The role of anger and sleep deprivation – both common features of PTSD hyperarousal – in any reports of ill health was also raised for consideration.

In relation to the first of these possible theories (a direct relationship between PTSD and poor health), it appears from the evidence reviewed in this paper that a significant association exists between trauma and poor health, with PTSD the critical mediating variable. Extensive research undertaken with veterans has provided evidence of both poorer self-reported health and more diagnosable medical conditions in those with lifetime PTSD, as compared with veterans without this condition (eg Wolfe et al, 1994; Kimerling et al, 2000; Litz et al, 1992; Boscarino, 1997; Boscarino and Chang, 1999b, and Beckham et al, 1998). More limited research undertaken with victims of other types of trauma also supports a significant relationship between PTSD and poorer self-reported health, with some evidence that immune function may be suppressed with lower natural killer cell levels and higher overall white blood cell counts found in survivors of natural disasters suffering from PTSD (Ironson et al, 1997; Inoue-Sakurai et al, 2000). Hence non-combat trauma victims may also be susceptible to disease and diagnosable medical conditions which may emerge in time. Further research is clearly needed on this front; however it is apparent that PTSD is associated with more than somatisation alone. Additionally, the negative impact upon health appears relatively consistent across a variety of different trauma types, suggesting that any injuries or medical problems sustained as a result of the trauma (as may be the case in combat or sexual assault) are not sufficient to explain later reports of poor health, and would impact upon both PTSD and non-PTSD groups.
It also appears that the relationship between PTSD and poor health is maintained when comorbid conditions or behaviours such as depression, smoking, or substance abuse are controlled for (e.g., Zatzick et al., 1997; Boscarino, 1997; Schnurr and Spiro, 1999; Clum et al., 2000; Clum et al., 2001; Zoellner et al., 2000). Consequently, negative health outcomes in trauma victims with PTSD cannot be explained primarily by behaviours such as smoking or alcohol abuse, or attributed solely to the negative impact of depression on health (such as coronary heart disease).

One must conclude from the majority of evidence reviewed in this chapter that PTSD arising from a variety of different traumatic events independently contributes to poor health and – according to recent evidence – impaired immune functioning. The means by which this might occur could involve any combination of increased physiological arousal as measured by higher baseline heart rates (e.g., Litz et al., 1992), fatigue and disrupted sleep (see for instance Falger et al., 1992; Clum et al., 2001; Mohr et al., 2003, and Van Velsen et al., 1996), altered neurochemical and immunological responses (e.g., Boscarino and Chang, 1999a; Ironson et al., 1997; Inoue-Sakurai et al., 2000), and self-neglect of health. Other factors such as increased levels of anger and hostility in individuals with PTSD may increase vulnerability to ill health, particularly cardiovascular disease (Williams, 1995). Research assessing the contribution of PTSD symptom clusters to poor self-reported health has tended to find a relationship between the hyperarousal cluster (which includes sleep problems and irritability among its symptoms) and physical health problems (Clum et al., 2001; Kimerling et al., 2000; Woods and Wineman, 2004; although see also Zoellner et al., 2000, for contrary findings). One thing is becoming clearer: the relationship between PTSD and poor health cannot be explained purely by somatisation or symptom over-reporting.

2.6 Hypotheses

The evidence reviewed in this chapter raises a number of issues relating to PTSD and physical health that have potential for further research, including the desirability for fairly objective measures of health and the impact individual PTSD symptom clusters
may have on physical health. Few studies have incorporated multiple measures of physical health; it would be useful therefore to obtain information about health outcomes through a number of avenues. These might include identification of specific symptoms, rating of general health before and after the trauma(s), and reporting the diagnosis of any medical problems. While it would be preferable to have medical practitioners provide objective assessments of the latter, this is an expensive and time-consuming process. Asking participants to provide details of medically diagnosed conditions according to a pre-determined list may be an acceptable compromise, although still subject to the vagaries of self-report. The impact that counselling might have on the self-reported health of individuals with PTSD would also be a useful area of research, as there is some evidence that even relatively simple interventions such as relaxation training can have positive impacts on white blood cell counts and minor wound healing (Kiecolt-Glaser et al, 2002). Based upon these observations, a study involving victims of crime in the ACT (see Chapters 3 - 5) was instigated with the following hypotheses:

1. That victims of crime with PTSD (controlling for depression, age, smoking, and other relevant factors) would report a greater number and frequency of negative health symptoms, compared with participants without PTSD.

2. That participants with PTSD would report significantly worse appraisals of their overall health since the traumatic event(s), compared with the non-PTSD group.

3. That participants with PTSD would report a greater number of medical problems diagnosed by a doctor, compared with participants without PTSD.

4. The hyperarousal symptom cluster of PTSD would be a significant predictor of lower (ie worse) health scores, given evidence that disrupted sleep and anger/irritability (components of the hyperarousal cluster) are significantly associated with abnormal immune system functioning and/or poorer overall
health

5. That those individuals with PTSD would report a marginal reduction in negative health symptoms after counselling, compared with those who did not receive counselling, while those not meeting criteria for PTSD would show little difference regardless of whether counselling was received or not.

6. Those participants diagnosed with PTSD in the first study who then received counselling by the time of the second study would report a reduction in PTSD symptom severity, as assessed using the Posttraumatic Diagnostic Scale (PDS).
Chapter Three: Methodology

3.1 Introduction

This chapter provides a description of the methodology used in this research, which consists of a longitudinal study conducted over a five to seven month period. Each of the two waves are reported separately, and will simply be referred to as Study One and Study Two, or the ‘first’ and ‘second’ part of the study. Details of participants and their recruitment, measures used to collect data, and the procedures followed in this research are outlined in this chapter.

3.2 Participants

Participants were individuals in the Australian Capital Territory (Canberra) who had experienced or witnessed a traumatic crime. It was decided to focus on victims of crime for the purposes of this study for a number of reasons. Firstly, it was expected that a significant proportion of this sample would meet criteria for PTSD, given results of previous research showing a high PTSD prevalence in assault victims (eg Kessler et al, 1995; Breslau et al, 1991). As it was anticipated that the overall sample size for this study may be relatively low, it was felt necessary to ensure that a reasonable number presented with significant PTSD symptomatology for the purposes of meaningful statistical analysis. Victims of crime were preferred over motor vehicle accident victims, who have relatively low rates of PTSD (see for example Mayou et al, 1997), and possible issues with compensation/insurance, and combat veterans, who have been surveyed/studied relatively extensively, and therefore may be less likely to volunteer for further research. Finally, victims of crime in the ACT area are entitled to certain free services and counselling, initially offered through two agencies (see Section 3.4 - ‘Procedure’ - for further details), and hence were relatively easy to locate.

The study sample consisted of three groups. One group included victims of crime who had presented for an intake interview or assessment session at one of two services set up
in Canberra to counsel or otherwise assist this population following exposure to a crime. Due to the relatively small sample size of this initial group (n=47), further participants were sought. Past or existing clients of these two services who were not currently seeking treatment formed a second group (n=44), while the third group comprised non-treatment seeking university students who responded to local noticeboard advertisements at the Australian National University asking for participants in a research project into victims of crime (n=6). In total, 99 victims of crime participated in the first part of the study. All participants were provided with written information about the study and gave written consent to participate in both the initial and latter parts of the research. The mean age of participants in Study One was 35.9 years (SD = 11.14 years, skewness = .302, kurtosis = -.421). All participants were followed up for the second part of the study, with 59 ultimately completing and returning questionnaires. The mean age of participants in Study Two was 37.8 years (SD = 11.6 years, skewness = .203, kurtosis = -.373).

3.3 Measures

3.3.1 Background

Data relating to health outcomes following exposure to trauma is generally collected in one of four ways:

- medical diagnoses of presenting problems
- self-report of particular symptoms and/or overall perception of health status
- use of medical services (such as number of visits to a doctor in a particular time period)
- review of mortality figures

The first of these data collection techniques is often considered to be the "gold standard" in empirical research, but is obviously time-consuming and expensive, thus limiting its use. Insufficient resources were available to utilize this mode of data collection in the
current studies. Self-reports of symptoms are inexpensive and relatively easy to obtain; however they cannot be objectively tested, and may be influenced by the individual’s psychological state. Some studies have demonstrated that medical examinations have failed to find any health problems in trauma survivors who report a myriad of health complaints (eg Centers for Disease Control, 1988; Shalev, Bleich, and Ursano, 1990). However, Friedman and Schnurr (1995) point out that self-reports can be a valid indicator of a person’s current health status, and are excellent predictors of mortality and disease type. Beckham, Moore, Feldman et al (1998) also note that, while use of general disease categories based on self-report in survey-based research may lead to some over-reporting of health problems, this was not the case for self-reporting of specific medical conditions. For this reason, the health questionnaires utilised in the current studies (described in Section 3.3.2.3) asked specific questions about both self-reported health symptoms, overall perception of health, and particular medical conditions that had been diagnosed by a medical practitioner.

Some researchers have also attempted to investigate the relationship between trauma and ill-health by examining it from the reverse perspective. For instance, Walker, Katon, Neraas, Jemelka and Massoth (1992) studied women with a history of chronic pelvic pain attending a health clinic and found a substantially greater likelihood that these women had a history of childhood sexual abuse compared with other clinic patients without pelvic pain.

Increased use of medical services may provide some indication of ill-health in trauma victims, although once again it is impossible to state definitively that high users have poorer health without knowing whether increased use relates to somatisation or diagnosed complaints. Hence, information of this nature was not sought in the current studies (the prospect of a complicated and time-consuming process to obtain such third-party data also mitigated against this).

Finally, mortality studies may offer some insight into a relationship between trauma, PTSD, and the ultimate consequence of ill health (such as death following heart disease).
However, this would require access to medical records of the deceased individuals, which may involve difficulties from a privacy perspective. Hence, very few mortality studies have looked at outcomes for trauma victims, with the exception of two that examined the mortality of Vietnam veterans (Centers for Disease Control, 1987, and Bullman and Kang, 1994).

3.3.2 Description of Measures

Participants were provided with three questionnaires to complete in each study for the purpose of this research (procedural details are provided in Section 3.4, while copies of the questionnaires are provided in the appendices).

3.3.2.1 Trauma and PTSD Measures

Exposure to direct or indirect trauma and PTSD symptoms were assessed using the Foa Posttraumatic Diagnostic Scale (PDS) (Foa, Cashman, Jaycox, & Perry, 1997). The PDS is a brief screening and diagnostic instrument used to identify the types of traumatic events the person may have been exposed to, while also assessing the severity of PTSD symptoms using questions based on the DSM-IV (1994) criteria. In addition, the PDS identifies the traumatic event that had the most impact on the person ("that bothers you the most") for those participants who identify more than one type of traumatic event. It also gathers information on how long ago the traumatic event occurred, how long the symptoms have been noticeable for, and to what extent they are interfering in various aspects of the person’s life. Responses to the DSM-IV associated questions are on a Likert type scale with answers of 0 (Not at all or only one time), 1 (Once a week or less/once in a while), 2 (2 to 4 times a week/half the time), and 3 (5 or more times a week/ almost always). The PDS has demonstrated good reliability and validity (Foa et al, 1997). Cronbach’s alpha in the first study was .92, indicating high internal consistency for questions 22 – 38 (the DSM-IV items). A PTSD severity score was calculated by summing the responses to the 17 DSM-IV items (score range 0 to 51). Additionally, participants were categorised as meeting DSM-IV criteria if they met
Criterion A (established by answers to questions 16-22, which assessed the nature of the individual's response to the traumatic event), indicated impairment in one or more area of their life (questions 41-49), and met each of the reexperiencing, avoidance, and hyperarousal clusters (questions 22–26, 27–33, and 34–38, respectively). The individual had to provide ratings greater than or equal to 2 for the required numbers of symptoms in each cluster in order to meet diagnostic cutoffs. Although this does not reflect a clinical diagnosis of PTSD, this descriptive method has been effectively utilised in previous research (Bernat, Ronfeldt, Calhoun, & Arias, 1998; Clum, et al., 2000; La Greca, Silverman, Vernberg, & Prinstein, 1996). A separate group of individuals who met criteria for partial PTSD was also established; the criteria was essentially the same as that described for the PTSD group, with the exception that the person fell short of meeting the full diagnostic criteria by one criterion, while still positively endorsing many of the symptoms of that particular criterion with at least one response greater than or equal to 2.

The PDS was chosen to assess PTSD symptoms because of its relative brevity, its good reliability and validity (Foa et al., 1997), and the fact that the questions directly mirror the DSM-IV PTSD symptoms, allowing both a provisional 'diagnosis' of PTSD and the calculation of a symptom severity score. The former was necessary for both studies as PTSD diagnostic status was an important grouping variable. In other words, it was necessary to establish PTSD status to test the hypothesis that victims of crime with PTSD would report worse health than those without PTSD. Similarly, it was necessary to establish severity of PTSD to evaluate whether counselling was beneficial in reducing PTSD symptomatology as well as health problems. While clinical interviews such as the PTSD Symptom Scale – Interview (PSS-I; Foa, Riggs, Dancu, and Rothbaum, 1993), the PTSD Interview (PTSD-I; Watson, Juba, Manifold, Kucala, and Anderson, 1991), the Diagnostic Interview Schedule (DIS, Robins and Helzer, 1985), the Clinician Administered PTSD Scale (CAPS, Blake, Weathers, Nagy et al., 1990), and the Structured Clinical Interview for the DSM-IV (SCID, First, Spitzer, Gibbon, and Williams, 1996) are often regarded as the most rigorous assessment instruments available for diagnosing PTSD, they suffer from two major drawbacks. Firstly, they are
relatively time-consuming and require a trained clinician to administer them to the client (resources were not available in this study to permit this). Secondly, the majority of psychometric studies used to assess their reliability and validity relied on combat veterans, raising questions on how valid they might be with other trauma groups (Foa et al, 1997).

3.3.2.2 Beck Depression Inventory, 2nd edition (BDI-II, Beck and Steer, 1987).

The Beck Depression Inventory is a 21-item, forced choice self-report scale that assesses cognitive and vegetative symptoms of depression (total score range 0 to 63). It is a reliable and valid measure of depressive symptoms (Beck, Steer, and Garbin, 1988; Beck and Steer, 1987) that categorises severity of depressive features using the following cut-off scores:

0 – 9: little or no depression
10 – 18: mild to moderate depression
19 – 29: moderate to severe depression
30+: severe depression

Cronbach’s alpha for the first study was .94, indicating high internal consistency among the BDI-II items.

The BDI-II was chosen as a measure of depression because of its good reliability and validity (Beck et al, 1988, and Beck and Steer, 1987), and relative ease of use. As depression was considered to be an important covariate for both studies, it was imperative that this was measured accurately. This measure allowed both continuous and dichotomous depression variables to be created, where a cut-off score of 19 or above suggested the presence of depression.
3.3.2.3 Health Questionnaire Parts 1 and 2

A self-report measure was designed specifically for this study to assess perceptions of health and somatic symptoms, in addition to drug, alcohol, and medication usage. Slightly modified forms of the same questionnaire were used for the second study. It was felt that existing standardized self-report measures of general health and well-being, such as the General Health Questionnaire (Goldberg and Williams, 1988) and the SF-36 (Ware, Snow, Kosinski et al, 1993) would not obtain the specific information sought about health problems and medical conditions, as well as substance use, and counselling history. Initial feedback from clinicians working with victims of crime indicated that the questionnaire appeared easy to understand and complete, and should not present any difficulties to the majority of participants.

The Health Questionnaire Part 1 required participants initially to tick any of 10 medical conditions that have been diagnosed by a medical doctor ('medical problems endorsed': heart disease, angina, high blood pressure, stomach/duodenal ulcers, diabetes, cancer, gynaecological problems, thyroid problems, rheumatism/arthritis, liver problems). Respondents were also asked to indicate approximately when these conditions were diagnosed, as applicable. Responses were scored as 0 = no and 1 = yes for each condition, and participants were ultimately identified for statistical purposes as either reporting at least one diagnosed medical condition (1) or not (0).

A separate question assessed 12 self-reported health symptoms (unexplained thirst, blurred vision, indigestion, headaches, nausea/stomach pain, chest tightness or pain, muscle weakness, pelvic pain, shortness of breath, frequent diarrhoea, coordination problems, and frequent urination). It also included an additional ‘other’ item for participants who wished to specify symptoms not included in this list (it should be noted that ‘fatigue’, if identified, was not included as an ‘other’ symptom to avoid duplicating the fatigue items in the BDI-II and PDS). Participants were asked to assess the frequency of each symptom occurring using a 6 point Likert-type scale (from 1 = occurs all the time through to 6 = never/not at all). As with the question about medical
conditions, respondents were asked to indicate approximately when they first noticed the problem. Specific medical conditions and health symptoms were scored as a 0 (‘no’) if the diagnosis or time that the symptom was first noticed preceded the onset of any traumatic event listed in the PDS. Health symptom responses were summed to provide a total health symptoms score (health score, range 0 – 78, with higher scores indicating fewer health problems). Cronbach’s alpha was .84 for these items, indicating good internal consistency and reliability.

The specific health symptoms and medical conditions included in the questionnaire were chosen on the basis of previous research in which some of these had been associated with either exposure to trauma and/or the presence of PTSD (Kimerling et al, 2000; Litz et al, 1992; White and Faustman, 1989; Boscarino, 1997; Falger et al, 1992; Hankin et al, 1996; Follingstad et al, 1991; Walker et al, 1992; Golding, 1994; Stein et al, 2004; Clum et al, 2001; Escobar et al, 1992; and Breslau and Davis, 1992).

Participants were also asked to rate their overall physical health prior to the crime or traumatic event that they experienced or witnessed on a 5 point Likert-type scale (from 1 = excellent through to 5 = very poor). They were then asked to rate their health since the traumatic event using the same rating scale. A difference score indicating change in perceptions of health from before to after the trauma was then calculated. While this measure may be highly subjective, with the perception of health prior to the event heavily influenced by the individual’s current mental state and health, it was felt that this would still be a useful ‘back-up’, or adjunct measure, to the other health outcome variables created from responses to this questionnaire (health score, and medical problems endorsed).

Other questions in the Health Questionnaire Part 1 cover whether a diagnosis (medical or otherwise) has been provided by a health professional for any symptoms or health problems, whether the person is using any medication (particularly antidepressant medication), drugs, or alcohol (current or past use), as well as questions relating to the quantity and duration of any substance use (including past or present cigarette smoking).
Respondents were also asked whether they are or have been receiving any counselling for symptoms relating to the traumatic event. Due to small cell sizes, some of these variables were collapsed into dichotomous categories. Past or present counselling was coded as 0 = no, 1 = yes, while cigarette smoking was coded as 0 = no if the person had never smoked or had only smoked lightly, and 1 = yes if the respondent reported current moderate to heavy tobacco use or recent past moderate to heavy smoking of at least 12 months duration. Moderate to heavy use was defined as smoking at least 10 cigarettes daily. Similarly, use of various illicit drugs (marijuana, heroin, amphetamines, etc) was coded as 0 = no (never or past/present occasional use) and 1 = yes (current or past moderate to heavy use, defined as at least weekly use of the substance). Alcohol use was similarly coded as 0 = no (never or past/present light use) and 1 = yes (past or present moderate to heavy use). Moderate to heavy use was defined as more than 14 units of alcohol per week for women, and more than 28 units per week for men, levels considered hazardous or harmful in Australia (Pols and Hawks, 1992).

Finally, demographic data relating to gender and age were collected, in addition to a question about whether the person had been engaged in any legal proceedings relating to the incident.

The Health Questionnaire Part 2 (used in the second study) was essentially similar to the one used in the first study (Part 1), but excluded the question relating to medically diagnosed conditions. An identical question asking about health symptoms was included, and scored in the same way as for the Part 1 questionnaire. An additional question was included about perceived physical health since receiving counselling or therapy (if applicable). The same questions relating to drug, alcohol, tobacco, and medication use were included, and scored in the way described earlier for the Part 1 questionnaire. Questions regarding counselling and involvement in legal proceedings were asked once again, although the counselling questions were more detailed, seeking information about the number of sessions received to date in the past six months, and the profession, if known, of the counsellor (psychologist, social worker, other).
3.4 Procedure

Applications to two separate human research ethics committees had to be submitted prior to the recruitment of participants for both studies. A finalised application (also known as a research protocol) was lodged with the Australian National University's Human Research Ethics committee in January 2003. Following various amendments and requests for additional information from the Committee, approval for the study was granted in July 2003. Similarly, an application had to be made to the ACT Department of Health's Ethics committee, given the fact that some participants would be recruited with the assistance of the Victims' Services Scheme (VSS, see Box 1 in Section 3.4.1), who fall under this department's portfolio responsibilities. Approval for the research by this latter committee was granted in August 2003.

3.4.1 Study One

Participants were initially recruited through two organisations offering counselling and support to victims of crime in the ACT, the Victims’ Services Scheme (VSS) and the Victims of Crime Assistance League (VOCAL). A brief description of these agencies is provided in Box 1.

3.4.1.1 Victims’ Services Scheme (initial clients)

The VSS was approached in November 2002 by the researcher and asked to distribute questionnaires to victims of crime presenting for their intake assessment after experiencing or witnessing a crime (secondary victims of crime, eg parents or friends, are also eligible for VSS services; however they did not form part of this study due to the likelihood that they would not technically meet the DSM-IV PTSD criterion A). While many of the criminal events would have occurred recently, some individuals presenting for assessment wished to have counselling for a traumatic event occurring some years ago (eg childhood sexual abuse). Distribution of the questionnaires commenced in October 2003, shortly after final approval was granted by both Ethics
committees. The initial aim was to gather a sample of treatment-seeking individuals who had not previously sought or received counselling or therapy for the traumatic event. VSS staff were provided with training and information about the questionnaires and the procedures involved in offering these to clients. They were asked to exclude individuals who exhibited evidence of active psychosis or suicidal ideation/intent, or a gross and disabling personality disorder.

**Box 1: Assistance for Victims of Crime in the ACT**

Victims of crime in the ACT can apply for free counselling, therapy, emotional support, and court support under the Victims' Services Scheme (VSS) and through the Victims of Crime Assistance League (VOCAL). The former is a section of the ACT Department of Health that assesses and refers individuals to counselling and other appropriate services (e.g., massage therapy) as needed. This service is free to people who have been a victim of a crime occurring in the ACT. Counselling is provided by qualified psychologists and social workers in private practice who are paid by the VSS for this service.

VOCAL is a voluntary organisation that receives some government funding from the ACT Department of Health to provide emotional support, court support, and help with practical issues relating to the crime. VOCAL also offers free counselling to individuals who cannot for some reason access the services provided by VSS (for instance, the crime occurred outside the ACT).

There is no requirement that a charge or conviction against the perpetrator(s) of the crime be laid/recorded for victims of crime to receive services through either VOCAL or VSS.

Questionnaires were photocopied and collated in ‘packs’. Each pack consisted of the Foa PDS, the BDI-II, the Health Questionnaire Part 1, two consent forms to be read and signed by the participant, and a detailed information sheet about the study, which also
included contact details for the researcher and both Ethics committees should questions or issues about the research arise. Names and addresses of participants were only included on the consent forms. The questionnaires were coded (eg BH001), with the VSS administrative officer maintaining a database cross-referencing the code to the individual’s personal details, hence enabling follow-up for the second study. For privacy reasons, the researcher was unable to see identifying information about participants prior to the questionnaires being distributed.

The questionnaire packs were offered to clients (with the exception of those excluded on the basis of the criteria outlined previously) at their intake interview by the VSS counsellor/intake officer. It was explained that participation in the research was entirely voluntary, and would not impact on the service they received through the VSS. Participants were given the option of completing the questionnaires at the time of intake (preferred by the researcher) or taking them away and returning the completed forms in a reply-paid envelope.

Questionnaires were given to 64 individuals, with 31 being completed and returned between October 2003 and October 2004. Given the small number of participants (compared with the numbers of clients presenting for intake interviews, usually around 40 – 50 per month), several meetings were held with VSS staff in an effort to improve questionnaire distribution. Some staff expressed reluctance or reservations about asking potentially traumatised clients to participate in a research project, citing frequent presentations of individuals who they felt to be ‘complicated’ (ie suicidal and/or with comorbid personality disorder traits). Staff also cited heavy workload and fatigue as a factor, either forgetting to offer the questionnaires to clients, or feeling too tired/overwhelmed to undertake this task. The researcher and staff agreed on a compromise solution (mailing questionnaires to new clients after the intake interview) while new ways of recruiting participants were explored. However, the mailout process did not result in any additional questionnaires being returned, and VSS staff did not record the number of questionnaires that were distributed in this way.
A new application to the ANU Human Research Ethics Committee was lodged in November 2004 after it became apparent that additional participants were needed from other sources for this research. The application was approved in December 2004, allowing the researcher to seek individuals affected by traumatic crime via advertisements placed on noticeboards at the Psychology department of ANU, mailing out questionnaires to past VSS clients who agreed to participate (see Section 3.4.1.3), and asking new and/or existing clients at VOCAL if they wished to participate in the research (see Section 3.3.1b). No financial inducements were offered, and procedures were similar to those followed in the initial part of the research. The researcher maintained a confidential database of contact details for non-VSS participants, and made herself available for any necessary de-briefing. Further procedural details are provided below.

3.4.1.2 VOCAL

The researcher contacted VOCAL in November 2004 to ask for their assistance with this study. They agreed to assist in distributing questionnaires to new and existing clients; this commenced in December 2004. A further 106 questionnaires were distributed by the researcher and VOCAL staff, with 16 being completed and returned by April 2005. The researcher contacted non-responders by phone and asked them to return the questionnaires if they were still interested in participating in the study. The overall response rate of 15% was low, and could be explained partly by the fact that some of the VOCAL clients had also received questionnaires through VSS, given the fact that the majority of clients eventually access both services (clients were instructed not to complete the questionnaires again, if they had already done so). In addition, the time of year was not conducive to research participation, as December/January is peak holiday season in the ACT, with many people going away to coastal locations (for instance) over summer. Another possible explanation for the low response rate is the fact that a substantial number of VOCAL clients are victims of domestic violence, often presenting during periods of upheaval (such as separating from their partner, and/or being engaged
in legal proceedings). Activities such as questionnaire completion could assume a low priority in this context.

3.4.1.3 VSS – past clients

VSS staff wrote to a random sample of 200 past clients in February 2005 to invite individuals to participate in this research. 180 questionnaires were distributed to those who agreed to participate, and 46 were returned. Of these, two were incomplete and hence not included in the dataset. No follow-up of non-responders was undertaken by VSS or the researcher (who did not have access to personal information such as names and addresses of those who had been sent questionnaires).

3.4.1.4 University students at ANU

Information about the study was placed on noticeboards in the ANU Psychology department inviting individuals who may have experienced or witnessed a traumatic crime to participate in the study. First year psychology students who participated (n = 3) were given credit for an hour’s research participation as part of their course requirements. In total, seven questionnaire packs were distributed to interested undergraduate and postgraduate students, with six packs being completed and returned. All students were given the opportunity to de-brief with the researcher if required and informed of appropriate options for counselling and support. None were treatment-seeking victims of crime.

3.4.2 Study Two

Participants from Study One were followed up five to seven months after completing the first set of questionnaires. They were mailed the Health Questionnaire Part 2, Foa PDS, and BDI-II (see description of measures in Section 3.3.2), along with a brief covering letter reminding them of their initial participation and asking for their assistance with the research once again through completion of this final set of questionnaires. Non-
responders were followed up with a phone call or letter reminding them of the need to return the questionnaires if they were still interested in participating. 98 sets of Study Two questionnaires were distributed, with 59 being returned by the close-off date for data entry (two more were subsequently returned, however it was too late to include these in the analysis).

The PDS and BDI-II were used once again to establish whether there was any variation in trauma or depressive symptomatology. Likewise, the Health Questionnaire Part 2 was very similar to the one used in the first study, as described earlier in this chapter, but asked more detailed questions about counselling received between completion of the first and second batch of questionnaires. The aim was to establish whether an intervention such as counselling (particularly for those who had not previously received any counselling for the trauma) resulted in any change in perceived health status or health symptoms. Those who did not receive any counselling during this period served as a control group.
Chapter Four: A Study Examining the Self-Reported Health of Victims of Crime

4.1 Results

4.1.1 Overview

This first study had the objective of examining the self-reported health of victims of crime in the ACT area, in terms of both symptoms (such as headaches and stomach pain) and any apparent medical diagnoses given by doctors since the first reported traumatic event. In addition, participants were asked to rate the state of their overall health before and since the crime or traumatic event(s). A discrete variable representing change in health perception was created from this latter question (a difference score). In summary, three outcome measures were utilised: “health score” (a continuous variable representing the sum of the health symptom scores), “medical problems” (a dichotomous variable indicating whether or not the participant endorsed one of the medical diagnoses in the questionnaire), and “difference score”, representing the change in health perception since the trauma(s). Data for the 99 participants was analysed using SPSS for Windows, Version 11.5 (a description of the analyses used follows, see Section 4.1.2).

Descriptive statistics are presented first, with chi squared analyses used where applicable to establish whether differences between those participants with PTSD and those without the condition are significant. A description of data screening techniques is provided next. This is followed by two hierarchical multiple regression analyses with the outcome measure “health score”, another hierarchical multiple regression analysis with the dependent variable “difference score”, and a hierarchical logistic regression analysis for the categorical outcome measure “medical problems”. Finally, interpretation and discussion of the results is provided.
4.1.2 Outline of Statistical Analysis

In relation to the descriptive statistics, chi squared analyses were undertaken to establish whether any significant differences existed between those who met criteria for PTSD (PTSD+) as determined through their responses on the PDS, and those who did not (PTSD-) on demographic and trauma-related variables. A univariate ANOVA was then undertaken to investigate whether significant differences in health scores existed between the PTSD+, PTSD- and PTSD partial (those who met two of the three B, C, and D criteria, and presented with significant impairment) groups. An outline of some of the issues associated with defining an individual as having ‘partial’ PTSD is found in Chapter One, Section 1.7. This ANOVA was followed up by a Kruskall-Wallis test as the homogeneity of variance assumption was violated. Two multiple hierarchical regression equations were then computed to determine the unique proportion of variance contributed by various predictors and PTSD to the outcome measure “health score”. One equation included predictors that were significantly correlated with the outcome measure, while the other explored the relative contributions of each of the PTSD symptom clusters to the health score variable. A further hierarchical multiple regression equation was computed examining the contribution of various predictor variables and PTSD to the outcome measure difference score, representing the change in overall health perception from before the trauma(s) to afterwards. Logistic regression was utilised for the categorical outcome measure “medical problems” endorsed (yes or no).

4.1.3 Preliminary Data Screening

Prior to analyses being conducted, data were screened for univariate outliers and missing data using the “Frequencies” and “Explore” commands in SPSS. No missing data were discovered, as only complete questionnaire packs were entered into the SPSS data file. One outlier was located, a low score on the health score variable (24, where the mean was 60.81, and standard deviation equalled 12.56). Despite the fact that this was two standard deviations outside the mean, it was decided to retain this case as it appeared to represent a valid health score for a highly distressed individual. In addition, there were
several scores in the low 30s, with the median score of 62.00 being fairly close to the mean of 60.81. This suggests that the score of 24 is not distorting the data substantially, and is relatively close to the next group of low health scores.

The distributions of data for the continuous and discrete variables were assessed using histograms and tests of skewness and kurtosis. While age appeared relatively normally distributed, with a slight positive skew reflecting the relative youth of participants (skewness .302, kurtosis -.421), health score had a moderate negative skew, indicative of the fact that the population was – in general – relatively healthy (skewness, -.659, kurtosis -.087). A reflect and square root transformation (see Tabachnik and Fidell, 1996) was undertaken to see if this improved the distribution. Skewness improved (-.211), however kurtosis became substantially worse (-.682). It was decided to retain the original non-transformed variable given the fact that the skewness was less than 1.0, and transformation did not produce a typically ‘normal’ distribution. The variable difference score was relatively normally distributed (skewness .061, kurtosis -.237).

Dichotomous variables were checked for any uneven splits between the categories. As gender consisted of only 11 males (out of a total 99 participants) a decision was taken to exclude this (as a variable) from multivariate analyses to avoid undue influence being granted to the smaller of the two categories.

### 4.1.4 Descriptive statistics

The mean age of participants was 35.9 years (sd = 11.14), with a range from 17 through to 65 years. The sample was predominantly female (n = 88), with 11 males participating. Table 4.1 provides details of the characteristics of the PTSD+ and PTSD- groups, with chi squared analyses used to establish any significant differences between them. The source from which participants were recruited was significant ($\chi^2 = 8.01$, df = 3, $p < .05$). New clients accessing VSS assistance were more likely to meet criteria for PTSD (19 in the PTSD+ group, as opposed to 12 PTSD- individuals), whereas past VSS clients were much more likely to not have a current diagnosis of PTSD (30 PTSD- and
16 PTSD+). This is not surprising given that many participants in the latter group had already received at least some counselling assistance, and it had often been months or years since the occurrence of the traumatic event(s).

4.1.4.1 Comorbidity and prevalence issues

Interestingly, Table 4.1 demonstrates that there were more smokers in the PTSD- group than the PTSD+ group, although the difference was not statistically significant, with non-smokers being equally distributed between the two groups. Similarly, past or current moderate-heavy users of alcohol were also fairly equally distributed across the PTSD+ and PTSD- groups.

Table 4.1: Characteristics of PTSD positive and PTSD negative groups

<table>
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<tr>
<th></th>
<th>PTSD+ (n)</th>
<th>PTSD- (n)</th>
<th>χ²</th>
<th>Significance</th>
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<td>&lt;40</td>
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<td>1.28 (df = 1)</td>
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<td>40+</td>
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<td>18</td>
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<td>0.00 (df = 1)</td>
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<td>7</td>
<td>8.01 (df = 3)</td>
<td>p &lt; .05</td>
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<tr>
<td>Smokers</td>
<td>15</td>
<td>26</td>
<td>2.22 (df = 1)</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Current/past alcohol use¹</td>
<td>11</td>
<td>12</td>
<td>0.07 (df = 1)</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Current illicit drug use</td>
<td>6</td>
<td>10</td>
<td>0.49 (df = 1)</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Past illicit drug use</td>
<td>9</td>
<td>17</td>
<td>1.67 (df = 1)</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Antidepressant medication</td>
<td>18</td>
<td>12</td>
<td>3.67 (df = 1)</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Depressed²</td>
<td>41</td>
<td>24</td>
<td>23.71 (df = 1)</td>
<td>p &lt; .001</td>
</tr>
<tr>
<td>Number of traumatic events</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>6</td>
<td>21</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2–3</td>
<td>20</td>
<td>22</td>
<td>9.83 (df = 2)</td>
<td>p &lt; .01</td>
</tr>
<tr>
<td>4+</td>
<td>19</td>
<td>11</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

¹ statistically significant at p < .05
² as defined in ‘Measures’ section in Methodology chapter, ie moderate to heavy alcohol consumption
³ ascertained via BDI scores, see ‘Measures’ section in Methodology chapter
More individuals in the PTSD+ group were currently taking antidepressant medication than those in the PTSD- group (17 as opposed to 12, $\chi^2 = 3.67$ (1df), $p = .055$).

Significantly more victims of crime with PTSD also met criteria for depression, as assessed using the BDI-II, than those without PTSD ($\chi^2 = 23.71$, df = 1, $p < .001$). Similarly, the more traumatic events experienced by an individual, the greater the likelihood of them meeting criteria for PTSD ($\chi^2 = 9.83$, df = 2, $p < .01$). Overall prevalence of PTSD in this sample was 44.3% ($n = 43$). A further 19.6% ($n = 19$) met criteria for partial PTSD; however this group was subsumed into the PTSD- category after a univariate ANOVA (followed by a Kruskall-Wallis test due to failure of the assumption of homogeneity of variance) showed no significant differences between the PTSD- and PTSD partial groups on one of the main outcome measures, health score.

The health score means for each group are displayed in Table 4.2, with standard deviations in parentheses. Higher scores represent less symptom reporting (ie better self-reported health).

Table 4.2: Mean health scores for each of the PTSD groups

<table>
<thead>
<tr>
<th></th>
<th>PTSD+</th>
<th>PTSD-</th>
<th>PTSD partial</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>54.78 (13.6)</td>
<td>66.46 (9.19)</td>
<td>64.68 (8.83)</td>
<td>60.81 (12.56)</td>
</tr>
</tbody>
</table>

Over one third of participants (36%) reported that the traumatic event which "mattered the most" to them had occurred over five years ago, while a further 39% stated that the trauma had happened from six months to five years ago. The type of trauma most likely to lead to a diagnosis of PTSD was sexual assault by a known person, with 67.6% of the participants experiencing this traumatic event meeting criteria for PTSD. It was also the second-most common traumatic event reported, with 37.4% of the sample acknowledging that they had experienced a sexual assault by someone they knew. This reflects the high proportion of domestic violence and child sexual abuse victims who participated in this study, ascertained by descriptions of the traumatic event in the questionnaire in addition to anecdotal advice from VSS and VOCAL staff. Some of these participants also endorsed an "other" traumatic event, often describing it as
emotional or verbal abuse. There was significant overlap between the two categories “sexual assault by someone you know”, and “sexual contact when you were younger than 18…”, due in some cases to individuals experiencing sexual assaults as both children and adults, but also because the sexual contact as a child was in many cases a sexual assault by a family member or other known person. Table 4.3 provides details of the types of traumatic events experienced by participants in this study, with chi squared analyses used to assess significant differences between the PTSD+ and PTSD- groups.

Table 4.3: PTSD diagnosis and different traumatic events

<table>
<thead>
<tr>
<th>Event</th>
<th>Total (n)</th>
<th>Total (%)</th>
<th>PTSD+ (n)</th>
<th>PTSD- (n)</th>
<th>(\chi^2) (df=1)</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accident</td>
<td>35</td>
<td>35.4</td>
<td>19</td>
<td>16</td>
<td>1.70</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Disaster</td>
<td>16</td>
<td>16.2</td>
<td>7</td>
<td>9</td>
<td>0.02</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Non-sexual assault (known person)</td>
<td>50</td>
<td>50.5</td>
<td>25</td>
<td>25</td>
<td>0.84</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Non-sexual assault (stranger)</td>
<td>31</td>
<td>31.3</td>
<td>15</td>
<td>16</td>
<td>0.16</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Sexual assault (known person)</td>
<td>37</td>
<td>37.4</td>
<td>25</td>
<td>12</td>
<td>11.65* (df=1)</td>
<td>p = .001</td>
</tr>
<tr>
<td>Sexual assault (stranger)</td>
<td>20</td>
<td>20.2</td>
<td>9</td>
<td>11</td>
<td>0.00</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Military combat</td>
<td>1</td>
<td>1.0</td>
<td>1</td>
<td>0</td>
<td>na</td>
<td>-</td>
</tr>
<tr>
<td>Sexual contact as child (known person)</td>
<td>35</td>
<td>35.4</td>
<td>19</td>
<td>16</td>
<td>1.70</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Imprisonment; being held hostage</td>
<td>4</td>
<td>4.0</td>
<td>2</td>
<td>2</td>
<td>na</td>
<td>-</td>
</tr>
<tr>
<td>Torture</td>
<td>5</td>
<td>5.1</td>
<td>4</td>
<td>1</td>
<td>na</td>
<td>-</td>
</tr>
<tr>
<td>Life threatening illness</td>
<td>18</td>
<td>18.2</td>
<td>8</td>
<td>10</td>
<td>0.01</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Other</td>
<td>31</td>
<td>31.3</td>
<td>17</td>
<td>14</td>
<td>1.60</td>
<td>p &gt; .05</td>
</tr>
</tbody>
</table>

* statistically significant at p = .001

The most commonly reported traumatic event was non-sexual assault by a known person (50.5% of the sample). Also of interest is the fact that traumatic experiences apparently unrelated to criminal events were also reported frequently, with 35.4% of the sample stating that they had experienced or witnessed an accident, fire, or explosion. Recent fatal bushfires which swept through Canberra, destroying over 450 homes and killing 4 people, may have had an impact here. A recently released study conducted by the Australian National University’s Centre for Mental Health Research indeed found that
79.2% of the 2085 young adults who were being studied as part of a longitudinal research project (PATH Through Life) reported at least one bushfire experience, and 5% met full criteria for current PTSD (Parslow, Jorm, and Christensen, 2005). 18.2% of the sample in this current study also reported experiencing or having someone close to them suffer a life-threatening illness. Together with data showing that a substantial minority of the sample (30.3%) had experienced or witnessed four or more traumatic events, this reflects the diversity and frequency of traumatic events in this largely treatment-seeking population.

Table 4.4 provides details of the degree to which various health problems that comprise the health score outcome measure were frequently reported by participants. Headaches, nausea, and indigestion/digestion problems comprised the majority of symptoms endorsed. There was insufficient data for each symptom to perform individual analyses of variance or regression analyses. Table 4.2 shows the mean health scores for each PTSD group, while hierarchical multiple regression analyses were performed to establish the unique contribution of each predictor to the overall outcome variable, health score. The mean health score for the sample was 60.81 (sd = 12.56), ranging from a low of 24 (indicating a poor perception of health through frequent endorsement of many symptoms) to a maximum of 78.
Table 4.4: Self-reported health problems endorsed by participants

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Endorsed (n)</th>
<th>Percentage of Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headaches</td>
<td>43</td>
<td>43.4</td>
</tr>
<tr>
<td>Nausea</td>
<td>39</td>
<td>39.4</td>
</tr>
<tr>
<td>Indigestion</td>
<td>37</td>
<td>37.4</td>
</tr>
<tr>
<td>Frequent urination</td>
<td>28</td>
<td>28.3</td>
</tr>
<tr>
<td>Muscle weakness</td>
<td>26</td>
<td>26.3</td>
</tr>
<tr>
<td>Breath shortness</td>
<td>26</td>
<td>26.3</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>24</td>
<td>24.2</td>
</tr>
<tr>
<td>Pelvic pain*</td>
<td>20</td>
<td>22.7</td>
</tr>
<tr>
<td>Coordination problems</td>
<td>22</td>
<td>22.2</td>
</tr>
<tr>
<td>Chest pain</td>
<td>21</td>
<td>21.2</td>
</tr>
<tr>
<td>Blurred vision</td>
<td>21</td>
<td>21.2</td>
</tr>
<tr>
<td>Unexplained Thirst</td>
<td>19</td>
<td>19.2</td>
</tr>
<tr>
<td>Other</td>
<td>10</td>
<td>10.1</td>
</tr>
</tbody>
</table>

* Females only

4.1.5 Multivariate analyses - multiple regression

Two hierarchical multiple regression analyses were used to assess firstly the relative contributions of various independent variables and PTSD on self-reported health symptoms (health score), followed by an examination of the extent to which the different PTSD symptom clusters predicted self-reported health. A further hierarchical multiple regression equation was then constructed to examine the unique contribution of PTSD to the outcome measure difference score (change in overall health perception since the trauma(s)), controlling for a number of other independent variables.

4.1.5.1 Multivariate data screening: Health Score regressed on PTSD and other predictors

An initial correlation matrix was produced to examine the relationships between seven predictor variables with each other and the outcome measure health score (see Table 4.5). Only one predictor (smoking) was not significantly correlated with the outcome variable; this was not included in the regression equation, leaving six predictors. While this is a relatively large number of variables in comparison to the number of cases, the
difference between $R^2$ and adjusted $R^2$ was not substantially different, indicating that the analysis was relatively robust.

Examination of the correlation matrix did not reveal any evidence of unacceptably high inter-correlations (eg > .6) among predictor variables. A residuals scatterplot was produced as part of the regression run in SPSS; this did not show any evidence of non-normality, non-linearity, or homoscedasticity of the residuals. Mahalanobis distance was computed for each case to check whether multivariate outliers were present. Critical $\chi^2$ at $\alpha = .001$ for 6 df is 22.458; there were no cases with values in excess of this, which would have indicated a multivariate outlier. The influence of the univariate outlier identified earlier in the health score outcome variable was not significant in the regression equation, as assessed using the Cook’s D measure of influence.

Table 4.5: Pearson $r$ correlations for PTSD, health score and associated variables

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. health score</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. smoking</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. alcohol use</td>
<td>-.107</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. BDI depression</td>
<td>-.225*</td>
<td>.314**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. PTSD diagnosis</td>
<td>-.347***</td>
<td>-.126</td>
<td>.146</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. antidepressant medication</td>
<td>-.441***</td>
<td>-.150</td>
<td>.026</td>
<td>.489***</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>7. age</td>
<td>-.204*</td>
<td>.129</td>
<td>.182*</td>
<td>.077</td>
<td>.155</td>
<td>-.015</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>8. no. traumatic events</td>
<td>-.273**</td>
<td>.054</td>
<td>.122</td>
<td>.369***</td>
<td>.286**</td>
<td>.206*</td>
<td>.157</td>
<td>1</td>
</tr>
</tbody>
</table>

* statistically significant at $p<.05$

** statistically significant at $p<.01$

*** statistically significant at $p<.001$
Checks for singularity and multicollinearity did not reveal any problems in this area, with acceptable levels of tolerance and VIF for each variable. The predictor with the lowest tolerance was depression (in the second step of the hierarchical regression) with a tolerance level of .648. While this is a little low, the standard errors for the regression coefficients were within an acceptable range (ie fairly low). No suppressor variables were found.

4.1.5.2 Correlation matrix interpretation and health score regressed on PTSD and other predictors

The correlation matrix in Table 4.5 shows a number of significant relationships between self-reported health symptoms (health score) and various independent variables. PTSD and depression were both significant negatively correlated with health score, indicating that a diagnosis (by means of the questionnaires used in this study) of either psychiatric condition is related to lower (worse) health scores. Similarly, substantial alcohol consumption and antidepressant use were also significantly negatively related to health score, suggesting that individuals who used these were more likely to report a greater number of health symptoms. Finally, age and number of traumatic events were also negatively correlated with health score. This would suggest that older people rate themselves as having poorer health, while experiencing a higher number of traumatic events is also significantly associated with perceptions of poor health.

Hierarchical multiple regression was undertaken to assess whether the addition of PTSD diagnosis improved the prediction of self-reporting of health symptoms (health score) over and above that predicted by the other five variables. In other words, the focus of this equation was to examine the unique contribution of age, followed by alcohol use, antidepressant medication, number of traumatic events, and depression diagnosis (entered in one block), and finally PTSD diagnosis. Table 4.6 displays the results of this regression equation, specifically the unstandardized regression coefficients (B), the standard errors of B, the standardized regression coefficients (β), the amount of variance
added to \( R^2 \) by each block of predictors when they enter the equation (Step \( \Delta R^2 \)), and adjusted total \( R^2 \) after each block of predictor variables is entered.

\( R^2 \) was significantly different from zero at the end of each step. At Step 1, age added significantly to the prediction of physical health symptoms, accounting for 4.2% of the variance. However, once alcohol use, antidepressant medication, number of traumatic events, and depression were added into the equation as a block in the second step, age was no longer a significant predictor of health symptoms. In fact, the only variable which accounted for a significant portion of the variance at this second step was the use of antidepressant medication (\( p < .05 \)). Antidepressant medication retained its significance when PTSD was entered into the equation in the final step. The addition of PTSD accounted for a further 8.4% of unique variance in health score, which was statistically significant at \( p = .001 \). At this final step, with all independent variables in the equation, \( R^2 = .322, F(6,92) = 7.26, p < .001 \).

Table 4.6: Hierarchical multiple regression for prediction of frequency of self-reported physical health symptoms, PTSD entered in final step

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE B</th>
<th>( \beta )</th>
<th>Step ( \Delta R^2 )</th>
<th>Total ( R^2 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1: Age</td>
<td>-0.23</td>
<td>0.11</td>
<td>-0.20*</td>
<td>.042</td>
<td>4.2%*</td>
</tr>
<tr>
<td>Step 2: Medication, trauma,</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>depression</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>-0.17</td>
<td>0.11</td>
<td>-0.15</td>
<td></td>
<td>.196</td>
</tr>
<tr>
<td>Alcohol use</td>
<td>-4.27</td>
<td>2.75</td>
<td>-0.14</td>
<td></td>
<td>23.8%**</td>
</tr>
<tr>
<td>Antidepressant medication</td>
<td>-6.47</td>
<td>2.63</td>
<td>-0.24*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. traumatic events</td>
<td>-2.99</td>
<td>2.79</td>
<td>-0.11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI Depression</td>
<td>-5.11</td>
<td>2.69</td>
<td>-0.19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Step 3: PTSD</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>.084</td>
</tr>
<tr>
<td>Age</td>
<td>-0.14</td>
<td>0.10</td>
<td>-0.12</td>
<td></td>
<td>32.2%**</td>
</tr>
<tr>
<td>Alcohol use</td>
<td>-4.93</td>
<td>2.62</td>
<td>-0.17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Antidepressant medication</td>
<td>-6.25</td>
<td>2.50</td>
<td>-0.23*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. traumatic events</td>
<td>-1.94</td>
<td>2.67</td>
<td>-0.07</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BDI depression</td>
<td>-1.19</td>
<td>2.81</td>
<td>-0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>-8.43</td>
<td>2.51</td>
<td>-0.34**</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*statistically significant at \( p < .05 \)
**statistically significant at \( p \leq .001 \)
4.1.5.3 Multivariate data screening: Health Score regressed on PTSD symptom clusters and antidepressant medication.

A second multiple hierarchical regression analysis was conducted to establish the relative contributions of each of the PTSD symptom clusters (reexperiencing, avoidance, and arousal) to self-reported health symptoms, after controlling for antidepressant use (the only significant variable apart from PTSD in the final step of the preceding regression equation). A matrix of Pearson r correlations for all variables in the equation is displayed in Table 4.7.

Table 4.7: Pearson r correlations for health score, PTSD symptom clusters, and antidepressant medication use

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Health score</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Antidepressant medication</td>
<td>-.331***</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. PTSD Reexperiencing</td>
<td>-.227*</td>
<td>.183*</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. PTSD Avoidance</td>
<td>-.423***</td>
<td>.223*</td>
<td>.376***</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>5. PTSD Hyperarousal</td>
<td>-.307**</td>
<td>.157</td>
<td>.503***</td>
<td>.470***</td>
<td>1</td>
</tr>
</tbody>
</table>

*statistically significant at p < .05  
**statistically significant at p < .01  
***statistically significant at p < .001

Examination of the correlation matrix did not reveal any evidence of unacceptably high inter-correlations among predictor variables. A residuals scatterplot was produced as part of the regression run in SPSS; this did not show any evidence of non-normality, non-linearity, or homoscedasticity of the residuals. Mahalanobis distance was computed for each case to check whether multivariate outliers were present. Critical $\chi^2$ at $\alpha = .001$ for 5 df is 20.515; there were no cases with values in excess of this, which would have indicated a multivariate outlier. Similarly, measures of influence such as Cook’s D did not reveal the existence of any problematic univariate outliers. Checks for singularity and multicollinearity did not reveal any problems in this area, with acceptable levels of
tolerance and VIF for each variable. The predictor with the lowest tolerance was PTSD arousal (in the second step of the hierarchical regression) with a tolerance level of .655. While this is a little low, the standard errors for the regression coefficients were within an acceptable range (ie fairly low). No suppressor variables were found.

4.1.5.4 Correlation matrix interpretation: health score regressed on PTSD symptom clusters

All three PTSD symptom clusters in the correlation matrix (Table 4.7) were significantly negatively correlated with health score, with the highest correlation being between the PTSD avoidance/numbing cluster and health score. In other words, individuals reporting meeting criteria for each symptom cluster are more likely to report health symptoms, particularly for those meeting the avoidance/numbing cluster. Table 4.8 displays the results of the regression equation examining the unique contribution of the PTSD symptoms clusters on health score (adjusted for antidepressant use), specifically the unstandardized regression coefficients (B), the standard errors of B, the standardized regression coefficients ($\beta$), the amount of variance added to $R^2$ by each block of predictors when they enter the equation (Step $\Delta R^2$), and adjusted total $R^2$ after each block of predictor variables is entered.

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE B</th>
<th>$\beta$</th>
<th>Step $\Delta R^2$</th>
<th>Total $R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1: Antidepressant medication</td>
<td>-9.00</td>
<td>1.43</td>
<td>-0.33**</td>
<td>.110</td>
<td>11%**</td>
</tr>
<tr>
<td>Step 2: PTSD symptom clusters and antidepressant medication</td>
<td></td>
<td></td>
<td></td>
<td>.140</td>
<td>25.0%**</td>
</tr>
<tr>
<td>A/dep medication</td>
<td>-6.58</td>
<td>2.51</td>
<td>-0.24**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD reexperiencing</td>
<td>-0.13</td>
<td>2.90</td>
<td>-0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD avoidance/numbing</td>
<td>-7.82</td>
<td>2.63</td>
<td>-0.31**</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD arousal</td>
<td>-3.39</td>
<td>3.10</td>
<td>-0.12</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**statistically significant at $p < .01$
R² was significantly different from zero at the end of each step. In step one, antidepressant medication use accounted for 11% of the variance in health symptoms; this was significant at p < .01. As with the previous regression equation, antidepressant medication retained its significance once the PTSD symptom clusters were introduced in the second and final step. The addition of these clusters accounted for a further 14% of the variance in health symptoms. However, the only symptom cluster that was statistically significant in its own right as a predictor was avoidance/numbing (p < .01). At this final step, with all independent variables in the equation, R² = .25, F(4,94) = 7.82, p < .001.

4.1.6 Multivariate analyses: multiple regression - difference score regressed on five independent variables

The discrete outcome variable difference score (change in health perception from before to after the trauma[s]) was calculated by subtracting participants’ perception of their health (rated on a 5 point Likert-type scale) before the trauma from their health perception (measured on the same scale) since the trauma. It is acknowledged at this point that this is a highly subjective variable: asking individuals at some point in time after a traumatic event to think back to and rate their health before the trauma will almost inevitably introduce some degree of recall bias. Nevertheless, it was considered that this might be a useful adjunct to the other dependent variables or outcome measures investigated in this study, notwithstanding its limitations.

Scores on this variable ranged from -3 (indicating the greatest change in health perception for the worst), to +2 (health somewhat better since the trauma). The majority of individuals (67 out of 99) indicated that they perceived their health to be worse since the trauma, while five felt it was better. 27 participants felt that their health had not changed.

An initial correlation matrix was produced to examine the relationships between seven predictor variables (age, smoking, antidepressant medication use, alcohol consumption,
number of traumatic events, depression, and PTSD) with each other and the outcome measure difference score. Smoking and antidepressant medication were not significantly correlated with difference score and hence were not included in the regression equation. Age was included, although its correlation with difference score fell just short of significance (-.159, p=.058). Pearson r correlations for the variables entered into the regression equation are displayed in Table 4.9.

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Difference score</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Age</td>
<td>-.159</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. Alcohol use</td>
<td>-.167*</td>
<td>.182*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. No. of traumatic events</td>
<td>-.169*</td>
<td>.188*</td>
<td>.122</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. BDI Depression</td>
<td>-.508***</td>
<td>.077</td>
<td>.146</td>
<td>.369***</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>6. PTSD</td>
<td>-.476***</td>
<td>.115</td>
<td>.026</td>
<td>.286**</td>
<td>.489***</td>
<td>1</td>
</tr>
</tbody>
</table>

*statistically significant at p ≤ .05  
**statistically significant at p < .01  
***statistically significant at p < .001

Examination of the correlation matrix did not reveal any evidence of unacceptably high inter-correlations among predictor variables. A residuals scatterplot was produced as part of the regression run in SPSS; this did not show any evidence of non-normality, non-linearity, or homoscedasticity of the residuals. Mahalanobis distance was computed for each case to check whether multivariate outliers were present. Critical \( \chi^2 \) at \( \alpha = .001 \) for 5 df is 20.515; there were no cases with values in excess of this, which would have indicated a multivariate outlier. Checks for singularity and multicollinearity did not reveal any problems in this area, with acceptable levels of tolerance and VIF for each variable. The predictor with the lowest tolerance was PTSD (in the second step of the hierarchical regression) with a tolerance level of .740. No suppressor variables were found.
4.1.6.1 Correlation matrix interpretation and multiple regression: difference score regressed on PTSD and other variables

The correlation matrix in Table 4.9 displays a number of significant relationships between difference score and various independent variables. Moderate significant correlations between the outcome variable and alcohol use (-.167, \( p = .05 \)) and number of traumatic events (-.169, \( p < .05 \)) suggest that higher rates of alcohol consumption and experiencing a greater number of traumatic events are associated with worse perceptions of overall health since the trauma(s) in this sample. Similarly, substantial negative correlations were present between difference score and diagnoses of depression (-.508, \( p < .001 \)) and PTSD (-.476, \( p < .001 \)), indicating that participants who meet criteria for these disorders (as assessed by the BDI and PDS, respectively) report substantially worse perceptions of health, overall.

Table 4.10 displays results of the regression equation examining the unique contribution of PTSD to overall perceptions of health since the trauma (difference score), controlling for age, alcohol use, number of traumatic events, and depression. Unstandardized regression coefficients (\( B \)), standard errors of \( B \), standardized regression coefficients (\( \beta \)), the amount of variance added to \( R^2 \) by each block of predictors when they enter the equation (Step \( \Delta R^2 \)), and adjusted total \( R^2 \) after each block of predictor variables is entered are displayed.

\( R^2 \) was significantly different from zero at the end of Steps 2 and 3, but not Step 1, indicating that age did not add significantly to the prediction of change in health perception (difference score). The only significant variable in the second step was depression, \( (p < .001) \), indicating that it alone of the variables in the second block accounted for a significant portion of the variance in the outcome variable. The addition of PTSD in the final step accounted for a further 7% of unique variance in difference score; this was statistically significant at \( p < .01 \). This shows that PTSD, over and above all other variables, explains a significant portion of the variance in change in health
perception. At the final step, with all independent variables in the equation, $R^2 = 0.349$, $F(5,93) = 7.62, p < .001$.

Table 4.10: Hierarchical multiple regression for prediction of change in health perception (difference score), PTSD entered in final step

<table>
<thead>
<tr>
<th>Predictor</th>
<th>B</th>
<th>SE B</th>
<th>$\beta$</th>
<th>Step $\Delta R^2$</th>
<th>Total $R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Step 1: Age</td>
<td>-0.02</td>
<td>0.01</td>
<td>-0.16</td>
<td>.025</td>
<td>2.5%</td>
</tr>
<tr>
<td>Step 2: Alcohol use, traumatic events, depression</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>27.9%***</td>
</tr>
<tr>
<td>Age</td>
<td>-0.01</td>
<td>0.01</td>
<td>-0.12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol use</td>
<td>-0.19</td>
<td>0.22</td>
<td>-0.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. traumatic events</td>
<td>0.11</td>
<td>0.23</td>
<td>0.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>-1.12</td>
<td>0.21</td>
<td>-0.51***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Step 3: PTSD</td>
<td></td>
<td></td>
<td></td>
<td>.070</td>
<td>34.9%**</td>
</tr>
<tr>
<td>Age</td>
<td>-0.01</td>
<td>0.01</td>
<td>-0.09</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol use</td>
<td>-0.24</td>
<td>0.21</td>
<td>-0.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No. traumatic events</td>
<td>0.20</td>
<td>0.22</td>
<td>0.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>-0.81</td>
<td>0.22</td>
<td>-0.37***</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>-0.65</td>
<td>0.21</td>
<td>-0.31**</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**statistically significant at $p < .01$
***statistically significant at $p < .001$

4.1.7 Multivariate Analyses: logistic regression – medical problems endorsed

Sequential logistic regression was used to assess the contribution of various independent variables on the categorical predictor variable, medical problems endorsed, yes or no (“medical problems”). Age, smoking, alcohol use, and antidepressant medication were entered first as a block into the logistic regression equation, followed by PTSD in the second block. An examination of the coefficients and standard errors resulting from this equation showed that these were of a relatively small magnitude, which indicated that the sample size was sufficient for the number of independent or predictor variables (Hosmer and Lemeshow, 2000), and also that no multicollinearity was evident. The least significant variables (as indicated by the Wald test) were eliminated, with reduced sequential models comprising age, depression, PTSD, and the dependent variable.
medical problems resulting. A search for outliers using standardized residuals and a measure of influence, Cook’s D, did not reveal any outliers with z residuals in excess of 3, hence no cases were deleted from the analysis. The logistic regression model showing standardized beta coefficients (β), the Wald test of significance, odds ratios, and 95% confidence intervals for the odds ratio for each variable is shown in Table 4.11, below.

Table 4.11: Logistic regression for medical problems endorsed, with age, depression, and finally PTSD entered as independent variables

<table>
<thead>
<tr>
<th>Variables</th>
<th>β</th>
<th>Wald test</th>
<th>Odds ratio (OR)</th>
<th>95% confidence interval for OR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.08</td>
<td>11.44**</td>
<td>1.08</td>
<td>1.03 - 1.13</td>
</tr>
<tr>
<td>Depression</td>
<td>0.03</td>
<td>0.00</td>
<td>1.03</td>
<td>0.36 - 2.90</td>
</tr>
<tr>
<td>PTSD</td>
<td>0.23</td>
<td>0.21</td>
<td>1.26</td>
<td>0.47 - 3.39</td>
</tr>
<tr>
<td>(Constant)</td>
<td>-3.17</td>
<td>13.72***</td>
<td>0.04</td>
<td></td>
</tr>
</tbody>
</table>

**statistically significant at p<.01

***statistically significant at p<.001

With respect to medical problems endorsed (yes or no), there was a good model fit on the basis of age and depression alone, χ² (2, n=99) = 118.46, p<.001. After addition of PTSD, χ² (3, n=99) = 118.25, a change of only .206 (p>.05) suggesting very little change or improvement in the model by the addition of PTSD. The only significant predictor variable was age, odds ratio 1.08 (p<.01). This indicates that the odds of reporting a medically diagnosed problem are 1.08 times greater for each one year increase in age.

4.2 Discussion

The current study sought to examine the impact of PTSD on specific self-reported health symptoms, medical diagnoses, and overall perceptions of health since the traumatic event(s) amongst a sample of victims of crime. The study sought to control for the
effects of variables that have been linked with poor health in the literature, including smoking, alcohol use, increasing age, and depression, as well as variables that might modify the effect of PTSD on health, such as use of antidepressant medication, and the number of traumatic events that the individual has been exposed to. The study also examined the differential impact of the three PTSD symptom clusters on self-reported health symptoms, given previous research showing that specific clusters (variously, hyperarousal, hyperarousal and avoidance, and reexperiencing) were associated with poorer perceptions of health (Clum et al (2001) and Kimerling et al (2000), Woods and Wineman (2004), and Zoellner et al (2000), respectively). A brief exploration of the characteristics of this particular population was also undertaken.

4.2.1 Sample characteristics

A current PTSD prevalence rate of 44.3% in this sample is consistent with that found in some previous studies of traumatised individuals, particularly involving samples where assault or domestic violence has been the primary trauma reported. For instance, Sharhabani-Arzy et al (2003) and Mertin and Mohr (2000) reported current prevalence rates for PTSD of 51.6% and 45%, respectively, in their samples of domestic violence victims. Golding (1999), in a meta-analysis of 11 studies, reported that between 31% and 84.4% of women in domestic violence situations met PTSD criteria. This compared with a current prevalence rate of 12.4% in the rape victims studied by Resnick et al (1993). The differences in PTSD rates reported in these studies are generally due to a number of factors, including the measurement instruments chosen, the nature of the trauma (ongoing violence perpetrated by a known other appearing to lead to higher rates of PTSD, compared with a one-off sexual assault), the recency of the trauma, and whether or not the sample was treatment-seeking.

A substantial proportion of the participants in this current study identified themselves as having experienced domestic violence and/or child sexual abuse. This is reflected in the fact that the most common traumatic events reported by participants were sexual and non-sexual assaults committed by a known person (50.5% and 37.4%, respectively).
Anecdotal feedback from staff at the VSS and VOCAL also indicated that many of the individuals presenting to these services reported domestic violence and/or childhood sexual abuse as the crime(s) for which they sought counselling or other assistance. Similarly, sexual assault by a known person (common amongst sufferers of domestic violence) was the trauma most likely to lead to a diagnosis of PTSD, with 67.6% of participants who had experienced this type of trauma meeting criteria for PTSD. The higher rates of PTSD among domestic violence and child sexual abuse victims, as compared with other traumatised populations, may reflect the ongoing nature of the trauma, the threat of injury or death to the individual and often their children (if applicable), and quite possibly a sense of helplessness and powerlessness in this situation. As outlined in Chapter One (under the sub-heading ‘Complex PTSD’, Section 1.10), Herman (1997) describes a type of “prolonged, repeated trauma” that may produce profound deformations of personality when individuals are exposed to such abuses in captivity. This captivity may take multiple forms in an abusive relationship: economic, social, psychological, and legal, in addition to fear and physical force. The person subjected to such captivity may regard themselves as being “imprisoned”, “tortured”, and at risk of often unpredictable violence aimed at themselves and their children. Victims of interpersonal trauma react with anxiety and may have strong feelings around loss of control, vulnerability, and self-blame (Resnick et al, 1993). Domestic violence victims with PTSD may also present with higher rates of comorbid suicidal ideation, major depression, and anxiety, compared with people exposed to violent situations (including domestic violence) without PTSD (Sharhabani-Arzy et al, 2003). Hence, one might expect a high rate of both PTSD and comorbid physical and emotional health problems in such a group, a prediction which was generally borne out in this study.

Other interesting characteristics included the high number of traumatic events (four or more) experienced by almost one third of this sample. This is consistent with the findings of Resnick et al (1993), who reported that over 50% of their sample who had been exposed to a crime had experienced multiple episodes of criminal events. Similarly, Kessler et al (1995) found that 10.2% of the men and 6.4% of the women in
their community sample who had experienced trauma reported more than four major traumas in their life.

Surprisingly, given the results of some previous research identifying high rates of smoking in individuals with PTSD (e.g., Beckham, Feldman, Vrana, et al., 2005; Schnurr and Spiro, 1999; Shalev et al., 1990), smoking was relatively uncommon amongst those with PTSD in this current study. One third of the group with PTSD reported themselves as being current or past smokers (moderate to heavy use of cigarettes), while 48.1% of the non-PTSD group identified that they had smoked moderately to heavily. The differences between the two groups were not statistically significant. Some studies also report higher rates of smoking amongst traumatised individuals when compared to the general population (e.g., Acierno, Kilpatrick, Resnick, Saunders, De Arellano, and Best, 2000; Weaver and Etzel, 2003), but no differences between the PTSD and non-PTSD groups. Beckham et al. (1998) did not find any differences in smoking (assessed by ‘pack year history’, calculated on the basis of average number of cigarettes per day times the years smoked, divided by 20) between male Vietnam veterans with and without PTSD. Similarly, Acierno et al. (2000) did not find an association between PTSD and an increased risk of smoking in their adolescent sample. This indicates that the literature is not always consistent on this point, with differences likely arising due to variations in measurement (e.g., whether past as well as current smoking is considered) and sample size. The questions assessing tobacco use in this study were thought to be relatively detailed, with participants asked explicitly about current and past smoking patterns (including quantity and duration of tobacco use), although the end variable (smoking yes or no) was by necessity dichotomized due to relatively small cell sizes.

4.2.2 Multiple Regression Analyses with (a) PTSD and (b) PTSD symptom clusters as predictors of Health Score

The results of the first hierarchical multiple regression analysis with health score as an outcome variable suggest that PTSD accounts for a significant proportion of the variance in self-reported health symptoms, after controlling for age, depressive symptoms,
number of traumatic events, alcohol use, and antidepressant medication use (factors which themselves are associated with poor physical health). PTSD symptoms accounted for a further 8.4% of the unique variance over and above the predictor variables previously listed, which was significant at \( p=.001 \), indicating that some factor associated with having PTSD is responsible to a certain extent for poor self-reported health. This finding is consistent with other research reporting a significant association between PTSD symptoms and self-reporting of specific health problems (Wolfe et al., 1994; Kimerling et al., 2000; Litz et al., 1992; Hankin et al., 1996; Solomon & Mikulincer, 1987; Shalev et al., 1990; Dobie et al., 2004; Clum et al., 2001; Woods & Wineman, 2004), and supports the hypothesis that a higher level of specific health complaints would be reported by victims of crime with PTSD, compared with those not meeting criteria for this disorder (as assessed using the PDS).

Interestingly, use of antidepressant medication was also a significant predictor of self-reported health problems \( (p<.05) \), while depression was not. Chest pain, breathlessness, nausea, and digestive problems, some of the specific health symptoms listed in the health questionnaire, are often present in anxiety disorders. It is therefore possible that medical practitioners are prescribing antidepressants for these particular symptoms, given the fact that SSRIs in particular are often a treatment of choice for anxiety. It is also possible that medical practitioners in busy practices who see patients presenting with a variety of physical health complaints may try antidepressant medication on the assumption that the person might be depressed. Research has shown that general practitioners (GPs) prescribe the vast majority of antidepressants in Australia (McManus, Mant, Mitchell, Britt, and Dudley, 2003), often for conditions other than major depression. For instance, one Canadian study found that antidepressants were commonly prescribed for chronic pain and insomnia (Thommasen, Baggaley, Thommasen, and Zhang, 2005). Several studies have also reported an association between increasing patient age and greater antidepressant use, despite evidence that depression is not significantly higher in older age groups (Percudani, Barbui, Fortino, and Petrovich, 2005; Thommasen et al., 2005), again suggesting that this group of medications is being prescribed on occasions for conditions that do not necessarily meet
the criteria for major depression. Alternatively, the association between health symptom reporting and anti-depressant medication use may also be related to side-effects from these treatments.

Previous research has established a clear association between depression and physical illness and/or immunological changes, including altered natural killer cell cytotoxicity and reduced lymphocyte proliferation (Miller, Cohen, and Herbert, 1999), heart arrhythmias (Boscarino and Chang, 1999b), and coronary heart disease (Glassman and Shapiro, 1998; Ward, Tueth, and Sheps, 2003). One would therefore have expected a clear association between depression and health problems in this study. This expectation was only partly supported by the results discussed here. There were significant negative correlations between depression and specific self-reported health problems, and depression and overall perception of health. However, depression did not emerge as a significant predictor of self-reported health problems in multivariate analyses. This may be due to the specific nature and diversity of some of the problems rated by participants. With a larger sample size, it would be possible to do separate regression analyses for each of the problems; one might then expect depression to predict certain specific health concerns commonly reported by depressed people. When a separate multiple regression analysis was undertaken with difference score (change in health perception from before to after the trauma) as the outcome variable, depression was a very significant predictor of this measure, more so than PTSD in fact (further discussion of this regression analysis follows shortly). This suggests that those individuals suffering from depression have a more negative impression of their current health status, and possibly a falsely optimistic view of how good their health was before the traumatic event(s). Finally, depression failed to be a significant predictor in the logistic regression equation of medical problems reportedly diagnosed by a medical practitioner. Without pre-empting the discussion to follow shortly on this analysis, the relative youth of this sample and recency of the traumatic event(s) may go some way towards explaining this result.

Another somewhat surprising finding was the fact that moderate to heavy smoking and alcohol use were not significantly correlated with the specific health symptoms in the
questionnaire used in this study, nor with participants' perception of their overall health. Neither was current or past drug use (not shown in the correlation matrix at Table 4.5), although the sample size for illicit substance use was quite small (23 current and 16 past users of drugs), which may negate against any findings one way or the other. Smoking has been associated with a number of often-fatal illnesses, including cardiovascular disease, lung and other cancers, chronic bronchitis, and emphysema (see Gold (1995), and Kozlowski, Henningfield, and Brigham (2001) for reviews). Hence one might have expected to see greater reporting of health problems among the smokers in this current study's sample, although it can take "decades" for smoking-related diseases to be identified (Kozlowski et al, 2001), and this is a relatively young sample.

Another explanation relates to how smokers were identified in this study: responses to the smoking-related questions in the health questionnaire were dichotomized, where "yes" indicated current or past moderate to heavy smoking of at least 12 months duration (where moderate to heavy equalled at least 10 cigarettes per day). Given evidence that individuals who cease smoking almost completely reduce their risk of cardiovascular disease and atherosclerosis within five years (Gold, 1995) it may be that any association between smoking and health problems in this study was diluted by the inclusion of these past smokers.

The relatively small sample size limited the number of variables that could be included in the regression equation. With more participants, this study could also have examined whether variables such as trauma type and duration of time since the trauma were significantly associated with poorer physical health. The specific impacts of sexual assault and childhood sexual abuse, in particular, could be examined.

The second hierarchical multiple regression equation aimed to evaluate the contribution of each of the PTSD symptom clusters to self-reported health symptoms, after controlling for the effect of antidepressant medication. Consistent with some of the findings of Woods and Wineman (2004), but contrary to those of Clum et al (2001), Kimerling et al (2000), and Zoellner et al (2000), the avoidance/numbing symptom
cluster was the only one that was a significant predictor of specific health symptoms. Interestingly, Woods and Wineman (2004) based their statement of significance on Pearson r correlations, rather than any form of regression model. If this same form of data analysis had formed the sole basis for assessing significant relationships between the PTSD symptom clusters and health symptoms in this study, then all three clusters would have been considered significantly related to the outcome variable (see Table 4.8 for correlations and levels of significance). A multivariate analysis (as in multiple regression) demonstrating which clusters best predict health problems is a stronger model: while hyperarousal and reexperiencing may be significantly associated with health problems in the bivariate correlation matrix, only the avoidance and numbing cluster remained as a significant predictor once these other clusters and anti-depressant medication were taken into account.

This finding was contrary to the hypothesis that the hyperarousal symptom cluster would be a significant predictor of health symptoms. It may be that avoidance and numbing reduce health compliance behaviours; by avoiding thinking about or going near any reminders of the traumatic event, individuals may also avoid medical practitioners and associated treatment, leading to an increase in health symptoms. Some support for this hypothesis exists in the literature. For instance, Epstein (1993) reported that the avoidant symptoms of PTSD prevented accurate diagnosis of this condition in seriously injured accident victims who were assertively followed up for nine months after their first admission to a community shock-trauma centre. Similarly, Pieper and Maercker (1999) noted that males in certain high-risk occupational groups (eg police, fire department, rescue teams) avoided seeking or accepting psychological help after traumatization.

In addition, suppression of reminders of the event may lead to an increase in somatic symptoms. Avoidance has been linked to increased levels of depression, anxiety, and somatisation symptoms (Tull, Gratz, Salters, and Roemer, 2004), and greater PTSD symptom severity in cancer patients (Jacobsen, Sadler, Booth-Jones, Soeys, Weitzner, and Fields, 2002). Another study of cancer patients found that avoidance was associated
with greater levels of psychological distress in patients with late-stage cancer; additionally a relationship between functional impairment and avoidance was reported among patients with early stage cancer (Manne, Glassman, and Du Hamel, 2000).

Finally, several studies have noted a close relationship between numbing (categorised as part of the avoidance cluster) and the hyperarousal cluster. Feuer, Nishith, and Resick (2005) conducted multiple regression analyses on data collected from a sample of 272 female rape survivors. They theorised that hyperarousal and numbing are functionally related mechanisms, and found that arousal accounted for the majority of the variance in numbing beyond that explained by avoidance and intrusion. Similarly, Tull and Roemer (2003) found that hyperarousal symptoms predicted emotional numbing symptoms above and beyond experiential avoidance. Hence, it is clear that there is a strong interrelationship between at least the numbing components of the avoidance/numbing cluster and the hyperarousal symptom cluster, which may go some way towards explaining why the former cluster was a significant predictor of health problems as opposed to hyperarousal. In other words, one might expect either or both symptom clusters to be associated with health problems if the two are closely related.

The final hierarchical multiple regression model sought to examine the relative contribution of PTSD to change in health perception from before to after the trauma(s), while controlling for age, alcohol use, number of traumatic events, and depression. Once again, it must be noted that this type of variable has its limitations, as it will most likely be affected by recall bias. The hypothesis in this case was that participants with PTSD would rate their general health as being substantially worse after the trauma, compared with those who did not meet diagnostic criteria for PTSD, while controlling for various factors including age, alcohol use, and depression. The results supported this hypothesis. PTSD was a significant predictor of a change in health perception for the worse (p<.01), accounting for 7% of the unique variance in this outcome variable after all other variables had been entered into the equation. This supports previous research findings that a diagnosis of PTSD is significantly associated with poorer perceptions of overall health (eg Kimerling et al, 2000; Kulka et al, 1990; Van Velsen et al, 1996).
Depression was a highly significant covariate \( (p < .001) \), again indicating that participants with a diagnosis of depression (as assessed using the BDI), were much more likely to rate their health as being poorer since the trauma(s). It may be that diagnoses of PTSD and depression lead to an increased – and negative – preoccupation with overall well-being, as well as reflecting the increased reporting of specific health complaints.

4.2.3 Logistic regression analysis: PTSD as a predictor of self-reported medically-diagnosed conditions

The sequential logistic regression analysis in this study examined whether PTSD predicted participants’ reporting of medically diagnosed conditions, while controlling for age and depression. Given work by Boscarino and his colleagues (eg Boscarino, 1997; Boscarino and Chang, 1999b) and Beckham et al (1998), demonstrating a relationship between PTSD diagnosis and medically diagnosed diseases and/or immunological suppression, it was predicted that the PTSD group would report a higher incidence of health conditions diagnosed by a medical doctor. However, the logistic regression analysis did not support this hypothesis, with age being the only predictor variable significantly associated with diagnosed medical conditions. It may be that the sample size in this study was too small for any meaningful trends to emerge. However, a more likely explanation is the fact that the sample is still relatively youthful, with a mean age of 35.9 years. Additionally, an average of 6 months to 4 years had passed since the traumatic event, which may not be enough time for obvious medical conditions to appear. In the Boscarino (1997) study, around 20 years had passed since the participants had been exposed to trauma (in the form of combat). It would be interesting to follow this current study’s sample up in another 10 – 20 years to establish whether PTSD was significantly related to medically-diagnosed conditions at that point. Alternatively, studies of natural killer cell activity and white blood cell counts may prove useful, given evidence of altered immunological responses in traumatised and/or stressed populations, as reviewed in Chapter Two of this thesis (see also Boscarino and Chang, 1999a; Ironson et al, 1997; Inoue-Sakurai et al, 2000; Kiecolt-Glaser et al, 2002).
4.2.4 Limitations

This study appears to be the first known Australian research examining the self-reported health of victims of crime with PTSD. It does have a number of limitations, however. These include the relatively small sample size (although samples of less than 100 are not unusual in PTSD research, see for example Kimerling et al, 2000; Litz et al, 1992; Hankin et al, 1996; Walker et al, 1992; Woods and Wineman, 2004; Zoellner et al, 2000), the inability to obtain objective medical diagnoses of health conditions, and the relatively short duration between occurrence of the trauma and inclusion of participants in this study. Collecting data on health over a lengthy period might establish more conclusively whether medically diagnosed conditions were more prevalent in those victims of crime with PTSD, compared to the non-PTSD group. Additionally, a larger sample size would enable health outcomes in this client group to be analysed separately for each trauma type, and possibly by the duration of time since the traumatic event occurred. These are important variables in their own right, with time since trauma in particular likely to be an important factor in the trajectory of symptoms.

4.2.5 Summary

The three hypotheses relating to health and PTSD in this study were that a PTSD diagnosis (controlling for certain other variables) would be a significant predictor of:

- higher reporting of specific self-rated health symptoms
- worse global health perception since the trauma, compared with prior to the trauma
- greater endorsement of specific medically diagnosed health problems in the health questionnaire

The first two hypotheses relating to this sample of victims of crime were supported by the data. This suggests that PTSD is significantly associated with self-report of specific
health symptoms and general perception of worse overall health since the trauma(s). However, PTSD was not found to be a significant predictor of medically diagnosed health problems, a result that may be partly explained by the small sample size, relative youth of the participants, and the fact that an average of only six months to four years had passed since the traumatic event. More time may be needed for any medical conditions to manifest themselves.

The final hypothesis concerned the impact of the various PTSD symptom clusters on self-reported health symptoms, with a prediction that the hyperarousal cluster would be a significant predictor of this health variable. While all three clusters were significantly negatively correlated with health symptoms, contrary to expectations the avoidance and numbing cluster was the only significant predictor. This may be due to the complex interrelationship between numbing and hyperarousal, and evidence that avoidance is related to increased reporting of somatic complaints and possibly a reluctance to seek medical or psychological assistance.

4.2.6 Conclusion

The results outlined in this chapter support previously documented international findings of a clear association between PTSD and self-reported health problems. PTSD predicted such health problems over and above other factors known to influence health, including age, smoking, alcohol use, depression, and the number of traumatic events. However, there was no evidence of an increase in (self-reported) medically diagnosed conditions among the PTSD group, compared to those without a PTSD diagnosis. Finally, it appeared that the avoidance/numbing cluster of the PTSD symptom categories predicted health problems most strongly, contrary to the hypothesis that the hyperarousal cluster would be most closely associated with self-reported health problems.
Chapter Five: An Examination of Self-Reported Health and PTSD Severity in Victims of Crime Following Counselling

5.1 Results

5.1.1 Objective

This chapter reports the results and interpretation of a follow-up study examining ACT victims’ of crime self-reported health and PTSD severity five to seven months after the initial study described in the preceding chapter. The goal of this follow-up study was to examine firstly whether the specific self-reported health symptoms measured in the first study were reported at a similar rate after counselling intervention, and secondly whether counselling (primarily provided through psychologists and social workers contracted by the Victims’ Services Scheme) resulted in any diminution of PTSD symptoms.

5.1.2 Overview

The first study described in Chapter 4 examined specific self-reported health symptoms, such as chest pain, headaches, etc (“health score 1”) measured on a 6-point Likert-type scale, self-reported medical diagnoses given by doctors since the traumatic event, and perception of health status before and since the traumatic event(s). This follow-up study included an identical continuous measure of self-reported health symptoms (“health score 2”), and another discrete measure of change in health perception, change in health status from after the traumatic event(s) to health status after counselling was received, if applicable (“change in health status”). As with the “difference score” measure utilised in the first study, this latter outcome variable is a highly subjective measure where one may expect some contamination, due to recall bias, of the initial measurement (health status after trauma but before counselling). It may, however, be a useful indicator of whether participants’ perceptions of their overall health are borne out by the more objective health score measure. Participants were not asked to report diagnoses of medical conditions in this second study, as it was felt that the five to seven month time
period that had elapsed between the two studies was insufficient to track any changes in diagnosable diseases or illnesses.

Of the 99 participants in the first study, 59 responded to the second set of questionnaires distributed five to seven months after the initial data collection – a response rate of 59.6%. Two of these did not complete the BDI-II or PDS, leaving 57 respondents for the analyses utilising PTSD severity scores as independent variables. Data were once again analysed using SPSS for Windows, Version 11.5.

An outline of the statistical analyses used in this study is provided next, followed by descriptive statistics and a description of appropriate data screening techniques. The remainder of the results section focuses on bivariate and multivariate data analysis, including Pearson $r$ correlations between variables of interest, and two repeated measures ANCOVAs with health score and PTSD severity score as dependent variables (with further ANOVAs examining the data broken down by counselling duration). Interpretation and discussion of the results is provided in Section 5.2 of this chapter.

### 5.1.3 Outline of statistical analysis

Chi squared analyses were undertaken to establish whether any significant differences existed between those who met criteria for PTSD (PTSD+) as determined through their responses on the PDS, and those who did not (PTSD-) on demographic and trauma-related variables. Independent samples t-tests were then utilised to compare the current study’s responders with non-responders on measures including age, depression, PTSD, and health score. Pearson $r$ correlations were produced to examine the relationships between the two dependent variables, health score 2, and change in health score, and independent variables of interest. A $2 \times 2 \times 2$ repeated measures analysis of covariance (ANCOVA, also often referred to as a split-plot ANCOVA, eg Coakes and Steed, 2005), with depression as covariate and PTSD, counselling status, and time as independent variables, was then undertaken to explore the impact of these variables on health score. T-tests were used to examine simple effects arising from this ANCOVA (post-hoc
analysis). Similarly, a 2x2 repeated measures ANCOVA with depression as covariate and counselling status and time as independent variables was undertaken to examine the effect these variables might have on PTSD severity score. T-tests were again used to explore simple effects, and further repeated measures ANOVAs were undertaken on two smaller groups of participants (categorised by number of counselling sessions), once more examining health score and PTSD severity.

5.1.4 Preliminary Data Screening

Prior to analyses being conducted, data were screened for univariate outliers, non-normal distributions, and missing data using the ‘Frequencies’ and ‘Explore’ functions in SPSS. As previously mentioned, missing data were confined to the non-completion of two PDS and BDI forms. The distributions of data for the continuous variables (PTSD severity score, BDI score, age, and health score) appeared relatively normal, with the Kolmogorov-Smirnov and Shapiro-Wilk tests of normality all being non-significant. A summary of skewness and kurtosis for these variables is provided in Table 5.1.

Table 5.1: Summary of skewness and kurtosis for PTSD severity score, BDI score, age, and health score

<table>
<thead>
<tr>
<th></th>
<th>Skewness</th>
<th>Kurtosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD severity score 2</td>
<td>.268</td>
<td>-.726</td>
</tr>
<tr>
<td>BDI score 2</td>
<td>.361</td>
<td>-.074</td>
</tr>
<tr>
<td>Health score 2</td>
<td>-.510</td>
<td>.025</td>
</tr>
<tr>
<td>Age</td>
<td>.203</td>
<td>-.373</td>
</tr>
</tbody>
</table>

One further discrete variable, change in health status, was abnormally distributed due to the majority of respondents indicating no change in their health from after the trauma to after counselling (0, on a scale from -1 to +3). Skewness was .703 and kurtosis -.229. Both statistical tests of normality were significant, indicating an abnormal distribution. In order to transform this variable using either square root or logarithmic functions, the original variable was recoded to remove the -1 and 0 labels, resulting in a variable with a scale ranging from 1 (change in health for the worse) through to 5 (substantial
improvement in health). However, neither the square root nor logarithmic transformations led to normal distributions that resulted in a non-significant Kolmogorov-Smirnov or Shapiro-Wilk statistic. A logarithmic transformation resulted in skewness of -.133 and kurtosis of .067. It was decided to use this latter transformed variable in the Pearson $r$ correlation matrix.

One outlier was identified for both health score 2 and BDI score 2 (the same participant had a very low health score of 19, and a high BDI score of 55). This participant also was present as an outlier on the first health score variable, recording the lowest health score in the initial study. A decision was taken to retain that case, as it was felt that this score represented genuine distress on the part of that individual after a review of their questionnaires. Similarly, it was decided to retain the data for that participant in this study, particularly given the relatively small number of cases. Re-running selected analyses (the two main ANCOVAs) without this case did not result in substantially different results.

Dichotomous variables were checked for any uneven splits between the categories. As gender consisted of only five males (out of a total of 59 respondents) a decision was taken to exclude this variable from statistical analyses to avoid undue influence being granted to the smaller of the two categories. Another problematic dichotomous variable was counselling (yes or no), with only eight respondents indicating that they did not receive counselling between the two studies. However, given that this was a variable of major interest, it was decided to include this in the analyses, adjusting for unequal sample sizes where relevant, but adding a precautionary note to the results that power (and hence interpretation) would be limited by this factor.
5.1.5 Descriptive Statistics

The mean age of participants who responded to this latter study was 37.8 years (sd = 11.6), with a range from 18 through to 65 years. As mentioned earlier, the sample was predominantly female (n = 54) with only five males responding. This is a similar gender split to that obtained in the first study (88 females and 11 males). A comparison of those participants with (PTSD+) and without PTSD (PTSD-) is provided in Table 5.2, with chi squared analyses used to establish any significant differences between these two groups.

Table 5.2: Characteristics of PTSD positive and PTSD negative groups at the time of this study

<table>
<thead>
<tr>
<th></th>
<th>PTSD+</th>
<th>PTSD-</th>
<th>( \chi^2 )</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;40</td>
<td>13</td>
<td>19</td>
<td>0.34 (df = 1)</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>40+</td>
<td>13</td>
<td>14</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>3</td>
<td>2</td>
<td>0.56 (df = 1)</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Female</td>
<td>23</td>
<td>31</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Source of Participants</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VSS new</td>
<td>4</td>
<td>12</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VOCAL</td>
<td>4</td>
<td>4</td>
<td>1.66 (df = 3)</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>VSS past</td>
<td>9</td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>University</td>
<td>1</td>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smokers (current)</td>
<td>7</td>
<td>13</td>
<td>1.01 (df = 1)</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Current/past alcohol use(^1)</td>
<td>5</td>
<td>4</td>
<td>0.57 (df = 1)</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>Antidepressant medication</td>
<td>16</td>
<td>9</td>
<td>6.99 ( ** ) (df = 1)</td>
<td>p &lt; .01</td>
</tr>
<tr>
<td>Depressed(^2)</td>
<td>21</td>
<td>11</td>
<td>14.04 ( *** ) (df = 1)</td>
<td>p &lt; .01</td>
</tr>
<tr>
<td>Number of traumatic events</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>3</td>
<td>11</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 – 3</td>
<td>8</td>
<td>16</td>
<td>0.95 (df = 2)</td>
<td>p &gt; .05</td>
</tr>
<tr>
<td>4+</td>
<td>7</td>
<td>12</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

\(^{1}\) as defined in ‘Measures’ section in Methodology chapter, ie moderate to heavy alcohol consumption
\(^{2}\) ascertained via BDI scores at time of this study, see ‘Measures’ section in Methodology chapter

Those with PTSD were significantly more likely (p < .01) to be taking antidepressant medication and be depressed at the time of this second study (\( \chi^2 = 6.99\) and 14.04,
respectively). Overall, 31.6% (n = 18/57) of this sample met current criteria for PTSD, as assessed by the PDS at the time of this study.

Table 5.3 provides details of the degree to which various health problems that comprise the health score outcome measure (health score 2) were reported by the participants. For comparison purposes, the data from the first study are also included. This reveals a relatively high rate of indigestion/digestion problems, frequent urination, headaches, chest pain, and breath shortness being reported by this sample, relative to their degree of endorsement in the first study. It is noteworthy that indigestion, in particular, was reported to be a significant problem by almost half of the participants, and appears to reflect greater endorsement of health problems by participants in this study. The following section explores whether there are any significant differences between the responders to this latter study and those participants in the first study who did not respond, either on health score or a number of other variables.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Endorsed (n)</th>
<th>Percentage of Total (Study 2)</th>
<th>Percentage of Total (Study 1)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indigestion</td>
<td>28</td>
<td>47.5</td>
<td>37.4</td>
</tr>
<tr>
<td>Frequent urination</td>
<td>26</td>
<td>44.1</td>
<td>28.3</td>
</tr>
<tr>
<td>Headaches</td>
<td>24</td>
<td>40.7</td>
<td>43.4</td>
</tr>
<tr>
<td>Nausea</td>
<td>22</td>
<td>37.3</td>
<td>39.4</td>
</tr>
<tr>
<td>Chest pain</td>
<td>20</td>
<td>33.9</td>
<td>21.2</td>
</tr>
<tr>
<td>Breath shortness</td>
<td>19</td>
<td>32.2</td>
<td>26.3</td>
</tr>
<tr>
<td>Diarrhoea</td>
<td>18</td>
<td>30.5</td>
<td>24.2</td>
</tr>
<tr>
<td>Unexplained thirst</td>
<td>17</td>
<td>28.8</td>
<td>19.2</td>
</tr>
<tr>
<td>Blurred vision</td>
<td>16</td>
<td>27.1</td>
<td>21.2</td>
</tr>
<tr>
<td>Muscle weakness</td>
<td>16</td>
<td>27.1</td>
<td>26.3</td>
</tr>
<tr>
<td>Coordination problems</td>
<td>14</td>
<td>23.7</td>
<td>22.2</td>
</tr>
<tr>
<td>Pelvic pain*</td>
<td>12</td>
<td>22.2</td>
<td>22.7</td>
</tr>
<tr>
<td>Other</td>
<td>10</td>
<td>16.9</td>
<td>10.1</td>
</tr>
</tbody>
</table>

* Females only
5.1.6 Comparison of responders with non-responders

Independent samples t-tests were undertaken to establish whether responders to the second package of questionnaires (i.e., participants in this current study) differed from non-responders in terms of the severity of PTSD, depression, or health symptoms, or their age. The results are summarised in Table 5.4.

Table 5.4: Independent samples t-tests comparing responders to this study (n = 59) with non-responders (n = 40) on initial mean PTSD severity score, BDI score, health score, and age

<table>
<thead>
<tr>
<th>Variable</th>
<th>Responder mean score (sd)</th>
<th>Non-responder mean score (sd)</th>
<th>df</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD severity score</td>
<td>24.58 (12.63)</td>
<td>25.18 (12.36)</td>
<td>97</td>
<td>0.23</td>
</tr>
<tr>
<td>BDI score</td>
<td>23.85 (12.36)</td>
<td>25.08 (14.83)</td>
<td>97</td>
<td>0.45</td>
</tr>
<tr>
<td>Health score</td>
<td>60.68 (12.73)</td>
<td>61.00 (12.46)</td>
<td>97</td>
<td>0.13</td>
</tr>
<tr>
<td>Age</td>
<td>37.78 (11.60)</td>
<td>33.10 (9.94)</td>
<td>97</td>
<td>-2.09*</td>
</tr>
</tbody>
</table>

Severity of depression and PTSD (as measured by the BDI and PDS, respectively) did not differ significantly between responders and non-responders. Similarly, health (assessed via the health score variable) also was not significantly different. Responders were, however, significantly older than non-responders (t = -2.09, p < .05). Based on these results, one can assume that the sample in this current study is representative of the participants sampled in the initial study, with the exception of age.

5.1.7 Bivariate and Multivariate Analyses

5.1.7.1 Bivariate relationships

Prior to conducting analyses of variance, a Pearson $r$ correlation matrix was produced to explore the relationships between each of the variables of interest in this study (see
Table 5.5). As expected, health scores 1 and 2 were highly positively correlated ($r = .622$, $p < .001$).

Table 5.5: Pearson $r$ Correlations between the main outcome measures and independent variables of interest

<table>
<thead>
<tr>
<th></th>
<th>Change health score logarithmic</th>
<th>health score 1</th>
<th>age</th>
<th>PTSD</th>
<th>depression</th>
<th>health score 2</th>
<th>antidepressant medication</th>
<th>smoking yes or no</th>
<th>alcohol use yes or no</th>
<th>counselling yes or no</th>
</tr>
</thead>
<tbody>
<tr>
<td>change health score</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>logarithmic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>health score 1</td>
<td>.068</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>age</td>
<td>-.325*</td>
<td>-.204*</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTSD</td>
<td>-.116</td>
<td>-.391**</td>
<td>.077</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>depression</td>
<td>-.233</td>
<td>-.365**</td>
<td>.149</td>
<td>.524***</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>health score 2</td>
<td>.078</td>
<td>.622***</td>
<td>-.257*</td>
<td>-.369**</td>
<td>-.450***</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>antidepressant meds</td>
<td>.041</td>
<td>-.345**</td>
<td>-.028</td>
<td>.185</td>
<td>.253</td>
<td>-.396**</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>smoking yes or no</td>
<td>.143</td>
<td>-.061</td>
<td>-.120</td>
<td>.026</td>
<td>-.084</td>
<td>-.413**</td>
<td>.111</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>alcohol use yes or no</td>
<td>.148</td>
<td>-.056</td>
<td>.086</td>
<td>.160</td>
<td>.052</td>
<td>-.279*</td>
<td>.018</td>
<td>.393**</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>counselling yes or no</td>
<td>.262*</td>
<td>-.071</td>
<td>-.049</td>
<td>.057</td>
<td>.254</td>
<td>-.219</td>
<td>.018</td>
<td>.105</td>
<td>.180</td>
<td></td>
</tr>
</tbody>
</table>

* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
*** Correlation is significant at the 0.001 level (2-tailed).

Both health score measures were also significantly negatively correlated with current PTSD and depression diagnoses, indicating lower health scores (ie more symptoms endorsed) in both studies for those with these psychiatric conditions as opposed to those not meeting the relevant cut-offs for these disorders. As with the first study, use of antidepressant medication was significantly negatively correlated with health score 2 ($r = -.396$, $p < .01$). Interestingly, a history of smoking at the time of this study was also
strongly negatively associated with the current health score measure ($r = -.413$, $p < .01$), a result contrary to that found in the first study where no such relationship was observed. Similarly, alcohol use was also negatively correlated with health score 2 ($r = -.279$, $p < .05$). These correlations suggest that lower health scores are associated with moderate to heavy tobacco and alcohol use. Finally, the transformed change in health perception score was positively associated with counselling status ($r = .262$, $p < .05$), although the latter variable was not significantly correlated with either of the health score measures. This suggests that those who received counselling perceived their overall health after counselling to be somewhat better, although caution should be used in interpreting this result because of the small number of non-counselling participants.

5.1.7.2 Repeated Measures Analysis of Covariance: Health Score 2

A repeated measures ANCOVA (with depression as a covariate) was undertaken to establish whether there were any substantial differences in health scores 1 and 2 between the PTSD+ and PTSD- groups who received counselling, as opposed to those who did not. This can be conceptualised as a $2 \times 2 \times 2$ factorial model, with depression as a covariate, and health scores as the dependent variable. This mixture of between groups and within groups factors is sometimes referred to as a split-plot ANOVA, or SPANOVA (eg Coakes and Steed, 2005). Other factors impacting on health were not included as covariates due to the relatively small sample size. Once again, caution needs to be observed in interpreting the results due to limited power with respect to the counselling status variable (with a small $n$ of 8 participants who did not receive counselling between the two studies). SPSS adjusted for these unequal sample sizes using Type III sums of squares. A graphical representation of the mean (unadjusted) health scores by PTSD and counselling group can be found in Figure 5.1. The lowest health scores in both studies (indicating greater endorsement of health problems) are evident for those participants with PTSD, as compared with the no-PTSD group. The graph also shows that health score appears to fall (worsen) slightly between the initial and current study for those without PTSD who received counselling. There is little apparent change in health score for any of the other
groups, and certainly no evidence of an improvement in health score over time in either the PTSD+ or PTSD- groups.

![Graphical representation of mean health scores](image)

Figure 5.1: Graphic representation of mean health scores for the PTSD and non-PTSD groups, by counselling status

Table 5.6 summarises the results of the repeated measures ANCOVA. Partial Eta squared is included as a measure of effect size. The assumption of homogeneity of variance was met, as was the assumption of homogeneity of variance-covariance matrices (assessed using the Box’s M statistic). The main effect for time is not significant (p > .05), indicating that there was no significant change in health score over time. However, there is a significant main effect for PTSD (F = 12.96, p < .01), indicating a significant difference in mean health scores between the PTSD and non-PTSD groups, and confirming that those with PTSD have lower (worse) health scores than those participants without PTSD. Counselling was not significant, suggesting that health scores were not affected by receipt or non-receipt of counselling. Once again, caution needs to be exercised in interpreting this result due to the small sample size of the non-counselling group. None of the interactions were significant.
Table 5.6: Results of repeated measures (Split-Plot) ANCOVA on health score, with PTSD, time, and counselling status as factors, and depression (study 2) as covariate

<table>
<thead>
<tr>
<th>Source</th>
<th>Type III SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>partial Eta²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>2.97</td>
<td>1</td>
<td>2.97</td>
<td>.04</td>
<td>.001</td>
</tr>
<tr>
<td>PTSD</td>
<td>1196.72</td>
<td>1</td>
<td>1196.72</td>
<td>12.96**</td>
<td>.200</td>
</tr>
<tr>
<td>Counselling</td>
<td>172.32</td>
<td>1</td>
<td>172.32</td>
<td>1.87</td>
<td>.035</td>
</tr>
<tr>
<td>Depression</td>
<td>165.98</td>
<td>1</td>
<td>165.98</td>
<td>1.80</td>
<td>.033</td>
</tr>
<tr>
<td>Time*PTSD</td>
<td>32.01</td>
<td>1</td>
<td>32.01</td>
<td>0.51</td>
<td>.010</td>
</tr>
<tr>
<td>Time*Counselling</td>
<td>30.17</td>
<td>1</td>
<td>30.17</td>
<td>0.49</td>
<td>.009</td>
</tr>
<tr>
<td>Time*Depression</td>
<td>86.90</td>
<td>1</td>
<td>86.90</td>
<td>1.40</td>
<td>.026</td>
</tr>
<tr>
<td>Counselling*PTSD</td>
<td>140.58</td>
<td>1</td>
<td>140.58</td>
<td>1.52</td>
<td>.028</td>
</tr>
<tr>
<td>Error (Time)</td>
<td>3247.80</td>
<td>52</td>
<td>61.28</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Error (BS effects)</td>
<td>9881.33</td>
<td>52</td>
<td>186.44</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

** Significant at p<.01

To explore in further detail whether the means for the various combinations of between-groups factors differed for health score 2, post-hoc analyses in the form of t-tests were undertaken (as per Coakes and Steed, 2005). Due to both limited power (small number of non-counselling participants) and multiple t-tests being conducted, a conservative p value of .01 was chosen to represent a significant result. Table 5.7 summarises these results: the headings ‘In group mean’ and ‘Not in group mean’ refer to whether the mean score for the unbracketed group was part of that group (eg PTSD yes = 49.95 where counselling = yes) or not.

Table 5.7: Simple effects analyses (t-tests) on health score 2 by groups

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>‘In group’ mean</th>
<th>‘Not in group’ mean</th>
<th>df</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTSD (counselling = yes)</td>
<td>50</td>
<td>49.95</td>
<td>60.54</td>
<td>48</td>
<td>3.12**</td>
</tr>
<tr>
<td>PTSD (counselling = no)</td>
<td>9</td>
<td>54.00</td>
<td>71.60</td>
<td>7</td>
<td>2.83*</td>
</tr>
<tr>
<td>Counselling (PTSD = yes)</td>
<td>26</td>
<td>49.95</td>
<td>54.00</td>
<td>24</td>
<td>0.58</td>
</tr>
<tr>
<td>Counselling (PTSD = no)</td>
<td>33</td>
<td>60.54</td>
<td>71.60</td>
<td>31</td>
<td>2.15*</td>
</tr>
</tbody>
</table>

** Significant at p<.01
* Significant at p<.05
The results show that there was a significant difference in health score 2 for the group who received counselling, by PTSD status. Those with PTSD who received counselling had substantially lower (worse) health scores than those participants without PTSD who also received counselling. One can also point to a possible trend towards lower health scores in the PTSD+ group who did not receive counselling (54), compared with those participants without PTSD who also did not receive any counselling (71.6), although a sample size of nine really is not sufficient to report trends with any confidence. These results are not surprising, and reflect the main effect for PTSD found in the ANCOVA. There did not appear to be any significant difference in health scores for the group with PTSD depending on whether or not they received counselling, although there was a trend for health scores to be lower (worse) for the group who did receive counselling (54 and 49.95, respectively). In fact, the PTSD+ counselling+ group had the lowest overall mean health score in this current study at 49.95. Similarly for those without PTSD, counselling appeared to result in lower health scores (60.54 for those who received counselling as opposed to 71.6 for those who did not). With the exception of the first t-test (examining health scores for those with and without PTSD who received counselling), the results for the latter three post-hoc analyses need to be treated as indicative of possible trends only due to issues with sample size.

5.1.7.3 Repeated Measures ANCOVA: PTSD severity score

A second repeated measures ANCOVA with depression as a covariate was conducted to establish whether the dependent variable PTSD severity score (taken from the PDS) varied over time according to counselling status. This may be represented as a 2 (counselling) x 2 (time) factorial ANCOVA. Again, this analysis has limited power due to the small size of the non-counselling sample, so results need to be interpreted with caution. The assumption of homogeneity of variance was met, as was the assumption of homogeneity of variance-covariance matrices (assessed using the Box's M statistic). A graph of the mean scores for each group at each time (adjusted for depression) is displayed in Figure 5.2. This appears to show a reduction in the severity of PTSD
symptoms (PTSD severity score) over time, with the most substantial decrease occurring in the non-counselling group.

This apparent reduction in scores over time was confirmed by the ANCOVA (see Table 5.8), which shows a main effect for Time ($F = 5.08, p < .05$). It should be noted, however, that a partial $\eta^2$ of only .087 indicates a low strength of association or effect size here. There was no main effect for Counselling, which suggests that counselling has little impact on PTSD severity score. The covariate (depression) was significant, an unsurprising result given the high comorbidity with PTSD. None of the interactions were significant.
Table 5.8: Results of repeated measures (Split-Plot) ANCOVA on PTSD severity score, with counselling status as a factor, and depression (time2) as covariate

<table>
<thead>
<tr>
<th>Source</th>
<th>Type III SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>partial Eta²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>309.87</td>
<td>1</td>
<td>309.87</td>
<td>5.08*</td>
<td>.087</td>
</tr>
<tr>
<td>Counselling</td>
<td>10.56</td>
<td>1</td>
<td>10.56</td>
<td>0.13</td>
<td>.002</td>
</tr>
<tr>
<td>Depression</td>
<td>1866.26</td>
<td>1</td>
<td>1866.26</td>
<td>23.16***</td>
<td>.304</td>
</tr>
<tr>
<td>Time*Counselling</td>
<td>4.11</td>
<td>1</td>
<td>4.11</td>
<td>0.07</td>
<td>.001</td>
</tr>
<tr>
<td>Time*Depression</td>
<td>0.87</td>
<td>1</td>
<td>0.87</td>
<td>0.01</td>
<td>.000</td>
</tr>
<tr>
<td>Counselling*Depression</td>
<td>143.76</td>
<td>1</td>
<td>143.76</td>
<td>0.89</td>
<td>.017</td>
</tr>
<tr>
<td>Time<em>Counselling</em>Depression</td>
<td>64.56</td>
<td>1</td>
<td>64.56</td>
<td>1.06</td>
<td>.020</td>
</tr>
<tr>
<td>Error (Time)</td>
<td>3235.41</td>
<td>53</td>
<td>61.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Error (BS effects)</td>
<td>4270.16</td>
<td>53</td>
<td>80.57</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Significant at p<.05
*** Significant at p<.001

Post hoc analyses of selected results (where mean differences appeared greatest) were conducted using t-tests. The first examined whether PTSD severity at time 2 (ie this current study) differed substantially between the counselling and non-counselling groups. This did not reveal any significant difference (t = -1.54, p > .05). The second investigated whether PTSD severity score differed over time for those participants who did receive counselling. The result was not significant (t = 1.59, p > .05). Together these results further suggest that counselling has little impact on PTSD severity scores.

5.1.7.4 Repeated measures ANOVAs examining health scores by PTSD diagnosis and number of counselling sessions

Given the results reported in Section 5.1.7.3, a decision was taken to explore whether PTSD diagnostic status had any impact on health score over time for those participants who received no or relatively few (less than eight) sessions of counselling, as opposed to more extensive counselling (eight or more sessions). Separate repeated measures ANOVA were undertaken for each scenario. Analysis of covariance was not utilised given the relatively small sample size. In both cases, the assumptions of homogeneity of
variance and homogeneity of variance-covariance matrices (assessed using the Box’s M statistic) were met. The relevant means are displayed in Table 5.9, below.

Table 5.9: Mean health scores by PTSD diagnosis and counselling duration

<table>
<thead>
<tr>
<th></th>
<th>0 – 7 sessions counselling (n=24)</th>
<th>8+ sessions counselling (n=34)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Health Score 1</td>
<td>Health Score 2</td>
</tr>
<tr>
<td>PTSD yes</td>
<td>50.89</td>
<td>56.22</td>
</tr>
<tr>
<td>PTSD no</td>
<td>66.60</td>
<td>64.13</td>
</tr>
</tbody>
</table>

The summary table for the repeated measures ANOVA run for 0 – 7 sessions of counselling is displayed in Table 5.10. The main effect for Time is not significant, indicating that there is no real difference in health score over time for the group as a whole. There is a significant main effect for PTSD (F = 8.45, p < .05), suggesting that – consistent with the previous analyses – those with PTSD have significantly lower (worse) health scores than those without this condition. Health score appeared to improve (ie increase) over time for the PTSD group, while worsening slightly for the non-PTSD group. Post-hoc analysis (paired samples t-tests) showed, however, that the improvement for the PTSD group was non-significant (t = -1.22, p > .05). Further repeated measures analysis of variance was undertaken excluding those participants who did not receive any counselling (ie, 1 – 7 sessions only; analysis not shown here); this supported the results reported above, with the PTSD group showing a trend towards improved health scores over time, while the non-PTSD group reported a more substantial reduction in health scores than is apparent when the no counselling participants are included (ie 0 – 7 sessions). There were no significant main effects for time or PTSD. Due to the small sample size, post-hoc analyses were not undertaken, however it should be noted that health scores for the PTSD group went from 49 at Study 1 to 58 at Study 2.
Table 5.10: Results of repeated measures ANOVA on health score for participants who received 0–7 sessions of counselling, with PTSD as a factor

<table>
<thead>
<tr>
<th>Source</th>
<th>Type III SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>partial Eta$^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>23.11</td>
<td>1</td>
<td>23.11</td>
<td>0.32</td>
<td>.014</td>
</tr>
<tr>
<td>PTSD</td>
<td>1569.40</td>
<td>1</td>
<td>1569.40</td>
<td>8.45$^*$</td>
<td>.278</td>
</tr>
<tr>
<td>Time*PTSD</td>
<td>171.11</td>
<td>1</td>
<td>171.11</td>
<td>2.34</td>
<td>.096</td>
</tr>
<tr>
<td>Error (Time)</td>
<td>1605.87</td>
<td>22</td>
<td>72.99</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Error (BS effects)</td>
<td>4085.91</td>
<td>22</td>
<td>185.72</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Significant at p<.05

The ANOVA summary table for those participants who received eight or more sessions of counselling is shown in Table 5.11. On this occasion, there was a significant main effect for Time (F = 13.34, p < .01), although not in the direction that one might have expected. Participants in both the PTSD and non-PTSD groups appeared to report more health symptoms at time 2, indicated by lower (worse) health scores. There was also a significant main effect for PTSD (F = 13.67, p < .01) indicating once again that participants with PTSD had lower health scores overall than those without PTSD. The interaction was not significant. Post-hoc analysis (using a paired sample t-test) comparing health scores over time for the PTSD group (n=16) indicated that the difference between the means was significant (t = 2.45, p < .05), with health score significantly lower (worse) at time 2 after 8 or more sessions of counselling had been received.

Table 5.11: Results of repeated measures ANOVA on health score for participants who received 8 or more sessions of counselling, with PTSD as a factor

<table>
<thead>
<tr>
<th>Source</th>
<th>Type III SS</th>
<th>df</th>
<th>MS</th>
<th>F</th>
<th>partial Eta$^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>587.50</td>
<td>1</td>
<td>587.50</td>
<td>13.34$^{**}$</td>
<td>.294</td>
</tr>
<tr>
<td>PTSD</td>
<td>2890.65</td>
<td>1</td>
<td>2890.65</td>
<td>13.67$^{**}$</td>
<td>.299</td>
</tr>
<tr>
<td>Time*PTSD</td>
<td>0.21</td>
<td>1</td>
<td>0.21</td>
<td>0.01</td>
<td>.000</td>
</tr>
<tr>
<td>Error (Time)</td>
<td>1409.56</td>
<td>32</td>
<td>44.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Error (BS effects)</td>
<td>6764.89</td>
<td>32</td>
<td>211.40</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

$^{**}$Significant at p<.01

Significant at p<.05
The overall interpretation of these additional ANOVAs, while bearing in mind the relatively small sample size, is that those individuals with PTSD who receive few or no sessions of counselling show a trend towards an improvement in their health score, while those who receive more counselling sessions actually appear to report more health symptoms, as shown by a lower health score.

5.1.7.5 Paired Samples t-tests for PTSD severity score by counselling duration

If health scores appear to worsen over time for participants who receive more counselling, one might ask what would happen to their PTSD severity scores (regardless of whether or not they meet diagnostic criteria for PTSD). Paired samples t-tests were run to investigate any differences in PTSD scores depending on whether participants received no or less than eight sessions of counselling, or eight or more sessions. The results are displayed in Table 5.12 below.

Table 5.12: Paired samples t-tests examining mean PTSD severity scores for the 0 – 7 and 8+ sessions of counselling groups

<table>
<thead>
<tr>
<th>No. counselling sessions</th>
<th>PTSD severity score 1 (sd)</th>
<th>PTSD severity score 2 (sd)</th>
<th>df</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 – 7 sessions</td>
<td>22.26 (15.18)</td>
<td>14.70 (12.03)</td>
<td>22</td>
<td>3.13**</td>
</tr>
<tr>
<td>8+ sessions</td>
<td>25.56 (10.61)</td>
<td>25.24 (12.92)</td>
<td>33</td>
<td>0.19</td>
</tr>
</tbody>
</table>

The t-tests show that, for those participants who receive no or up to seven sessions of counselling, their PTSD severity score drops significantly by time 2, indicating an improvement in their PTSD symptoms (t = 3.13, p < .01). However, there appears to be no change in PTSD severity scores for those who receive eight or more sessions of counselling, which suggests that a longer duration of counselling does not result in a reduction in an individual’s PTSD symptoms. Once again, further analysis (paired samples t-tests, results of which are not shown here) conducted on the short-term counselling group, excluding those who did not receive any counselling, supported the
results reported above, with a substantial drop in PTSD severity score over time (23.5 to 15.6), although this was a trend with $p = .058$.

5.2 Discussion

5.2.1 Initial hypotheses and summary of results

This second study sought to follow up participants in the initial health study, in order to examine the impact of counselling on PTSD symptomatology and specific self-reported health symptoms. Two particular issues were examined via repeated measures analyses of covariance:

- Whether those individuals who received counselling reported any change in specific health symptoms at time two (the time of this current study), with particular attention given to the impact counselling has on the PTSD group. It was expected that health scores might improve marginally (given the short time-frame of 5 – 7 months between the studies) for the PTSD group who received counselling, with little change evident for the non-PTSD group, for reasons canvassed at the end of Chapter 2. Briefly, these related to evidence that PTSD is associated with an underlying pathological process and impaired immunological responses – in effect, real health problems as opposed to somatisation alone. Although PTSD symptoms might improve following counselling (see next point), it may take some time for health symptoms to show a corresponding improvement. This prediction was partially supported by the results (which should be interpreted with a degree of caution, given the small sample size), with those meeting criteria for PTSD who received shorter-term counselling (up to 7 sessions) appearing to report a slight improvement in health symptoms over time, while those participants without PTSD reported more symptoms. However, longer-term counselling (over 8 sessions) was associated with a worsening of health scores over time for both the PTSD and non-PTSD groups. Possible reasons for this will be explored shortly.

- Whether the severity of PTSD symptoms (as measured through the PDS) is reduced to a greater extent in the counselling group as opposed to the group who did not
receive counselling. One would expect that appropriate counselling would lead to a reduction in PTSD symptoms, given extensive evidence that CBT-based treatment with an exposure component is most effective at treating PTSD (see Foa and Meadows, 1997, and Resick and Calhoun, 2001, for reviews). Again, this expectation was met partially, in an unexpected way, with a substantial improvement in PTSD severity scores evident in the short-term, but not long-term, counselling group. It should also be noted, however, that a similar improvement in PTSD severity was noted in the small number of participants who did not receive any counselling, although the majority of these were non-treatment seeking individuals without PTSD.

In an attempt to control for the effect of depression, which has a clear and well documented impact on both physical health and health perception, while often co-morbid with PTSD, this factor was included as a covariate in the two ANCOVAs.

5.2.2 Sample characteristics

This sample had much in common with the original, larger group sampled in study one, but also differed from this latter group in a number of interesting ways. The current PTSD prevalence rate was 31.6%, which compared with 44.3% for the entire sample in the first study. This suggests that the number of individuals diagnosed with PTSD has actually declined over time, particularly when t-tests did not find any differences between responders to this latter study and non-responders from the original sample on PTSD severity score. Such a decline is consistent with research showing that – even without therapeutic intervention – a certain proportion of individuals with PTSD will recover, or no longer meet DSM-IV criteria, within twelve months of symptom onset (eg Rothbaum et al, 1992). In other words, this can be attributed to natural remission of symptoms.

In relation to other characteristics of this group, there was a non-significant trend for smoking to be more prevalent amongst those without PTSD than those with this
disorder, while approximately one third of the sample reported experiencing four or more traumatic events in their lifetime. These statistics are consistent with findings from the first study.

An examination of the specific health symptoms reported by respondents in this current study shows that certain symptoms were endorsed more frequently now compared with the first study. These include indigestion or digestive problems (47.5% compared with 37.4% in the first study), frequent urination (44.1% compared with 28.3%), and chest pain (33.9% compared with 21.2%). This is an interesting finding, and is consistent with the overall greater endorsement of health symptoms in this sample, as will be discussed shortly in the context of an analysis of covariance (see Section 5.2.3). It is somewhat surprising given the reduction in the percentage of individuals meeting criteria for PTSD, as there appears to be a strong relationship between the two variables. It may be the case that some health symptoms become more pronounced during counselling (given that the majority of individuals in this sample received some form of counselling between the two studies), due to an increased awareness of bodily responses through behavioural monitoring and relaxation training, for instance. It could also be that an initial increase in anxiety and PTSD symptoms may result from desensitisation (and hence re-exposure) to the original trauma, leading again to greater anxiety-related physical symptoms such as chest pain and digestive problems. Individuals may simply become more focussed on their symptoms, with a relatively short time-frame of 5 – 7 months between the two studies being insufficient to track any improvement in this area. However, this does not necessarily explain the reasons for greater endorsement of ‘frequent urination’, although this may also be an anxiety-related phenomenon.

Finally, correlations between variables of interest appeared to show that the transformed health perception variable which examined the change in an individual’s perception of their health from before to after counselling (if applicable) was not correlated with the health score variable in this current study. This would suggest that there is no direct relationship between the two. However, the transformed health perception variable was positively associated with counselling, which may indicate that those individuals who
receive counselling perceive their health to be better, overall, than those who did not receive counselling. This is at odds with the increased reporting of specific health problems between studies one and two mentioned in the previous paragraph, and may reflect a subjective “feel-good” factor associated with receiving counselling. Caution needs to be exercised on two fronts: firstly, the size of the non-counselling sample is very small, and secondly the health perception variable is highly subjective and potentially effected by recall bias. It is worth bearing this result in mind, however, when considering the outcomes of the analysis of covariance examining health score.

Interestingly, smoking and alcohol use were negatively correlated with health score 2, a result expected but not forthcoming in the first study. One would expect moderate to heavy use of alcohol and tobacco to have deleterious effects on health. It is unclear why a relationship between use of these substances and poor health is present in this follow-up study (consisting of a smaller sample size), but not the initial slightly larger one, particularly given the fact that identical measures were used to assess smoking and alcohol use. It may simply be a vagary of the overall fairly small sample size in both studies.

5.2.3 Interpretation of ANCOVA/ANOVA results

The results of the two ANCOVAs were somewhat surprising, and at first glance contrary to the hypotheses that counselling would a) lead to marginally improved health scores, and b) result in a substantial reduction in PTSD symptom severity. In brief, it appears from this follow-up study that longer-term counselling not only has a negligible effect on health symptom reporting, but also that PTSD severity is improved more by shorter-term counselling than assistance provided over a longer duration. These findings need to be interpreted in the context of a small sample size, however, and hence viewed with caution. More detailed interpretations of these results follow.

Counselling overall did not appear to lead to improved health scores for either those individuals with PTSD, or those who did not meet criteria for this condition. In fact,
there was a trend towards lower (worse) health scores for all participants who received counselling. A post-hoc hypothesis that this result may be due to the majority of counselling being relatively short-term (and therefore yet to have an impact) was tested by conducting separate repeated measures ANOVAs for no or few sessions of counselling (up to 7 sessions) and more extensive counselling (8 or more sessions). The results of these analyses (and further analyses undertaken excluding those participants who did not receive any counselling) did not support the post-hoc hypothesis, rather its reverse. In other words, those participants with PTSD receiving up to 7 sessions of counselling reported a trend towards improved health scores, while those without PTSD reported more health symptoms, which is line with the original hypothesis for counselling regardless of duration. These gains were lost once participants received 8 or more sessions of counselling, with the PTSD group reporting significantly more health symptoms (in other words, a lower health score) at time 2 (p = .027). Health score also declined for the group without PTSD who received longer-term counselling.

The interpretation from this is that a longer duration of counselling leads to a worsening of self-reported health, while shorter-term or no counselling is associated with marginally improved health scores for the PTSD group. However, one limitation of these data is the size of the sample (n = 34 for the longer duration of counselling group, and n = 32 for the no or few sessions of counselling group). Replication of this study with a substantially larger sample would be useful to ascertain if the same results were reproduced. Notwithstanding this, there may be a number of other reasons for these surprising results. One of the most likely possibilities is that longer-term counselling may actually sensitize individuals' perceptions of their health, causing them to focus on and potentially magnify their symptoms as counselling progresses. The increased reporting of specific health symptoms by the time of Study Two discussed earlier (see page 126) supports this theory to some extent, with participants possibly focussing on their physical health problems to a greater degree. Shorter-term counselling may be more focussed, structured, and ultimately more effective in dealing with participants' presenting problems than counselling over a greater time-frame, which could potentially
lose focus as individuals (particularly those with more complex problems, see next paragraph) dwell on other issues or earlier problems.

Another possible factor relates to the nature of this sample, comprising a large proportion of domestic violence, child sexual abuse, and other assault victims who may have been subjected to repeated acts of terror and trauma. The descriptive statistics show that around one third of this sample reported experiencing four or more traumatic events. As such, this client group may be fairly complex, having been exposed to multiple traumatic incidents and potentially having comorbid psychiatric conditions such as personality disorders and other anxiety disorders. Unfortunately (to avoid deterring potential participants by giving them more questionnaires or screening procedures), this study only assessed whether depression was present in addition to PTSD. Regardless, it may be the case that this group presents particular challenges and may require extensive counselling before a lasting improvement in health or PTSD symptomatology is noticed. It is possible that those who benefited from shorter-term counselling were the minority who reported only one or two traumatic events in their lifetime. Resick and Calhoun (2001) note that most of the research on treatment for PTSD has been done with single trauma survivors, and suggest that ‘more complicated’ individuals could take longer to improve, particularly if child sexual abuse is a past factor (as is the case with a substantial minority of this sample). Some researchers go further in suggesting that the traditional PTSD framework is ill-suited in the context of unrelenting violence against women, with consequences for developing appropriate interventions (eg Mechanic, 2004). However, a number of recent treatment outcome studies focussing on complex clients with PTSD have returned encouraging results for brief cognitive-behavioural treatments. Hembree, Cahill, and Foa (2004) found that female rape or assault survivors (including those who had suffered childhood sexual abuse) with comorbid personality disorders reported a similar reduction in PTSD symptoms by the end of a CBT-based course of counselling as those rape or assault victims without these comorbid disorders. The duration of therapy appeared to be nine weeks (this was unclear from the description; however the control group was assigned to a nine week ‘wait-list control’ condition). One might therefore expect health outcomes to also improve along with
PTSD symptomatology. Hembree et al (2004) also noted that there were no differences in outcomes between those who were treated by CBT experts compared with community-based therapists, with both receiving identical training in the therapy utilized.

Similarly, Resick, Nishith, and Griffin (2003) reported that CBT-based treatment (either cognitive-processing therapy or prolonged exposure) was effective in reducing PTSD, depression, and complex PTSD symptoms in female rape victims with extensive trauma histories. Those reporting a history of child sexual abuse improved at the same rate as those without such a history, suggesting that cognitive-behavioural therapies are effective for clients with complex trauma histories and symptom patterns.

Notwithstanding the results reported from the previous two studies (above), it is also possible that a client group of this nature may still be in unstable or dangerous environments, and/or experiencing other stressful situations such as custody battles, accommodation problems, difficulties with violent ex-partners, and so forth. In these circumstances, counselling would often need to address the immediate crises to ensure stability and some degree of safety for the individual in order for them to benefit from counselling. This does not explain, however, the initial trend towards an improvement in health scores for those who received fewer sessions of counselling.

Another explanation that needs to be considered is the effectiveness of the counselling provided. ‘Counselling’ was not defined in this study, and could have comprised any form of counselling or therapy (or even ‘ventilation’ with little therapist input), rather than the evidence-based CBT with an exposure component that is known to be effective in treating PTSD and its associated problems. Counselling was provided by independently practising psychologists and social workers, with the former group comprising the majority of the therapists, contracted by the Victims’ Services Scheme (VSS) to treat victims of crime who were assessed by the VSS as needing some form of psychological intervention. The VSS do not mandate the type of therapy provided. Once again, it may the case that short-term counselling could be more evidence-based,
structured, and effective, while counselling over the longer term may lose specificity and focus. Participants may ‘lose’ the initial early gains by becoming sensitized to their health symptoms.

A simpler and related explanation – borne out by the second repeated measures ANCOVA – is that many participants are still suffering from PTSD even after counselling has been provided. Findings from the first study supported previous research linking poor self-reported health with PTSD, rather than the experience of trauma itself. Hence, if participants were still experiencing PTSD symptoms, it is unlikely that their health would undergo any noticeable improvement. Results from this second study appear to show a significant reduction in PTSD severity scores between the two studies, particularly for the non-counselling group (although no main effect was evident for counselling, and the main effect for Time was weak, with a small effect size). However, paired samples t-tests examining PTSD scores by counselling duration revealed that the most substantial reduction in these scores occurred for the 0 – 7 sessions counselling group (maintained when the ‘no counselling’ individuals were removed from this analysis), with no change in severity scores for the group who received longer counselling. These results might help to explain the lack of impact that more extensive counselling appears to have on self-reported health: PTSD symptoms appear to improve initially, then worsen to the extent that they are very similar in severity to those that existed before counselling. The reasons why this might be mirror those explored above, specifically:

- Small sample size
- Counselling over the longer-term may lead to an increased focus on the person’s current condition, with a corresponding increase in PTSD symptoms
- Shorter-term counselling may be more focussed and effective in treating PTSD and associated symptoms
- Complexity of this client group
- The counselling/assistance provided – particularly over a longer period of time - is not effective in reducing PTSD symptoms
5.2.4 Summary and conclusions

In summary, the results from this follow-up study suggest that the counselling received by these participants is only beneficial in terms of reducing PTSD and self-reported health symptoms over time if it is short-term in nature. Paradoxically, it even appears as if longer-term counselling produces worse outcomes than no or few sessions of counselling in this sample of traumatised individuals. These results are potentially attributable to the complex nature of the client group, the majority of whom have experienced multiple traumas, and the type of counselling provided, which either may not be evidence-based or best-practice therapy for PTSD, or may lead to an increased focus on PTSD and health symptoms to the detriment of the individual’s overall well-being. Interestingly, a significant positive correlation between the transformed health perception variable and counselling indicates that participants generally feel that their health and well-being is better after receiving counselling. This suggests that counselling has an intrinsic “feel-good” factor associated with it, where individuals perceive their health to be better afterwards, without necessarily resulting in an improvement in more objective measures, namely health and PTSD severity scores. The need to focus on specific, quantifiable measures in seeking feedback about the efficacy of counselling is highlighted through this result.

A number of limitations of this study may also impact upon interpretation of the results. These relate primarily to the sample size, which overall was relatively small given the number of data cells and statistical analyses. In particular, it is difficult to compare outcomes for those who received counselling with those who did not as the number of participants in the non-counselling category was very small, at $n = 9$. This reflects the difficulties associated with accessing victims of crime who may have PTSD, but do not present for counselling or other forms of assistance. Ethically, it was not possible to place some of the participants in a ‘wait-list’ control group, given the fact that the Victims’ Services Scheme is expected to assign treatment-seeking individuals to a counselling provider as soon as practicably possible.
The other limitation concerns the inability to ascertain (or in some way control) the nature of the counselling being provided, as may occur in more controlled studies. This makes it difficult to establish whether the worsening of symptoms as counselling duration increases is due to the quality of the counselling, or the complex nature of the client group. Additionally, it was not possible to gather information about the number of clients dropping out of counselling for whatever reasons (felt better, therapy too confronting, not ready, etc) which could have impacted on the health and PTSD severity scores. Further research administered through a setting which strictly controls the type of counselling and therapy provided would be useful.
Chapter Six: General Discussion

This thesis has outlined and investigated a number of issues relating to the self-reported health of victims of crime with posttraumatic stress disorder (PTSD). The first two chapters reviewed the literature on this topic, focussing initially on the concept of PTSD as being an often chronic condition that may develop following exposure to a traumatic event. Studies such as those undertaken by Kessler et al (1995) have demonstrated that exposure to trauma is relatively common, and in fact many individuals go on to experience multiple traumatic events. The lifetime prevalence of PTSD following exposure to trauma appears to vary according to the nature of the event, with rape, domestic violence, and torture appearing to lead to diagnoses of PTSD in around 30% - 50% of exposed individuals (Van Velsen et al, 1996, Resnick et al, 1993, Gleason et al, 1993). Given these statistics, it was decided in this dissertation to focus on victims of crime, who often present for treatment after physical and sexual assault, including in the context of domestic violence. A high incidence of PTSD was therefore expected, allowing a reasonable sample size to be obtained for the PTSD condition.

The second chapter focussed on the possible health consequences of having PTSD, a disorder characterised by continued cognitive re-experiencing, hyperarousal, and consequent elevation in some stress hormones. Pioneering research by Wolfe, Schnurr and their colleagues (eg Wolfe et al, 1994; Friedman and Schnurr, 1995) demonstrated that PTSD – rather than exposure to trauma per se – was the prime mediating variable in the development and/or reporting of physical health problems following trauma. Issues then canvassed in subsequent research included whether the health compromising behaviours often associated with PTSD (such as smoking, alcohol abuse, poor lifestyle habits) were in fact primarily responsible for any health problems, or indeed whether the reported health symptoms were indicative of a medical problem at all, as opposed to somatisation. More sophisticated studies with larger sample sizes that controlled for possible confounding variables (primarily health-compromising behaviours and depression) continued to find a direct relationship between PTSD and poor self-reported

Research by Boscarino and colleagues (Boscarino, 1997, Boscarino and Chang, 1999a and 1999b), again controlling for an extensive number of confounding variables, established that Vietnam veterans with a current or past diagnosis of PTSD were more likely to present with specific diseases and elevated white blood cell counts than veterans without this disorder. The latter finding has also been reported in survivors of natural disasters (Ironson et al, 1997, and Inoue-Sakurai et al, 2000). Finally, several studies using medical practitioners to establish the presence or absence of ill health in participants with PTSD have found a direct link between the diagnosis of medical conditions and concurrent PTSD (eg Deykin et al, 2001, Boscarino, 1997, Beckham et al, 1998), although some have not (Shalev et al, 1990, McFarlane et al, 1994).

The mechanisms by which PTSD might be implicated in poorer health functioning are unclear, but may be associated with the ongoing reexperiencing and hyperarousal symptoms which form part of this disorder, including sleep problems and increased anger and irritability. Some studies have found that the reexperiencing and/or hyperarousal symptom clusters account for the majority of health problems reported by individuals with PTSD (Woods and Wineman, 2004, Clum et al, 2001, Zoellner et al, 2000, Kimerling et al, 2000). There is also the likelihood that the additive effect of tobacco and alcohol use, comorbid depression, and other health-compromising behaviours would also impact negatively on health. This is explored later in this chapter when discussing the results reported in this thesis.

Interestingly, no published studies were able to be located examining the health of individuals with PTSD following a period of psychological counselling or other treatment. Additionally, no published Australian studies exploring the health of individuals with PTSD could be found using relevant databases (PsychInfo, Medline). The studies in this thesis were designed to go some way towards overcoming these apparent gaps in research. Specifically, the current studies set out to explore the
prevalence and nature of self-reported health symptoms in victims of crime with and without PTSD in the Canberra region, prior to and following counselling delivered as part of assistance provided through the Victims’ Services Scheme (VSS). The first of these studies also investigated which of the PTSD symptom clusters were most closely associated with self-reported health problems, as well as examining whether PTSD symptom severity was reduced after a period of counselling. It was expected that victims of crime with PTSD would report more specific health symptoms, worse appraisals of their health, and a greater number of medically diagnosed problems than those without PTSD, while controlling for the impact of variables such as depression, age, and smoking (hypotheses one through to three). Another prediction was that the hyperarousal symptom cluster of PTSD would best predict greater health symptom reporting, as opposed to the other two symptom clusters (hypothesis four). Finally, it was also anticipated that counselling provided in the five to seven months between the two studies would be associated with a marginal reduction in self-reported health symptoms and a more substantial diminution of PTSD severity scores in the PTSD group as opposed to the non-PTSD group (hypotheses five and six).

A health questionnaire was designed specifically for both studies (see Chapter 3 for description, and Appendix for a copy) in order to evaluate these hypotheses, in addition to the PDS and BDI-II. Information about specific medical conditions (first study only) and health symptoms, perceptions of overall health before and after the trauma, and substance/medication use was obtained, as well as details of the duration of any counselling received between the two studies.

The first two hypotheses were supported, with a PTSD diagnosis accounting for a significant and unique proportion of the variance in the health score variable (representing health symptoms) after controlling for other significant variables in a hierarchical multiple regression analysis. A diagnosis of PTSD was significantly associated with frequent reporting of more health symptoms ($r = -.441$, $p < .001$). Similarly, participants with PTSD also reported a significantly worse appraisal of their overall health since the trauma, compared to those without PTSD ($r = -.476$, $p < .001$).
Once again, hierarchical multiple regression confirmed that PTSD accounted for a unique proportion of the variance in this change in health perception variable over and above other factors such as depression, alcohol use, and number of traumatic events experienced. These findings are consistent with other research reporting a significant association between PTSD symptoms and self-reporting of poorer health (Wolfe et al., 1994; Kimerling et al., 2000; Litz et al., 1992; Hankin et al., 1996; Solomon and Mikulincer, 1987; Shalev et al., 1990; Dobie et al., 2004; Clum et al., 2001; Woods and Wineman, 2004), and demonstrate that this sample of Australian victims of crime is comparable in this respect to the primarily veteran and sexual assault victims studied in the USA.

A number of variables are implicated in the relationship between exposure to trauma and poor health (refer also back to Chapter 2, Section 2.1). The relationships existing between these variables are complex, and not necessarily mutually exclusive. For instance, exposure to a violent crime can result in physical injuries and pain, as well as being associated with greater depression, PTSD, and substance use. However, pain can worsen depressive symptoms, while both depression and PTSD may result in an increased awareness of pain. Injury, depression, PTSD, and substance use have each individually been associated with a greater risk of ill health, possibly through some combination of immunological suppression, sleep disturbance, anger, and avoidance behaviours. In addition, Resnick et al. (1997) note that certain types of pain and medical problems identified in traumatized individuals can be at least partly explained in the context of the learning theory model of how PTSD develops following exposure to a stressor. According to learning theory, PTSD symptoms are classically conditioned responses to a highly distressing event. The traumatic event is the unconditioned stimulus (UCS) which evokes extreme fear, horror, or helplessness (the unconditioned response, UCR), as well as cognitive responses (eg “I’m going to die”) and behavioural reactions. Physiological “fight-flight” responses also occur, such as increases in heart rate, respiration, muscle tension, and digestion. At the same time, the trauma (UCS) may become associated with previously neutral cues that are present during the event (eg being alone, certain smells, and the weather at the time). These cues then become
conditioned stimuli (CS), and exposure to them in the future may be sufficient to elicit a conditioned response (CR), possibly including both emotional and physiological reactions. The conditioned responses are maintained – and sometimes strengthened – by avoidance of the cues that have become conditioned stimuli. Importantly, some of the initial physical reactions at the time of the traumatic event, such as pain received from injuries suffered, and anxiety-related physical distress, may become learned conditioned responses to the cues, leading to chronic pain in some instances and longer-term health problems such as irritable bowel syndrome or pain during sexual intercourse (Resnick et al, 1997; Leserman et al, 1996). As mentioned earlier, chronic pain and distressing physical conditions may then increase PTSD and depressive symptoms, which in turn increases the probability of poor health.

It is clear that clients with PTSD not only report more health symptoms, but have an overall poorer perception of their health now as opposed to before the trauma(s) occurred. The impact of such a perception cannot be overlooked by clinicians: a negative view of one’s health and well-being may in effect produce a self-fulfilling prophecy, whereby clients increasingly utilise more health services and report ill health, ruminating on their health and current (often undesirable) situation and perhaps engaging in a vicious cycle of PTSD-depressive-poor health symptomatology. Ironically, they may seek solace in cigarette smoking, alcohol use, and drugs (legal or otherwise), while not engaging in health-promoting behaviours such as exercise, pleasurable activities, and proper nutrition, which may further contribute to poor health. Some clients may pay greater attention to somatic problems, possibly as part of a conditioned response as described above, and misinterpret anxiety-related symptoms (eg chest pain), worrying about them and heightening the perception of poor health, over and above any underlying pathology. Indeed, the results of these studies show an increase in reporting of certain health symptoms between Study One and Study Two, despite an overall decline in the percentage of individuals with PTSD.

The third hypothesis, predicting that individuals with PTSD would report a greater number of medically diagnosed conditions than those without PTSD, was not supported.
This may be due to the relative youth of this sample (mean age 35.9 years), and relative recency of the traumatic event(s) – six months to four years, on average. It is worth noting that studies reporting a greater number of diagnosed medical conditions in participants with PTSD have generally been with older individuals who have had this psychiatric disorder for many years (eg Vietnam veterans – see Boscarino, 1997). While immunological suppression may occur fairly soon after the onset of PTSD symptoms, it could be years before this translates into diagnosable medical conditions. Similarly, chronic anger/hostility and sleep disturbance, as well as avoidance and numbing, may lead to alterations in catecholamines and white blood cell counts (eg Boscarino and Chang, 1999a and 1999b), increasing the risk of coronary heart disease over a period of years.

Similarly, the fourth hypothesis, that the PTSD hyperarousal symptom cluster would be a significant predictor of poorer self-reported health (assessed by regressing the symptom clusters on the health score variable), was not supported. Despite this hyperarousal symptom cluster being significantly associated with greater health symptom endorsement (in fact, all three symptom clusters were significantly negatively correlated with the health score variable), it was not a significant predictor of health problems in the hierarchical regression analysis. In fact, avoidance/numbing was the only cluster to predict a significant proportion of the variance in health problems. This is contrary to the findings of Clum et al (2001), Kimerling et al (2000), and Zoellner et al (2000), who variously found that the hyperarousal or reexperiencing symptom clusters accounted for the greatest proportion of variance in self-reported health. This inconsistency with previously documented research may be a factor of the relatively small sample size, or it may signify an association between avoidant cognitions and behaviours and health problems. Avoidance in general has been linked to increased levels of depression, anxiety, and somatisation symptoms (Tull et al, 2004), greater PTSD symptom severity in cancer patients (Jacobsen et al, 2002), and avoidance of health practitioners (Epstein, 1993; Pieper and Maercker, 1999). It has also been associated with functional impairment among patients with early stage cancer (Manne et al, 2000). Another interesting interpretation relates to recent research findings which
document a close inter-relationship between the numbing components and the hyperarousal cluster (Feuer, Nishith, and Resick, 2005; Tull and Roemer, 2003): if the two are functionally related, one might expect either or both to predict self-reported health problems.

The final two hypotheses were partially supported in an unexpected way, with shorter-term (or no) counselling associated with a significant reduction in PTSD symptoms, and a non-significant trend towards improved health scores for those who met criteria for PTSD. These results need to be interpreted with some degree of caution, however, given the small numbers of participants. Counselling overall was positively associated with a perception of a general improvement in health and well-being, compared to retrospective perceptions of health before counselling, although as discussed in Section 5.2.4 of Chapter 5, this may reflect a subjective “feel-good” factor which is not supported by the more objective measures of health and emotional well-being.

Overall, the provision of counselling did not result in any significant change in health symptom reporting for those with or without PTSD, with the exception of shorter-term assistance which may be more focussed or structured, and hence more effective. While PTSD severity scores declined over time, longer-term counselling did not appear to have a significant impact on these, although this conclusion is limited somewhat by the relatively small number of individuals (15.3% of the total) who did not receive counselling between the two studies. Such an improvement may be attributed to clients simply getting better, a natural occurrence in a significant proportion of PTSD sufferers (Rothbaum et al, 1992), and may also be related to the improvement found in the short-term counselling group. The finding that longer-term counselling appeared to lead to a significant reduction in health scores, equating to more symptom reporting, and no change in PTSD severity scores, is concerning and may suggest either that the counselling being provided is ineffective in reducing PTSD and associated health symptoms (and may in fact sensitize individuals to their bodily symptoms), or that the complexity of this client group (some of whom may still be in unstable or unsafe environments, and/or suffering from comorbid personality disorders or substance abuse...
issues) is such that a different approach is required. However, Hembree et al (2004) and Resick et al (2003) have demonstrated that brief cognitive behavioural treatment for women with PTSD resulting from rape or nonsexual assault was equally effective in reducing the incidence of PTSD at the end of treatment in those participants with and without complex trauma backgrounds. However, neither of these studies appeared to include participants who are or were domestic violence victims, potentially remaining in unstable and violent environments. Excluded from the Hembree et al (2004) study were individuals with concurrent substance abuse problems, alcohol dependence, actively suicidal and self-harming clients, and those in current domestic violence situations. While victims of crime expressing prominent suicidal ideation were also excluded from the studies reported in this thesis, it is clear that some participants had drug and alcohol dependence issues as well as potential personality disorder characteristics. It is unfortunate that it was not possible to identify the type of counselling or therapy provided by the VSS counsellors in this instance. Similarly, data relating to the numbers of participants who dropped out of therapy was not able to be gathered: some of the improvement in the shorter-term counselling group may have reflected individuals who simply improved to the point that they did not feel further counselling was warranted. However, it is also possible that short-term counselling in this instance is simply more effective in reducing health and PTSD symptoms than assistance provided over the longer-term.

The findings reported in this thesis add to the growing literature on the health impact of PTSD, and point to a new research direction, specifically whether self-reported health problems will diminish along with PTSD symptoms after the provision of timely, evidence-based therapy. Limited research investigating the impact of counselling upon health problems suggests that even relatively straightforward interventions such as relaxation training, as well as behaviour therapy, can have beneficial effects in terms of improving immunological responses or reducing the likelihood of cancer or coronary heart disease developing (Kiecolt-Glaser et al, 2002; Eysenck, 1991).
Clinicians treating traumatised individuals will have to tackle these health issues sensitively in order to avoid alienating their clients by implying that their physical problems (and/or perceptions of such) are simply a product of somatisation. A need for appropriate liaison with other health professionals, particularly GPs, is also warranted in order to both raise awareness about the association between PTSD and poor health, and to monitor and investigate any health problems. Concurrent anti-depressant treatment may be warranted, particularly if the PTSD and/or comorbid depression is causing significant distress, although the association found in the first study between such medication and health symptoms may also reflect side-effects from the former. Clinicians will also need to be aware that the avoidance and numbing symptoms of PTSD may not only maintain the posttraumatic symptoms overall (through avoidance of cues unless and until some form of exposure therapy is instituted), but – on the evidence presented in this thesis – contribute to reports of ill health. Assertive follow-up and persistence with therapy (client permitting) is critical.

The other major implication of this study for clinicians is the need to plan appropriate, timely, and evidence-based interventions for clients with PTSD. The best outcomes arise from cognitive-behavioural therapies with an exposure component (Foa and Meadows, 1997; Resick and Calhoun, 2001). However, realistically clinicians often need to balance this against an imperative to deliver treatment in a limited number of sessions (often dictated by outside funding sources), while dealing with various crises and safety issues raised by clients during therapy sessions. The results reported in this study suggest that this is possible, although more information about the type of participants benefiting most from shorter-term therapy would be useful. Careful planning and structuring of sessions, appropriate scheduling of interventions, and an empathic client-centred approach will be necessary to deliver positive outcomes for this client group.

The studies described in this thesis have a number of strengths. As previously mentioned, they appear to be the first Australian studies examining the health of victims of crime with and without PTSD. The use of multiple measures of health (medical
problems endorsed, reporting of specific health symptoms, and change in perception of health) is important in this context as positive results in one area (e.g., perception that health is better after counselling as opposed to beforehand) may not necessarily reflect the outcomes of more objective measures. Likewise, the second study is the only known research into the self-reported health of trauma victims following counselling, and highlights some important issues associated with the efficacy of counselling provided to victims of crime in a community setting by seeking specific information about changes in PTSD and health scores. Another strength is the breakdown of counselling provided by number of sessions, which highlighted that shorter-term counselling was associated with better outcomes than 8 or more counselling sessions.

There are also a number of limitations to this study that are worth noting. It would have been preferable to use a more objective measure of health problems, such as diagnoses of disease/illness arising from medical examinations of each individual. Additionally, there were issues with small sample sizes, particularly for the second study. However, the number for both were not inconsistent with other studies in the trauma/PTSD and health area that have utilised samples of between 30 and 100 (e.g., Kimerling et al., 2000; Litz et al., 1992; Hankin et al., 1996; Walker et al., 1992; Woods and Wineman, 2004; Zoellner et al., 2000). Unfortunately, the size of the group who did not receive counselling (n = 9, or 15.3% of the total) was smaller than desirable, with a larger sample necessary to have sufficient power to interpret findings with confidence. Other limitations included the fact that some of the participants reported receiving counselling in the past for their trauma. It would have been preferable if all participants had started in the same ‘no counselling’ condition, although realistically this was not feasible in the context of seeking a sufficiently large sample of victims of crime. It should be noted, though, that receipt of prior counselling was not significantly associated with the health score variable in the first study, which suggests that this did not act as a confounding variable. Ideally, it would have been desirable to have had some level of control over (or at least understanding of) the type and duration of therapy provided to participants between the first and second studies. However, this would have necessitated a more complex study design with identification of clinicians most likely required. In addition,
it was not possible to ascertain the number of individuals who dropped out from therapy. Finally, the use of a more rigorous interview-based measure of establishing a definitive PTSD diagnosis (such as the SCID [First et al. 1996] or the PSS-I [Foa et al., 1993]) would have been desirable; however as noted in Chapter 3, Section 3.3.2.1, resources for this study did not permit the use of a measure which would have been much more time-consuming to administer. Additionally, the requirement for participants to have been interviewed by the researcher would have added another layer of complexity to the study, and possibly further reduced the number of willing participants (who by completing a questionnaire in private maintained anonymity from the researcher).

The studies reported in this dissertation have highlighted several areas for possible future research in the PTSD and physical health field. These current studies represent the first known attempt to empirically study the self-reported physical health of individuals in Australia who meet criteria for PTSD. Replication of these results with a larger sample, allowing a greater number of covariates to be controlled for, would obviously be useful. In addition, this may permit a longitudinal study over a more significant period of time (e.g., 10 years) to be carried out, allowing researchers to track any development of medically diagnosed conditions in those with PTSD as the sample aged. A larger scale project of this nature may also be able to investigate more thoroughly the impact of cognitive-behavioural therapy for PTSD on health outcomes.

If undertaken in a controlled clinical setting, researchers may be able to place some participants in a ‘wait-list’ control group (ethics committees permitting) while allocating others to the therapy, or treatment, condition. Standardized therapy could then be applied using clinicians who have received relevant training in the type of CBT-based counselling used (e.g., Prolonged Exposure, Stress Innoculation Training, Cognitive Processing Therapy, or some combination of these – see Resick and Calhoun, 2001, or Foa and Meadows, 1997, for reviews of outcomes from these types of therapy). Sessions could be taped and evaluated by peers or supervisors to ensure consistency with therapeutic protocols.
In summary, the studies presented in this dissertation confirm the finding from international research that victims of crime with PTSD tend to report worse health outcomes than individuals exposed to trauma who do not develop PTSD. Contrary to previously reported findings, the first study found that the avoidance/numbing symptom cluster, rather than the hyperarousal cluster as predicted, was a significant predictor of self-reported health problems. No increase in self-reported medically diagnosed conditions was found, which may be a factor of sample age and relative recency of the majority of traumatic events. The second study established that community-based counselling (particularly of a longer term nature) did not appear to reduce either PTSD symptoms or self-reported health problems, despite participants reporting an improved perception of their overall health and well-being following receipt of counselling. However, no or shorter-term counselling resulted in a significant reduction in PTSD symptom severity, and a non-significant improvement in the health of those with PTSD. It would be useful to conduct a similar longitudinal study with a larger sample over a substantially longer period of time to see if these results were replicated, and whether medically diagnosed conditions were also reported more frequently among the PTSD group. Similarly, controlled treatment conditions consisting of specified evidence-based therapy after exposure to trauma would help to establish whether therapy might be beneficial in reducing self-reported and/or medically diagnosed health problems (presumably through a reduction in PTSD symptoms).
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Appendices

Copies of the following forms used in these studies are attached:

1. Health Questionnaire Part 1
2. Posttraumatic Diagnostic Scale (PDS)
3. Beck Depression Inventory, 2nd Edition (BDI-II)
4. Health Questionnaire Part 2