Personality and Adaptation to Military Trauma

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Personality and Adaptation to Military Trauma

Persoonlijkheid en aanpassing aan militair trauma
(met een samenvatting in het Nederlands)

PROEFSCHRIFT

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Contents

"What you see and hear depends a good deal on where you are standing:
it also depends on what sort of person you are"

C.S. Lewis (1955). The Magician's Nephew.

Chapter 1

Introduction, general design and outline

Introduction

Exposure to severe stressors, such as war, abuse, violence, and natural or technological disasters, can have a profound and lasting deleterious effect on physical health and psychological functioning. Although the risk of experiencing such events may appear slim, many people will be confronted with a potentially traumatic event at some point in life. Adversity and hardship are parts of life. But for some this is more true than for others. Some people are more frequently exposed to stressful life-events and may therefore be at risk of developing trauma or stress-related health problems.

By virtue of their profession, soldiers are one of the populations at risk for exposure to traumatic events. The dangers of the military profession have become painfully clear in recent peacekeeping operations in Afghanistan. Between 2006 and 2008, sixteen Dutch soldiers were killed, including the son of the Commander in Chief of the Dutch Armed forces. Deployed soldiers run the risk of being killed or injured whether or not they leave the base. They run the risk of hitting an improvised explosive devise (IED) buried alongside the road, or they may come into enemy contact with Taliban troops every time they leave camp; there are frequent mortar attacks both inside and outside the base, and there is always the risk of a suicide attack. In addition to the physical threat associated with the mission, soldiers may be confronted with other people's suffering and are at risk of witnessing or experiencing the death of a colleague or friend. These stressors, combined with the strain of being away from home and loved-ones, puts soldiers at increased risk for developing stress-related symptoms and disorders.

Stress and trauma related symptoms are of a transient nature in most people. For some however, the distress that arises after exposure to traumatic events may develop into disorders like depression or posttraumatic stress disorder (PTSD). Differences in susceptibility for stress-related disorders can be explained through multiple pathways. Whether or not problems arise depends on a combination of personal and situational factors. Stress does not have the same effect on all people, and not everyone perceives the same experiences as stressful or threatening. Moreover, people may perceive stress differently at different time-points. Nonetheless, trauma victims often display similar symptom patterns, irrespective of the nature of the trauma (Weisæth & Eitinger, 1993).

So, why does one person develop PTSD while another is able to carry on with life seemingly unaffected by life's adversities? Which factors determine the outcome? Increasing the understanding of individual differences in vulnerability and resilience to trauma is of vital importance to troops that are sent to war. Not only because this knowledge can aid in treatment of trauma related disorders, but also because a better understanding of factors that increase resilience may improve prevention strategies. To answer the aforementioned questions, a number of factors have to be examined; the pathways that lead to successful adaptation as well as the ones that lead to posttraumatic illness and disease have to be explored. This dissertation will focus on both 'routes' and will examine them from a personality perspective, and with particular relevance to the Dutch armed forces.

This chapter describes the background of the thesis and provides the framework from which the studies were undertaken. The history and background as well as diagnostic features of posttraumatic stress disorder (PTSD) are reviewed in the next paragraph. To provide insight into prevalence and incidence of posttraumatic stress disorder in military populations, epidemiological aspects are also reviewed in this chapter. Additionally, this chapter provides the conceptual framework central to this dissertation and it describes the possible interplay between trauma, personality and PTSD. The chapter is concluded by a general description and outline of the studies that form the body of this dissertation.

Posttraumatic stress disorder

An impressive volume of textbooks and scientific papers has accumulated that describes the potentially detrimental effect of trauma exposure on mental and physical health. The aggregated scientific knowledge from trauma research as well as anecdotal descriptions and clinical observations has shown that trauma can give rise to a variety of physical, psychological and social problems. After trauma exposure, many people suffer from sleep disturbances, nightmares, increased anxiety and anger. Often a heightened startle response is present as well as increased irritability. People may actively try to avoid recalling the events that took place or avoid stimuli that are associated with the stressful event. At other times vivid memories or flashbacks may suddenly occur. Victims may show amnesia for substantial parts of the traumatic experience and they often report difficulties in thinking and concentrating. If these symptoms persist for a longer period, they may develop into a mental disorder, most notably posttraumatic stress disorder (PTSD).

The last decade has also shown an increase in studies describing the other side of the coin, showing an increased interest in factors that promote the ability to thrive or persevere in the face of adversity and despite the presence of risk factors (Kleber, 1999; Richardson, 2002; Rutter, 1987). This shift in attention from clinical symptoms to indicators of well being, which can be observed in other areas as well (Seligman, Steen, Park, & Peterson, 2005), may reflect a change in paradigm: from sickness models to wellness models. The stress-resilience framework has introduced new perspectives on coping with trauma. It has lead to the identification of inter- and intrapersonal factors that facilitate recovery, and to the development of concepts that describe positive outcomes such as posttraumatic growth.

Historic overview

PTSD is a relatively young psychiatric diagnosis. It was introduced in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III) almost 30 years ago, in 1980. However, the consequences of trauma exposure have been described throughout the ages, and can be traced back to several centuries before Christ. For instance, accounts of traumatic stress can be found in ancient Greek tragedies such as Sophocles' 'Ajax' and 'Electra', both staged in the aftermath of the Trojan War. However, it was not until the middle of the 19th century that various traumatic phenomena were studied scientifically and described in more detail.

Around this time, a syndrome was described in Britain following a series of train accidents and the proposed 'excessive' amount of strain that was inflicted on the human body when travelling by train. A number of articles appeared in *the Lancet*, describing this syndrome, which was later coined 'Railway Spine' or 'Concussion of the Spine' (Erichsen, 1867). The syndrome consisted of irritability, restlessness, memory loss and malaise. In special sections of *the Lancet* (1862), it was stated that the violent shocks and jolting, not just in a collision but typical in any train ride, could lead to paralyses by damaging the nervous system and spinal cord. The organic nature and cause of the afflictions of railway passengers was subject to fierce debate however, and others suggested that psychological aspects were more important determinants of this type of morbidity than spinal cord damage (e.g., Page, 1885). Similarly, Seguin (1890, in McFarlane, 2000) proposed that the term railway spine be dropped in favour of the more appropriate designation 'Traumatic Neurosis' which was first used by Oppenheim (1892) to describe symptom patterns in victims of traumatic incidents.

Across the Atlantic Ocean, DaCosta (1871) described a cluster of symptoms reported by combatants of the American Civil War. This syndrome, which was later referred to as 'Soldier's Heart', 'Irritable Heart' or 'Effort Syndrome', included symptoms like shortness of breath, palpi-

tations, chest pains, fatigue, diarrhoea and headache, which DaCosta attributed to an organic nature. Around the middle of the 19th century in France, several psychiatrists described the psychological consequence in trauma victims (Van der Hart, 2003). For instance, Charcot described the occurrence of hysterical afflictions in males. He attributed the occurrence of this affliction to physical exertion, emotional strain and toxic environmental influences in most cases, but also observed a relationship with physical accidents. Moreover, he noted that the type and severity of the injury could not sufficiently explain nature and intensity of these 'traumatic hysterias' (Micale, 1990). The importance of psychological aspects to explain the symptoms reported by victims of railroad accidents was also stressed by Janet, who posited that the symptoms reported by these patients should be perceived as a form of neurosis (Van der Kolk & Van der Hart, 1989).

The large amount of psychiatric casualties of WW I sparked an increased interest in trauma-related (psycho)pathology and lead to the description of syndromes like 'combat fatigue' and 'shell shock'. The relative importance of physical and psychological factors in the etiology of this type of disorder continued to be subject to debate throughout the first half of the 20th century. It was not until after WWII that syndromes like 'shell shock', 'post-concentration camp syndrome', and 'survivor syndrome' were more readily described in terms of psychological and psychiatric phenomena (Kinzie & Goetz, 1996). Finally, when the first edition of DSM was formulated (APA, 1952), it included the diagnosis 'Gross Stress Reaction' that described acute psychological responses to extreme stressor, like combat or a catastrophe, in otherwise normal individuals. Surprisingly however, the diagnosis was dropped in the second edition (APA, 1968), which only included the diagnosis 'Transient Situational Disturbance'. After the war in Vietnam the diagnosis PTSD was first formulated in DSM III (APA, 1980).

Diagnostic features

In the most recent version DSM-IV-TR (APA, 2000), the core symptoms of PTSD are clustered in three categories: symptoms pertaining to re-experiencing the traumatic event, in dreams, flashbacks or intrusive memories; avoidance of stimuli associated with the experience, social alienation and emotional numbing; and symptoms of 'hyper-arousal' like irritability, sleeping disorders, vigilance and heightened startle responses (See Box 1). In most cases PTSD is accompanied by other disorders. American and Australian epidemiological studies showed that around 85% of individuals with PTSD meet criteria of additional disorders, comorbid depressive and anxiety disorders, and alcohol abuse/ dependence being most common (Creamer, Burgess, & McFarlane, 2001; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Additionally, dissociative symptoms are often reported (Vermetten, Dohary, & Spiegel, 2007) and feelings of shame may be present after exposure to various forms of trauma. Exposure to trauma can also lead to changes in self-perception and worldview (Janoff-Bulman, 1992). Finally, studies of veterans with chronic PTSD have demonstrated high rates of comorbid personality problems (Bollinger, Riggs, Blake, & Ruzek, 2000; Dunn et al., 2004; Southwick, Yehuda, & Giller, 1993).

Box I: DSM-IV-TR Criteria for PTSD

A. The person has been exposed to a traumatic event in which both of the following were present:
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. The person's response involved intense fear, helplessness, or horror.

B. The traumatic event is persistently reexperienced in one (or more) of the following ways:
recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions recurrent distressing dreams of the event 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) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event 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:
efforts to avoid thoughts, feelings, or conversations associated with the trauma efforts to avoid activities, places, or people that arouse recollections of the trauma inability to recall an important aspect of the trauma markedly diminished interest or participation in significant activities feeling of detachment or estrangement from others restricted range of affect (e.g., unable to have loving feelings) sense of a foreshortened future (e.g., 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:
difficulty falling or staying asleep irritability or outbursts of anger difficulty concentrating hypervigilance 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.

Prevalence of PTSD

A large epidemiological study in the USA estimated the life-time prevalence of PTSD in the general population at 6 to 8% (Kessler et al., 2005; Kessler et al., 1995). A large European study reported considerable lower life-time prevalence rates of 1.9% (Alonso et al., 2004). However, as Kleber and Brom (1989) showed, incidence rates of PTSD in samples of trauma-exposed individuals may be significantly higher. They reported incidence rates between 10 and 30% in their review of studies of victims of violence, accidents and disasters, and combatants. As soldiers are more often exposed to potentially traumatic events, the occurrence of PTSD may be more common in military populations. On the other hand, it is also possible that the military profession attracts individuals who are more resilient to stress. So how many soldiers develop PTSD?

Available prevalence rates vary markedly across studies. The National Vietnam Veterans Readjustment Study (NVVRS; Kulka et al., 1990) reported that 31% of the male Vietnam veterans and 27% of female veterans met lifetime criteria for PTSD. However, when Dohrenwend et al. (2006) analyzed the data some 10 years later using more strict criteria, they reported life-time prevalence rates of 'only' 19%. These figures correspond more closely to data from Australian Vietnam veterans where life-time prevalence rates were estimated in the range of 12 to 21% (O'Toole et al., 1996).

The last decades have shown an increase in joint military peace-keeping and peace-enforcement operations around the globe, including multiple regions on the African continent, the Balkans, the Middle East and Afghanistan. These missions bring with them a whole set of different stressors that may nonetheless be equally related to morbidity (Shigemura & Nomura, 2002). Again, prevalence and incidence rates vary across studies. Data from recent operations in Iraq and Afghanistan showed estimated incidence rates around 12-20% 4 months after deployment in male US soldiers who participated in operation Iraqi Freedom in 2003. The prevalence of PTSD in soldiers participating in operation Enduring Freedom in Afghanistan was estimated at 6-12% (Hoge et al., 2004). Unwin et al. (1999) reported that 5% of British soldiers displayed posttraumatic stress reactions after deployment to Bosnia versus 13% in Gulf War veterans. However, in a later study prevalence rates between 4 and 5% were reported for British peacekeepers who participated in peacekeeping missions during the nineties (Greenberg, Iversen, Hull, Bland, & Wessely, 2008). A large scale study of U.S veterans from the first Gulf war estimated prevalence of PTSD at 10% (Kang, Natelson, Mahan, Lee, & Murphy, 2003). Eight percent of U.S. soldiers deployed to Somalia were shown to meet diagnostic criteria for PTSD after deployment (Litz, Orsillo, Friedman, Ehlich, & Batres, 1997).

Prevalence rates of PTSD in the Dutch peacekeepers range from 2 to 8%. In veterans deployed to Lebanon (UNIFIL) between 1979-1985, PTSD rates were estimated at 5% in one study (Bramsen, Dirkzwager, & Van der Ploeg, 1997). Prevalence rates of 2-3% were reported in veterans who participated in a humanitarian mission to Cambodia in 1992-1993 (UNTAC; De Vries, Soetekouw, Bleijenberg, & Van der Meer, 1998). Estimates for veterans who participated in the UN peace-keeping operations in former Yugoslavia between 1992 and 1996 range from 3 to 8% (Bramsen et al., 1997; Mulder & Reijneveld, 1999). Finally, a recent study of Dutch soldiers deployed to Iraq in 2004 and 2005 showed that 5 months after deployment incidence rates of PTSD varied across groups with estimates ranging from 3 to 12 %, depending on prior experiences, pre-deployment psychopathological symptoms and deployment experiences (Engelhard et al., 2007).

Adaptation to trauma

The previous paragraph showed that only a relatively small percentage of individuals develop PTSD. How can we account for these differences between trauma exposed individuals? Any theory on the etiology of PTSD has to take into account that different pathways exist for individuals exposed to similar circumstances; one leading to adjustment and one leading to maladjustment and mental disorders (Jones & Barlow, 1990). Several different theories have been forwarded to account for the development of trauma related psychopathology. Perhaps one of the most influential models comes from Horowitz. In his book *Stress Response Syndromes* (1976, 2001), Horowitz described a sequence of phases and associated symptoms that may occur after exposure to a traumatic stressor. First, as the realization of the nature and extent of the traumatic event sinks in, this is accompanied by an emotional outcry. After the outcry, efforts are directed at reconsolidating prior memories and experiences with the new (traumatic) information and at integrating the new information into available cognitive schemas.

This process is characterized by alternating stages of denial and avoidance versus intrusions of trauma-related images. As this process evolves, existing cognitive schemas are accommodated and new ones develop until the individual 'works through' the traumatic event. PTSD is proposed to occur when an individuals is unable to complete this process. Although this model provides a clear account for the occurrence of intrusive and avoidance symptoms, it does not sufficiently address the effects of social support and other environmental influences on posttraumatic morbidity, nor does it provide insight into individual differences in resilience or vulnerability to stress-related disorders (Brewin & Holmes, 2003; Kleber & Brom, 1992).

Ehlers and Clark (2000) described a cognitive model of PTSD in which the interplay between environmental factors and individual characteristics is proposed to affect the appraisal and processing of trauma-related information. Leaning heavily upon earlier work (e.g., Foa & Rothbaum, 1998; Janoff-Bulman, 1992; Jones & Barlow, 1990), Ehlers and Clark note that idiosyncratic negative appraisals of self and external world can be observed in individuals who develop PTSD. They argue that exposure to trauma may cause unrealistic expectancies of future threats, and that these appraisals may lead to avoidance behaviour which in turn sustains the (irrational) sense of fear. Later studies have provided evidence for the role of cognitions and appraisal in the etiology of PTSD. For instance, in assault victims, cognitive aspects measured shortly after trauma predicted PTSD at 4 months follow-up (Dunmore, Clark, & Ehlers, 2001). Also, Bryant and Guthrie (2005) showed that pre-trauma negative (catastrophic) appraisal predicted PTSD severity 20 months later in a prospective study of fire-fighters.

According to the A2 criterion in DSM-IV, the experience of a traumatic stressor must be accompanied by intense emotions in order for an individual to meet criteria for PTSD, and research has shown that emotional response and perceived threat are important predictors of PTSD (Ozer, Best, Lipsey, & Weiss, 2003). The cognitive model of PTSD formulated by Ehlers and Clark, provides a framework for understanding the role of emotional responses like threat and other intense peritraumatic emotions in the etiology of PTSD as it postulates how these emotions may shape future behaviour and cognition.

In addition to (cognitive) psychological models of PTSD, several 'biological' paradigms have been forwarded to account for observed associations between PTSD and specific brain regions, neurobiological and immunological changes, changes in the hypothalamic–pituitary–adrenal (HPA) axis and adrenocortical arousal (De Kloet, 2007; Vermetten, 2003). These include neurobiological applications of stress and trauma paradigms and conditioning models like 'failure of extinction' and 'stress sensitization' (Bremner, Krystal, Southwick, & Charney, 1995; Wessa & Flor, 2007).

Risk factors

Evidently, the traumatic stressor is an important determinant of posttraumatic morbidity. However, there are more determinants of posttraumatic adjustment. Kleber and Brom (1992) clustered the factors implicated in posttraumatic morbidity in three groups: 1) situation and context aspects; 2) person characteristics, and 3) social and cultural determinants. The importance of stressor characteristics was demonstrated by Brewin, Andrews and Valentine (2000), who a showed robust relationship between trauma severity and PTSD in their meta-analysis. Others have provided evidence for a dose-response relationship between trauma exposure and PTSD (e.g., Dohrenwend et al., 2006). Intentional interpersonal violence especially, including sexual assault and combat, is associated with increased risk of PTSD compared to accidents or disasters (Creamer et al., 2001; Kessler et al., 1995). Further, in combat samples, physical injuries during deployment have been shown to increase the risk of subsequent depression and PTSD (e.g., Grieger et al., 2006).

Social support (or lack thereof) and prior exposure to trauma, including childhood trauma, have also been found to be important determinants (Ozer et al., 2003). Demographic and other personal variables can also affect the outcome of trauma exposure. Women are more likely to develop PTSD, even though men appear to be more frequently exposed to trauma (Breslau, Chilcoat, Kessler, Peterson, & Lucia, 1999; Kessler et al., 1995). Other demographic determinants of vulnerability for posttraumatic disturbances include age, socioeconomic status, education, and (family) psychiatric history. The relative importance of the factors may vary across samples (Brewin et al., 2000). However, demographic variables are little insightful when it comes to explaining *why* certain individuals develop psychopathology while others do not, that is: 'The fact that my age, sex, social status help form my outlook on life does not change the fact that the outlook is a functioning part of me' (Allport, 1966, p. 2). In the next section we will elaborate on the role of specific cluster of psychological attributes in posttraumatic morbidity: personality.

Personality

In 1947, Abram Kardiner already pointed towards the importance of personality in the etiology of posttraumatic disturbances. He pointed out that traumatic neuroses were distinctly different from 'ordinary' neurosis and that the only way to evaluate this type of pathology was by examining the personality functions involved, and their function in the individual's adaptation to the external world (Kardiner & Spiegel, 1947). Moreover, because PTSD is the only mental disorder that is so explicitly linked to an external cause, examining the relationship between personality and PTSD offers a unique opportunity for evaluating diathesis-stress models of psychopathology. It enables us to examine individual differences in mental disorders. Specifically, research into the relationship between personality and PTSD may explain why only a relatively small percentage of individuals exposed to trauma go on to develop PTSD on the one hand (Miller, 2003), and enables us to determine which psychological factors promote successful adaptation to extreme circumstances. But what is personality?

The term personality originates from the Latin 'Persona', meaning 'mask'. This suggests that personality is closely related to the way we present ourselves to others. But that is not all. Personality can also been described as 'what a man really is' (Allport, 1937, p. 48). Neither description is particularly informative however, and both definitions fail to reveal why or how individuals differ from each other. A more useful definition can be found in DSM-IV, where personality is described as 'enduring patterns of perceiving, relating to, and thinking about the environment and oneself that are exhibited in a wide range of social and personal contexts' (APA, 2000, p. 686). This description is not dissimilar from Allport's (1937) definition that personality is the 'dynamic organization within the individual of those psychophysical systems that determine his unique adjustment to his environment' (p.48). Although these definitions provide more insight into the range and scope of personality, they also show that personality is a particularly complex construct. It comprises of clusters of traits associated with various domains, including perception, cognition, behaviour and affect. Although personality is perceived as being relatively stable over time it is by no means 'set like plaster' (Srivastava, John, Gosling, & Potter, 2003) and individuals may behave differently in different situations.

Personality is sometimes used interchangeably with character and temperament. However, the term temperament is generally reserved for the hereditary, neurobiological origin of personality whereas character is usually coined to describe the developmental aspects of personality (Cloninger, Svrakic, & Przybeck, 1993; Eysenck, 1961). Personality then refers to the combination of inborn and acquired characteristics (Akiskal, Hirschfeld, & Yerevanian, 1983).

Personality as a vulnerability factor for PTSD

So how can personality psychology aid in the understanding of PTSD and other stress-related disorders? The questions of if and how personality increases the risk of PTSD taps on a classic theme in psychology: the relationship between personality and psychopathology. The idea that personality or temperamental factors predispose an individual to certain illnesses dates as far back as 400 BC, with Hippocrates' doctrine of the four humours (Maher & Maher, 1994). According to Hippocrates, the balance of four essential bodily fluids - black bile, yellow bile, blood and water - could essentially determine whether a person was healthy or ill. Later Galen proposed a personality taxonomy based on these humours that consisted of personality types which he labelled sanguine, choleric, melancholic and phlegmatic, each with a specific set of psychological attributes (Maher & Maher, 1994).

In modern science, several decades of research have provided ample empirical evidence to sustain the notion that personality and psychopathology are closely related. For example, schizotypal personality disorder has been conceptualized as an attenuated form of schizophrenia. The typical cluster of cognitive, perceptual and interpersonal disturbances, and disorganized behaviour in schizotypal personality, that are also frequently observed in relatives of schizophrenics, may represent a premorbid or prodromal stage of schizophrenia (e.g., Raine, 2006). The association between schizotypal personality features and schizophrenia underlines the notion that some mental disorders might be conceptualized as extreme manifestations of personality characteristics. With respect to the relationship between personality and PTSD, several prospective studies, in combat populations, have provided evidence for the dispositional effects of personality in the etiology of PTSD (Bramsen, Dirkzwager, & van der Ploeg, 2000; Schnurr, Friedman, & Rosenberg, 1993; Sutker, Davis, Uddo, & Ditta, 1995). However, although these studies show that personality is implicated in the etiology of PTSD, they fail to explain why or how.

A coping model

The association between trauma, personality and PTSD can be explained by looking at it from a person-situation perspective as described by Lazarus and Folkman (1984). According to their transactional-model, any potential stressor is first evaluated in terms of its significance and whether it composes a threat or challenge (primary appraisal). Second, the availability of coping resources is appraised (secondary appraisal). The subsequent coping efforts and behaviours then determine the outcome. Diverging from the original contextual approach to coping such as proposed by Lazarus and colleagues, Aspinwall (2004) pointed out that personality characteristics can be expected to affect this process at various stages.

Personality may affect the attention that is directed at any potential stressor, the way it is appraised, the subsequent coping behaviour, and ultimately the outcome of the coping process. As Lewis (1955, p. 136) noted: 'what you see and hear depends a good deal on where you are standing: it also depends on what sort of person you are'. Individuals with characteristically high 'baseline' levels of distress (i.e. high on neuroticism or negative emotionality) may appraise more situations as demanding or particularly stressful and may become more easily aroused by relatively mild stressors (Watson & Clark, 1984). If this is the case, then more coping efforts will be directed at regulating the (intrapersonal) emotional state than at effectively confronting the situation at hand (Aspinwall, 2004).

Personality may also affect the nature and amount of (other) resources that are available. As personality and social situations may be reciprocally related (Bandura, 1978; Mischel & Shoda, 1998), personality can affect the availability of social support. More extraverted and sociable individuals can be expected to have a more extensive social support network and

thus be more resilient to stressors. By contrast, hostility has been associated with increased exposure to stressors and reduced levels of social support (Smith, Glazer, Ruiz, & Gallo, 2004). As shown in Figure 1, which displays the schematic association between trauma, personality and PTSD, personality can increase the risk of PTSD through various pathways.

The outcomes of the coping process may also result in changes in personality. The outcome and the evaluation of the coping response may have an effect on personality and other resources and assets. For instance, the inability to effectively cope with a traumatic event may affect an individual's level of self-esteem and other self-schemas as well as the availability of resources to manage this persistent stressors as well as other (new) stressors. The coping process does not end there however. When the presence of PTSD is perceived as a chronic stressor, the figure above shows how personality can affect the longitudinal course of the disorder. That is, dealing with a mental disorder like PTSD may affect personal resources that are available to adapt to additional (new) stressors. Also, as PTSD is often accompanied by marked social alienation, external resources may be reduced. The transactional model displayed in Figure 1 forms the framework for the studies described in this dissertation.

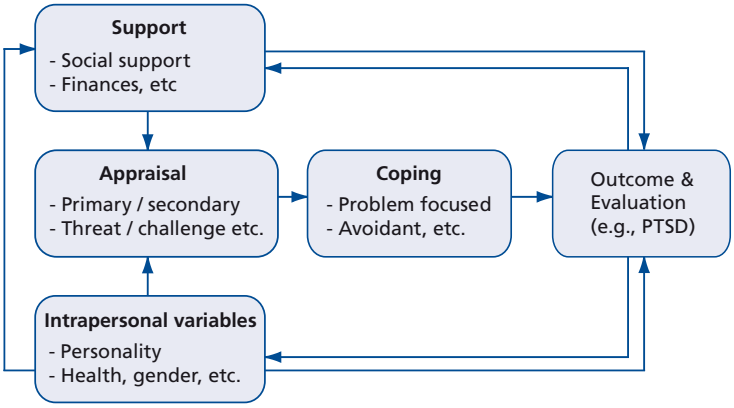

Figure 1: *Transactional coping model*

Effects of trauma on personality

As noted before, Kardiner (1947) concluded that nature of posttraumatic morbidity was determined by pre-trauma personality. Kardiner also noted the possible toxic effects of traumatic neurosis on personality functioning. More recently, Judith Herman (1992) gave a compelling description of the detrimental effects of prolonged exposure to stress and repeated trauma like childhood abuse, war, and torture on identity, affect-regulation, self-perception, and other personality domains. Similarly, Janoff-Bullman (1992) described how trauma can alter an individual's sense of security and faith, and that it may interfere with or challenge basic needs. The effects of prolonged stress are also recognized by the World Health Organization (WHO) as the diagnostic category "enduring personality changes following exposure to catastrophic experience or prolonged stress" was included in ICD-10 (WHO, 1992). Although effects of trauma on personality have been described by several authors, many questions remain unanswered. For one thing, the effects of trauma exposure on personality have hardly been examined empirically in longitudinal studies. It is unclear which personality domains are affected by prolonged stress or exposure to trauma and whether changes in personality are permanent or whether personality functioning may 'normalize' when PTSD and other posttraumatic symptoms abate. More importantly, the fact that trauma exposure and / or PTSD

can affect personality composes an important methodological problem for research aimed at disentangling the relationship between trauma, PTSD and personality.

Resilience

The transactional model depicted in Figure 1 showed how personality can affect the risk of PTSD. It also illustrates how personality can be associated with reduced vulnerability. On the one hand, if high levels of a certain characteristic are associated with reduced coping ability and increased risk of PTSD, then low levels of that same characteristic might be related to more effective coping and / or decreased risk of PTSD. On the other hand, there may be characteristics that are specifically related to enhanced endurance or resilience. So, what is resilience and how should we define it?

Resilience might be defined as the ability to thrive despite the presence of risk factors (Richardson, 2002; Rutter, 1987). In adult trauma literature the term resilience is generally used to describe the process of coping with adversities as well as those intra-individual qualities and process that enable people to 'bounce back' in the wake of stress or high risk situations (Mancini & Bonanno, 2006; Richardson, 2002; Rutter, 1987). The construct encompasses a 'complex repertoire of behavioural tendencies […] with identifiable patterns of thinking, perceiving and decision making across different types of situations' (Agaibi & Wilson, 2005, p. 197). This broad description has many commonalities with the definition of personality mentioned earlier. It is therefore not surprising that various personality attributes have been associated with resilience.

An impressive list of personality traits that may increase resilience has accumulated over the years. These include, but are not limited to, self-esteem, self-efficacy, hardiness, altruism, optimism, humour, locus of control, positive emotionality, and hope (Agaibi & Wilson, 2005; Bowman, 1999; Richardson, 2002; Southwick, Vythilingam, & Charney, 2005). However, research on how the presence of resilient qualities (vs. absence of risk factors) aid in overcoming hardship in adult populations has not received sufficient attention in clinical research (Bonanno, 2004). Moreover, the interplay between personality risk and resilience factors in the etiology of PTSD remains poorly understood.

Goal and general design

Although personality may be an important determinant in posttraumatic adjustment, research on how personality contributes to enhanced resilience or increased vulnerability has not received sufficient attention. The purpose of this dissertation is to examine how personality may enhance vulnerability or resilience to trauma related psychopathology in Dutch soldiers. To do so, we examined several pathways and mechanisms that may underlie the relationship between personality and trauma. Specifically the aims were to:

- *Investigate the relationship between trauma in early life and adult personality;*
- *Examine temperamental differences in biological systems associated with stress-related disorders like PTSD;*
- *Test whether personality affects coping;*
- *Assess whether personality risk and resilience factors independently predict PTSD;*
- *Examine resilience in the aftermath of PTSD by looking at the effects of treatment on personality and coping.*

These themes were investigated in a series of studies that zoomed in on specific aspects of this relationship in military and veteran samples at different timeframes. Data were gathered

from active military personnel during preparation for deployment to Afghanistan, peacekeeping veterans who were referred to the psychiatric department of the Dutch Central Military Hospital for treatment, and a random, non-clinical sample of veterans who served in the first Dutch peacekeeping operation to Lebanon in the late seventies/ early eighties.

Outline

The studies in this dissertation are divided into three sections. The first sections contains studies that focus on pathways through which personality may account for increased vulnerability or enhanced resilience to posttraumatic morbidity. The second section focuses on clinical studies in Dutch military samples presenting with posttraumatic stress symptoms. The findings are summarized and integrated in the final section, which also contains the Dutch summary.

Chapter 2 provides a review of the literature on evidence for a relationship between PTSD and neuroticism. Neuroticism is one of the most extensively studied personality traits. There is evidence for a relationship between neuroticism and various mental disorders, most notably mood and anxiety disorders (Bienvenu & Stein, 2003), and neuroticism has been proposed to be an important risk factor for the development of PTSD. Focussing on neuroticism therefore provides us with sufficient empirical data to explore the potential pathways between personality and PTSD.

In *Chapter 3* we examine how early adverse experiences are related to personality development in active soldiers. Both personality and childhood trauma have previously been marked as risk factor for adult PTSD. The study described in this chapter explores the possibility that personality acts as the 'vessel' between early trauma and adult combat- or deployment-related psychopathology. It examines the association between early life trauma and adult personality in a healthy, non-clinical sample of male soldiers.

Chapter 4 focuses on biological correlates of personality. Specifically, we examine the relationship between temperament and the cortisol response to awakening (ACR). The ACR can be seen as an indicator of the reactivity of the hypothalamic-pituitary-adrenal (HPA)-axis, which plays a key role in stress-related disorders like PTSD. The relationship between ACR and personality is investigated in a sample of healthy male soldiers.

The complex interplay between trauma exposure, coping and personality is examined in close detail in *chapter 5*. This study focuses on dispositional resilience factors of optimism, hardiness and locus of control and investigates whether these personality aspects predict coping, social support and PTSD symptoms, independently of neuroticism. To do so, test scores from a random sample of Lebanon veterans are analyzed using path analyses.

Chapter 6 focuses on the personality profiles of treatment seeking peacekeeping veterans. This study investigates the clinical symptom presentations of Dutch peacekeepers as measured with the MMPI-2. The MMPI-2 is possibly one of the most widely used psychological tests in clinical settings. A great number of papers have been published on MMPI-2 scores in trauma samples, especially Vietnam veterans. The relationship between PTSD symptoms and MMPI-2 scores is also examined in this chapter. Test scores of veterans with PTSD symptoms are compared with a control group of deployed soldiers screening negative for PTSD. Also, MMPI-2 scores of soldiers that served in peacekeeping operation in the Balkans are compared to those of veterans who served in the first Dutch UN mission to Lebanon in the late seventies / early eighties, and MMPI-2 profiles of peacekeeping veterans are compared to available literature from Vietnam veterans.

Chapter 7 reports the outcomes of an intensive and long-term group treatment on PTSD and associated symptoms, as well as on personality and coping. This case study of veterans

suffering from chronic PTSD and comorbid disorders, illustrates the potential lasting and deleterious effects of PTSD on personality on the one hand, and provides some insight in which habitual coping styles and personality features may be associated with recovery from PTSD on the other. Finally, in *chapter 8* the findings from these empirical studies are integrated and discussed.

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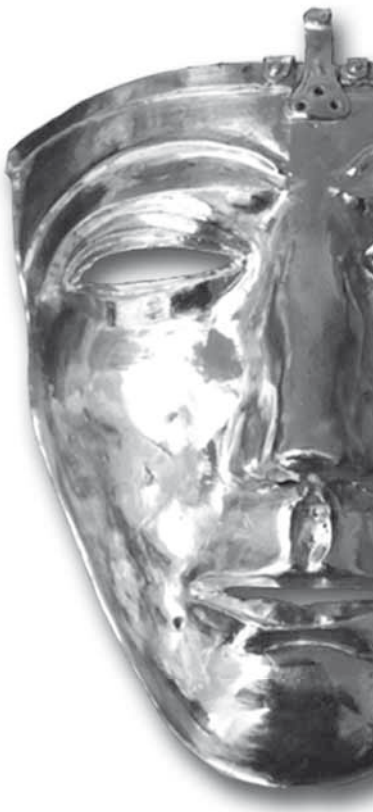
Section 1

Non-clinical studies



Chapter 2

Trauma, neuroticism, and PTSD: A review

Trauma, neuroticism and PTSD: A comprehensive review
Arthur R. Rademaker, Rolf J. Kleber, & Eric Vermetten
Revised submission under review

Introduction

Most people who are confronted with extreme life stressors or traumatic events adapt to these experiences without major problems. Only a relatively small proportion of all exposed individuals develop serious disturbances such as posttraumatic stress disorder (PTSD). The life-time prevalence of this disorder in the general population is estimated at around 8%. Although there is a causal relation between the traumatic event and PTSD, the occurrence of the stressor is a prerequisite, not a guarantee, for the occurrence of PTSD. Studies into the etiology of PTSD point to multiple causeways (Brewin, Andrews, & Valentine, 2000; Kleber & Brom, 1992). To explain individual differences in susceptibility, research has focused on the influence of vulnerability factors and predispositions on the development of PTSD. As such, researchers have also investigated the relationship between personality factors, trauma exposure and PTSD.

In 1947, Abram Kardiner already stressed the importance of studying personality factors and their associations to posttraumatic disturbances. He concluded that war illnesses were distinctly different from 'ordinary neurosis' and that the only way to evaluate this type of pathology was by examining the personality attributes involved, as well as their function in the individual's adaptation to the world (Kardiner & Spiegel, 1947, p. 9). Kardiner stated that the nature of posttraumatic morbidity was determined by pre-trauma personality, but also recognized that personality was subject to change as a result of the traumatic neurosis.

To date, research has provided ample empirical evidence to sustain the notion that certain personality characteristics increase the risk of PTSD and may influence the development over time (e.g., Paris, 2000). Prospective studies, mostly in combat populations, provide compelling evidence for the dispositional nature of personality in the etiology of PTSD (Bramsen, Dirkzwager, & Van der Ploeg, 2000; Schnurr, Friedman, & Rosenberg, 1993; Sutker, Davis, Uddo, & Ditta, 1995). Some have even suggested that personality may be a better predictor of posttraumatic morbidity than the traumatic event itself (Bowman, 1999; McFarlane, 1989). By contrast, two major meta-analyses of predictors of PTSD did not include any personality factors (Brewin et al., 2000; Ozer, Best, Lipsey, & Weiss, 2003). Clearly, the predisposing role of personality in the etiology of PTSD is not equally supported by all studies.

Even though many studies have reported an association between personality and PTSD, the nature of this relationship remains unclear. One problem is the complexity of personality assessment. Many different perspectives on personality are available; differing in theoretical premises as well as in focus on specific areas of functioning. In general, personality can be described as 'enduring patterns of perceiving, relating to, and thinking about the environment and oneself that are exhibited in a wide range of social and personal contexts' (APA, 2000). The personality construct encompasses a range of domains including perception, cognition, behavior and affect, and there exists an abundance of tools to measure (specific aspects of) personality. As a result, the body of literature describing the relation between personality and PTSD has become very heterogeneous.

Neuroticism

One of the most well known and possibly one of the most extensively studied personality traits is neuroticism. Neuroticism pertains to an individual's emotional reactivity, tendency to worry and susceptibility to negative moods. The origins can be traced back to the early psychodynamic theories of Freud and Jung, in which neuroticism was theoretically linked to introversion (Eysenck & Eysenck, 1969). The trait can also be found in other early personality models. In one of the first five-factor taxonomies, described by Fiske (1949), it was called 'Emotional control'; in Catell's (1957) model it was coined 'General integration factor', and in

Norman's (1963) personality model, it was labeled 'Emotional stability'.

Neuroticism is a key trait in Eysenck's (1947, 1960) personality theory as well as in the more contemporary Big Five personality taxonomies (Costa & McCrae, 1985; Goldberg, 1990). Although the theoretical background of the Big Five is quite different from Eysenck's model, the correlation between the Big Five and Eysenck's measures of neuroticism tend to be so high that, for practical purposes, they can be considered equivalent (Pervin, 1993). Due to the emotional component incorporated in most, if not all, descriptions, the term neuroticism is sometimes used interchangeably with negative affectivity and/ or negative emotionality (Watson & Clark, 1984).

Neuroticism is a relatively stable and enduring trait (Roberts, Walton, & Viechtbauer, 2006; Santor, Bagby, & Joffe, 1997). Its dimensional attributes make it a useful personality factor in clustering normal personality. In addition, neuroticism is related to a broad range of mental disorders, in particular mood and anxiety disorders (e.g., Bienvenu & Stein, 2003). Furthermore, it can act as a vulnerability factor for a wide range of disturbances (Costa & McCrae, 1992; Khan, Jacobson, Gardner, Prescott, & Kendler, 2005). The most common personality measures of neuroticism are Eysenck's Personality Questionnaire (Eysenck & Eysenck, 1975; Eysenck, Eysenck, & Barett, 1985), the NEO Personality Inventory-Revised (NEO-PI-R) and the Five Factor Inventory (FFI; Costa & McCrae, 1985; 1992).

Aims and selection of studies

The purpose of this paper was to review papers describing a relationship between neuroticism, trauma, and PTSD, and to explore how neuroticism might be related to the etiology of PTSD. We performed a search in PsychInfo and PubMed databases for empirical articles published in (peer reviewed) journals in English language between 1987 and June 2009 by combining the keywords 'traumatic stress', 'PTSD' and / or 'post traumatic stress disorder' with the search term 'neuroticism'. This yielded 142 unique hits. These included 18 reviews/ theoretical papers, 6 commentaries, 2 case studies, 1 reprint, and 1 animal study, which were excluded. Of the remaining 114 articles, studies focusing exclusively on children or childhood trauma were excluded, as were articles that did not examine the relationship between neuroticism and trauma or PTSD. Relevant papers that were cited by other authors were also included in the study, resulting in a total number of 70 reviewed articles.

Table 1: Summary of studies reporting an association between neuroticism and PTSD, grouped by research design (E, C, L, P) †.

Study	Measure		Sample	ΔT	Design†	Result
	Neuroticism	PTSD				
Davies & Clark, 1998	EPQ	-	*N* =90 (52% ♂); Students.	N.A.	E	N did not predict intrusive recollections.
Boelen, 2009	EPQ	PSS-SR	*N*= 254 (11% ♂); Bereaved adults.	± 3.5 y	C	Correlation with total PSS-SR = .49; N predictor of PTSD, ns when controlling for concomitant symptoms.
Bramsen, Van der Ploeg, Van der Kamp & Ader, 2002	?	SRIP	*N* = 455 (80% ♂); Dutch WW II survivors.	> 45 y	C	N predictor of PTSD symptoms in path model.
Breslau, Davis, Andreski & Peterson, 1991	EPQ-R	DIS	*N* = 1007 (38% ♂); Community sample of young adults.	?	C	N predictor of PTSD, OR = 1.53.
Brodaty, Joffe, Luscombe & Thompson, 2004	EPI-SF	DSM-IV	*N* = 100 (41% ♂); Holocaust survivors.	> 50 y	C	N predictor of PTSD; OR = 2.63.
Carr, Lewin, Kenardy, Webster, Hazell, Carter, et al., 1997	EPI-SF	IES	*N* =680 (?); Earthquake survivors.	+6-24 mts	L[1]	Correlation with total IES = .49; N predictor of PTSD, partial correlation = .42.
Chung, Berger, Jones & Hanah, 2006	Neo FFI	PDS	*N* = 96 (81% ♂); Elderly MI patients.	> 1 mts	C	N predictor of all PTSD symptom clusters.
Chung, Dennis, Easthope, Werret & Farmer, 2005	EPQ-R-SF	IES	*N* = 148 (34% ♂); Civilians exposed to a plain/ train crash.	+6-7 mts	C	Correlation with intrusions = .40; avoidance = .36; N predictor of intrusion and avoidance symptoms in path model.
Chung, Easthope, Chung & Clark-Carter, 1999	EPQ-R-SF	IES	*N* = 82 (35% ♂); Civilians exposed to an airplane crash.	+6 mts	C	N predictor of PTSD symptoms, explaining 8% variance intrusion, 9% avoidance.

Study	Measure		Sample	ΔT	Design†	Result
Chung, Easthope, Farmer, Werrett & Chung 2003	EPQ-R	IES	*N* = 66 (35% ♂); Civilians exposed to a train crash.	+7 mts	C	N predictor of PTSD, explaining 12% of variance intrusion, 13% avoidance symptoms.
Chung et al., 2002	EPQ-R	IES	*N* = 60 (40% ♂); Adults after relationship ending.	+0-24 mts	C	N predictor of PTSD symptoms explaining 3% of variance.
Cox, MacPherson, Enns & McWilliams, 2004	Goldberg‡	DIS	*N* = 3238 (53% ♂); Community sample.	?	C	N predictor of PTSD, OR (♀) = 1.85, OR (♂) = 1.93.
Hyer et al., 1994; 2003	Neo-PI	Miss, IES	*N* = 80 (100% ♂); Vietnam veterans.	> 15 y	C	Correlation with IES = ns; Miss total = .32.
Inoue, Tsukano, Muraoka, Kaneka, & Okamura, 2006	EPQ-R	IES-R	*N* = 141 (20% ♂); Nurses.	?	C	Correlation with t otal IES-R = .45
Jaycox, Marshall, & Orlando, 2003	Neo FFI‡	PCL	*N* = 267 (100% ♂); Victims of community violence.	+0-3 w	C	Correlation with t otal PCL = .36
Lauterbach & Vrana, 2001	EPQ-R	PPTSD-R	*N* = 402 (56% ♂); Student sample.	?	C	Correlation with total PPTSD-R = .39
Matsuoka, Inagaki, Sugawara, Imoto, Akechi & Uchitomi, 2005	EPQ-R	SCID	*N* = 155 (0% ♂); Breast cancer patients after surgery.	+3-15 mts	C	N after surgery predicted intrusive symptoms, OR = 1.27.
Morgan, Matthews, & Winton, 1995	EPQ-R-SF	DSM-III-R rated	*N* = 44 (24% ♂); Flood victims.	?	C	Correlations with intrusions: ns; avoidance = .54; arousal = .36.
Pedersen & Denollet, 2004; Pedersen, Middel, & Larsen, 2002, 2003	EPQ-SF	PDS	*N* = 112 (70% ♂); MI patients.	+4-6 w	C	Correlation with PDS intrusions = .30; avoidance = .41; arousal = .44. N predictor of PTSD symptoms OR = 1.36.
Sembi, Tarrier, O'Neill, Burns, & Faragher, 1998	EPQ-R-SF	PENN, IES	*N* = 61 (?); TIA/ stroke patients.	+18 mts	C	Correlation with PENN total =.64; IES avoidance = .46.
Stewart, Conrod, Samoluk, Pihl & Dongier, 2001	Neo FFI	PSS-SR	*N* = 294 (0% ♂); Substance abusers.	?	C	Correlation with PSS-SR total = .26; PSS-SR scores predicted N.

Study	Measure		Sample	ΔT	Design†	Result
Suar, Mandal & Khuntia, 2002	MPI‡	DSM-IV	*N* = 65 (69% ♂); Indian cyclone victims	+3 mts	C	N did not predict PTSD over exposure status.
Thompson & Solomon, 1991	EPQ	IES	*N* = 31 (90% ♂); Body recovery workers.	+7-15 mts	C	Correlation with IES total = .39.
Watson, Gamez & Simms, 2005	SNAP	SCID, PCL	*N* = 573 (100% ♂); Gulf war veterans.	> 6 yrs	C	Correlations with SCID diagnosis = .35; PCL total = .44; intrusions = .32; avoidance = .28; arousal = .30.
Weiss, Marmar, Metzler, & Ronfeldt, 1995	HPI	Miss, IES-R	*N* = 154 (88% ♂); Emergency workers.	1.5 yrs	C	Correlation with Miss total = .43; IES-R intrusions = .24; avoidance = .24; arousal = .32.
Van Zelst, De Beurs, Beekman, Deeg & Van Dijk, 2003	DPI	CIDI	*N* = 422 (43% ♂); Community sample of older persons.	?	C	N predictor of 6 month prevalence of PTSD, OR = 13.4.
Bennett, Owen, Koutsakis, & Bisson, 2002	PANAS	PDS, IES	*N* = 75 (78% ♂); MI patients.	T1: +0 T2: +3 mts	L	Correlations N (T1) and PDS (T2): avoidance = .27; arousal = .30; intrusions = .40; N (T1) predicted PDS (T2) intrusions and arousal after controlling for IES (T1).
Holeva & Tarrier, 2001	EPQ-R	PENN	*N* = 265 (42% ♂); Victims of road traffic accidents.	T1: +2-4 w T2: +4-6 mts	L	Correlation between N (T1) and PTSD (T1/ T2): T1 = .50; T2 = .44; N (T1) predicted PTSD (T2).
Lawrence & Fauerbach, 2003	Neo FFI	DTS	*N* = 158 (84% ♂); Adult burn survivors.	T1: +0 T2: +1 mts T3: +6 mts	L	N (T0) predicted acute (T0) PTSD symptoms only, not at T2 or T3.
Lee, Vaillant, Torrey, & Elder, 1995	Neo-PI	DSM-III	*N* = 107 (100% ♂); WW II veterans.	T1: +1 yr T2: +43 yrs	L	Correlation between N (T2) and DSM-III symptoms: T1: ns; T2: .20.

Study	Measure		Sample	ΔT	Design†	Result
Mason, et al., 2009	EPI	IES-R	N = 832 (60% ♂); Patients at hospital emergency department.	T1: +0 T2: +1 mts T3: +3 mts	L	N (T1) ns predictor of PTSD (T3), when controlling for psychiatric history and PTSD symptoms (T2).
Ribi, Vollrath, Sennhauser, Gnehm & Ladolt, 2007	Neo FFI	PDS	N = 139 (100% ♂); Fathers of pediatric patients.	T1: +4-6 w T2: +6 mts	L	Correlation between N (T2) and PDS total (T1/ T2): T1: .23; T2: .33; N (T2) predictor of PTSD (T2).
Engelhard, Huiding, Van den Hout & De Jong, 2007	EPQ	SCID, PSS-SR	N = 169 (100% ♂); Soldiers deployed to Iraq.	T1: -1.5 mts T2: +5 mts T3: +15 mts	P	Correlations N (T1) and PSS/ SCID: T2: .29/ .18; T3: .28/ ns; Pre-trauma N ns predictor of PTSD after controlling for perceived vulnerability scores (T2)/ prior symptoms (T3).
Engelhard & Van den Hout, 2007	EPQ-SF	SCID, PSS-SR	N = 383 (97% ♂); Soldiers deployed to Iraq.	T1: -1.5 mts T2: +5 mts	P	Correlation N (T1) and total PSS (T2) = .03; SCID symptoms (T2) = .25.
Engelhard, Van den Hout & Kindt, 2003	EPQ		N = 118 (0% ♂); Women exposed to pregnancy loss.	T1: -1 mts[2] T2: +1 mts	P	Correlation N (T1) and PSS (T2) total PSS = .25; intrusions = ns; avoidance = .19; arousal = .34; N ns predictor of PTSD when controlling for pre-trauma arousal.
Engelhard, Van den Hout, Kindt, Arntz & Schouten, 2003; Van den Hout & Engelhard 2004	EPQ	PSS-SR	N = 118 (0% ♂); Women exposed to pregnancy loss.	T0: -1 mts[2] T1: + 1mts T2: +4 mts	P	Correlation N (T0) and PSS (T1/ T2): T1 .25; T2 = .25; Pre-trauma N predictor of PTSD symptoms.
Engelhard, Van den Hout, & Schouten, 2006	EPQ	PSS-SR	N = 117 (0% ♂); Women exposed to pregnancy loss.	T0: -1 mts[2] T1: +1mts	P	High N (dichotomized) increased risk of PTSD symptoms, OR = 2.2.

Study	Measure		Sample	ΔT	Design†	Result
Knezevic, Opacic, Savic, & Priebe, 2005	Neo-PI	IES	N = 70 (11% ♂); Students exposed to air raids.	T0: -1 to 2 yr T1: +0 T2: +1 yr	P	Correlations N (T0) and IES (T1/ T2): intrusions = ns/ ns; avoidance = ns/ ns; N (T0) did not predict PTSD (T2).
Parslow, Jorm & Christensen,2006	EPQ-R-SF	TSQ	N = 2085 (47% ♂); Community sample exposed to bushfires.	T0: -3.4 yrs T1: +38 wks	P	Pre-trauma N (T0) predictor of intrusion and avoidance (T1), (adjusted) incidence rate ratio = 1.03.

Note. † *E = Experiment, C = cross-sectional, L = longitudinal study including only post-trauma personality data, P = prospective study including pre-trauma personality measure;* ‡ *= adapted version; ? = not reported;* [1] *= correlation based on the average N score over two occasions and average IES score over four occasions;* [2] *= time-frame before exposure to trauma not specified; ΔT = elapsed time since trauma; BFI = Big five inventory; CIDI = Comprehensive international diagnostic interview; DSM = Diagnostic and statistical manual; DPI = Dutch personality inventory; DTS = Davidson trauma scale; EPI(-SF) = Eysenck personality inventory (Short Form); EPQ(-R; -SF) = Eysenck personality questionnaire (Revised; Short Form); HPI = Hogan personality inventory; IES(-R) = Impact of event scale (Revised); Miss = Mississippi scale for combat related PTSD; MPI = Maudsley personality inventory; N = neuroticism; Neo-PI = Neo personality inventory; Neo FFI = Neo five factor inventory; ns = non-significant; PCL = PTSD checklist; PANAS = Positive and negative affect schedule; PDS = Posttraumatic diagnostic scale; PENN = Penn inventory of PTSD; PPTSD-R = Purdue PTSD scale revised; PSS = Posttraumatic symptom scale (self-rating); PTSD = Posttraumatic stress disorder; SCID = Structured clinical interview for DSM; SNAP = Schedule for non-adaptive and adaptive personality; SRIP = Self-rating inventory for PTSD; TIA = transient ischemic attack; TSQ =Trauma screening questionnaire.*

Trauma, neuroticism, and PTSD

High neuroticism ratings have been reported in patients suffering from PTSD compared to controls across studies, including fire-fighters (McFarlane, 1988), burn-victims (Fauerbach, Lawrence, Schmidt, Munster, & Costa, 2000), HIV-diagnosed bi- and homosexual men (Kelly et al., 1998), and shipwreck survivors (Thompson, Chung, & Rosser, 1994). Increased neuroticism ratings were also observed in a people meeting life-time criteria for PTSD in a large epidemiological study in the U.S. (Lauterbach, Vora, & Rakow, 2005), in treatment seeking offspring of WW II survivors (Mook et al., 1997), and combat veterans from various war era's (Casella & Motta, 1990; Davidson, Kudler, & Smith, 1987; Dimic, Tosevski, & Jankobic, 2004; Kozaric-Kovacic et al., 2000; Talbert, Braswell, Albrecht, Hyer, & Boudewyns, 1993).

Moreover, as shown in Table 1, cross-sectional studies generally report small to moderately strong correlations between neuroticism and measures of PTSD. Therefore, despite some contradicting findings (Hyer et al., 1994; 2003) the majority of papers show a relationship between neuroticism and PTSD irrespective of trauma type, gender, age, time since trauma, and measures used. Several explanations can be forwarded to account for this association (see also Clark, Watson, & Mineka, 1994). First, it may be that that exposure to trauma leads to increased neuroticism, parallel to and independent of the development of PTSD. This association was previously described as a common-cause model (e.g., Lyons, Tyrer, Gunderson, & Tohen, 1997). Second, higher pre-trauma neuroticism levels may have been present in subjects who later developed PTSD and neuroticism may have acted as a risk factor for trauma exposure. Third, the relationship between neuroticism and PTSD can be explained from a diathesis-stress or vulnerability model for psychopathology. That is, neuroticism might increase the vulnerability for PTSD after exposure to extreme circumstances, either by affecting the way individuals deal with the traumatic sequelae or independent of the relationship between trauma exposure and PTSD (Ormel & Wohlfarth, 1991, in Carr et al., 1997). Fourth, as described in spectrum models, it may be that PTSD and neuroticism are part of the same continuum and reflect the same underlying processes. Finally, PTSD may lead to increased neuroticism, as described in 'scar' or 'complication' models (Akiskal, Hirschfeld, & Yerevanian, 1983).

Studies that provide more insight into the validity of the hypotheses mentioned above are described in more detail in the following sections. First, in the next paragraph, available studies on the association between neuroticism and exposure to trauma are reviewed. Empirical evidence for respectively the common cause model and the potential role of neuroticism as a risk factor for trauma exposure are discussed. The subsequent section focuses on the relationship between neuroticism and PTSD. It reviews available evidence for the hypothesis that neuroticism may act as vulnerability factor in the development and maintenance of PTSD. The potential role of coping and appraisal in the relationship between neuroticism and PTSD is discussed as well as the available evidence for a spectrum model. Additionally, the possibility that PTSD may give rise to increased neuroticism will be discussed briefly. Finally, we address the possibility that the relationship between neuroticism and PTSD is tautological.

Neuroticism and trauma exposure

As can be seen in Table 2, several studies reported a relationship between exposure to traumatic events and neuroticism. Two explanations can be forwarded to account for the possible association between neuroticism and exposure to trauma. First, although personality dimensions are generally assumed to be relatively stable over time, it is possible that they are affected by exposure to traumatic events (WHO, 1992). Alternatively, it is possible that neuroticism predisposes individuals to trauma exposure.

Table 2: Summary of studies reporting an association between neuroticism and exposure to adverse events and trauma, grouped by research design (C, L, P) †.

Study	Neuroticism Measure	Sample	ΔT	Design†	Association with neuroticism
Avdibegović & Sinanović, 2006	CMI	*N* = 215 (0%♂); Abused women.	?	C	Correlation with severity of respectively physical abuse = .47; sexual abuse = .37; psychological abuse = .47.
Bramsen, Van der Ploeg, Van der Kamp & Ader, 2002	?	*N* = 455 (80% ♂); Dutch WW II survivors.	> 45y	C	Relationship between trauma exposure and N mediated by appraisal of meaning.
Breslau, Davis, Andreski & Peterson, 1991	EPQ-R	*N* = 1007 (38% ♂); Community sample of young adults.	?	C	N exposed > N non-exposed; N predictor of life-time exposure, OR = 1.20.
Bunce, Larsen & Peterson 1995	EPQ	*N* = 58 (32% ♂); Students.	?	C	N exposed > N non-exposed
Chung, Dennis, Easthope, Werret & Farmer, 2005	EPQ-R-SF	*N* = 148 (34% ♂); Civilians exposed to a plain/ train crash.	+6-7 mts	C	Correlation with being away or at home during crash = .23; distance from crash site = ns.
Chung, Easthope, Farmer, Werrett & Chung 2003	EPQ-R	*N* = 66 (35% ♂); Civilians exposed to a train crash.	+7mts	C	Residents who lived closer to crash site did not display higher N scores than residents who lived further away.
Jaycox, Marshall & Orlando, 2003	Neo FFI‡	*N* = 267 (100% ♂); Victims of community violence	+0-3 wks	C	Correlation with life-time exposure to violence = .35; past year life-events = .32; injury severity = ns.
Lauterbach & Vrana, 2001	EPQ-R	*N* = 402 (56% ♂); Students.	?	C	Correlation with life-time exposure = ns, trauma intensity = .21.
Talbert, Braswell, Allbrecht, Hyer & Boudewyns, 1993	Neo-PI	*N* = 267 (100% ♂); Male Vietnam veterans with PTSD.	> 15 yrs	C	No relation with level of combat exposure.

Study	Neuroticism Measure	Sample	ΔT	Design†	Association with neuroticism
Engelhard, Van den Hout, Kindt, Arntz & Schouten, 2003	EPQ	*N* = 118 (0% ♂); Women exposed to pregnancy loss.	?	P*	Non-significant correlation between life-time trauma exposure and neuroticism.
Carr et al., 1997	EPI-SF	*N* =680 (?); Earthquake survivors.	+6-24 mts	L	Correlation with disruption as a result of trauma = NS; level of threat = .22; life events = .36; ongoing disruptions = .23.
Lee, Vaillant, Torrey & Elder, 1995	Neo-PI	*N* = 107 (100% ♂); WW II veterans.	+1, +40 yrs	L	Non-significant correlation between combat exposure and late life N.
Breslau, Davis & Andreski, 1995	EPQ-R	*N* = 979 (38%♂); Community sample of young adults.	+0-3yrs	P	N predictor of trauma exposure in three year follow-up, OR = 1.28.
Engelhard & vd Hout 2007	EPQ-SF	*N* = 383 (97%♂); Soldiers deployed to Iraq.	0, +5 mts	P	Correlation with pre-deployment N and minor stressor = .28; major stressors = .12.
Löckenhoff, Terracciano, Patriciu, Eaton, & Costa, 2009	Neo-PI-R	*N* = 458 (36%♂); Community sample.	0-2 yrs	P	N Post-trauma > N Pre-trauma in individuals exposed to extreme event(s); Pre-trauma N did not predict exposure.

*Note. ? = not reported; ‡ = adapted version; † C = cross-sectional, P = prospective study including pre-trauma personality measure, L = longitudinal study; * only cross-sectional data are reported; ΔT = elapsed time since trauma; CMI = Cornell Medical Index; EPI (-SF) = Eysenck personality inventory (Short Form); EPQ(-R; -SF) = Eysenck personality questionnaire (Revised; Short Form); N = neuroticism; Neo-PI(-R) = Neo personality inventory (revised); Neo FFI = Neo five factor inventory; ns = non-significant.*

Common cause model

Exposure to trauma might affect an individual's belief-system and cognitive schemas (Janoff-Bulman, 1992; McCann & Pearlman, 1990), and it is possible that it affects other personality domains as well. Some evidence for a relationship between trauma and neuroticism can be found in studies that reported a correlation between trauma exposure and neuroticism (Avdibegovic & Sinanovic, 2006; Chung, Dennis, Easthope, Werrett, & Farmer, 2005; Jaycox, Marshall, & Orlando, 2003). Additionally, cross-sectional studies by Breslau et al. (1991) and Bunce et al. (1995) reported higher neuroticism ratings in trauma-exposed individuals relative to non-exposed controls. Similarly, in a study in a large community sample, Bramsen and colleagues (Bramsen, Van der Ploeg, Van der Kamp, & Ader, 2002) observed that, relative to non-exposed individuals, respondents who reported more wartime exposure, scored significantly higher on several (neurotic) personality traits.

To explain these findings, Bramsen et al. (2002) proposed that experiencing a traumatic stressor might change a person's belief system, and that these changes could lead to changes in trait neuroticism. They investigated this hypothesis by examining whether 'attribution of meaning' mediated the relationship between war time exposure and neuroticism, using structural equation modeling on data obtained from a sample of 455 subjects who had experienced high amounts of wartime stress. Results showed that wartime stressors were directly related to victims' attributions, and that these in turn statistically predicted neuroticism ratings. No direct relationship was found between wartime stress and neuroticism; only an indirect relationship was observed, mediated by negative appraisals (meaning) of wartime stressors. Unfortunately, this study was limited by the fact that it relied on retrospective data that were collected 50 years after the war. Therefore, it is unclear to what extent attributions of meaning were affected by pre-trauma characteristics and post war influences

These results are contrasted by a number of studies that failed to find any evidence for an association between trauma exposure and neuroticism. Engelhard et al. (2003b) found no association with life-time trauma exposure in women who had recently experienced pregnancy loss. Similarly, Lauterbach and Vrana (2001) found no evidence for an association between life-time exposure to trauma and neuroticism in a student sample. Chung et al. (2003) reported that individuals that lived closer to a crash site did not display higher neuroticism ratings then those who lived further away, and Lee et al. (1995) found no significant correlation between level of combat exposure during WW II and neuroticism 40 years after the war. Similarly, Talbert et al. (1993) found no relationship between neuroticism and combat exposure in a cross-sectional study of 100 Vietnam veterans suffering from PTSD. However, it is possible that the (potential) relationship between exposure and neuroticism was obscured by the fact that all veterans in this study were diagnosed with PTSD in his study. Additionally, as the veterans were characterized by extremely high neuroticism scores, ceiling effects may have obscured the association between neuroticism and trauma exposure. Finally, in a large longitudinal study of earthquake survivors, Carr et al. (1997) did not find an association between post-trauma neuroticism and level of disruption caused by the earthquake although small correlations were reported between neuroticism and life-events (r = .36) as well as ongoing disruptions (r = .22) in the years following the disaster.

Despite these contradicting results, compelling evidence in support of a common cause model was recently provided in a large prospective and longitudinal study by Löckenhof et al. (2009). In this study neuroticism was assessed twice within an average time frame of 8 years, and respondents were asked whether or not they had experienced any trauma in the two years prior to the second assessment. Results showed that exposed adults displayed a greater increase in neuroticism as compared to non-exposed individuals. Unfortunately however, the

authors did not report more information on trauma characteristics. Nevertheless, this study demonstrates that neuroticism may be subject to change following exposure to trauma. It may be especially sensitive to interpersonal violence (Avdibegovic & Sinanovic, 2006; Jaycox et al., 2003), although additional research is needed to confirm the differential effects of specific trauma types on neuroticism.

Neuroticism as a risk factor

An alternative explanation for the relationship between neuroticism and trauma exposure as observed in cross-sectional studies is that neuroticism somehow predisposes individuals to encounter more events that are traumatic. Although a prospective study by Löckenhoff et al. (2009) did not show an association between neuroticism and exposure, evidence for this hypothesis was provided in several other studies. For instance, in a cross-sectional study Breslau et al. (1991) examined the potential predisposing effect of neuroticism on trauma exposure in a community sample of young adults. Neuroticism was found to predict self-reported lifetime exposure to trauma in regression analyses, with an odds ratio of 1.20. More importantly however, the authors later replicated these findings in a prospective study. In a three year follow-up, neuroticism was found to predict subsequent trauma exposure with odds ratios similar to those obtained in the retrospective ratings (Breslau, Davis, & Andreski, 1995).

Additional evidence was provided by a longitudinal study of Carr et al. (1997) that linked life-events and disruptions following an earthquake to victims' neuroticism levels as measured after the events. More compelling evidence was provided in a prospective study by Engelhard and Van den Hout (2007), who showed that in Dutch soldiers deployed to Iraq, small correlations were present between neuroticism ratings obtained prior to military deployment and stressors encountered during the subsequent deployment.

Results from genetic and twin studies may corroborate these findings. Studies have shown that genetic factors can predict combat exposure (Lyons et al., 1993) as well as exposure to interpersonal violence (Stein, Jang, Taylor, Vernon, & Livesley, 2002). Koenen et al. (2008) attributed these findings to personality. As research has shown that neuroticism is about 40 to 50% heritable (Floderus-Myrhed, Pedersen, & Rasmuson, 1980; Jang, Livesley, & Vernon, 1996), it is possible that trait neuroticism is one of the temperamental traits associated with increased risk of exposure trauma. At least with respect to self-reported stressful life events, there appears to be proof for this hypothesis (e.g., Van Os & Jones, 1999). Therefore, these findings show neuroticism can add to the risk of developing PTSD by increasing the risk of exposure to stress and trauma.

Overall, the available studies on the relationship between trauma and PTSD suggest that neuroticism ratings are themselves subject to change following exposure to trauma. Additionally, the studies described in this section demonstrated that neuroticism can increase the risk of exposure to stress and trauma. Although it may be hard to imagine that highly neurotic individuals are at increased risk of experiencing natural disasters, a relationship between personality and interpersonal trauma seems plausible. Nonetheless, before we can conclude that neurotic individuals somehow cause traumatic events to happen to them, we have to consider alternative explanations for these findings. Perhaps highly neurotic individuals subjectively experience more events as traumatic rather than that they encounter more (objective) traumatic events. This possibility will be addressed in the next section.

Neuroticism and PTSD

Neuroticism is related to trauma exposure. It may also determine who develops PTSD after exposure to stress and trauma and who does not. For instance, McFarlane (1988) observed that even though all firefighters in his study had been exposed to similar levels of stressor intensity, only those with high levels of neuroticism developed PTSD. Perhaps, neuroticism mediates or moderates the relationship between trauma and response. It is possible that the trait affects the way a stressor is perceived and the way it is handled. Further, neuroticism may reflect an inherent vulnerability factor or diathesis for PTSD. That is, individuals high in neuroticism may perceive more events as stressful; they may be more sensitive to the effects of stressors and/ or less proficient in coping with (traumatic) stress, and they may be more prone to experiencing negative emotions like anxiety, dysphoria, guilt and anger (Watson, Clark, & Harkness, 1994).

Neuroticism as a vulnerability factor

The potential direct effect of neuroticism on PTSD following trauma exposure has received considerable attention. As can be seen in Table 1, despite some contradicting results (e.g., Suar, Mandal, & Khuntia, 2002), the majority of cross-sectional studies shows that neuroticism predicted PTSD severity and symptoms across a wide variety of trauma samples. Although only a limited number of prospective studies is available, most of these, though not all (Knezevic, Opacic, Savic, & Priebe, 2005), also provide evidence to support a diathesis-stress model in which neuroticism increases vulnerability for PTSD.

In a large prospective community study, Parslow et al. (2006) showed that neuroticism assessed prior to the outbreak of fires in Australia, significantly increased the odds of meeting PTSD criteria after 3-18 months, independent of the effects of other variables like gender, age, trauma characteristics, perceived threat, and social support. Likewise, a study of Australian Vietnam veterans showed that neuroticism ratings at enlistment were higher in soldiers who later developed PTSD (O'Toole, Marshall, Schureck, & Dobson, 1998a, 1998b). Finally, a prospective study of females who experienced pregnancy loss, reported a significant association between pre-trauma neuroticism and post-trauma avoidance and arousal symptoms (Engelhard, Van den Hout, & Kindt, 2003a).

The relationship between neuroticism and PTSD appears to depend on other variables as well. Results from a cross-sectional study of male victims of community violence showed that the relationship between neuroticism and acute PTSD was no longer significant after controlling for distress (Jaycox et al., 2003). Similarly, Boelen (2009) and Mason et al. (2009) observed that post-trauma neuroticism no longer significantly predicted PTSD scores after statistically controlling for psychiatric history and prior PSTD symptoms (Mason et al. 2009) or concomitant PTSD symptoms (Boelen, 2009). By contrast, several studies demonstrated that post-trauma neuroticism remained a significant predictor of PTSD symptoms after controlling for prior PTSD in fathers of pediatric patients (Ribi et al., 2007), and MI patients (Bennett et al., 2002). Morgan et al. (1995) also reported that in flood survivors, correlations between neuroticism and total number of self-reported PTSD symptoms remained significant after controlling for symptom severity (partial correlation .34).

In a prospective study of Dutch peacekeepers, the effect of pre-trauma neuroticism on PTSD symptoms 5 months after deployment failed to reach significance when post-trauma vulnerability assumption and beliefs were entered simultaneously, although a trend ($p = .06$) could be observed in the follow-up assessment 15 months after deployment (Engelhard, Huijding, Van den Hout, & De Jong, 2007). Furthermore, although Engelhard and coworkers showed that neuroticism prospectively predicted PTSD symptoms in women who experienced pregnancy

loss (Engelhard et al., 2003a; Engelhard et al., 2003b; Engelhard, Van den Hout, & Schouten, 2006; Van den Hout & Engelhard, 2004), they also reported that this relationship was no longer significant after controlling for pre-trauma arousal (Engelhard et al., 2003a).

Overall, studies that did find neuroticism to prospectively predict PTSD showed that the contribution of the trait to the increased risk is relatively small. Pre-trauma neuroticism accounted for 3-6% of explained variance in PTSD scores in women who experienced pregnancy loss (Engelhard et al., 2003a; Van den Hout & Engelhard, 2004) and Parslow et al. (2006) reported an increased risk ratio of 1.06. Therefore, it appears that the direct effect of neuroticism on PTSD is modest at most. Furthermore, controlling for (pre-existing) arousal, distress or clinical symptoms can attenuate the strength of the relationship between neuroticism and PTSD. Controlling for these factors may impose an important conceptual problem however, as it could be argued that they constitute core features of trait neuroticism. We will elaborate on this issue below.

Moderating the dose-response relationship

In addition to the direct - albeit modest - effect of neuroticism on PTSD severity, neuroticism may interact with trauma intensity. The role of neuroticism as a moderator in posttraumatic adjustment was examined by Lauterbach and Vrana (2001). In a sample of 402 students they found a significant interaction between trauma intensity and neuroticism. Trauma intensity was only marginally related to PTSD in subjects low in neuroticism, whereas a strong relationship was found between trauma intensity and PTSD scores in persons high in neuroticism. Further, a regression analysis showed that neuroticism alone, did not predict self-reported PTSD symptoms, but that the interaction-term of trauma intensity and neuroticism was significantly related to the explained variance in PTSD severity over the effects of trauma intensity (squared semi partial correlation = .02), increasing the amount of explained variance by 12%.

Appraisal

As stated in DSM-IV (APA, 1994) the experience of a traumatic stressor is a prerequisite for the development of PTSD. However, DSM-IV also states that this stressor must be accompanied by sufficient levels of emotional distress. Therefore, subjective experience is an equally important determinant of the posttraumatic reaction, and neuroticism, with its strong affective component, may affect the way potential stressors are appraised.

In line with earlier cognitive models (e.g., Creamer, 1995; Horowitz, 1976), Ehlers and Clark (2000) proposed a model for PTSD in which individual differences in appraisal were considered to be predictors of persistent morbidity. They suggested that individuals who develop PTSD are characterized by idiosyncratic negative appraisal of either themselves or the external world which would lead them to appraise more situations as stressful or harmful. Idiosyncratic appraisals may reflect cognitive styles that are linked to neuroticism. If so, high neuroticism may elicit negative appraisals of the self and external world, resulting in increased perceived threat and increased vulnerability for PTSD.

If there is indeed a relationship between neuroticism and appraisal, this would provide an alternative explanation for the findings of an association between neuroticism and trauma exposure. This would suggest that neurotic individuals perceive more situations as stressful, rather than or in addition to the increased risk of encountering traumatic events. This hypothesis fits well with data from Carr et al. (1997) who reported that (post-trauma) neuroticism was unrelated to 'objective' levels of trauma exposure (i.e., disruption) in earthquake survivors, whereas a small but significant correlation (r = .22) was observed with levels of threat. Similarly, Lauterbach and Vrana (2001) reported a correlation of the same magnitude

between neuroticism and a measure of trauma intensity (perceived danger, stressors severity and injury severity), but no association with exposure to traumatic events. Chung et al. (2005) who focused on the potential relationship between neuroticism and threat appraisal in a study of 238 subjects exposed to a plane or train crash, reported correlations ranging from .31 to .39 between neuroticism and death anxiety.

Engelhard and Van den Hout (2007) have provided the only available prospective evidence to support the notion that neuroticism affects the appraisal of external stressors. In a study of Dutch soldiers deployed during the last Gulf War they reported an association between pre-deployment neuroticism and the appraisal of both minor (e.g., climate, trouble getting e-mail, reduced life-space) and major (e.g., being shot at, going on patrol) deployment stressors. They showed that the appraisal of major stressors was largely independent of pre-war neuroticism as controlling for prewar neuroticism reduced the explained variance in PTSD scores by (only) 4 to 7 %. However, controlling for pre-deployment neuroticism affected the association with minor stressors rather drastically, as it reduced the amount of explained variance by 22 to 31%. This suggests that neuroticism is especially salient to the appraisal of minor and (possibly also ambiguous) external stressors.

These studies show that neuroticism can affect the appraisal of external stressors and that it is one of the pathways through which neuroticism increases the vulnerability for PTSD. Interestingly, and contrary to the hypotheses formulated by Ehlers and Clark (2000), Engelhard et al. (2007) demonstrated that negative appraisals about the self did not prospectively predict the occurrence of PTSD symptoms in Dutch soldiers. Neurotic individuals may thus be more vulnerable for PTSD because they display idiosyncratic negative appraisals of (relatively mild) external stressors: they may perceive stressors as more threatening and they can be expected to report more daily hassles.

Relationship to coping

Aspinwall (2004) pointed out that personality characteristics can be expected to affect the coping process at various stages. In addition to an association between personality and appraisal, she proposed that neurotic individuals experience more negative emotional arousal when confronted with stressors, which leads them to focus their (initial) coping efforts on regulating these emotions rather than on effectively dealing with the problem at hand. Thus, neuroticism would be expected to be positively related to avoidant and emotion-focussed coping styles and inversely to active or problem oriented coping. Since active or problem oriented coping is usually associated with positive health outcomes whereas passive or avoidant coping can be linked to poor health outcomes and low resilience to stress (Campbell Sills, Cohan, & Stein, 2006; Folkman & Moskowitz, 2004), this provides an additional explanation for the association between neuroticism and PTSD.

Support for this hypothesis comes from a cross-sectional study by Morgan et al. (1995) who reported a moderately strong association between neuroticism and emotion-focused coping, which in turn was related to greater symptom severity in flood survivors. Morgen et al. found no relationship between neuroticism and problem oriented coping however. Chung et al. (2005) examined the relationship between neuroticism, coping and PTSD after exposure to an aircraft or train crash. After testing several models, they concluded that a model in which PTSD was caused and maintained by both direct effects of coping and neuroticism as well as the interaction between these two, provided the best fit. Specifically, the results provided evidence for partial mediation because both coping and personality were directly related to scores on Impact of Event Scale (IES; Horowitz, Wilner, & Alvarez, 1979), although neuroticism also affected the IES ratings indirectly through coping. However, contrary to what would

be expected, Chung et al. reported that neuroticism was positively related to both emotion-focused coping and problem focused coping - even though the association with problem-oriented coping was rather small -, and that both coping styles were related to PTSD symptoms.

Lawrence and Fauerbach (2003) adopted a similar research strategy in their study of burn-survivors. They used structural equation modeling to predict PTSD levels from personality and coping the first six months after hospitalization. At hospitalization, a model that included both a direct and an indirect relation between neuroticism and PTSD, partially mediated by avoidant coping, accounted for 46% of the variation in PTSD scores. At follow-up one month later, only the relationships between neuroticism and coping, and between coping and self-reported PTSD symptoms, remained significant. At this time, the amount of explained variance dropped to 29% and the relationship between neuroticism and PTSD was fully mediated by avoidant coping and social support. After six months neither avoidance coping nor neuroticism significantly predicted PTSD, but the lack of significance may have been caused by subject attrition.

These results are corroborated by those of Carr et al. (1997) who reported a moderately strong association between post-trauma neuroticism and avoidance coping versus a negligibly small (positive) association with active coping. They also demonstrated that avoidance coping significantly added to the amount of explained variance in PTSD scores over the effects of neuroticism in their longitudinal study of earthquake survivors. Moreover, this study also reported a significant interaction effect of neuroticism and avoidance coping on PTSD symptoms.

Taken together, these studies demonstrate a relationship between neuroticism, emotional and/ or avoidance coping, and PTSD. There is evidence for (partial) mediation of the relationship between neuroticism and PTSD by avoidance coping behavior. However, the available data also show that the relationship between avoidance coping and PTSD cannot be fully explained by the effects of neuroticism as both coping and neuroticism are also uniquely associated to PTSD.

The course of PTSD

Since neuroticism can affect vulnerability to PTSD, it may also affect the course of the disorder. It may determine, at least in part, who recovers from PTSD and who does not. Only a handful of studies are available that provide insight into the validity of the pathoplasty model (Clark et al., 1994). Persistent morbidity was associated with higher neuroticism in fire fighters (McFarlane, 1989), and a study by Lewin, Carr and Webster (1998) also showed that persistent morbidity in earthquake survivors was associated with higher neuroticism. In a similar vein, patients treated for PTSD symptoms with anti-depressant medication (amitriptyline) were shown to have a better therapeutic prognosis when baseline levels of neuroticism were lower (Davidson et al., 1993). Thus, there appears to be some evidence to support the pathoplasty hypothesis, but clearly, more research is needed here.

Complication or scar models

Personality disturbances can be a sequel to psychiatric disorders (Akiskal et al., 1983), therefore the association between neuroticism and PTSD may represent the (toxic) effects of this disorder on personality. In a cross-sectional study of female substance abusers Stewart et al. (2000) examined this hypothesis and demonstrated that PTSD symptoms as measured with the PTSD Symptom Scale-Self-report (PSS-SR; Foa, Riggs, Dancu, & Rothbaum, 1993), statistically predicted neuroticism levels in regression analyses.

Treatment studies would be particularly insightful to examine potential scar effects: if personality changes would persist when PTSD is in remission, this would support the scar

hypothesis (see also, Lilenfeld, Wonderlich, Riso, Crosby, & Mitchell, 2006). We found only two studies that measured neuroticism before and after treatment of PTSD symptoms. One study reported that that post-treatment neuroticism ratings remained unchanged after 6 to 10 weeks of pharmacological treatment of PTSD symptoms in war veterans (Davidson, Kudler, & Smith, 1987). By contrast, a larger (N=112) randomized trial that assessed the effectiveness of brief psychotherapy (hypnotherapy, trauma desensitization or psychodynamic therapy) in treating PTSD, showed that between 14 and 18 sessions resulted in significantly reduced post-treatment neuroticism ratings in civilians exposed to various traumata (Brom, Kleber, & Defares, 1989).

It is possible that the differences between these results are due to differences in testing materials between studies. For instance, the personality measure used by Davidson et al. (1987) may have been less sensitive to state effects and (transient) levels of distress than the one used by Brom et al. (1989). In addition, differences in treatment modality may have lead to contradicting results. A -relatively short- pharmacological treatment may have little effect on personality characteristics whereas psychotherapy might affect personality functioning more rapidly or more profoundly. In addition, the fact that different samples were examined across studies limits comparability of results. For instance, treatment effects reported in studies of combat veterans are usually smaller than in civilian samples (Bradley, Greene, Russ, Dutra, & Westen, 2005).

Finally, it may be that the results reported by Davidson et al. reflect scar effects. As they included veterans from World War II, the Korean War, and the Vietnam War, the subjects in this study appear to have suffered from more chronic forms of PTSD than the participants in the study by Brom et al. (1989), who developed PTSD following trauma that occurred no more than five years before. If the scar hypothesis is valid, than it seems plausible that the longer people suffer from PTSD, the more pronounced the effects on personality would be and the more deeply the trauma would become ingrained in personality. Additional research is needed to assess whether or not trait neuroticism ratings are affected by the presence of (chronic) PTSD.

Tautology?

The reviewed papers have shown a relationship between neuroticism, trauma exposure and PTSD. How should these findings be interpreted? A large number of studies concluded that neuroticism increases the risk of PTSD. Overall however, available prospective studies showed lower rates of explained variance and lower odds ratios than studies that focused on post-trauma data exclusively. A possible explanation for the disparity in these results is that content overlap between neuroticism and PTSD measures artificially increased the strength of the association in post-trauma samples. Measures of neuroticism typically include items that assess negative mood, including anxiety and depressive symptoms, worry, anger and irritability (Costa & McCrae, 1992; Eysenck & Eysenck, 1975). These items are also included in measures of PTSD. For example, it could be expected that individuals who would endorse Neo-PI-R (Costa & McCrae, 1992) items like - *I am easily frightened* -, and - *Even minor annoyances can be frustrating to me* -, would also respond positively to questions like - *Have there been times when you felt especially irritable?* - and - *Have you had any strong startle reactions* -, which are part of the CAPS interview for PTSD (Blake et al., 1995). In fact, it could be expected that a PTSD patient would endorse almost all of the neuroticism items in the Neo-PI-R Angry hostility (N2) subscale, and at least one item from the Anxiety (N1), Depression (N3), Self-consciousness (N4), and Vulnerability (N6) subscale respectively, which would amount in an expected score of around 11 out of 48 neuroticism items. If we focus on the short form, the

Five Factor Personality Inventory, it could be expected that a PTSD patient would endorse approximately 3 to 8 out of 12 items, including items referring to uneven temperament, being a worrier, feelings of being unable to cope with one's problems, feeling lonely, feeling self-conscious around other people, and anxiety. The overlap with the Eysenck neuroticism measures is even more striking as it can be expected that a PTSD patient would endorse around 50% of neuroticism items in the EPI and EPQ-R (Eysenck & Eysenck, 1991).

On a theoretical level, the hypothesis that arousal symptoms account for the association between neuroticism and PTSD would fit well within Eysenck's original conceptualization of neuroticism in which arousal was a key feature. He stated that 'there seems to be little doubt that emotionality is linked to activity of the autonomic system' (Eysenck & Eysenck, 1969, p. 50). This is in accordance with results from Roca and colleagues (Roca, Spence, & Munster, 1992) who found increased neuroticism in burn victims who presented more hyper-arousal symptoms. Perhaps then, the relationship between neuroticism and PTSD is tautological.

Importantly, the association between PTSD and neuroticism is not limited to arousal symptoms but appears to extend to other symptoms as well. Studies have shown that the predictive value of neuroticism was reduced when controlling for psychiatric history and prior PTSD symptoms (e.g., Mason et al., 2009), perceived vulnerability (Engelhard et al., 2007), and distress (Jaycox et al., 2003), as well as arousal (Engelhard et al., 2003a). As Duncan-Jones et al. (1990) argued that neuroticism reflects an individual's characteristic pattern of psychiatric symptoms, the tautology appears to pertain to the arousal symptoms as well as to symptoms that are indicative of (generalized) levels of distress.

This point was previously raised by Ormel, Rosmalen and Farmer (2004; p. 906) who stated that 'even prospective associations of neuroticism with mental health outcomes are basically futile, and largely tautological since scores on any characteristic with substantial […] stability will predict, by definition, that characteristic and related variables at later points'. Consequently, the overlap between neuroticism and measures of PTSD (as well as other 'distress' disorders) might reflect more than a methodological issue related to content overlap. Perhaps it should be explained from a dimensional view on personality and psychopathology (Watson et al., 1994). Individuals high on neuroticism, who experience more baseline levels of distress (and arousal), may be more sensitive to relatively small increases in distress. That is, only modest increases in distress would be necessary in order for symptom levels to reach clinically significant levels. This 'threshold' hypothesis fits well with the data reported by Engelhard et al (2003a), although it fails to explain the results from studies that demonstrated that neuroticism may interact with trauma severity to produce different outcomes for individuals exposed to various levels of trauma severity (Lauterbach & Vrana, 2001).

Some studies reported an associations between neuroticism and specific PTSD symptom clusters of intrusions and avoidance (Carr et al., 1997; Chung et al., 2005; Chung et al., 1999; Pedersen & Denollet, 2004; Watson et al., 2005; Weiss et al., 1995). If the spectrum / dimensional hypothesis is valid, then these findings might indicate that PTSD intrusions and avoidance symptom clusters tap on avoidant and ruminative tendencies that are part of trait neuroticism. However, the spectrum hypothesis fails to explain indirect associations, i.e., when the effects of personality are mediated by other variables. It also fails to explain the predisposing role of neuroticism in trauma exposure. Therefore, although the spectrum hypothesis seems valid, it does not cover all aspects of the association between PTSD and neuroticism.

Methodological issues

Several methodological aspects need to be mentioned that hinder comparison of the reviewed papers and limit the generalizability of the reported results across studies. First, the outcome

measures used across studies vary. Second, the fact that different trauma populations were investigated must be taken into consideration when comparing results. Third, the possible influence of temporal effects on the strength of any reported associations is an important factor that must be considered. Each of these methodological issues will be discussed briefly.

Measures

Both PTSD and neuroticism have been measured using different instruments across studies. PTSD measures in particular, varied considerably across studies. One difference between self-report measures and clinical interviews is that most self-report measures for PTSD focus on symptoms or clusters exclusively, without providing any information on whether the other DSM criteria are met, in particular criterion A and F. For example, in Dutch soldiers15 months after deployment to Iraq, Engelhard et al. (2007) found a significant association between neuroticism and PTSD symptoms as measures with the PSS (Foa, 1995), which focuses specifically on cluster B, C and D symptoms, but not with the SCID (First, Spitzer, & Williams, 1997), which incorporates all DSM criteria. Moreover, even when the presence of PTSD is established following all DSM criteria, different DSM versions were used across studies, including DSM-III (APA, 1980), DSM-III-R (APA, 1987), and DSM-IV (APA, 1994). Seeing that symptoms have been reshuffled across DSM versions this could have affected the correlations between neuroticism and specific symptom clusters.

Additional differences exist between self-report measures for PTSD. For example, studies that used the IES (Horowitz et al., 1979) provide information on levels of intrusion and avoidance symptoms only, not on the number and/ or severity of hyper-arousal symptoms. By contrast, the Mississippi combat scale (Keane, Caddell, & Taylor, 1988) also includes items related to feelings of shame. The fact that the Mississippi addresses a broader range of symptoms might explain why Hyer et al. (1994; 2003) found a relationship between neuroticism and this PTSD scale only, but not with the IES.

Similarly, different neuroticism measures were used across studies. Some used the Neo-PI-R (Costa & McCrae, 1992), which consists of 48 items to measure neuroticism, versus 23 items in the revised Eysenck Personality Questionnaire (EPQ-R; Eysenck & Eysenck, 1991) and 12 items in the Neo-FFI (Costa & McCrae, 1992). These differences can be expected to represent variations in the range and scope of the neuroticism construct measured across studies. Not only does this limit the comparability of results, but different outcome measures can also be expected to yield differences in the strength of the relationship between neuroticism and PTSD. For instance, as demonstrated in the previous section, the correlation between PTSD and neuroticism could be expected to be considerably larger when the later is measured using one of the Eysenck questionnaires relative to the Neo-PI-R. Differences between neuroticism measures across studies may also explain why in the prospective study by Engelhard et al. (2007), who used the EPQ, a significant relationship was found with PTSD symptoms, whereas the prospective study by Knezevic et al. (2005), who used the Neo-PI-R, failed to find a significant association.

Trauma type

Previous studies have shown that intentional interpersonal violence, including sexual assault and combat, is associated with increased risk of PTSD compared to accidents or disasters (Creamer, Burgess, & McFarlane, 2001; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). In combat samples physical injuries during deployment have been shown to further increase the risk of subsequent PTSD (e.g., Grieger et al., 2006). As not all trauma types are equally related to PTSD, it is possible that the relationship between neuroticism and PTSD is co-dependent on the nature of the traumatic stressor.

Although correlations of the same magnitude (small to moderate) have been reported across trauma samples, results from studies using regression analyses varied somewhat across studies. For instance, lower amounts of explained variance were found in bereaved individuals (Chung et al., 2002; Van den Hout & Engelhard, 2004) compared to victims of technological disasters (Chung, Easthope, Chung, & Clark-Carter, 1999; Chung et al., 2003). Large scale community studies reported odds ratios associated with neuroticism between 1.6 and 1.9 for PTSD following life-time exposure to various traumata (Breslau et al., 1991; Cox, MacPherson, Enns, & McWilliams, 2004) and similar results were found in nurses exposed verbal and physical abuse (Inoue, Tsukano, Muraoka, Kaneko, & Okamura, 2006). Lower odds ratios were reported in myocardial infarction patients (e.g., Pedersen, Middel, & Larsen, 2002) whereas considerably higher odds ratios were found in Holocaust survivors (Brodaty, Joffe, Luscombe, & Thompson, 2004), and older adults (Van Zelst, de Beurs, Beekman, Deeg, & van Dyck, 2003). These data suggest that the strength of the association between neuroticism and PTSD may be moderated by trauma type. Additional prospective studies in different trauma samples are needed to confirm this hypothesis, especially since the available prospective data suggest that the predictive value of neuroticism may be limited (Parslow et al., 2006).

Temporal effects

Another important methodological issue with respect to the predictive validity of any variable in the etiology of PTSD is the influence of time between the stressor and data acquisition. The elapsed time since the occurrence of trauma has a direct effect on the number and intensity of reported symptoms. Although a small proportion of individuals develop chronic PTSD, most show a steady decrease of symptoms over time. Results from the National Comorbidity Survey reported by Breslau and colleagues (Breslau, Chilcoat, Kessler, Peterson, & Lucia, 1999), showed a steep decline in the proportion of individuals meeting PTSD criteria within the first 12 months of onset. They estimated that the median time of remission of PTSD is 24.9 months (Breslau et al., 1999). Since distress levels and posttraumatic stress symptoms can vary over time, associations between PTSD and other variables may also vary. Furthermore, stress symptoms that are present shortly after trauma may not be predictive of the development of PTSD (Shalev, 1992). Moreover, neuroticism ratings that are obtained shortly after the occurrence of a traumatic event may be inflated. Data collected shortly after trauma may therefore yield limited information on relevant factors in the etiology of PTSD.

Therefore, as Regehr et al. (2000) also pointed out, when the aim is to filter out characteristics that predict the occurrence of posttraumatic disturbances, it is advisable to gather data after the acute phase has passed. All individuals may display some stress symptoms shortly after the stressor, and individual differences in vulnerability or resilience will only become visible after a longer period has passed. Several studies support this notion as they showed stronger effects of personality on long-term morbidity when both were measured some time after the trauma (e.g., Brodaty et al., 2004; McFarlane, 1989; Van Zelst et al., 2003). Obviously, the most solid approach to examine the relationship between trauma, PTSD and personality would be to include pre-trauma data, but as this is often impossible, it is important to be aware of temporal effects.

Conclusion and future directions

The aim of this paper was to review papers describing a relationship between neuroticism, trauma, and PTSD, and to explore how neuroticism might be related to the etiology of PTSD. The reviewed papers demonstrated that neuroticism can be linked to PTSD through multiple pathways. First, prospective studies in civilian as well as combat samples showed that neuroti-

cism increases the risk of exposure to trauma. The most compelling evidence was provided by Breslau et al. (1995), who showed in a prospective study that high neuroticism increased the odds of exposure to traumatic events.

Second, since several studies provided evidence for a diathesis-stress model, there is evidence that neuroticism reflects an inherent vulnerability to PTSD. The reviewed papers showed that neuroticism can have direct as well as indirect effects in the etiology of PTSD, mediated by coping and appraisal. Studies in Vietnam veterans and fire fighters suggested that trauma severity in and of itself did not sufficiently predict PTSD as only subjects high on neuroticism developed PTSD (Casella & Motta, 1990; McFarlane, 1988). Also, one study reported that the interaction between neuroticism and trauma severity was a better predictor of PTSD than either factor alone (Lauterbach & Vrana, 2001). Additional evidence for the validity of a diathesis-stress model was provided by epidemiological and cross-sectional studies that showed that neuroticism was an important predictor of PTSD. Evidence for a vulnerability model was also provided by prospective studies (e.g., Parslow et al., 2006). However, prospective studies also showed that the direct and unique effects of neuroticism on PTSD were relatively small. Moreover, studies that examined the validity of a vulnerability model to account for the association between neuroticism and PTSD suggest that the association is tautological as both constructs tap on the same (theoretical) domain.

Issues that were insufficiently addressed in the reviewed papers pertain to the potential effects of trauma exposure and/ or PTSD on neuroticism ratings on the one hand, and to the possible interaction between neuroticism and PTSD in the progression of the disorder on the other hand. We were unable to find sufficient studies that assessed neuroticism prior to as well as after trauma exposure. Although we found one prospective study that underscored the validity of a common cause model, these results need to be replicated. Also, more longitudinal treatment studies are needed that assess neuroticism at different time points in order to assess the extent to which distress and clinical symptomatology affect neuroticism ratings, to determine the validity of the scar hypothesis, and to investigate whether neuroticism affects treatment outcome.

Further, it is important to remain aware of other variables that have been implicated in the etiology of PTSD. Although the reviewed papers show that neuroticism is related to the etiology of PTSD, research should try to demonstrate the *interplay* between personality and other relevant variables in the etiology of the disorder. For example, childhood trauma has been shown to increase the risk of PTSD (Ozer et al., 2003), and personality factors other than neuroticism may also add to the increased risk of trauma exposure and / or PTSD (Clark et al., 1994; Paris, 2000). As childhood trauma has also been associated with maladaptive personality functioning (e.g., Glaser, van Os, Portegijs, & Myin-Germeys, 2006), the relative importance of personality and early trauma on adult PTSD can only be established when both are assessed simultaneously.

The fact that neuroticism is associated with a range of mental disorders underscores the necessity to investigate the pathways more thoroughly (Ormel et al., 2004). Because of its largely non-specific relationship to psychopathology, establishing whether neuroticism predisposes an individual to PTSD reveals limited information on the mechanisms involved. Therefore, when the aim is to establish how personality predisposes an individual to PTSD, or *how* personality moderates the development of PTSD, and whether these mechanisms are specific to the relationship between neuroticism and PTSD, additional research is required.

One potentially fruitful line of research could be one that 'deconstructs' trait neuroticism. It would be useful to investigate if and how different aspects of neuroticism can be linked to the development and maintenance of different types of disorders. For instance, it seems

plausible that certain facets of the Neo-PI-R neuroticism scale (Costa & McCrae, 1992) would be more strongly related to PTSD than to depressive disorder. At face value, the subscales of anxiety, hostility and impulsiveness would appear more closely related to PTSD than the self-consciousness or vulnerability subscales. In addition, as neuroticism may increase the risk of PTSD by affecting the potential to effectively engage in adequate coping behavior, it would be useful to examine whether this holds for other disorders as well.

Finally, research into the 'biological' mechanisms underlying trait neuroticism may increase the understanding of the relation between personality and psychopathology (Ormel et al., 2004; Paris, 2000). For instance, research into shared neurobiological factors between high neuroticism and depressive disorders points towards commonalities in hypothalamic–pituitary–adrenal (HPA) functioning as well as functioning of specific brain areas (Foster & MacQueen, 2008, for a review). It would be useful to examine whether a similar relationship exists with PTSD. In conclusion, although a number of important issues remain unresolved, this review has highlighted the importance of examining personality aspects in the etiology of PTSD.

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Chapter 3

Self-reported early trauma as a predictor of adult personality

Self-reported early trauma as a predictor of adult personality: A study in a military sample.
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Introduction

There is ample empirical evidence that childhood trauma has a significant impact on the prevalence of various (mental) health problems in adults (Anda et al., 2006; Mulvihill, 2005; Whiffen & Macintosh, 2005). Childhood trauma has been linked to mental disorders, including mood disorders (e.g., Weiss, Longhurst, & Mazure, 1999; Widom, DuMont, & Czaja, 2007), attention-deficit/hyperactivity disorder (Rucklidge, Brown, Crawford, & Kaplan, 2006), pathological gambling and substance abuse (Kausch, Rugle, & Rowland, 2006), psychosis (Read, van Os, Morrison, & Ross, 2005), and posttraumatic stress disorder (PTSD) in both adult civilians (Twaite & Rodriguez-Srednicki, 2004; Yehuda, Halligan, & Grossman, 2001), and war veterans (Bremner, Southwick, Johnson, Yehuda, & Charney, 1993; Clancy et al., 2006; Zaidi & Foy, 1994). Additionally, childhood abuse has been associated with maladaptive personality functioning, including increased neuroticism (Glaser, van Os, Portegijs, & Myin-Germeys, 2006; Wilson et al., 2006) and personality disorders (Herman, Perry, & van der Kolk, 1989; Lee, 2006; Sansone, Pole, Dakroub, & Butler, 2006).

From a developmental perspective, the relation between early trauma and increased risk for adult psychopathology can be explained by the harmful effects of adverse early experiences on core developmental tasks including the formation of stable attachments, affect regulation, impulse control, development and integration of self-concepts, and socialization from childhood to adulthood (Cole & Putnam, 1992; Herman, 1992; Hildyard & Wolfe, 2002; Toth & Cicchetti, 1998). Several studies have supported this hypothesis, showing that early trauma affects defense styles, self-esteem, social functioning, memory, and aggression (Briere & Runtz, 1990; Finzi-Dottan & Karu, 2006; Liem & Boudewyn, 1999; Lynch & Cicchetti, 1998). Additionally, to explain the increased risk for psychopathology in survivors of childhood trauma, some researchers have posited that early trauma affects biological systems underlying stress regulation (De Bellis, 2001), and emerging evidence has indicated that exposure to stress in early life may result in long-term changes in neurobiology (e.g., Teicher et al., 2003).

A useful approach to study the psychobiological effects of early trauma on self-concepts and other personality variables is the model proposed by Cloninger (1988; Cloninger, Svrakic, & Przybeck, 1993). This model proposes a neurobiological basis for personality, or temperament, which, in conjunction with environmental variables, determines the development of additional character traits. Studies have shown that Cloninger's constellation of basic temperament and character dimensions can be related to social behavior and anxiety (Hofmann & Loh, 2006; Sigvardsson, Bohman, & Cloninger, 1987), defense styles (Kennedy, Schwab & Hyde, 2001), and mental disorders including mood and anxiety disorders (Matsudaira & Kitamura, 2006; Richter, Eisemann, & Richter, 2000), alcohol abuse (Meszaros et al., 1999), and personality disorders (Joyce et al., 2003). This model may therefore provide an adequate framework to integrate psychological and biological determinants of adult psychopathology following childhood trauma.

The aim of the present study was to investigate how early adverse experiences may relate to personality in a military sample. Since both personality and early trauma have been shown to constitute risk factors for the development of adult PTSD in soldiers (Bramsen, Dirkzwager, & van der Ploeg, 2000; Sutker, Davis, Uddo, & Ditta, 1995), investigation in this area is especially relevant to this population at risk. In Cloninger's model, the temperament dimensions represent automatic responses and emotional impulses that are thought to be heritable whereas the character dimensions reflect individual differences related to self-awareness, abstract deductions, and cognitive schemas that are developed through environmental influences and social learning experiences (Cloninger, 1999; Cloninger, Pryzbeck, Svrakic, & Wetzel, 1994). Because early traumatic experiences may be conceived as particularly toxic examples of early learning

experiences, we hypothesized that effects of early trauma would be observable only on the character scales and not the temperament scales of the Temperament and Character Inventory (TCI; Cloninger et al., 1994). To account for the potential effects of previous deployments on current personality, we examined this relationship controlling for prior deployments.

Additionally, to provide an estimate of the generalizability of the findings from the present study, we compared the TCI scores in the present sample to data from a normative civilian sample collected and reported by Duijsens, Spinhoven, Verschuur, and Eurelings-Bontekoe (1999). In this way, we also were able to explore possible idiosyncrasies of the present sample. Soldiers' personality scores may differ from those obtained in the general (male) population. For instance, one might expect soldiers to be less inclined to avoid potential harmful situations, and more prone to seek out new and challenging situations. Surprisingly, however, we were able to retrieve only a handful of recent studies examining personality in healthy, nonclinical, military samples. None of these used the TCI. Therefore, it is unclear how the test performs in military settings and whether soldiers and civilians actually differ.

Method

Participants

Participants for the study were drawn from a large prospective study into risk factors for the development of deployment-related disorders, such as PTSD (Prospective Research in Stress during Military Operations; Veenman, Vermetten, Kloet, Unck, & Westenberg, 2005) in the Dutch Armed Forces. Participants volunteered for this study prior to a 4-month U.N. deployment to Afghanistan. Written consent was obtained from all participants after a complete written and verbal description of the study.

The present study reports on data collected between March 2005 and August 2006. In total, data from 246 male soldiers were available for the current study. The data of 4 participants had to be omitted due to incomplete test scores, resulting in a sample of 242 soldiers with a mean age of 31.4 years (SD = 10.3). Demographic data are displayed in Table 1.

Materials

For this study, participants were asked to fill out a packet of "paper-and-pencil" questionnaires on demographic variables, prior deployments, current health status, and personality. Personality was assessed using the Dutch short-form version of the TCI (Cloninger et al., 1994). This short version of the TCI (TCI-SF; Duijsens et al., 1999; Duijsens & Spinhoven, 2002) consists of 105 "true" or "false" questions measuring four temperament scales: Harm Avoidance (HA), Reward Dependence (RD), Novelty Seeking (NS), and Persistence (P), and three character scales: Self-Directedness (SD), Cooperativeness (CO), and Self-Transcendence (ST). Cronbach's α of the Dutch TCI-SF range from .69 (RD) to .85 (HA) (Duijsens & Spinhoven, 2002).

Exposure to early trauma was assessed using the short form self-report version of the Early Trauma Inventory (ETISR-SF; Bremner, Bolus, & Mayer, 2007; Bremner, Vermetten, & Mazure, 2000). The ETISR-SF contains 27 "true" or "false" items designed to assess whether someone has been exposed to potential traumatic experiences before the age of 18 years. The inventory is divided into four scales measuring: General Trauma (11 items; e.g., exposure to natural disasters, deaths in the family, and exposure to violence), Physical Punishment (five items; e.g., being slapped in the face), Emotional Abuse (five items; e.g., often being put down or ridiculed), and Sexual Abuse (six items). Scores on each scale represent the number of items that were endorsed.

The ETISR-SF was translated specifically for the present study. The list was translated to Dutch and back-translated to English by an independent professional translator. The translation was then modified to ensure face validity, and this process was repeated until no disagreement existed between the original and the translation.

Table 1: Demographic variables of the present sample.

		Count (%)
Marital status	Married	83 (34.4)
	Cohabitating	31 (12.8)
	Other relationship	36 (14.9)
	Single	81 (33.5)
	Divorced	4 (1.7)
Rank	Private	84 (34.7)
	Corporal	46 (19.0)
	NCO	76 (31.4)
	Officer	34 (14.1)
Education*	Low	92 (38.0)
	Moderate	84 (34.7)
	High	62 (25.6)
Previously deployed	Yes	No
	95 (39.9)	143 (59.1)

*Note. * Education: Low = equivalent to some years of high school; Moderate = equivalent to finished high school; High = equivalent to some years of college or university education or more.*

Data analyses

First, the psychometric properties of the TCI-SF and the translated ETISR-SF were examined by computing Cronbach's α for the subscales. A canonical correlation analysis was then performed to explore the association between early traumatic experiences and personality, followed by multiple regression analyses to predict personality on the basis of early trauma ratings. To explore potential differences between the present sample and the general population, scores on the TCI-SF scales in the present sample were compared to data from a previously collected normative sample of 282 male civilians (Duijsens et al., 1999; Duijsens & Spinhoven, 2002) using t tests.

Results

Psychometrics

As shown in Table 2, internal consistency of the ETISR-SF proved adequate for the Physical Abuse (.76) and Emotional Abuse (.83) scales, but not for the General Trauma (.48) and Sexual Abuse (.53) scales. The internal consistency ratings for the physical and emotional abuse dimensions are comparable to those reported by Bremner et al. (2007) whereas Cronbach's α of the General Trauma and Sexual Abuse scales were considerably lower in the present sample.

Cronbach's α for the TCI-SF scales were .58 for NS, .79 for HA, .65 for RD, .71 for P, .76 for SD, .82 for CO, and .79 for ST. These coefficients were comparable to the values mentioned

in the TCI-SF manual (Duijsens & Spinhoven, 2002), except for the NS scale for which a value of .75 was reported in the manual versus .58 in the current sample. Table 2 displays the mean scores of the present sample on the ETISR-SF and TCI-SF as well as Cronbach's α of the scales. Data from Duijsens and Spinhoven (2002) and Bremner et al. (2007) are added for comparison.

Table 2: Mean scores and internal consistency ratings (Cronbach alpha) for the TCI-SF and the ETISR-SF in the present sample. TCI data from Duijsens and Spin-hoven (2002) and ETISR-SF data from Bremner et al. (2007) added for comparison.

	Present sample			Normative data		
	Mean	***SD***	**Alpha**	**Mean**	***SD***	**Alpha**
Early Trauma Inventory (ETISR-SF)						
General trauma	2.31	1.67	.48	1.6	1.8	.70
Physical abuse	1.71	1.64	.76	1.0	1.3	.75
Emotional abuse	0.48	1.14	.83	0.8	1.3	.86
Sexual abuse	0.12	0.43	.53	0.2	0.6	.87
Temperament and Character Inventory (TCI-SF)						
Novelty Seeking	7.89	2.72	.58	6.6	3.4	.75
Harm Avoidance	2.89	2.83	.79	4.9	3.8	.85
Reward Dependence	8.32	2.83	.65	8.3	3.1	.69
Persistence	9.97	2.83	.71	8.9	3.0	.71
Self-directedness	13.59	2.00	.76	12.5	2.8	.78
Cooperativeness	12.00	3.10	.82	12.2	2.9	.79
Self-transcendence	2.92	2.82	.79	3.8	3.3	.81

Relationship between early trauma and personality

First, a canonical correlation analysis was performed to explore the association between early trauma as measured by the ETISF-SR and personality as measured by the TCI-SF. Canonical correlation analysis can be used to describe the number and nature of mutually independent relationships that exist between two sets of variables. It creates uncorrelated pairs of linear combinations that result in the additive partitioning of variance (Stevens, 2002). A significant canonical correlation was found between the two sets, Wilks's $\lambda = .816$; $F_{(28, 809)} = 1.68$, $p = .016$, with only the first canonical correlation reaching significance (eigenvalue = .136). The canonical correlation was .35, explaining 12% in shared variance. The standardized canonical coefficients of the ETISR-SF scales were -.02, -.06, -.97, and -.04 for General Trauma, Physical Abuse, Emotional Abuse, and Sexual Abuse scales, respectively. The correlations between these scales and the canonical variable were -.39, -.36, -.99, and -.11, respectively. This shows that emotional abuse is most important in explaining the canonical function and that it is most strongly related to the canonical variable.

The standardized coefficients for the TCI-SF were -.07, .00, -.16, -.11, .44, .74, and -.01 for NS, HA, RD, P, SD, CO, and ST, respectively. The correlations between these scales and the canonical variable were -.22 (NS), -.18 (HA), .11 (RD), .04 (P), .77 (SD), .90 (CO), and -.27 (ST),

respectively. This shows that the SD and CO scales of the TCI-SF are the most important for interpreting the canonical function and the most strongly related to the canonical variable. Therefore, self-reported early emotional trauma can be linked to the character scales SD and CO. Figure 1 displays the correlations between all variables and canonical variates.

Next, two multiple regression analyses were performed to predict scores on the SD and CO scales, respectively, from ETI scores, after controlling for the effects of previous deployments. As can be seen in Tables 3 and 4, both previous deployments and emotional abuse in childhood were significantly associated with personality. After controlling for the effects of prior deployment, emotional abuse explained 9% of variance in the SD and 11% of variance in the CO scores.

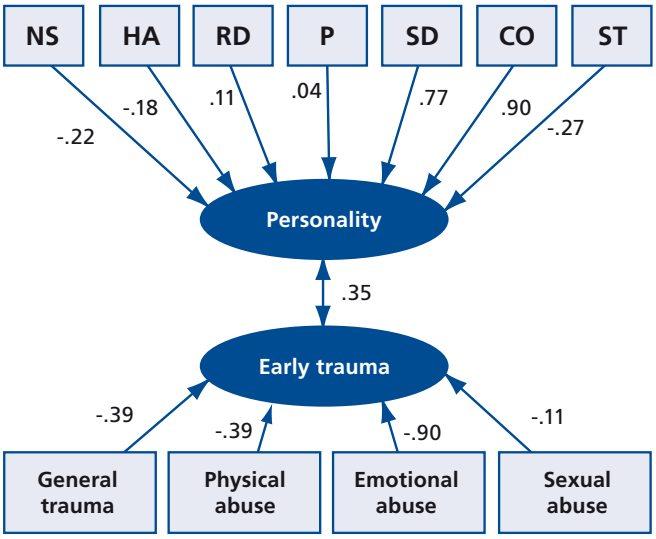

Figure 1. *Schematic representation of the first function in the canonical correlation analysis. Arrows represent correlations of respectively personality domains Novelty seeking (NS), Harm avoidance (HA), Reward dependence (RD), Persistence (P), Self directedness, Cooperativeness (CO), and Self transcendence (ST), and self-reported exposure to early trauma, to each canonical variate.*

Military personality idiosyncrasies

The scores in the present sample were compared to data from a normative male sample of 282 male civilians collected and reported by Duijsens et al. (1999). These data were collected as part of a representative sample from the Dutch population and provided to us for the present study. Mean age in the total normative sample was 43.7 (*SD* = 15.7). More detailed information is described elsewhere (Duijsens et al., 1999; Duijsens, Spinhoven, Goekoop, Spermon, & Eurelings-Bontekoe, 2000). Comparing TCI-SF scores of the male soldiers in the present sample to the normative civilian sample yielded significant differences between groups on several scales (Bonferroni corrected $\alpha = .007$). Soldiers scored significantly higher on NS, t (518) = -4.74, $p < .001$; P, t (522) = -4.45, $p < .001$; and SD, t (507) = -5.48, $p < .001$, and lower on HA, t (509) = 7.22, $p < .001$; and ST, t (519) = 3.33, $p < .001$. Mean scores of the present sample as well as the reference group are displayed in Table 2.

Table 3: Multiple regression of early traumatic experiences (ETISR-SF) on cooperativeness (TCI-SF) after controlling for prior deployments.

	Variable	B	S.E.	β
1	(Constant)	11.71	.25	
	Prior deployments	1.10	.39	.18**
2	(Constant)	12.38	.37	
	Prior deployments	1.06	.38	.17**
	General trauma	-.04	.12	-.02
	Physical abuse	-.09	.12	-.05
	Emotional abuse	-.78	.17	-.30***
	Sexual abuse	-.00	.44	.00

Note. $R^2 = .14$; *Adjusted* $R^2 = .12$; $\Delta R^2 = .03$ *for step 1;* $\Delta R^2 = .11$ *for step 2;* * = $p < .05$; ** = $p < .01$; *** = $p < .001$.

Table 4: Multiple regression of early traumatic experiences (ETISR-SF) on self-directedness (TCI-SF) after controlling for prior deployments.

	Variable	B	S.E.	β
1	(Constant)	12.60	.21	
	Prior deployments	.95	.33	.19**
2	(Constant)	12.63	.32	
	Prior deployments	.96	.32	.19**
	General trauma	.09	.10	.06
	Physical abuse	.08	.11	.05
	Emotional abuse	-.70	.15	-.31***
	Sexual abuse	-.28	.38	-.05

Note. $R^2 = .12$; *Adjusted* $R^2 = .10$; $\Delta R^2 = .04$ *for step 1;* $\Delta R^2 = .09$ *for step 2;* * = $p < .05$; ** = $p < .01$; *** = $p < .001$.

Discussion

The present study showed that adverse childhood experiences were negatively associated with certain aspects of personality. Self-reported exposure to emotional abuse before 18 years of age was related to the character dimensions of both self-directedness and cooperativeness. This was consistent with our hypothesis, as we expected the character scales of the TCI to be more sensitive to the potential effects of early trauma than would be the temperament scales. The fact that this relationship appeared in a healthy, nonclinical sample underscores the potential detrimental effects of trauma on personality development and illustrates how early trauma may establish vulnerability to adult psychopathology.

Low scores on the SD and CO scales may reflect difficulties in accepting responsibility, lack of long-term goals, low self-esteem, poor impulse control, inadequate social functioning, identity problems, interpersonal hostility, intolerance, egocentrism, and opportunism (Cloninger, 1999). Low cooperativeness and self-directedness appear to be related to more use of

avoidant coping and less use of social coping strategies (Ball, Smolin, & Shekhar, 2002; Duijsens & Spinhoven, 2002). Moreover, low scores on both the CO and the SD scales have been reported in psychiatric outpatients (Duijsens et al., 2000). They have been associated with recurrent depressive episodes (Richter et al., 2000) as well as with increased risk for depression and anxiety (Matsudaira & Kitamura, 2006), and appear to be common in personality disorders (Svrakic et al., 2002; Svrakic, Whitehead, Przybeck, & Cloninger, 1993).

These characteristics correspond to adverse effects of early trauma on core developmental tasks as outlined by Herman (1992) and Cole and Putnam (1992), who described the potential detrimental effects of early trauma on identity development and socialization. Extensive research in the field of child development has shown a relationship between emotional trauma and interpersonal difficulty as well as problems with affect regulation, impulse control, and self-concepts (Finzi-Dottan & Karu, 2006; Kim & Cicchetti, 2006; van der Kolk & Fisler, 1994). Integrating the present results with evidence from this field therefore illustrates how early trauma may become ingrained in personality.

Some limitations to the current study need to be taken into account when interpreting the results. First, the design of the study limits the inferences that can be made from the data obtained. Although a significant association was established, we cannot be certain that there is any causal relation between early trauma and personality on the basis of the present study alone. It may be possible that the relationship is better explained by a third (i.e., unmeasured) variable. For example, we did not control for the possibility that military personnel who had been previously deployed suffered from PTSD symptoms or other complaints as a result. Both prior deployments and the presence of pathology may have affected personality ratings. Although the relationship between early trauma and personality remained significant after controlling for prior deployments, we cannot exclude the possibility that the amount of explained variance in personality ratings was influenced by the presence of PTSD symptoms. This limits the reliability of the proportion of explained variance that was attributed to early trauma. To reduce the possible confounding effects of prior deployments and subsequent PTSD symptoms, we repeated the analyses after excluding soldiers who had already been deployed (Data not shown.) This did not substantially alter the results. Therefore, we expect effects of deployment-related psychopathology on the present results to have been modest at most.

Second, because we relied on self-report data exclusively, the present results may have been biased. The accuracy of recall of adverse experiences has been subject to debate, and several studies have raised questions about the stability and reliability of self-reported trauma-exposure ratings (for a review, see Loftus & Davis, 2006). Some studies have shown that the reliability of recall of trauma exposure can be seriously flawed (e.g., Southwick, Morgan, Nicolaou, & Charney, 1997). Mood or emotional state may be one potential source of bias in self-reported childhood experiences (Brewin, Andrews, & Gotlib, 1993).

On the other hand, the fact that the soldiers were assessed prior to a deployment may have led to a positive response bias in which the participants were inclined to paint a more favorable picture. It is possible that in an attempt to minimize feelings of insecurity or apprehension about the mission, the soldiers tended toward denial of items that were associated with emotional distress and unfavorable characteristics whereas items that reflected more (socially) desirable aspects of functioning were more frequently endorsed. The potential influence of response bias, which is inherent in the use of self-report measures, cannot be excluded.

Third, we did not measure the severity of the traumatic experiences or the subjective distress. Contrary to many other measures of childhood trauma, the original interview (ETI) and the complete self-rating version (ETISR) also included assessment of impact of traumatic experiences on the individual. Different scoring schemes, including weighted scoring indi-

ces, were developed (Bremner et al., 2000), but these did not provide additional information above the more parsimonious and easy method of adding the number of events that occurred (Bremner et al., 2007). Nonetheless, the severity of trauma as well as the duration can be expected to be of importance on various aspects of functioning. Several studies have reported a dose–response relationship between trauma exposure and the prevalence of mental disorders including PTSD (e.g., Dohrenwend et al., 2007; for a review, see Murthy, 2007). Including a trauma intensity rating is therefore recommended for future studies.

The question remains whether the present findings can be generalized to the general (male) population. The soldiers scored differently from civilians on various scales of the TCI-SF. Army personnel scored higher on novelty seeking, persistence, and self-directedness, and scored lower on harm avoidance and self-transcendence. This may indicate that on the whole, they were more inclined to seek out new situations and less inclined to avoid aversive circumstances, and that they were more confident, more perseverant, goal oriented, and controlling (Cloninger et al., 1993). Taken together, the lower score on the HA scale and the higher score on the NS scale may represent the tendency to seek out novel and challenging situations and the willingness to take risks for the sake of such activities. This was originally described by Zuckerman (1994) as sensation seeking. This may have been part of the motivation for joining the army in the first place, but it also may increase the risk of exposure to traumatic experiences. Research has shown that novelty seeking is indeed related to vocational interest, but not necessarily to engaging in risk behavior (e.g., Mallet & Vignoli, 2007). Moreover, research has shown that high harm avoidance and low novelty seeking, not vice versa, increased the risk of PTSD after trauma exposure (Gil, 2005). Therefore, the personality scores of the soldiers in the present sample may reflect increased resiliency to stress.

Even though the differences between the present sample and a civilian sample were statistically significant, the soldiers scored within the "average" range on all scales except HA according to the Dutch manual (Duijsens & Spinhoven, 2002). The HA score was in the "below average" range for males. Additional research is needed to verify whether the scores in the present sample are representative for the male military population and to construct new military norm scores if needed. We did not find any significant association between early sexual or physical trauma and personality, nor was any relation found with general trauma. This is somewhat surprising as we would expect sexual and physical abuse especially to be at least equally detrimental to development. With respect to sexual trauma, a possible explanation for the absence of an association with personality can be found in the fact that items pertaining to sexual abuse were rarely endorsed in the present sample. The participants possibly were less inclined to disclose information of a sexual nature. By contrast, the occurrence of sexual abuse may have actually been low in this all-male sample. Compared to the scores reported by Bremner et al. (2007), the soldiers' mean score on the SA scale was somewhat lower; however, part of the sample described by Bremner et al. was included because they were victims of childhood abuse. Therefore, it is plausible that the low(er) score on the SA subscale actually reflects a lower incidence of this type of abuse in the present study. By contrast, general trauma and physical abuse were more common in the present sample. The lack of association may therefore indicate that these trauma types are not specifically linked to personality dimensions in Cloninger's model. Additional research is needed to elaborate on this issue.

The present study showed that exposure to emotional trauma in early life was associated with lower self-directedness and cooperativeness. Although the notion that (early) experiences shape personality is central to most personality theories, the present study adds to the understanding of which environmental influences are related to specific personality traits or characteristics. To our knowledge, this is one of the first studies providing empirical evidence to link

early trauma to Cloninger's personality model. More importantly, the present study illustrates a potential pathway through which early trauma may increase the vulnerability to adult psychopathology. Early emotional trauma may increase the risk of adult psychopathology through its effects on personality traits associated with self-esteem, impulse control, empathy, and interpersonal behavior. The finding of a significant relationship between early trauma and maladaptive personality features in a healthy, nonclinical sample underscores the importance of including early environmental influences in etiological models of psychopathology.

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Chapter 4

Personality and the cortisol response to awakening

Personality dimensions harm avoidance and self-directedness predict the cortisol awakening response in military men.
Arthur R. Rademaker, Rolf J. Kleber, Elbert Geuze, & Eric Vermetten
Biological Psychology, 81, 177-183 (2009).

Introduction

Dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis has been associated with mental disorders like posttraumatic stress disorder (PTSD) and depression (Charlton & Ferrier, 1989; de Kloet et al., 2006; Yehuda et al., 1996), and may compose a vulnerability factor for these disorders (Holsboer, 2000). Psychological factors can have a pronounced effect on HPA activity (Mason, 1968), but the relationship between personality and HPA activity in healthy individuals remains unclear (Van Eck et al., 1996). Personality traits like harm avoidance and neuroticism have been demonstrated to increase vulnerability for stress-related psychopathology (Battaglia et al., 1996; Bienvenu & Stein, 2003; Clark et al., 1994; Khan et al., 2005). It is possible that these personality traits reflect increased sensitivity for stressors because they are related to HPA-axis functioning (Tyrka et al., 2006).

To examine the association between HPA activity and personality, studies investigated the relationship between personality and cortisol using different research methodologies. Cortisol is a glucocorticoid hormone secreted by the adrenal cortex that can serve as a marker for HPA-axis functioning. In humans, cortisol levels usually, though not always (Smyth et al., 1997; Stone et al., 2001), show a distinct circadian rhythm with levels rapidly rising early in the morning and then steadily decreasing during the day (Weitzman et al., 1971). Laboratory studies have found the cortisol response to be sensitive to acute (psychosocial) stressors and uncontrollable threats to the self (Dickerson & Kemeny, 2004). There is evidence that the cortisol response to stressors is related to personality traits such as self-esteem and locus of control (Kirschbaum et al., 1995; Pruessner et al., 1999b; Seeman et al., 1995), as well as trait anxiety, neuroticism and / or extraversion (Jezova et al., 2004; Oswald et al., 2006; Phillips et al., 2005), although conflicting results have also been reported (Kirschbaum et al., 1995; Schommer et al., 1999; Van Eck et al., 1996).

So far, relatively few studies examined the relationship between personality and unstimulated, baseline cortisol concentrations in healthy adults. Studies that have been performed are plagued by ambiguous findings. For instance, Polk et al. (2005) linked high negative affectivity (aggregated from daily measures and thought to reflect the tendency to experience negative emotions and emotional distress) to higher total daytime salivary cortisol in adult men. Low positive affectivity was related to a high but flat daytime cortisol profile in men, and high positive affect was related to a low and flat diurnal cortisol profile in women. By contrast, Schommer et al. (1999) found no evidence of an association between diurnal cortisol and neuroticism or extraversion. Similarly, Vedhara et al. (2006) failed to find a significant association between trait anxiety and diurnal cortisol in women.

Disparity between study results may be caused by factors such as small sample size, state effects, gender differences, and differences in assessment procedures. Because the HPA-axis is characterized by high inter- and intra-individual variability, development of reliable and easily assessable markers has composed a challenge in research (Wilhelm et al., 2007). Recently, attention has shifted towards measuring the free fraction of cortisol in saliva after awakening, or cortisol awakening response (CAR), as an indicator of adrenocortical responsiveness and HPA activity. In most individuals, cortisol levels peak within the first hour after awakening. This cortisol awakening response is a sensitive, robust and reliable index of HPA and adrenocortical activity (Edwards et al., 2001a; Pruessner et al., 1997; Wüst et al., 2000b). Due to its high intra-individual stability and because the CAR appears to be linked to genetic influences, the CAR can be perceived as a trait measure of HPA (re)activity (Wüst et al., 2000a).

Early emerging evidence has linked the CAR to personality dimensions. Portella et al. (2005) reported that individuals high in neuroticism showed significantly greater levels of salivary cortisol after awakening than individuals low in neuroticism. Similarly, Polk et al. (2005) reported

that negative affectivity (NA) - a trait related to neuroticism - was positively related to the CAR in healthy men. By contrast, female subjects in this study displayed an inverse relationship between NA and CAR. Additionally, the CAR may be related to self- esteem and locus of control (Pruessner et al., 1999a). Although the CAR may be a useful marker to investigate the relationship between personality and HPA-axis functioning, additional research is needed.

The aim of this study was to investigate the relationship between the CAR and personality as measured by Cloninger's Temperament and Character Inventory (TCI; Cloninger et al., 1994). This psychobiological personality model describes four temperament dimensions: novelty seeking, harm avoidance, reward dependence, and persistence. These are thought to represent underlying biological systems associated with behavior activation, behavior inhibition, and behavioral dependence respectively. Additionally, it comprises of three character dimensions which are proposed to develop through environmental influences and social learning experiences: self-directedness, cooperativeness and self-transcendence, reflecting individual differences in self-awareness, abstract deductions, and cognitive schemas.

The present study focused exclusively on soldiers. Since soldiers are frequently exposed to stressful and potentially traumatic circumstances, they are at increased risk for developing stress related psychopathology. Examining the mechanisms involved in the etiology of stress-related disorders is of vital importance to them, as it may help to pinpoint risk and resilience factors on the one hand and because it may facilitate the development of adequate treatment programs on the other hand. Previous studies in military samples have shown that military training can affect morning (Clow et al., 2006), as well as evening cortisol levels (Hellhammer et al., 1997); that awakening cortisol levels are sensitive to stress of military training (Clow et al., 2006), and that the cortisol response to stress is related to social status in army recruits (Hellhammer et al., 1997). We found no studies that examined the association between the CAR and personality in military samples. However, and as noted before, certain personality traits may enhance the vulnerability for stress-related disorders. The purpose of the study was to examine a potential pathway to account for the association between personality and stress-related disorders (i.e., dysregulation of the HPA-axis) in a population at risk.

Temperament trait harm avoidance in particular has been shown to compose a vulnerability factor for mental disorders associated with HPA-dysregulation. Therefore, the main focus of the present study was to examine the relationship between harm avoidance and the CAR. Harm avoidance is related to trait neuroticism (De Fruyt et al., 2000), and it reflects the tendency to avoid new situations and aversive stimuli (Cloninger et al., 1993). Like neuroticism, harm avoidance has been (prospectively) linked to the development of mood and anxiety disorders (Battaglia et al., 1996; Cloninger et al., 2006; Gil, 2005). Also, there is evidence that harm avoidance may reflect a genetic predisposition for certain mental disorders (Ono et al., 2002). We expected to find higher morning cortisol levels in subjects higher in harm avoidance. Additionally, we explored the possible association of the CAR with other scales of the TCI.

Methods

Subjects

Participants for this study were selected from a large prospective study on the development of stress- and trauma-related psychopathological symptoms following military deployment in the Dutch armed forces. The sample consisted of 107 healthy male soldiers between 19 and 57 years old. The mean age was 32.6 years (*SD* = 10.9). The study was approved by the Institutional Review Board of the University Medical Centre in Utrecht, the Netherlands. Written informed consent was obtained from all soldiers who participated in the study after a

complete written and verbal description of the study. All participants passed standard army medical examinations prior to deployment, and were perceived healthy and mentally fit by their military physicians. None of the respondents used psychotropic medication or steroids. Demographics are displayed in Table 1.

Table 1: Sample characteristics

	M	*SD*
Age	32.60	10.92
BMI	25.32	3.08
	Count	**%**
Smoker	47	54.4
Previously deployed	48	45.7
Rank*		
I	43	39.8
II	48	44.4
III	17	15.7

*Note. * I = enlisted and corporal; II = non-commissioned officers; III = officers and staff-officers; BMI = Body mass index.*

Psychological measures

All participants were asked to fill out a series of questionnaires that included questions on demographic variables and prior deployments. Participants also completed the Dutch short-form Temperament and Character Inventory (Duijsens et al., 1999). The Dutch short-form TCI has been shown to adequately measure Cloninger's seven personality dimensions (Duijsens et al., 1999). The list consists of 105 'true' or 'false' items measuring seven personality scales (15 items per scale) which include: four temperament dimensions: harm avoidance (HA), reward dependence (RD), novelty seeking (NS) and persistence (P), and three character scales: self-directedness (SD), cooperativeness (CO) and self-transcendence (ST). Cronbach alpha's of the Dutch TCI short-form range from .69 for reward dependence to .85 for harm avoidance (Duijsens & Spinhoven, 2002), and comparable rates were recently observed in a military sample for all scales except novelty seeking (Rademaker et al., 2008). Mean scores on the TCI-SF are presented in Table 2.

Cortisol assessment

Salivary cortisol sampling is a non-invasive and reliable assessment procedure to estimate HPA-axis functioning (Aardal-Eriksson et al., 1998). Participants were instructed to collect salivary cortisol samples by means of salivettes (Sarstedt Inc., Newton, NC, USA). Each participant received a set of salivettes along with verbal and written instructions on the sampling procedure. Subjects were instructed to collect saliva samples on a work day, directly after awakening, i.e., 'the moment you can open your eyes', and then again 15, 30 and 60 min after awakening. Participants were also instructed to abstain from food or drink intake, smoking, and brushing their teeth within the first hour. Samples were collected with reference to time of waking rather than at fixed clock-time as this procedure has been shown to provide a more robust and reliable cortisol measurement (Pruessner et al., 1997).

After collection, samples were sent to the research centre by mail. Salivettes were then shipped to the Biological Psychology Laboratory at the Technical University of Dresden, Germany, for analysis. Although the shipping procedure may have exposed saliva samples to varying conditions, i.e., temperature changes and movement, previous studies suggest that these conditions did not impose a limitation on the reliability of cortisol assessments in the present study (Clements & Parker, 1998; Garde & Hansen, 2005). Salivary free fraction of cortisol was measured using a time-resolved immunoassay with fluorescence detection as described in detail by Dressendorfer et al. (1992). Intra- and inter-assay coefficients of variability were below 6.0 and 9.0%, respectively.

Statistical analyses

Data were analyzed using SPSS version 15.0 for Windows. Results were considered significant when p values were smaller than .05. First, data were checked for violations of multivariate normality. Box-Cox transformations were computed using Minitab Statistical Software when appropriate. Total cortisol levels after awakening were computed as previously recommended (AUCg; Pruessner et al., 2003). Mean cortisol increase (MnInc) was computed with the following formula: MnInc = (CORT15 + CORT30 + CORT60) / 3 - CORT0 (Wüst et al., 2000a). Previous research has demonstrated that the MnInc is virtually identical to the AUC with reference to the first awakening sample (Edwards et al., 2003) or AUCi (Pruessner et al., 2003).

Hierarchical regression analyses were performed with potential confounders added in the first block using forward selection (p to enter = .05; p to remove = .10) and scores on all seven TCI-SF scales forced into entry in the second block, to predict (a) awakening cortisol levels, and (b) mean cortisol increase. Since a number of participants in the present study had previously been deployed, and because previous studies have shown that deployment status can affect cortisol levels (de Kloet et al., 2007), deployment status was also entered in the first block. Next, to illustrate the association between CAR and personality factors that significantly added to the regression analyses results, the total sample was divided into subgroups by a median split on relevant personality scales. Subgroups were compared using chi-squared analyses or student's t-test. Cortisol concentration over time was analyzed using repeated measures ANOVA with group (dichotomized personality trait) as between-subject variable, and time (4 time points) as within subject variable, controlling for potential confounders.

Results

Data manipulation

Cortisol data were sufficiently normally distributed but scores on several scales of the TCI-SF were highly skewed. To control for violations of multivariate assumptions, Box-Cox transformations were applied. Box-Cox transformation estimates the lambda value that minimizes the standard deviation of a standardized transformed variable. The transformed variable $Y' = Y^{\lambda}$ when $\lambda \neq 0$, and Log (Y) when $\lambda = 0$. Table 2 displays the computed lambda values and corresponding transformations.

Table 2: Mean scores on the short-form Temperament and Character Inventory (TCI-SF) and Box-Cox estimated lambda values and transformations.

TCI-SF Scale	Mean	SD	λ	Trans.
Novelty Seeking	7.36	10.92	1.0	N.A.
Harm Avoidance	2.83	2.72	0	Ln Y
Reward Dependence	8.39	2.82	1.0	N.A.
Persistence	10.15	2.86	2.0	Y^2
Self-directedness	13.68	2.39	-1.0	1/ Y
Cooperativeness	12.56	2.93	0	Ln Y
Self-transcendence	3.04	2.84	0	Ln Y

Note. ln = natural logarithm; N.A. = not applicable; Trans. = transformation; Y = original variable.

Awakening cortisol response

Mean (*SD*) cortisol concentrations were observed of 16.29 nmol/l (8.61) directly after awakening (CORT0), 20.77 nmol/l (10.67) after 15 min (CORT15), 22.92 nmol/l (11.81) after 30 min (CORT30), and 19.17 nmol/l (11.76) 60 min after awakening (CORT60). To explore the association between morning cortisol levels and personality, scores on all TCI-SF scales were regressed onto total salivary cortisol concentrations after awakening (AUCg), computed over 4 time points. Age, BMI, military rank, smoking-status, and prior deployment status were entered in the first block using forward selection, and TCI-SF scores in the second block, using forced entry. As shown in Table 3, only harm avoidance significantly predicted cortisol levels after awakening, accounting for 9% in variance, $F_{(7,92)} = 2.46$, $p = .024$. None of the potential confounders were significantly related to AUCg. Next, all TCI-SF scales and potential confounders were regressed onto mean cortisol increase[1]. As can be seen in Table 4, harm avoidance and self-directedness accounted for 10% explained variance in the mean increase in free salivary cortisol, $F_{(7,92)} = 2.56$, $p = .019$. Again, none of the potential confounders significantly added to the proportion of explained variance.

Table 3: Multiple regression of personality (TCI-SF) on baseline cortisol levels after awakening (AUCg).

Variable	B	S.E.	β
Novelty seeking	10.18	21.54	.05
Harm avoidance†	223.81	85.67	.29*
Reward dependence	-10.86	21.78	-.05
Persistence†	-1.35	1.16	-.13
Self-directedness†	192.01	186.74	.11
Cooperativeness†	-167.07	88.50	-.22
Self-transcendence†	43.53	84.89	.05

*Note. † = Box-Cox transformed data; $R^2 = .16$; Adjusted $R^2 = .09$; * = $p < .05$.*

[1] *The MnInc correlated almost perfectly ($r = .998$, $p < .001$) with the area under the curve with respect to increase (AUCi; Pruessner et al., 2003) in the present study.*

Table 4: Multiple regression of personality (TCI-SF) on mean cortisol increase within 60 minutes after awakening

Variable	B	S.E.	β
Novelty seeking	.26	.32	.08
Harm avoidance†	3.06	1.28	.27*
Reward dependence	.50	.32	.17
Persistence†	.01	.02	.03
Self-directedness†	9.09	2.79	.36**
Cooperativeness†	.91	1.32	.08
Self-transcendence†	.65	1.27	.05

*Note. † = Box-Cox transformed data; R^2 = .16; Adjusted R^2 = .10; * = p <.05; ** = p < .01.*

A median split on harm avoidance scores (Mdn = 2.00) dichotomized the sample into two subgroups: a 'low' HA (M = .87, SD = .81), and 'moderate' HA (M = 5.29, SD = 2.20) subgroup. The subgroups did not differ in age or any other demographic variable, only the military rank distribution differed between subgroups, with more participants with higher ranks in the low HA subgroup ($\chi^2 = 5.88$, $p = .05$). ANOVA for repeated measures with HA as between-subjects variable ('moderate' vs. 'low'), controlling for military rank, showed significant group differences in cortisol concentrations, $F = 9.16$, $p = .003$, as well as a main effect of time, $F_{(3,98)} = 3.59$, $p = .016$, and a significant time by group effect, $F_{(3,98)} = 4.16$, $p = .008$. No significant time by rank interaction was present, $F_{(3,98)} = 1.01$, $p = .391$. Figure 1 displays the mean cortisol levels after awakening in both subgroups.

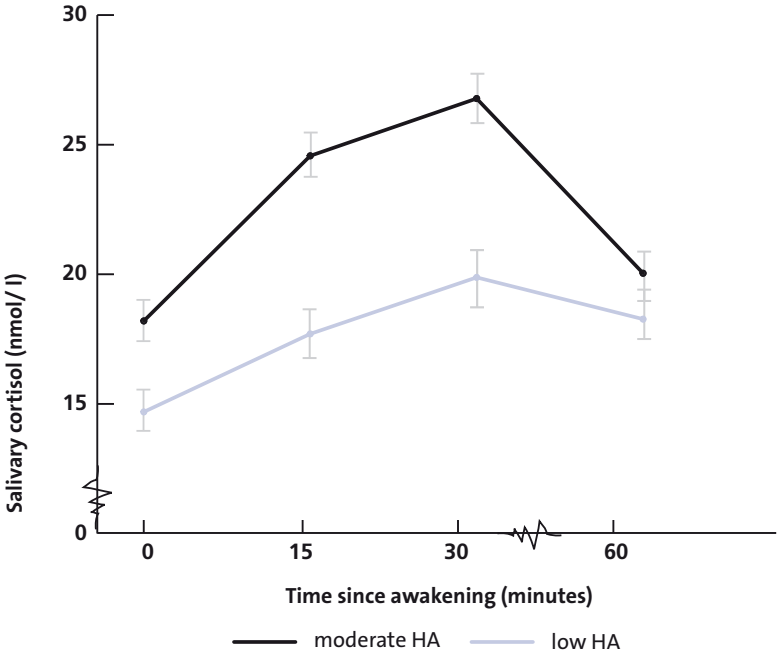

Figure 1. *Mean salivary cortisol levels after awakening of soldiers in 'moderate' and 'low' harm avoidance (HA) subgroups. Vertical bars represent standard error.*

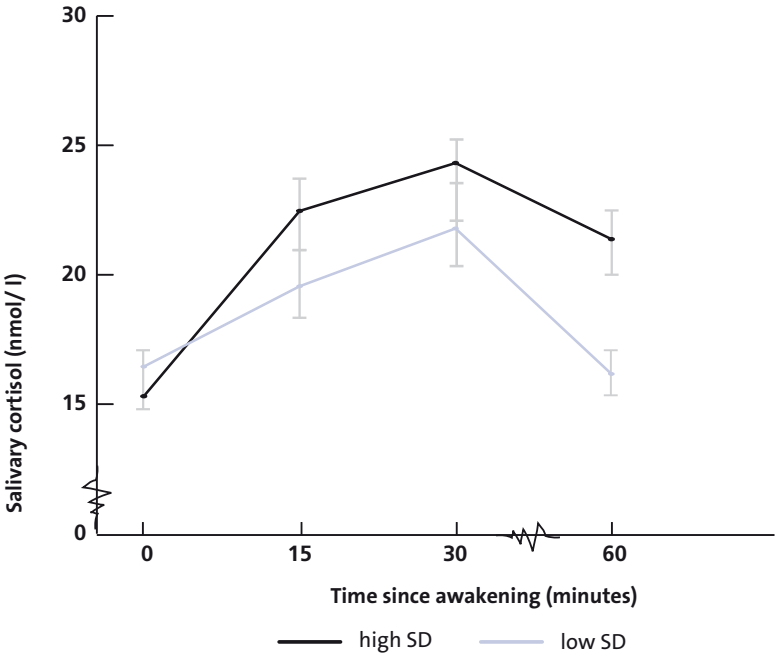

Figure 2. *Mean salivary cortisol levels after awakening of soldiers in 'low' and 'high' self-directedness subgroups. Vertical bars represent standard error.*

Next subjects were divided into two groups using a median split on scores on TCI-SF scale self-directedness (Mdn = 15). The high SD subgroup was older ($t = 3.71$, $p < .001$), contained more high ranking soldiers ($\chi^2 = 10.87$, $p = .004$) and fewer smokers ($\chi^2 = 6.39$, $p = .011$). ANOVA for repeated measures with SD ('high' vs. 'low') as between-subjects variable, controlling for age, rank, and smoking-status, showed a significant main effect of time, $F_{(3,91)} = 3.26$, $p = .025$, and a significant time by group interaction, $F_{(3,91)} = 5.92$, $p = .001$, as well as a significant time by smoking-status interaction, $F_{(3, 91)} = 4.26$, $p = .007$. Figure 2 displays the cortisol levels after awakening in both groups.

Discussion

This study showed an association between harm avoidance and overall cortisol levels after awakening, and between the mean cortisol increase after awakening and personality dimensions harm avoidance and self-directedness. No relationship was found between the CAR and previous deployments. To our knowledge, this was the first study to show a relationship between personality as measured with the TCI and the CAR. In line with previous findings (Edwards et al., 2001b; Pruessner et al., 1997), mean cortisol levels peaked 30 min after awakening. Harm avoidance predicted 10% of variance in cortisol levels after awakening, computed as the area under the cortisol curve with reference to zero (AUCg). A median split on harm avoidance scores illustrated that individuals low on harm avoidance displayed a flattened awakening cortisol curve. These findings are similar to the results by Portella et al. (2005), who reported an attenuated CAR in subjects low on neuroticism, albeit that in the Portella et al. study scores in the extreme range were dichotomized (high vs. low). In the present sample, the low harm avoidance subgroup had a mean score that was in the below average range as compared to scores in the general population, whereas the 'moderate' harm avoidance sub-

group scored within the average range (Duijsens et al., 1999).

Edwards et al. (2001a) stressed the importance of examining both the static and the dynamic aspect of the CAR. They argued that the dynamic aspect of the CAR, the relative cortisol increase, might be under a different regulatory mechanism than the static, baseline awakening cortisol levels. This study demonstrated a relationship between personality and the mean cortisol increase as well as overall cortisol levels after awakening. High harm avoidance and high self-directedness were related to a greater mean cortisol increase after awakening, together accounting for 10% in variance. By contrast, overall cortisol levels after awakening were related to harm avoidance only. As overall cortisol levels after awakening are related to day time cortisol (Edwards et al., 2001a), it is possible that harm avoidance is also related to diurnal cortisol levels, although we did not test this hypothesis.

Harm avoidance reflects a personality dimension associated with inhibition of behavior. It is positively correlated to trait anxiety (Jiang et al., 2003) and neuroticism (De Fruyt et al., 2000). High harm avoidance constitutes a risk factor for depression and PTSD (Cloninger et al., 2006; Gil, 2005), whereas low harm avoidance has been linked to increased resilience in healthy adults (Simeon et al., 2007). Self-directedness taps on personality aspects related to identity, responsibility and achievement motivation and high scores on self- directedness can be seen as indicative of mature and well-integrated personality functioning (Cloninger et al., 1993). Self-directedness can be conceptually linked to (internal) locus of control and is negatively correlated to trait anxiety and neuroticism (De Fruyt et al., 2000; Jiang et al., 2003). Similarly, this trait showed a moderate inverse correlation (r = -.32, p = .001) with harm avoidance in the present study.

So far, only few studies have examined personality aspects in cortisol increase after awakening. Whitehead et al. (2007) reported a positive association between the CAR and type-D personality in acute coronary syndrome patients and found that that type-D personality accounted for approximately 8% of the variance in the cortisol increase. Polk et al. (2005) explored the relationship between cortisol increase after awakening and trait positive and negative affectivity. They found that low trait negative affectivity was related to reduced morning cortisol increase in men; trait positive affectivity was not related to awakening cortisol increase. Although contradicting results have also been reported (Quirin et al., 2008), these results suggest that individuals with an anxious disposition (i.e., neurotic and / or high harm avoidant) will display an increased cortisol awakening response and high cortisol levels after awakening, whereas people low on these traits can be expected to show flattened awakening cortisol curves.

Parallel to that, the present study showed that high self-directedness was related to increased adrenocortical activity after awakening. As high self-directedness is generally associated with positive outcomes and successful adaption, this finding contrasts the association between increased CAR and harm avoidance and / or neuroticism. It shows that the CAR can be affected by both adaptive and maladaptive personality traits. As noted by Schulz et al. (1998), cortisol availability has an adaptive function as it facilitates increased vigilance and provides energy to meet upcoming (external) demands. Therefore, it is possible that the cortisol increase after awakening is affected by anticipation of upcoming problems and challenges (Schlotz et al., 2004). Individuals with a 'prospective orientation' (Schönpflug, 1985, as cited in Schulz et al., 1998), who expect a particular demanding day, would then exhibit a greater cortisol increase after awakening. This may be true not only for people experiencing work-related stress, but also for individuals who are highly engaged in their (occupational) activities (Langelaan et al., 2006). Therefore, the association between high self-directedness and mean cortisol increase may reflect increased cortisol reactivity related to achievement related prospective orientation. Individuals who are characterized by personality traits associated with enhanced vigi-

lance and / or anxiety (i.e., neuroticism, harm avoidance) as well as those with a dispositional prospective orientation (i.e., high self-directedness) could then be expected to display a greater cortisol increase after awakening.

Although the CAR can be linked to psychological variables and personality, the association between the CAR and (mental) health is unclear. Both increased cortisol and a decline in cortisol output after awakening have been implicated in illness processes. Available studies have failed to univocally link good health and well being to an increased or reduced cortisol awakening response (Clow et al., 2004). For instance, studies have reported an increased (Schulz et al., 1998), or attenuated CAR in burn-out patients, or no significant association at all (Österberg et al., 2009). Some studies report an association between increased CAR and depressive symptomatology (e.g., Pruessner et al., 2003), whereas lower awakening cortisol levels have been reported in PTSD patients (Neylan et al., 2005; Rohleder et al., 2004; Wessa et al., 2006).

Whereas acute stress is generally related to increased cortisol secretion, a large body of research is available, which shows that chronic stress is associated with blunted HPA activity and reduced cortisol output (Heim et al., 2000). Similarly, the CAR appears to be related to stress, but contradicting results have been published. Both an increased (e.g., Schulz et al., 1998) and a reduced (e.g., Pruessner et al., 1999a) CAR have been reported in (chronically) stressed individuals. A recent meta-analysis showed that exposure to chronic stress was associated to morning cortisol concentrations across different studies, but that the strength of the association diminished as more time elapsed since the onset of the stressor, suggesting that, over time, cortisol output might rebound to below baseline levels (Miller et al., 2007). The nature of the stressor was shown to be an important determinant of the direction and strength of the association with morning cortisol. Traumatic stressors and loss, as well as stressors that were uncontrollable or stressors that involved threat to the physical self, were associated with lower morning cortisol. By contrast, stressors that were (potentially) controllable or that reflected a threat to the social self, were associated with higher morning cortisol (Miller et al., 2007). Consequently, the positive association between self-directedness and CAR might reflect the tendency of individuals high on self-directedness

to perceive more stressors as controllable. Longitudinal studies are needed to determine whether a reduced or increased CAR composes a risk or vulnerability factor for the development of stress-related disorders.

Reduced awaking cortisol levels in PTSD patients are thought to be the results of an enhanced negative feedback of the HPA-axis, i.e., enhanced cortisol suppression, typical in PTSD (e.g., Yehuda, 2002). Reduced awakening cortisol levels may also be related to trauma exposure, not PTSD per se. Results from de Kloet et al. (2007) showed that soldiers who had been deployed and were exposed to traumatic events, but who had not developed PTSD, also displayed lower morning cortisol levels than healthy, nonmilitary subjects. However, in the present study we found no association between deployment status and morning cortisol. Therefore, the present results suggest that a flattened CAR is not necessarily indicative of trauma exposure or PTSD, and that an attenuated cortisol awakening response may also occur in healthy subjects.

Combining the present findings with previous results leads us to the following observations: First, low scores on harm avoidance are related to a lower CAR in soldiers. Second, inter-individual variation in the CAR is to be expected in healthy individuals. Third, low cortisol levels after awakening are not necessarily the result of hyper-suppression associated with PTSD or prior deployments. Fourth, the CAR may serve as a biomarker for harm avoidance. Fifth, because low scores on harm avoidance are indicative of being less inclined to avoid new situations and being more confident, more perseverant, goal oriented and controlling (Cloninger et al., 1993), we have to consider the possibility that a flattened CAR may reflect

increased resilience to stress and novelty. Sixth, the association between self-directedness and mean cortisol increase after awakening, suggests that the CAR may be related to intrapersonal motivational processes like goal orientation, and achievement motivation.

Some limitations to the current study need to be taken into account when interpreting the results. First, although CAR has been reported to be fairly robust to factors like age, weight or smoking (Pruessner et al., 1997), the present results may have been influenced by state effects (Hellhammer et al., 2007), individual differences in sleep duration (Wüst et al., 2000b) and / or (expected) awakening time (Born et al., 1999; Edwards et al., 2001b). Cortisol levels were measured on one day only, even though it has been recommended to obtain samples on at least two consecutive days in order to minimize state influences on cortisol levels (Hellhammer et al., 2007). The fact that the association between personality and CAR emerged despite this limitation suggests that the relationship with harm avoidance and self-directedness may actually be quite robust. Further, we did not control for the potential effects of current mental disorders and psychiatric history. Although all participants were assumed to be mentally and physically fit, no formal clinical assessments were conducted to examine (mental) health status for this study.

Finally, we did not monitor compliance with saliva sampling procedure. Previous studies have shown that noncompliant individuals may produce flattened cortisol awakening responses compared to compliant subjects (Kudielka et al., 2003). We feel that this confounder may pose less of a problem for the present study as data were gathered from soldiers who can be expected to be well disciplined and compliant. Focusing exclusively on male soldiers may have introduced a source of systematic error however, and might limit generalizability of the results. All soldiers in the present sample were preparing for a deployment to Afghanistan and expectancies and stress associated with possible apprehension about the oncoming mission may have affected the cortisol levels (e.g., Wüst et al., 2000a). Expectancies about the mission may also have affected personality ratings. As noted elsewhere, low scores on harm avoidance for instance, may be the result of a response bias (Rademaker et al., 2008). The oncoming deployment may have evoked attempts to minimize feelings of insecurity or apprehension about the mission, which may have resulted in denial of items associated with emotional distress and / or unfavorable characteristics whereas items that reflect more desirable characteristics may have been more frequently endorsed. Additional studies are needed to address these limitations and to examine how gender affects the relationship between personality and the CAR.

Examining the pathways through which personality might add to the increased or reduced risk for psychopathology is of vital importance to populations at risk for trauma and stress-related disorders like the armed forces. As soldiers are frequently deployed to regions of conflict around the globe, understanding the processes involved in the development of (mental) health problems will continue to be of great importance to them and to the health care professionals who treat them. The present study illustrates that research into the CAR to explain individual differences in susceptibility to stress-related health problems may prove fruitful. As research has shown that that both the CAR (Wüst et al., 2000a) and harm avoidance (e.g., Ono et al., 2002) are related to genetic factors, it would be useful to examine whether the association between them could be perceived as being indicative of a vulnerable endophenotype for stress-related disorders (see also Gottesman and Gould, 2003).

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Chapter 5

Pathways to resiliency: Personality, coping, and social support

Pathways to resiliency: An analysis of personality, coping, and social support in Dutch veterans.
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Introduction

Individuals exposed to traumatic events may develop trauma related psychopathology. The more severe the trauma is, the greater the risk of posttraumatic disturbances like posttraumatic stress disorder (PTSD). Fortunately, only a relatively small percentage of all people exposed to traumatic events develop mental disorders as a result (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Thus, individual differences exist in vulnerability for and resilience to posttraumatic disturbances. With respect to PTSD, an impressive body of literature has accumulated over the years, examining individual risk and vulnerability factors (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003). Parallel to that, the last decade has shown a growing interest in factors that increase resilience (Richardson, 2002 for a review).

Resiliency may 'merely' reflect the absence of vulnerability (Hoge, Austin, & Pollack, 2007). This would mean that vulnerability and resilience are at opposite ends of the same dimension. Since such a view could be contended (e.g., Duckworth, Steen, & Seligman, 2005), resilience might be better defined as the ability to thrive *despite* the presence of risk factors (Richardson, 2002; Rutter, 1987). However, research on how the presence of resilient qualities (vs. absence of risk factors) aid in overcoming hardship in adult populations has not received sufficient attention in clinical research (Bonanno, 2004). Moreover, available studies on resilience typically assessed only a small number of factors so that the interplay between risk and protective factors remains unclear. The aim of the present study was to expand the understanding of resiliency by examining the interplay between dispositional risk and resilience factors, coping, social support and posttraumatic stress symptoms in a sample of Dutch peacekeeping veterans.

Background

In adult trauma literature the construct of resilience is used to describe the process of coping with adversities as well as those individual - rather than external - qualities, processes and motivational aspects that enable people to 'bounce back' in the wake of stress, high risk situations or setbacks, without notable complaints or impairment (Mancini & Bonanno, 2006; Richardson, 2002; Rutter, 1987). Resilience can be attained through multiple pathways and encompasses various aspects of human behavior including thinking, perceiving and decision making across different types of situations (Agaibi & Wilson, 2005).

By definition, coping is an important determinant of resilience. Resilient individuals may adopt more adaptive coping behavior. For instance, task or problem oriented coping is usually associated with positive health outcomes whereas passive or avoidant coping has been frequently linked to poor health outcomes (Campbell-Sills, Cohan, & Stein, 2006; Folkman & Moskowitz, 2004; Smith, 2003). Furthermore, resilient individuals may be better at seeking and getting support from others (Hoge et al., 2007). Social support is a protective factor to many forms of distress (Taylor, 2007), while lack of social support is an important risk factor for PTSD (Brewin et al., 2000).

Personality factors can affect the coping process and coping outcomes. According to Lazarus and Folkman (1984), coping is preceded by the appraisal of stressors. When a stressor is appraised as controllable, it will be perceived as a challenge rather than a threat. Sense of control therefore seems vital to problem focused coping behavior. By contrast, avoidant coping styles have been associated with a sense of lack of control (Brown, Mulhern, & Joseph, 2002). Accordingly, studies have shown perceived control to be related to well-being (Skinner, 1996), whereas (perceived) lack of control may increase the risk of depression and anxiety (Chorpita & Barlow, 1998). Similarly, generalized perceived lack of control, or external locus of control (LoC; Rotter, 1966), has been associated with PTSD (Chung, Preveza, Papandreou, & Prevezas,

2007; McKeever, McWhirter, & Huff, 2006)

Perceived control is also a key feature in one of the most well-known dispositional resilience factors: hardiness (Kobasa, 1979). Hardiness encompasses three dimensions: control, or the belief that one can influence the course of events; commitment, reflecting the appraisal of one's efforts as worthwhile and the willingness to engage oneself in purposeful actions; and challenge, which pertains to the notion that change rather than stability represents the normative way of life. Hardiness can be perceived as the personality aspect of coping and appraisal (Kobassa, 1979). It can be linked to both problem-focused coping and seeking social support (Florian, Mikulincer, & Taubman, 1995; Williams, Wiebe, & Smith, 1992), and several studies have demonstrated that hardiness is a resilience factor for PTSD (Agaibi & Wilson, 2005).

Dispositional optimism may also increase resiliency through its effects on appraisal and coping. Optimism, or the ability to maintain hope about future outcomes, has been linked positively to health and well being (Kubzansky et al., 2002; Scheier & Carver, 1987; Segerstrom, Taylor, Kemeny, & Fahey, 1998). Studies have shown that coping may mediate the relationship between optimism and health (Carver et al., 1993), as optimists engage in more effective coping, have a more extensive social support network, and are more adept in switching between coping strategies (Brissette, Scheier, & Carver, 2002; Solberg Nes & Segerstrom, 2006, for a review). Also, optimism can act as a resilience factor in adaptation to (posttraumatic) stress (Ai, Evans-Campbell, Santangelo, & Cascio, 2006; Connor & Davidson, 2003).

By contrast, one of the most important personality factors associated with increased risk for a wide range of psychological problems is neuroticism (Bienvenu & Stein, 2003; Kahn, Jacobson, Gardner, Prescott, & Kendler, 2007; Watson & Clark, 1984). Neuroticism has been reported to be negatively related to social support and positively to avoidance coping (Lawrence & Fauerbach, 2003). Moreover, both hardiness and dispositional optimism have been shown to be closely related to neuroticism (Funk, 1992; Williams, 1992). With respect to optimism, some studies have shown that it accounts for a unique proportion of variance in health outcomes and coping (Peterson, 2000; Scheier, Carver, & Bridges, 1994), whereas others reported that it did not significantly predict psychological adjustment over neuroticism and social support (e.g., Boland & Cappeliez, 1997).

To further the understanding of how dispositional risk and resilience factors are interrelated and how they affect coping and health outcomes, research is needed that assesses these factors simultaneously. Investigating how risk and resilience factors are related to health outcomes and trauma related disorders like PTSD is especially relevant to populations at risk, like military personnel. Numerous studies have shown that deployment experiences can have a long-term negative impact on physical and psychological health (Fikretoglu, Brunet, Poundja, Guay, & Pedlar, 2006; Gray, Bolton, & Litz, 2004; Wolfe, Erickson, Sharkansky, King, & King, 1999). Examination of the mechanisms involved the etiology of posttraumatic morbidity may lead to recommendations to improve treatment and prevention.

In this study, we explored the impact of traumatic stressors during military operations and related risk and resilience factors on posttraumatic stress symptoms. Specifically, we examined whether dispositional resilience factors (optimism, locus of control, and hardiness) predicted coping, social support, and PTSD independently of the effects of neuroticism. To do so, we used path analysis on data obtained from peace-mission veterans. We expected resilience factors to be negatively related to neuroticism and positively to social support and active / problem oriented coping styles. Further, we expected an inverse relationship between PTSD and dispositional resilience factors, problem focused coping and social support respectively. A positive relationship was expected between posttraumatic stress symptoms and neuroticism as well as avoidant coping.

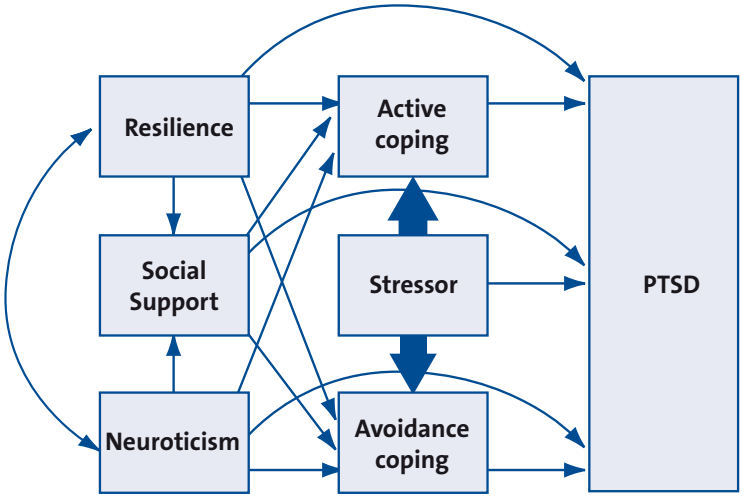

Figure 1. *Hypothesized model.*

Hypothesized model

In accordance with the coping model of Lazarus and Folkman (1984) and the reviewed literature, we expected coping styles to mediate the relationship between trauma exposure and health outcomes. To allow for the possibility of partial mediation, the direct effect of wartime exposure on post deployment morbidity was also included in our initial model. Because dispositional risk and resilience factors may be related to appraisal and subsequent coping behavior, a direct relation was expected between coping and optimism, hardiness, locus of control and neuroticism respectively. Direct effects of personality on PTSD symptoms were also expected and were included in the initial model. Similarly, to reflect the effects of social support on coping, a direct relation between social support and coping was expected. A direct relation between social support and PTSD symptoms was also included. To test if neuroticism, optimism, hardiness and locus of control predicted the amount of social support, these effects were added to the initial model. Finally, to account for correlation between personality factors, covariance arrows were drawn between all personality factors. Figure 1 displays the hypothesized model.

Method

Participants

Subject for this study were 138 male peace-mission veterans aggregated from two sources. The first group consisted of a random sample of 102 veterans registered with the Dutch Veterans Administration. The second group consisted of a convenience sample of 26 healthy veterans. The mean age of the total sample was 46.1 years (*SD* = 7.15). Most (ex)-soldiers (83%) had been deployed one time only, to Lebanon in the late seventies/ early eighties (84%). These and other demographic data are reported in Table 1.

Measures

Locus of Control was measured using Rotter's Internal-External control scale (Rotter, 1966). Optimism was measured with the revised Life Orientation Test (LOT-R; Scheier et al., 1994). Hardiness was measured using items from Kobasa's (1979) hardiness scale. The neuroticism

subscale of the revised NEO Personality Inventory (NEO-PI-R; Costa & McCrae, 1992) was used to measure trait neuroticism.

Table 1: Demographic variables and reported deployment stressors.				
Marital status	Married	Single	Cohabit.	Div./ wid.
Count (%)	104 (75.4)	16 (11.6)	11 (8.0)	7 (5.0)
Children	Yes	No		
Count (%)	105 (76.1)	33 (23.9)		
Number of deployments	1	2	3	More
Count (%)	115 (83.3)	14 (10.1)	5 (3.6)	4 (2.8)
Years since last deployment	15+	10-15	5-10	0-5
Count (%)	116 (84.1)	10 (7.2)	7 (5.1)	3 (3.5)
Rank	Priv./ corp.	NCO	Officer	
Count (%)	84 (60.9)	34 (24.6)	20 (14.5)	
Reported deployment stressors	Count	%		
Enemy fire	121	87.0		
Witnessed people suffering	111	80.4		
Personal danger	84	60.9		
Incoming fire	77	55.8		
Insufficient means to intervene	74	53.6		
Colleague injured or killed	69	50.0		
Insufficient control over situation	68	49.3		
Held at gunpoint	65	47.1		
Witnessed dead	63	45.7		
Witnessed wounded	58	42.0		
Colleague held hostage	54	39.1		
Motor vehicle accident	45	32.6		
Rejected by locals	41	29.7		
Witnessed others injured/ killed	40	29.0		
Mission felt useless	39	28.3		
Heard people screaming	37	26.8		
Held hostage	19	13.8		
Physical injuries	7	5.1		
Memories of earlier deployments	7	5.1		

Coping was assessed using the Utrecht Coping List (UCL; Schreurs, Willige, Van de Brosschot, Tellegen, & Graus, 1993) which measures coping styles. Respondents are asked to indicate how often a particular coping strategy is adopted by rating statements on a four-point likert scale ranging from 1 'never or seldom' to 4 'almost always'. Scores on scales measuring active / problem oriented (A) and avoidant (Av) coping styles were included. Active coping

reflects the tendency to engage in instrumental and problem solving coping whereas avoidance taps on a passive and avoidant coping style. The UCL has been shown to be a reliable and valid tool to assess habitual coping with test-retest reliability ratings over six weeks of .62 and .66 for active and avoidance coping respectively (Schreurs et al., 1993).

The amount of social support during and after deployment was assessed by adding the scores on sections F and L respectively of the Deployment Risk and Resilience Inventory (DRRI; King, King, & Vogt, 2003). Section F pertains to the social support received from the military during the mission. Section L measures emotional and instrumental support in civilian life and after the homecoming. Both sections were translated to Dutch specifically for the present study. The scales were translated to Dutch and back translated to English by an independent translator to ensure face validity.

Posttraumatic stress symptoms were measured with the Self-rating Inventory for PTSD (SIP; Hovens, Bramsen, & Van der Ploeg, 2000). The SIP is a Dutch self-rating scale for PTSD symptoms. It contains 22 items corresponding to symptoms in cluster B, C and D (re-experiencing, avoidance and hyper-arousal respectively) of DSM-IV (APA, 1995) diagnostic criteria for PTSD. Respondents are asked to rate the severity of PTSD symptoms over the month prior to testing on a 4-point scale ranging from 1 'not at all' to 4 'very much'. The SIP possesses good psychometric properties with internal consistency ratings (Cronbach alpha) above .75 for all subscales, and a two-week test-retest reliability of .92 for the total scale (Hovens et al., 2000). Also, the SIP has shown good concurrent validity with other PTSD measures like the Clinician Administered PTSD Scale (CAPS), and the Mississippi scale for PTSD with correlations of .73 and .82 respectively (Hovens et al., 1994). Internal consistency ratings of all tests are displayed in Table 2.

Procedure

We sent out 200 questionnaires to a random sample of veterans registered with the Dutch Veterans Administration (VA) who served in UN peacekeeping missions to Lebanon. Questionnaires were sent out through the VA to inform potential participants about the nature and purpose of the study and to ask them to anonymously fill out the questionnaire and return it using the enclosed return envelope. A total number of 102 questionnaires were returned. Additionally, 35 veterans were approached that previously participated as trauma controls in another study from our group, and who had consented to being contacted for future research. These were contacted by phone before questionnaires were sent out. Of these veterans, 26 filled out the questionnaire and returned it by mail.

Statistical analyses

Data were entered in SPSS version 15.0 and checked for missing values and normality. Missing item scores were extrapolated from individual test scores. One participant failed to answer items pertaining to exposure to stressors during deployment. Another veteran had omitted too many items on the coping questionnaire. Sample means were imputed to control for these missing data. Box-Cox transformations were computed using Minitab Statistical Software when appropriate. Path analyses were performed using AMOS 7.0. Model fit was assessed using the chi-square test statistic, Comparative Fit Index (CFI), Root-Mean-Squared Error of Approximation (RMSEA), and Tucker-Lewis Index (TLI). A p value greater than .05 for the χ^2 statistic suggests good model fit. Values greater than .95 for CFI and TLI, and around .06 for RMSEA, indicate good model fit (Byrne, 2001; Hu & Bentler, 1999). Bollen-Stine bootstrap analysis was performed on 2000 bootstraps as recommended by Nevitt and Hancock (2001) to assess overall model fit and to control for violations of multivariate normality. Finally, a standard maximum likelihood (ML) estimator bootstrapping procedure was performed.

Table 2: Means, standard deviations, Cronbach alpha's and Pearson correlations of variables in the present study.										
Variable		**N**	**H**	**O**	**LoC**	**AC**	**AvC**	**SOC**	**Exp**	**PTSS**
Neuroticism										
Hardiness		-.608**								
Optimism		-.733**	.558**							
Locus of Control†		.417**	-.541**	-.470**						
Active coping		-.557**	.538**	.470**	-.323**					
Avoidant coping		.424**	-.413**	-.300**	.288**	-.374**				
Social support		-.467**	.456**	.442**	-.345**	.336**	-.349**			
Exposure		.077	-.024	-.131	.096	-.080	-.054	-.172*		
PTSD symptoms		.752**	-.528**	-.730**	.379**	-.386**	.370**	-.508**	.239**	
	Mean	117.43	60.38	15.62	33.88	20.16	14.49	71.43	7.56	32.74
	SD	23.56	7.68	3.99	4.05	3.58	3.67	13.26	3.54	13.35
	Cronbach	.90	.75	.84	.78	.77	.73	.86	.76	.97

*Note. † = high scores reflect external locus of control; * = $p < 0.05$ level (2-tailed); ** = $p < 0.01$ level (2-tailed).*

Results

Parameter estimates and association between variables

Table 2 displays means, standard deviations, Cronbach alpha's and Pearson correlations between measures used in the present study. As shown, all scales had adequate to excellent internal consistency ratings with Cronbach alpha's ranging from .73 to .97. All variables, except exposure to deployment stressors, showed moderate to strong correlation with other variables.

Fitting the model: predicting posttraumatic stress symptoms

First, we tested the fit of the initial, hypothesized model. This yielded a χ^2 of 9.38 with six degrees of freedom and a p value of .153. Model fit indices indicated good model fit: CFI = .993; RMSEA = .064 and TLI = .958. Upon closer inspection of the regression weights and covariances, it was found that locus of control did not significantly predict posttraumatic stress ratings, social support, or coping style. Therefore, we modified the model by removing LoC. This model provided good fit, χ^2 (5) = 7.23, p = .204; CFI = .995; RMSEA = .057 and TLI = .970. Bollen-Stine bootstrap results showed that the model provided adequate fit and should not be rejected (p = .298). However, bootstrapping results showed that one path in the adjusted model may not adequately reflect the association between variables in the population from which the present sample was derived, as the path from neuroticism to social support was not significant in the bias-corrected bootstrapping results (p = .084).

As can be seen in Figure 2, which displays the standardized estimates for the adjusted model, the model predicted 64% of variance in posttraumatic stress symptoms. Additionally, the model explained 28 % of variance in social support; 23% variance in avoidant coping, and 37% variance in problem-focused coping. Hardiness, optimism and neuroticism were highly interrelated but each accounted for a unique proportion of variance in several outcome variables.

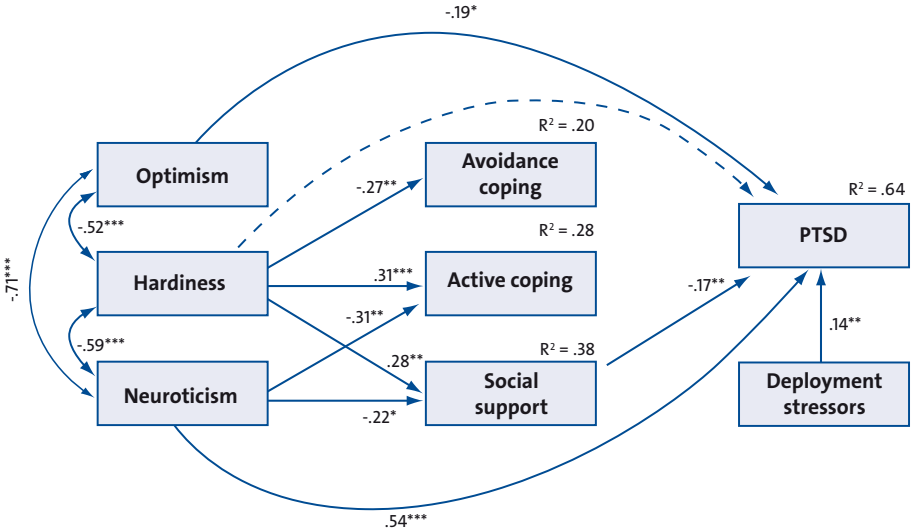

Figure 2. *Final path model explaining the relationships between deployment related stressors, coping and social support, neuroticism, hardiness, and posttraumatic stress symptoms. Dashed lines represent indirect effects. All numbers represent standardized coefficients (regression weights and correlations). *** = p < .001; ** = p < .01; * = p < .05.*

Trauma exposure was a direct predictor of posttraumatic stress symptoms. Social support and optimism also predicted posttraumatic stress symptoms but neuroticism accounted for the largest proportion of explained variance in PTSD scores. Hardiness independently and directly predicted social support, active coping and avoidance. Furthermore, hardiness was found to be an indirect (mediated) predictor of posttraumatic stress symptoms with a standardized indirect effect of -.035.

Optimism did not significantly predict coping or social support. Neuroticism was a direct predictor of active coping only, not of avoidance coping. The results pertaining to the association between neuroticism and social support were inconclusive as the path between them was not significant in the bootstrapping results. Neither problem-oriented nor avoidance coping were related to posttraumatic stress symptoms in the present sample.

Discussion

The present study showed that neuroticism, hardiness and optimism were distinct variables; each accounting for unique proportions of explained variance in different outcome measures. Stressors encountered during a peacekeeping mission more than 20 years ago predicted current posttraumatic stress symptoms in Dutch veterans. Neuroticism explained the largest amount of self-reported PTSD symptoms in this study. Social support and optimism were inversely related to posttraumatic stress symptoms. Locus of control did not add to the amount of explained variance in any of the outcome measures. Therefore, the other dispositional factors in the model appear to be better suited to predict posttraumatic adjustment than generalized control expectancies.

As expected and consistent with previous findings (Campbell-Sills et al., 2006), all dispositional resilience factors in our study were negatively related to neuroticism. The finding of an direct and independent relationship of optimism and neuroticism respectively, with posttraumatic stress symptoms, provides evidence for the uniqueness of the optimism concept relative to trait neuroticism (Scheier et al., 1994). Similarly, the significant relationships of hardiness with coping styles and social support, after statistically controlling for the effects of neuroticism, underscores the fact that hardiness must be regarded as an independent personality factor. Furthermore, both hardiness and neuroticism were related to posttraumatic morbidity via different pathways; neuroticism was a direct predictor of posttraumatic stress symptoms while the effect of hardiness on PTSD was mediated by other variables.

With respect to coping and social support, the results only partially confirmed our hypotheses. Surprisingly, neither coping style significantly predicted posttraumatic stress symptoms. This is in sharp contrast to studies that have shown problem oriented and / or avoidance coping to be a predictor of PTSD (Chung, Dennis, Easthope, Werrett, & Farmer, 2005; Kanninen, Punamäki, & Qouta, 2002; Lawrence & Fauerbach, 2003) albeit not all studies have found such a relationship (Yehuda & Flory, 2007). Possibly, coping styles exert their influence more strongly shortly after trauma exposure as compared to many years later. This is in line with findings from Kanninen et al. (2002) who reported that the acuteness of trauma exposure was an important determinant of the association between coping and symptoms. Additionally, memories of events that took place over 20 years ago may have become so deeply ingrained in memory that they are no longer under the direct influence of any particular coping style.

Hardiness accounted for a unique proportion of explained variance in coping styles and social support. This shows that hardiness may affect adjustment through these pathways. This is in agreement with previous studies (Florian et al., 1995; King, King, Fairbank, Keane, & Adams, 1998). By contrast, high neuroticism was negatively related to problem oriented coping strategies. Whether or not high neuroticism also predicts reduced social support cannot be

determined on the basis of the present study as the results were inconsistent. Optimism did not predict social support or coping. However, we cannot rule out the possibility that optimism may increase resilience by enhancing the potential to effectively switch between coping strategies (Solberg Nes & Segerstrom, 2006), as we did not examine this possibility.

An inverse relationship was expected between posttraumatic stress symptoms and dispositional resilience factors, active coping, and social support, versus a positive relationship with neuroticism and avoidance coping. Deployment related stressors were found to predict posttraumatic stress symptoms. Further, neuroticism, optimism and social support were direct predictors. These results converge with a great number of studies that have shown neuroticism to be a predictor of PTSD on the one hand (e.g., Paris, 2000), and with the existing body of evidence on the beneficial effects of social support on morbidity on the other hand (Taylor, 2007). Also, this study highlights the potential of optimism as a predictor of health outcomes in posttraumatic adjustment (Sumer, Karanci, Berument, & Gunes, 2005). The ability to maintain hope about future outcomes may directly, and independent of neuroticism, ameliorate symptom severity.

In contrasts to some studies, locus of control did not significantly predict PTSD symptoms. This was unexpected as lack of perceived control, a key aspect of both external locus of control and low hardiness, can elicit fear and distress (Hull, Van Treuren, & Virnell, 1987). Moreover, other studies did find locus of control (e.g., Chung et al., 2007) and hardiness (e.g., King et al., 1998) to be predictors of PTSD. Several explanations may account for these findings. First, there may be another variable, a mediator or common predictor, that explains the association between these personality traits and posttraumatic stress. With respect to hardiness, this seems to be the case, as social support mediated the relationship between hardiness and posttraumatic stress symptoms. Second, lack of control was incorporated in the model as part of deployment related stressors. Veterans were specifically asked whether they had experienced feelings of insufficient control, insufficient means to intervene, and whether they had at times felt that the mission was useless. Therefore, it is possible that we found no direct relationship between hardiness or locus of control and posttraumatic stress symptoms because the relationship was better accounted for by the path from deployment stressors to posttraumatic stress symptoms. Finally, previous studies on the relationship between hardiness and PTSD typically did not include optimism and neuroticism, leaving open the possibility that the association between hardiness and PTSD or between locus of control and PTSD, could be explained by overlap of these construct with other dispositional factors.

The findings of the present study have to be viewed in light of some limitations. First, the data were cross-sectional, limiting inferences on actual causality of relationships between variables on the hand, and on the strength of these relations on the other. Also, retrospective ratings of events that took place more than 20 years ago may have become biased. Life events and other variables may have obscured the relationships. Additionally, relying exclusively on self-report data may have introduced an extra form of bias. Second, the present sample consisted almost entirely of veterans who were deployed to Lebanon, which limits the generalizability of the findings. For one thing, the stressors encountered during peace-keeping operations are different from traditional combat so that findings from the present study may not be applicable to operations involving more combat. In a similar vein, the present study focused on males exclusively, therefore additional research is needed to verify whether the present results can be extended to female populations. Finally, and with special respect to the use of structural equation modeling, it should be noted that whether or not a model fits the data says nothing about the validity of any such model. An alternative model with substantially different relationships might fit the data equally well, therefore it is of paramount importance that any model under investigation has a solid theoretical basis (Byrne, 2001).

It is unclear how the resilience aspects under investigation in the present study would measure up against neuroticism in predicting positive health outcomes like posttraumatic growth. For instance, Karademas (2007) showed that optimism was a predictor of both positive and negative health outcomes whereas neuroticism predicted negative well-being only. Also, the present study captured only a fraction of the vast domain of potential resilience factors (Richardson, 2002; Southwick, Vythilingam, & Charney, 2005). Additional research is needed to address these issues.

Despite these limitations, the strength of the present study is that it assessed resilience in a non-clinical sample of soldiers who had all been exposed to the stressors of military deployment. This enabled us to examine resilience factors as they occurred in a healthy sample as opposed to patients suffering from trauma- or stress-related psychopathology. Although only a limited number of variables were included in the analyses, the amount of explained variance in PTSD scores was considerable. Moreover, the present study has provided additional evidence to support the notion that resiliency not merely reflects the absence of vulnerability: Even though the personality constructs of hardiness and optimism share overlapping variance with neuroticism, they were demonstrated to be distinct constructs that can increase resilience through various pathways.

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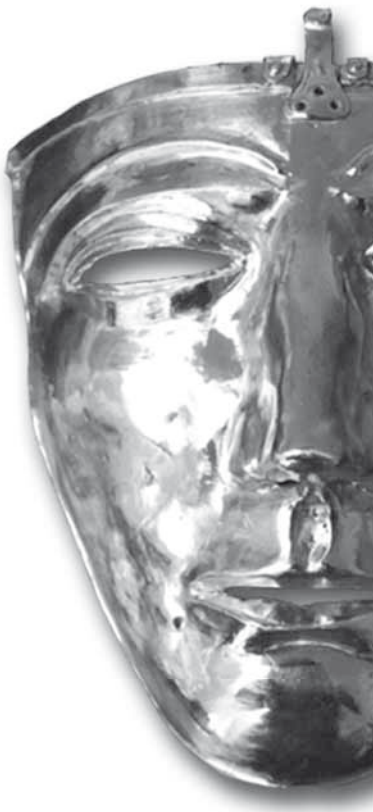
Section 2

Clinical studies



Chapter 6

MMPI-2 scores in treatment-seeking peacekeepers

Investigating the MMPI-2 trauma profile in treatment seeking peacekeepers
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Introduction

Posttraumatic Stress Disorder (PTSD) is a debilitating mental disorder that can occur after exposure to an extreme stressor. In the most recent edition of the Diagnostic and Statistical Manual of Mental Disorders, DSM-IV-TR (APA, 2000), the core symptoms are clustered in three categories: symptoms pertaining to re-experiencing the traumatic stressor, in dreams, flashbacks or intrusive memories; avoidance of stimuli associated with the traumatic event, social alienation and emotional numbing; and symptoms of increased arousal including irritability, sleeping disorders, hyper vigilance and exaggerated startle response. However, patients suffering from PTSD often display other symptoms as well. Dissociative symptoms may be present (Vermetten, Dorahy, & Spiegel, 2007), as well as problems in impulse control (e.g., Casada & Roache, 2005), substance abuse (e.g., Deering, Glover, Ready, Eddleman, & Alarcon, 1996), and personality problems (e.g., Bollinger, Riggs, Blake, & Ruzek, 2000). Therefore, in clinical practice, a multi-dimensional approach assessing the broad scope of psychopathology is recommended (Lyons, Gerardi, Wolfe, & Keane, 1988). The Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1951) has been purported to aid in such an approach.

The MMPI and its successor, the MMPI-2 (Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989), are among the most widely used self-report measures of psychopathology and personality (Greene, 2000). Extensive research, resulting in more than 10,000 published studies (Groth-Marnat, 1999), has provided a vast body of normative data for the test. Considerable research has focused on the use of the MMPI/ MMPI-2 in the assessment of PTSD. These studies have laid the foundation for the development of additional MMPI/ MMPI-2 scales, designed to differentiate between patients with and without PTSD like the Post Traumatic Stress Disorder scales PK (Keane, Malloy, & Fairbank, 1984) and Ps (Schlenger & Kulka, 1989). However, several authors have suggested that the PK and Ps scales measure general psychological maladjustment or emotional distress rather than PTSD (e.g., Moody & Kish, 1989; Wise, 1996). In addition, the predictive validity of scale PK, which is more widely used than scale Ps (Miller, Goldberg, & Streiner, 1995), appears to be limited (Adkins, Weathers, McDevitt-Murphy, & Daniels, 2008; Scheibe, Bagby, Miller, & Dorian, 2001).

Studies in traumatized individuals, mostly Vietnam veterans, have demonstrated marked similarities in MMPI/ MMPI-2 scale configuration. These have led to the identification of a prototypical PTSD profile (Wise, 1996). Patients suffering from PTSD often show high scores on validity scale F, and low scores on scales L and K. Scale L measures the tendency to present oneself in a favorable light. The K-score is an indicator of more subtle attempts to deny psychopathology. The score on scale F is an indicator of emotional distress and atypical experiences. Together this configuration may reflect high levels of emotional distress and insufficient ego-mastery (Butcher et al., 1989; Graham, 1993). This scale configuration, also referred to as the "cry-for-help" configuration, has been identified in civilian (Gaston, Brunet, Koszycki, & Bradwejn, 1996) as well as military (Wise, 1996) PTSD populations.

Studies with the original MMPI usually showed marked elevations on many of the ten clinical scales in combat PTSD samples, reflecting the presence of diffuse symptom patterns (Frueh, Hamner, Cahill, Gold, & Hamlin, 2000). Peak elevations were often reported on scale 2 (Depression; D), measuring depressive symptoms and restricted affect, and scale 8 (Schizophrenia; Sc), measuring social alienation, difficulties in thinking or concentrating, and possible intrusive symptoms (Lyons & Wheeler-Cox, 1999). These profiles are usually coded as a 2-8 or 28/ 82 code type (Fairbank, Keane, & Malloy, 1983; Lyons et al., 1988; Orr et al., 1990; Talbert et al., 1994; Wilson & Walker, 1990; Wise, 1996).

Studies in Vietnam veterans that used the successor to the MMPI, the MMPI-2, showed that the mean score on scale 7 (Psychasthenia; Pt) is often slightly higher than the score on scale

2 (D) (Albrecht et al., 1994; Baldrachi, Hilsenroth, Arsenault, Sloan, & Walter, 1999; Forbes, Creamer, & McHugh, 1999; Litz et al., 1991; Wetter, Baer, Berry, Robinson, & Sumpter, 1993; Weyermann, Norris, & Hyer, 1996). This suggests that the mean MMPI-2 profile for trauma-related pathology in combat veterans may be best characterized as an 87/ 78 code type with scale 2 (D) following closely; also described as an 872 code type. Although peak elevations on scales 2 (D), 7 (Pt) and 8 (Sc) are sometimes reported in civilian samples as well (e.g., Scott, Knoth, Beltran-Quiones, & Gomez, 2003; Wise, 1996), other studies suggest that the clinical scale configuration frequently observed in Vietnam veterans cannot be extended to civilian trauma victims (e.g., Elhai, Frueh, Gold, Gold, & Hamner, 2000; Engels, Moisan, & Harris, 1994; Gaston et al., 1996; Morrell & Rubin, 2001).

Most studies in military samples focused on Vietnam veterans. Relatively few studies have examined MMPI/ MMPI-2 scores in other military samples. A study in Gulf war veterans reported a mean 18/ 81 code type (Glenn et al., 2002), whereas in Croatian war veterans, highest mean elevations were observed on scales 1 (Hs), 2 (D), and 3 (Hy) (Begic & Jokic-Begic, 2007). Interestingly, different mean code types have also been observed in Vietnam veterans (Elhai et al., 2000; Franklin, Repasky, Thompson, Shelton, & Uddo, 2002). Furthermore, several studies have demonstrated a discrepancy between mean code type and the most frequent occurring code types in individual cases. For instance, Munley et al. (1995) demonstrated that although scales 2 (D) and 8 (Sc) were the highest in the mean profile, only 14% of individual cases displayed the 28/ 82 code type. Similarly, Mozley et al. (2005) reported that scales 2 (D) and 8 (Sc) were the highest in their mean profile, while only 8.9% of the total of 210 veterans in their sample had this code type.

Heterogeneity in MMPI/ MMPI-2 profiles within and across studies may be due to sample and stressor characteristics. Specific traumatic experiences have been reported to be associated to different symptom clusters. For instance, a study in a random sample of Australian Vietnam veterans demonstrated that specific combat experiences were differentially related to PTSD symptom patterns (O'Toole, Marshall, Schureck, & Dobson, 1999). Combat exposure in general, was associated to all three PTSD clusters. Exposure to mutilation was associated with current avoidance only, whereas exposure to civilian harm was related to avoidance and arousal but not current diagnosis. Another study demonstrated that soldiers who felt that they had failed to prevent death or injury in others reported more general psychiatric symptoms than PTSD symptoms (Fontana, Rosenheck, & Brett, 1992). As particular trauma types can be linked to specific symptom patterns in veteran samples, it is possible that MMPI/ MMPI-2 scores are related to exposure characteristics (Fontana et al., 1992; Green, 1990; O'Toole et al., 1999). Furthermore, several studies have demonstrated a relationship between stressor intensity and PTSD severity. In a meta-analysis of risk-factors for PTSD, Brewin et al. (2000) reported a weighted effect size of $r = .23$ for the association between trauma severity and PTSD severity. Therefore it is possible that more severe trauma could lead to more elevated MMPI/ MMPI-2 scores.

Research is needed to examine whether findings from Vietnam veterans can be extended to other military trauma populations or whether specific (clusters of) trauma types are related to specific MMPI-2 scores and code types. In addition, while the MMPI-2 is commonly used in many different countries, only a limited number of papers are available that report on the use of MMPI-2 in non-English speaking countries. The purpose of the present study was to investigate the relationship between posttraumatic stress symptoms and MMPI-2 scores in treatment seeking peacekeeping veterans. Specifically, we wanted to examine whether the MMPI-2 trauma profile could be identified in peacekeeping veterans with PTSD symptoms. In general, the stressors during peace missions differ from traditional combat. Whereas traditional combat is usually associated with an increased risk of casualties due to enemy fire, peacekeepers

are generally more frequently exposed to other people's suffering as they are often deployed to regions of conflict to provide humanitarian aid or to maintain safety of the local population. At the same time however, they have to be prepared to respond to life-threatening situations while the rules of engagement associated with peace missions are usually stricter, as the principle of non-use of force except for self-defense is central to most peacekeeping missions (Dirkzwager, Bramsen, & Van der Ploeg, 2005; Litz, King, King, Orsillo, & Friedman, 1997). These and other stressors put the soldiers at risk for developing health problems and mental difficulties, such as PTSD (Bolton, Litz, Glenn, Orsillo, & Roemer, 2002; Litz et al., 1997). Consequently, research has shown that among Dutch peacekeeping veterans, prevalence rates of PTSD can be found in the range of 5 to 15% (Bramsen, Dirkzwager, & van der Ploeg, 1997; Dirkzwager et al., 2005; Mouthaan et al., 2005).

The present study examined MMPI-2 scores of Dutch peacekeeping veterans with posttraumatic stress symptoms. The first Dutch participation in a United Nations (UN) peacekeeping operation dates back to operation UNIFIL in Lebanon in 1979. Since then, about 80,000 Dutch soldiers have been deployed to various regions of conflict, including former Yugoslavia, the Middle East and Afghanistan. The purpose of the present study was to provide a body of data for peace mission veterans with PTSD and to examine whether findings from previous studies in combat samples could be extended to former peacekeepers. If a mean 872 code type is indicative of (chronic) PTSD, we would expect to find a similar profile in former peacekeepers. On the other hand, differences in sample and stressor characteristics between Vietnam veterans and peace mission veterans might result in different MMPI-2 scores in the present study. We aim to answer the following questions: (a) what are the mean scores and mean code type in peacekeeping veterans with PTSD symptoms; (b) which code type occurs most often in individual cases; (c) how are PTSD symptoms and MMPI-2 scores related, and (d) what is the convergent validity of scale PK? Because scale PK is more widely used than scale Ps, we examined supplemental scale PK only.

Method

Participants

Cases were selected retrospectively from test files of soldiers and veterans who were deployed to various regions of conflict under UN or NATO mandate and who were referred to the psychiatric unit of the Dutch Central Military Hospital for assessment and treatment between 1998 and 2002. The military psychiatry department is a treatment facility that provides service to soldiers suffering from psychiatric disorders. It also offers specialized care in in- and outpatient settings for soldiers and veterans suffering from PTSD. The MMPI-2 is routinely administered to soldiers and veterans who are referred for treatment. If referrals were previously deployed, a self-report measure for PTSD is included in the standard assessment procedure. Data were available from 142 veterans who completed these self-report questionnaires as part of the intake procedure.

Cases were excluded when T-scores on MMPI-2 Variable Response Inconsistency Scale (VRIN) or True Response Inconsistency Scale (TRIN) were equal to or greater than 80, as this could indicate the presence of an invalid response-set (Derksen, de Mey, Sloore, & Hellenbosch, 1997; Graham, 1993). Exclusion of invalid and incomplete test files yielded a final sample of 120 male veterans. Cases were assigned to the PTSD group (vs. non-PTSD) when scores exceeded the recommended cut-off on a self-rating inventory for PTSD (see below). The PTSD group consisted of 90 veterans with a mean age of 35 years (SD = 7.0). This group consisted of veterans deployed to Lebanon between 1979 and 1985 (n = 34, mean age = 40

years, *SD* = 2.8), veterans who served in former Yugoslavia after 1990 (n = 40, mean age = 32 years, *SD* = 6.5), and soldiers deployed to areas like Angola, Iraq and Cambodia (*n* = 16, mean age = 34 years, *SD* = 8.5). The reference group consisted of 30 veterans (mean age = 35 yrs, *SD* = 8.7) who were referred for treatment of mental problems but who screened negative for PTSD. All had previously been deployed to various regions of conflict.

Measures

PTSD was assessed using the Self-rating Inventory for PTSD (SIP; Hovens, Bramsen, & van der Ploeg, 2000). The SIP consists of 22 items that correspond to cluster B, C and D of DSM-IV (APA, 1994) diagnostic criteria for PTSD (re-experiencing, avoidance and arousal respectively). Respondents are asked to rate the severity of PTSD symptoms over the month prior to testing on a 4-point scale ranging from 1 *(not at all)* to 4 *(very much)*. A total score equal to or greater than 52 suggests the presence of PTSD. The SIP has been validated against several other measures for PTSD and has good psychometric properties with internal consistency ratings above .75 (Cronbach alpha) for all subscales, and a two-week test-retest reliability of .92 for the total scale (Hovens et al., 2000). Also, the SIP has shown good concurrent validity with other PTSD measures like the Clinician Administered PTSD Scale (CAPS) and Mississippi scale for PTSD with correlations of .73 and .82 respectively (Hovens et al., 1994).

Additionally, all subjects completed the MMPI-2 (Butcher et al., 1989) as part of the intake procedure. The Dutch version of the MMPI-2 consists of 567 items. Items are scored "true", "false" or "don't know". Raw scores are coded into T-scores with a mean of 50 and a standard deviation of 10 for all clinical scales apart form scale 5 (M/ f) and 0 (Si). T-scores greater then 65 correspond to scores in the 9th decile (Derksen et al., 1997; Graham, 1993). For the present study, T-scores on MMPI-2 validity scales: L, F, K, all clinical scales, and raw scores on supplemental scale PK (Keane et al., 1984) were included. For each respondent, the 2- and 3-point code type was derived from the Welsh-code.

Analyses

Multivariate and subsequent univariate analyses of variance, (M)ANOVA, were conducted to compare the PTSD group to the non-PTSD group on the basis of T-scores on MMPI-2 validity and clinical scales. Frequency analysis of individual 2- and 3-point code types was performed to investigate whether any code types were predominant in the PTSD sample. To explore the association between MMPI-2 clinical scales and PTSD symptoms as measured by the SIP, bivariate correlations were computed. Similarly, convergent validity of scale PK was examined by computing bivariate correlations between this MMPI-2 PTSD scale and scores on the SIP.

Results

Data considerations

Prior to running analyses, data were checked for outliers and other potential violations of assumptions. All variable were sufficiently normal distributed and multivariate normality was observed. Levene's test for equality of error variances showed that the assumption of homogeneity of variance was met. The Variance Inflation Factor (VIF) of each scale was evaluated to assess multi-collinearity. A commonly applied rule of thumb is to consider VIF > 5 as a cause for concern and VIF > 10 as indicative of serious multicollinearity problems (O'Brien, 2007); although others have suggested that this rule of thumb is too lenient (Cohen, Cohen, West & Aiken, 2003). The highest VIF value was found on scale 8 (VIF = 7.5). A VIF of 5 was

observed on MMPI-2 scales 1 (VIF = 5.3) and 3 (VIF = 5.0). Scales 2 and 7 showed a VIF of 4.2, whereas the other scales yielded VIF values ranging from 1.7 (SIP re-experiencing) to 2.4 (MMPI-2 Scale 0). The VIF values of scales 1 (Hs), 3 (Hy) and 8 (Sc) indicate that collinearity might compose a problem.

Group characteristics and mean scores

Mean scores and standard deviations of MMPI-2 scales in the total PTSD group (n = 90) and reference group (n = 30) are reported in Table 1. Veterans screening positive for PTSD showed mean elevations greater than T = 65 on all clinical scales apart from scale 5 (M/f), 9 (Ma), and 0 (Si). Highest mean elevations were found on scales F and 2 (D), followed by scales 8 (Sc) and 7 (Pt). The non-PTSD group (n = 30) showed elevations on validity scale F, and clinical scales 3 (Hy), 4 (Pd), and 2 (D).

MANOVA comparing the PTSD and non-PTSD group on MMPI-2 scales yielded significant differences between groups, $F_{(13,106)} = 4.90$, $p < .001$, with a moderate effect size (Cohen's $d = 0.48$). Table 1 displays the F values and effect sizes (Cohen's d) of subsequent ANOVA's. Veterans screening positive for PTSD scored significantly higher (Bonferroni corrected alpha $p < .004$) on scales F, 2 (D), 4 (Pd), 6 (Pa), 7 (Pt), 8 (Sc), and 0 (Si), and lower on scale K. The mean profile of the PTSD group is presented in Figure 1. Data from Wetter et al. (1993), Forbes et al. (1999), and Litz et al. (1991) are added for comparison.

Table 1: MMPI-2 Data for Dutch Peacekeepers Screening Positive and Negative for PTSD

Scale	PTSD (n = 90)		Non-PTSD (n = 30)				
	M	*SD*	*M*	*SD*	*F*	*p*	*d*
L	44.24	8.66	49.37	9.91	7.32	.008	0.57
F	77.50	21.22	62.40	13.71	13.30	< .001	0.77
K	36.79	8.80	48.07	11.88	30.72	< .001	1.17
1 (Hs)	70.46	14.42	62.10	14.32	7.58	.007	0.58
2 (D)	77.24	14.46	66.10	13.73	13.69	< .001	0.78
3 (Hy)	74.09	16.44	67.00	14.57	4.42	.038	0.44
4 (Pd)	74.80	12.87	66.63	12.25	9.27	.003	0.64
5 (Mf)	51.80	8.85	50.40	10.44	0.51	.475	0.15
6 (Pa)	70.98	13.87	60.47	10.60	14.40	< .001	0.80
7 (Pt)	75.39	10.93	62.47	10.42	32.19	< .001	1.20
8 (Sc)	75.71	12.28	64.50	9.20	21.03	< .001	0.97
9 (Ma)	63.71	12.90	62.70	12.94	0.14	.711	0.08
0 (Si)	60.44	12.31	50.50	11.46	15.19	< .001	0.82
Pk (rs)	27.99	8.45	15.73	7.06	51.12	< .001	1.51

Note. L = Lie; F = infrequency; K = correction; 1 (Hs) = Hypochondriasis; 2 (D) = Depression; 3 (Hy) = Hysteria; 4 (Pd) = Psychopathic Deviate; 5 (M/f) = Masculinity-femininity scale; 6 (Pa) = Paranoia; 7 (Pt) = Psychastenia; 8 (Sc) = Schizophrenia; 9 (Ma) = Hypomania; 0 (Si) = Social introversion; rs = raw score.

As can be seen in Figure 1, the mean profile showed similarities with those of Vietnam veterans with PTSD. Peacekeeping veterans that screened positive for PTSD showed mean elevations on scales 1 (Hs), 2 (D), 3 (Hy), 4 (Pd), 6 (Pa), 7 (Pt) and 8 (Sc). Scores on the validity scales were indicative of a cry-for help configuration. An inverted V-shape could be identified in the mean scores on scales 1 (Hs), 2 (D) and 3 (Hy) and the profile showed high scores on scales 6 (Pa), 7 (Pt), and 8 (Sc). Also similar to what has been previously been reported in other trauma samples, scales 2 (D), 8 (Sc) and 7 (Pt) were highest in the mean profile.

According to Graham (1993) this configuration is indicative of mixed pathology in people experiencing anxiety and depressive symptoms, blunted affect, difficulty concentrating and a tendency towards a schizoid lifestyle. Use of alcohol or other drugs may be present as a way of coping. This description aptly depicts most of the clinical features of chronic PTSD. Because scores on scales 1 (Hs), 3 (Hy), 4 (Pd) and 6 (Pa) were within the same range however, items pertaining to somatic complaints, fatigue, alienation, interpersonal difficulty, impulsiveness, vigilance and anger, were as often endorsed and are therefore equally relevant to the clinical description of the mean profile.

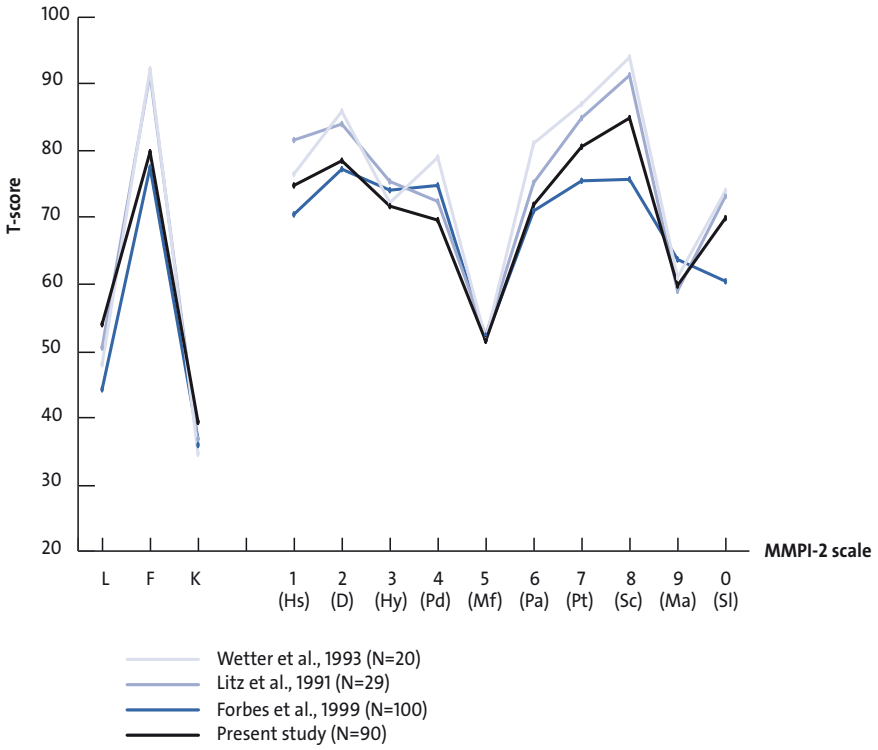

Figure 1. *Mean MMPI-2 profile of treatment seeking peacekeeping veterans screening positive for PTSD. Data from Vietnam veterans with PTSD from studies by Wetter et al. (1993), Litz et al. (1991) and Forbes et al. (1999), added for comparison.*[1]

[1] *Adapted from the MMPI-2 Validity and Clinical Scales Profile, MMPI ®-2 (Minnesota Multiphasic Personality Inventory®-2) Manual for Administration, Scoring, and Interpretation, Revised Edition. Copyright © 2001 by the Regents of the University of Minnesota. All rights reserved. Used by permission of the University of Minnesota Press. "MMPI-2" and "Minnesota Multiphasic Personality Inventory-2" are trademarks owned by the Regents of the University of Minnesota.*

Code types

Scales 2 (D), 7 (Pt) and 8 (Sc) were the highest in the mean profile, but only 5 veterans showed highest elevations on all three scales. Frequency analysis of individual code types in the total PTSD group revealed 25 different two-point code types. Scale 2 (D) occurred most frequently as single high-point (27.8 %). As shown in Table 2, the 27/ 72 two-point code type was most often found (13.3 %) in the total PTSD group, followed by the 24/ 42 and 34/ 43 code, both in 8.9 % of the cases. A 78/ 87 code type was present in 4.4 % of the veterans screening positive for PTSD.

Table 2. Summary of 2- and 3-point Code Types in Dutch Veterans Screening Positive for PTSD (n = 90). †

Code type	count	%	3-point (f)
1-3/ 3-1	5	5.6	132 (1); 312 (3); 318 (1)
2-3/ 3-2	6	6.7	231 (1); 236 (1); 237 (2); 238 (1); 321 (1)
2-4/ 4-2	8	8.9	241 (1); 243 (2); 247 (3); 249 (1); 427 (1)
2-7/ 7-2	12	13.3	270 (2); 273 (3); 278 (3); 721 (1); 723 (1); 724 (1); 726 (1)
2-8/ 8-2	6	6.7	281 (1); 283 (2); 287 (1); 824 (1); 827 (1)
3-4/ 4-3	8	8.9	341 (1); 342 (1); 346 (1) ; 432 (1); 437 (3); 439 (1)
4-6/ 6-4	4	4.4	462 (2); 468 (2)
4-7/ 7-4	3	3.3	472 (2); 473 (1)
4-9/ 9-4	5	5.6	497 (2); 940 (1); 946 (1); 947 (1)
6-8/ 8-6	5	5.6	682 (1); 861 (1); 863 (1); 864 (2)
6-9/ 9-6	3	3.3	694 (1); 968 (2)
7-8/ 8-7	4	4.4	789 (1); 872 (1); 874 (1); 879 (1)

Note.† Only 2-point code types occurring three or more times are displayed.

Association between MMPI-2 scores and PTSD symptoms

To explore the association between MMPI-2 scores and PTSD symptoms, bivariate correlations were computed. Table 3 shows that moderate correlations were present between the SIP total score and MMPI-2 scales 1 (Hs), 2 (D), 6 (Pa), 7 (Pt), and 8 (Sc). The association with scale 1 (Hs) most likely reflects physiological reactivity and somatic problems associated with increased anxiety, typical to PTSD. The association between scale 2 (D) and PTSD symptoms can be accounted for by depressive symptoms including sleeping problems, restricted affect, and lack of interest which are often reported by PTSD patients. The relationship with scale 6 (Pa) most likely reflects the presence of aggressive acting-out behavior and heightened irritability and hostility. MMPI-2 scale 7 (Pt) encompasses symptoms of anxiety, feeling tense, irritability and concentration difficulties. The association found between scale 8 (Sc) and PTSD symptoms most likely taps on symptoms of social alienation and emotional numbing as found in cluster C (avoidance) of PTSD, and intrusive and/ or dissociative symptoms. Additionally, it is possible that generalized distress may account for part of the association between MMPI-2 clinical scales and PTSD symptoms. Patients suffering from (chronic) PTSD usually report marked impairment and distress, and the presence of a general distress or maladaptation factor underlying scores on the MMPI-2 clinical scales is well established (Archer, 2006).

Table 3. Correlations between the Self-rating Inventory for PTSD (SIP) and MMPI-2 Scales (N = 120).

	Scale	T	R	AV	HA	1	2	3	4	5	6	7	8	9	0
SIP	Tot. (T)	-													
	Re-exp. (R)	.76**	-												
	Avoidance (AV)	.83**	.52**	-											
	Arousal (HA)	.85**	.57**	.67**	-										
MMPI-2	1 (Hs)	.43**	.35**	.32**	.37**	-									
	2 (D)	.42**	.26**	.40**	.40**	.65**	-								
	3 (Hy)	.29**	.30**	.19*	.24**	.85**	.65**	-							
	4 (Pd)	.31**	.19*	.25**	.30**	.44**	.48**	.42**	-						
	5 (Mf)	.13	.06	.11	.07	.09	.27**	.19*	.11	-					
	6 (Pa)	.47**	.38**	.39**	.37**	.47**	.46**	.42**	.53**	.27**	-				
	7 (Pt)	.56**	.37**	.47**	.47**	.57**	.73**	.53**	.54**	.34**	.57**	-			
	8 (Sc)	.56**	.37**	.47**	.46**	.68**	.69**	.58**	.66**	.24**	.70**	.83**	-		
	9 (Ma)	.15	.11	.10	.12	.17	-.22*	-.01	.18*	-.03	.30**	.00	.24**	-	
	0 (Si)	.36**	.18*	.38**	.30**	.32**	.62**	.21*	.26**	.31**	.36**	.60**	.58**	-19*	-
	PK	.69**	.41**	.64**	.61**	.52**	.71**	.40**	.62**	.25**	.67**	.77**	.82**	.22*	.64**

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

Correlations in the range of .60 to .69 were observed between scale PK[2] and the SIP total score and two of its subscales, avoidance and arousal. A correlation of .41 was observed with re-experiencing symptoms. Notably, correlations of .71, .77, and .82 were observed between scale PK and MMPI-2 clinical scales 2 (D), 7 (Pt), and 8 (Sc) respectively. As these exceeded the correlations of scale PK with SIP scores, the discriminant validity of scale PK may be limited.

Discussion

Similar to what has been reported in combat veterans with PTSD (Frueh et al., 2000), peacekeepers with posttraumatic stress symptoms showed mean elevations on many of the MMPI-2 clinical scales, revealing diffuse levels of psychopathology. Scores on scales 2 (D), 8 (Sc) and 7 (Pt) were highest in mean profile but only marginally so, and high scores on MMPI-2 clinical scales 1 (Hs), and 6 (Pa) were also shown to be moderately correlated with PTSD symptoms. Scale PK showed marked correlations with avoidance and arousal symptoms and correlated moderately with intrusive symptomatology. These findings correspond to results reported by Wolf et al. (2008).

As Figure 1 showed, the mean profile of the peacekeepers in the present study resembled that of Vietnam veterans with PTSD. However, there were also differences between peacekeepers and combat veterans. Consistent with the notion that, in general, peacekeeping operations are associated with lower stressor intensity than traditional combat (Dirkzwager, Bramsen, & Van der Ploeg, 2003), the present sample showed less severe levels of psychopathology on the MMPI-2, compared to Vietnam veterans. It is possible that cultural factors account for (part of the) differences in overall scale elevations between Dutch peacekeepers and US and Australian Vietnam veterans. However, an evaluation of cross-cultural aspects in psychiatric morbidity is beyond the scope of the present paper.

The present sample showed marked heterogeneity in individual code types. Only five veterans had a three-point code type combining scales 2 (D), 7 (Pt) and 8 (Sc). The "typical" 28/ 82 code type was found in only 7% of the cases. A 78/ 87 code type was observed in even fewer veterans. Because code types are proposed to reflect specific symptom patterns, the observed diversity in individual code types suggests that the veterans presented heterogeneous psychopathological symptom patterns. Also, mean elevations on many of the MMPI-2 clinical scales were within the same range so no code type could be defined that adequately reflected the profile presented by the majority of veterans in the present study. These results underscore the notion that a mean code type cannot be expected to accurately reflect individual code types (Baldrachi et al., 1999; Glenn et al., 2002; Mozley et al., 2005; Munley et al., 1995). In fact, clinicians and researchers should expect a variety of code types and profiles to emerge in trauma populations, irrespective of the presence of PTSD (Wise, 1996). Therefore, we have to conclude that the code type cannot be used to screen for the presence of PTSD.

As noted before, it is possible that the diversity in code types reflects variations in trauma exposure. According to Bramsen et al. (1997), Dutch peacekeepers frequently reported encountering enemy fire and being confronted with other people's suffering during their missions. They were less likely to be confronted with atrocities or to have witnessed their colleagues get injured or killed than veterans from the Vietnam War. Therefore, the observed heterogeneity in code types may reflect diffuse psychiatric symptoms associated with the stressors encountered during peacekeeping missions.

The disparity between the present results and those reported by other researchers (Albrecht et al., 1994; Forbes et al., 1999; Litz et al., 1991; Wetter et al., 1993) may also be due to differences

[2] *A T-score of 65 corresponds to a raw score of 15 in the Dutch MMPI-2 whereas in the US version it corresponds to a raw score of 17 for males (Graham, 1993), even though scale PK comprises of 46 items in both versions.*

in MMPI-2 versions. As noted before, the T-score transition of scale PK in the Dutch version differs from the US version. A related effect can be observed in the K-correction algorithm. To control for a defensive response style, the K-correction is an algorithm that adds a fraction of the score on validity scale K to raw scores on clinical scales. This results in higher T-scores on the scales that are affected by the correction. However, we discovered that when low scores on validity scale K are present, applying the K-correction will result in lower T-scores on the affected clinical scales. Using K-corrected T-scores will yield lower scores on several clinical scales, and will increase the relative elevation of the other scales in the profile. This effect is especially relevant to PTSD populations, as both previous research and the present study have shown that low K-scores are characteristic for patients suffering from PTSD.

Because the K-correction affects clinical scale elevations, it can also alter the code type. Therefore, differences between reported results across studies may be attributed to whether or not the K-correction was applied. Moreover, according to the manual by Graham (1993, p.19) the K-correction affects scores on scales 1 (Hs), 4 (Pd), 7 (Pt), and 9 (Ma) in the US version, whereas scales 2(D) and 8 (Sc) are unaffected. In the Dutch version however, the K-correction also affects scale 8 (Sc). As K-corrected T-scores were used in the present study, this may have resulted in lower T-scores on scale 8 (Sc) compared to studies using the English version. This might explain why instead of scale 8 (Sc), scale 2 (D) was found to be the most occurring high-point.

There are several limitations to the present study that need to be taken into account. Cultural aspects may limit the generalizability of the present results and may have biased the comparison between the present study and previous results. Also, we did not control for the influence of comorbid psychiatric disorders. Therefore, differences in MMPI-2 scores between peacekeepers and Vietnam veterans may reflect differences in psychiatric (co)morbidity, one the one hand, and may be related to cultural and demographic aspects on the other hand. Further, we did not control for potential effects of compensation-seeking on MMPI-2 scores. A confounding factor in the group comparisons is the fact that PTSD was measured with a self-report instrument, the SIP (Hovens et al., 2000). Because the respondents were categorized in PTSD and non-PTSD subgroups on the base of the scores on this self-report measure, the results relied upon the accuracy and reliability of the SIP in predicting the presence of PTSD.

Furthermore, the MMPI-2 is known for its highly inter-correlated clinical scales (e.g., Simms, Casillas, Clark, Watson, & Doebbeling, 2005), and this was demonstrated in the present study as well. Therefore, for future research it would be interesting to see how the restructured clinical scales (Tellegen et al., 2003) are related to PTSD symptom clusters. As these were constructed to reduce inter-scale correlations, investigating them using more sophisticated multi-variate analyses might provide insight in the unique contribution of each scale to the association with PTSD.

Despite these limitations, the present study adds to the body of literature evaluating the use of the MMPI-2 in PTSD samples. We provided more insight into the association between MMPI-2 scores and PTD symptoms and we showed that peacekeeping veterans presenting with posttraumatic stress symptoms display similarities with other (military) trauma samples. However, it must be concluded that the available literature on MMPI-2 scores in Vietnam veterans with PTSD cannot be extended to former peacekeepers as important differences were observed. Although scales 2(D), 7 (Pt) and 8 (Sc) emerged as the highest in the mean profile, neither the 28/ 82 nor the 78/ 87 (or any other code type) adequately described the symptom patterns presented by the majority of the peacekeepers. Because different code types should be expected in trauma populations, irrespective of the presence of PTSD, the code type and mean profile were of limited diagnostic use in the assessment of PTSD. Furthermore, although scale PK was correlated with PTSD symptom clusters and total score, it appears that other instru-

ments may be better suited to differentiate between PTSD and non-PTSD individuals. That being said, we feel that the MMPI-2 can contribute to the assessment of trauma-related morbidity as it can provide information on the severity of posttraumatic morbidity, and because it adequately assessed the broad range of symptoms typically present in trauma populations.

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Chapter 7

Evaluation of a multi-modal group treatment program

Multimodal exposure-based group treatment for peacekeepers with PTSD:
A preliminary evaluation
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Introduction

Over the last decade several treatments have been proposed for posttraumatic stress disorder (PTSD), varying in setting (e.g., inpatient vs. outpatient), modality (e.g., exposure vs. relaxation), timeframe, and most importantly, therapeutic focus. Most effective therapies have been found to be cognitive behaviour therapy (CBT), incorporating elements of exposure, and more recently eye movement desensitization and reprocessing therapy (EMDR; Bisson & Andrew, 2005; Bradley, Greene, Russ, Dutra, & Westen, 2005; Davidson & Parker, 2001; Van Etten & Taylor, 1998). Cognitive behaviour therapy generally focuses on reduction of PTSD symptoms and improvement of quality of life despite the presence of, often chronic, symptoms. Although the efficacy of psychotherapy in treatment of PTSD has been established in various trauma populations (Foa, Keane, & Friedman, 2000), there is evidence that many patients still suffer from clinically significant symptoms and interpersonal difficulties after treatment (Bradley et al., 2005; Brom, Kleber, & Defares, 1989; Lyons & Wheeler-Cox, 1999). Combat-related PTSD especially appears to be a difficult disorder to treat (Johnson, Fontana, Lubin, Corn, & Rosenheck, 2004; Solomon, Gerrity, & Muff, 1992).

Group treatment for veterans with disorders related to war trauma became popular in the 70s, after the Vietnam War (Shatan, 1973). This form of therapy was deemed especially effective to treat Vietnam veterans who, because of their pathology and post-war adjustment problems, had to cope with alienation and isolation (Allen & Bloom, 1994). Most of the early group programs were of a supportive nature, but since then other variants have been developed. On the whole, available studies indicate that these are effective in reducing PTSD symptoms irrespective of trauma type and differences in sample characteristics (Bisson & Andrew, 2005; Foy et al., 2000). The majority of the available studies are based on individualized programs however. Most of the available research, though not all (Bolton et al., 2004), has focussed on the effects of treatment on PTSD symptoms only, not on associated features or disorders like depression and anxiety, work performance, or quality of life (Glynn et al., 1999). This is somewhat surprising given the usually high rates of comorbid disorders in patients suffering from PTSD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Magruder et al., 2005; Owens, Baker, Kasckow, Ciesla, & Mohamed, 2005). Furthermore, hardly any studies have examined the efficacy of group-based therapy programs that combine exposure with other therapeutic means or modules.

We found only a few studies that evaluated multimodal group therapy programs for combat-related PTSD that reported effects on PTSD as well as associated problems and disorders (Frueh, Turner, Beidel, Mirabella, & Jones, 1996; Schnurr et al., 2003). Frueh et al. (1996) described the effects of a multimodal group therapy in a sample of chronic combat-related PTSD patients. Their program combined individual psycho-education and exposure with group sessions for social skills training and anger management. The authors observed a significant improvement in PTSD symptoms, general anxiety, and overall social functioning. Although the study did not include a control group, the authors concluded that the additional therapeutic elements provided a program that was superior to exposure alone as it targeted both PTSD and associated problems.

Schnurr et al. (2003) investigated the therapeutic effects of a program described by Foy et al. (2000): a manualized exposure-based group therapy for combat-related PTSD in Vietnam Veterans. This program was based on systematic exposure, cognitive restructuring, psychoeducation and coping skills training. It was aimed at improving quality of life and increasing control over chronic PTSD symptoms, as well as preventing relapse. That is, therapy was intended to reduce symptoms, and more importantly, to teach patients how to cope with the lifelong risk of symptom exacerbation. Also, the program aimed at challenging veterans to adopt

realistic goals to obtain higher levels of quality of life whilst managing their vulnerability to relapse. Therapy consisted of 30 weekly sessions of 90-120 minutes, followed by 5 monthly 'booster' sessions. The program was evaluated in a randomized clinical trial that showed that therapy only had modest effect. Moreover, the effects were no better than those obtained in a control group of non-trauma focused group therapy (Schnurr et al., 2003).

Although the empirical evidence supporting the use of multimodal group programs is limited, from a therapeutic viewpoint, group treatment remains attractive because it can provide several benefits over individual therapy. These are described in great detail by Yalom (1995). One of the more salient advantages of group therapy for combat veterans is that it provides veterans with social support of people with whom they closely identify. Successful alliance to the group can create a sense of safety, which facilitates self-disclosure. In addition, a sense of safety is necessary to engage optimally in exposure therapy. Moreover, group therapy can help to recreate some of the (narcissistic) identifications, which were present during military training and the mission, thereby helping to reconstruct the context in which the trauma occurred (Van der Kolk, 1985). Additionally, group exposure provides the opportunity for vicarious exposure to trauma (Schnurr et al., 2003), and as Yalom (1995) pointed out, the group may enable its members to provide feedback on each other's functioning. This enables group members to become aware of maladaptive patterns and allows patients to learn from each other. Finally, the group structure can help to normalize symptoms and the group environment can become a place to try out new behaviour and newly acquired coping and social skills (Yalom, 1995).

In line with increased efforts to help veterans with deployment related pathology in the Netherlands in the nineties, the Department of Military Psychiatry of the Dutch Central Military Hospital started an outpatient group treatment program for veterans with chronic PTSD. The program was multimodal in nature. It consisted of CBT, creative arts therapy, psychodrama, psychomotor therapy, socio-therapy, psycho-education and case management. These were provided as day treatment on a weekly basis with an average duration of 21 months. The aim of the present study was to evaluate this program with respect to the effects on PTSD and related symptoms, as well as with respect to social and work related problems, personality functioning and coping. Specifically, we assessed the effects of treatment on PTSD and depressive symptoms, level of anxiety, personality, somatic complaints, social functioning, work problems, as well as self reported active, avoidant and social coping behaviour.

Method

Subjects

Data were collected retrospectively from veterans who participated in the outpatient group therapy program. As part of the intake procedure, participants were extensively screened by trained clinicians and with the use of different instruments to assess psychopathology, coping and personality functioning. Diagnosis and therapeutic plan were assigned by clinical staff consensus on data gathered during the intake procedure. Veterans meeting criteria for psychotic disorders were excluded from participation, disorders in substance or alcohol abuse/dependence had to be in (early) remission before enrolling in the program.

We performed a retrospective evaluation of the test files of those veterans who had successfully completed the treatment program as well as the pre- and post-treatment assessments. Post treatment assessments were conducted on average 7 weeks upon completion of treatment. Between August 2003 and March 2006, 26 former peacekeepers finished the program. All subjects were male and all had participated in UN peacekeeping missions (e.g. Lebanon,

Balkans). However, we were only able to retrieve test-files from 22 veterans as either intake (1) or post-treatment (3) assessments were missing from 4 veterans.

All subjects met DSM-IV (APA, 1994) criteria for chronic PTSD and almost all participants had received treatment prior to enrolling in the day-treatment program. About 30% of participants had previously received treatment for substance abuse / dependence and an equal amount of veterans had been inpatients at civilian or military settings. Additionally, several participants had received individual treatment. Veterans meeting criteria for psychotic disorders were excluded from participation, disorders in substance or alcohol abuse/ dependence had to be in (early) remission before enrolling in the program was possible. The most commonly reported traumata were incoming enemy fire, being held at gunpoint/ held hostage, and witnessing other peoples suffering. Additionally, several participants reported being confronted with death or injuries of colleagues, being under mortar attack, witnessing bombings, seeing people getting killed or injured, and witnessing mass graves and/ or charred bodies.

Treatment

The program was devised in the 90's with the aim to provide treatment for a growing group of UN-veterans presenting with complex forms of PTSD (Meijer, 2002). Therapy was provided in semi-closed groups of up to 8 patients who received treatment one day from 9:00 am to 4:00 pm each week. To ensure group cohesion, new participants enrolled in therapy on only four occasions each year, with a maximum of 2 new participants per occasion. The program was based on a phase-oriented approach that followed a set path of phases comprising of stabilization, exposure and integration (Ford, Courtois, Steele, Hart, & Nijenhuis, 2005; Van der Kolk & van der Hart, 1989).

Each day of treatment consisted of multiple modules and included exposure-based CBT, psycho-education, socio-therapy, case-management, creative arts therapy, psychodrama, and psycho-motor therapy. Socio-therapy focused on specific problems in everyday situations pertaining mainly to self-esteem and self-perception, family, social support, work and finances. Psychodrama, creative therapy and psycho-motor therapy were incorporated to facilitate therapeutic progress as they addressed and sought to resolve emotional problems that are common to PTSD. The case-managers coordinated care between patient, care providers, and the patient's social system. Additionally, every 6 weeks the partners or important family members (based on the choice by the participant) were invited to participate in the program to ensure partner involvement. On these days, the therapeutic progress of the patients and the problems the spouses or significant others encountered in dealing with the patients' problems were the focus of attention.

Measures

Before and after treatment all patients completed a battery of self-report measures including: The *Self-rating Inventory for PTSD*, SIP (Hovens, Bramsen, & van der Ploeg, 2000). The SIP is a Dutch self-rating scale for PTSD symptoms. It comprises of 22 items that correspond to cluster B, C and D of the DSM-IV (APA, 1994) diagnostic criteria for PTSD (re-experiencing, avoidance and hyper-arousal respectively). Respondents are asked to rate the severity of PTSD symptoms during the month prior to testing on a 4-point scale ranging from 1 *(not at all)* to 4 *(very much)*. The list has been validated against several self-report measures for PTSD and the psychometric properties have been rated adequate to excellent (Evers, Vliet-Mulder, & Groot, 2002; Hovens, 1994; Hovens et al., 2000)

The Symptom Checklist, SCL-90 (Derogatis, Lipman, & Covi, 1973). The SCL-90 is a 90-item self-report symptom inventory, designed to measure psychological symptom patterns

and levels of distress. The total score can be seen as an indicator of the overall level of distress. The SCL-90 is often used to study therapeutic efficacy and several studies have shown the SCL-90 to be sensitive to detect therapeutic effect (Arrindell & Ettema, 1986; Arrindell & Ettema, 2003).

The Utrecht Coping Scale, UCL (Schreurs, Willige, van de Brosschot, Tellegen, & Graus, 1993). This scale was designed to measure the ways in which people react when confronted with problems or daily hassles. It is one of the most commonly used coping questionnaires in the Netherlands. The list comprises of 47 items which are answered on a 4-point Likert scale (1 = seldom or never, 2 = sometimes; 3 = often, 4 = very often). Scores on scales (A) Active coping, (Av) Avoidance, and (S) Social support were included in the present study.

The Minnesota Multiphasic Personality Inventory, MMPI-2 (Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989). The Dutch version of the MMPI-2 (Derksen, de Mey, Sloore, & Hellenbosch, 1997), consists of 567 items. T-scores on MMPI-2 validity scales: L, F, K, as well as K-corrected T-scores on all clinical scales, except scale 5 (M/f) measuring gender (a) specific behaviour, were included. MMPI-2 content scales Anx, Dep, Lse, Wrk, Sod, and supplemental scale Pk were included as a measure of self-reported levels of anxiety, depressive symptoms, self-esteem, work problems, social problems, and PTSD symptoms respectively.

Data analyses

Two MANOVA's for repeated measures were conducted as omnibus tests to compare pre- to post treatment scores on MMPI-2 scales, and SCL-90, SIP and UCL scales respectively. Subsequent paired samples t-tests were performed to examine pre- to post treatment scores in more detail.

Results

Sample description

The mean age of the participants that were included in this analysis was 41.7 years (*SD* = 5.6) and most were married or cohabitating. Most of the veterans had been deployed once, to either Lebanon or former Yugoslavia. Only two veterans had been deployed twice. The average duration of therapy was 1 year and 9 months (*SD* = 5.3). These and other background variables are displayed in Table 1.

Table 1: Background variables

	Mean	*SD*	Range	
Age (years)	41.68	5.64	32 - 48	
Duration of therapy (mts)	20.91	5.34	13 - 33	
Marital status	Married	Divorced	Cohabitating	Single
Count (%)	11 (50.0)	6 (27.3)	4 (18.2)	1 (4.5)
Education level	Low	Average	Higher	Unknown
Count (%)	8 (36.4)	10 (45.5)	2 (9.1)	2 (9.1)
Area of deployment	Lebanon '78-'83	form.Yugo. '93-'99	Other	
Count (%)	14 (63.6)	6 (27.3)	2 (9.1)	

Table 2: Comorbid DSM-IV Axis I and Axis II classification at intake		
	Count	%
Axis I disorders		
Depressive/ dysthymic	12	54.5
Alcohol/ substance abuse	6	27.3
Anxiety	6	22.7
Axis II Classification		
Personality disorder NOS	3	13.6
Narcissistic PD	1	4.5
Avoidant features	4	18.2
Schizoid features	2	9.0
Dependent features	1	4.5
Borderline features	1	4.5
Histrionic features	1	4.5
Antisocial features	1	4.5

Note. NOS = not otherwise specified; PD = personality disorder.

At intake 19 veterans met diagnostic criteria for a comorbid axis I disorder and 4 patients also met criteria for a comorbid personality disorder. Half of all patients met diagnostic criteria for a comorbid mood (depressive or dysthymic) disorder. Other comorbid disorders at intake were anxiety disorders (generalized anxiety disorder, social phobia, panic disorder) and substance or alcohol abuse. Also, many patients presented with typical personality features, avoidant features being the most often observed. Table 2 presents the comorbid DSM-IV (APA, 1994) axis I and axis II classification at intake.

Data considerations

Before analyzing pre- and post treatment data, individual scores were explored. Closer examination of individual test scores revealed that two veterans displayed scores on the MMPI-2 validity scale F which raised serious questions to the validity of their test scores. They both scored above T = 109 after treatment (F = 115 and F = 119 respectively) which should be considered as an indication of symptom over-reporting or a so called 'fake bad' response set (Derksen et al., 1997). Therefore the analyses were run twice: first with these cases included, and again after removing the data for these two.

Treatment effects

MANOVA for repeated measures showed significant pre- to post-treatment differences on MMPI-2 scales, $F_{(14,8)} = 4.31$, $p = .022$, as well as SCL-90, SIP and UCL scales, $F_{(7,15)} = 7.46$, $p < .001$. Subsequent paired t-test showed significant differences on MMPI-2 scales 2 (D) and Dep, all PTSD scales of the SIP, total score on the SCL-90, and active, social and avoidant coping scales of the UCL. After removing the aforementioned two participants, MANOVA's remained significant for scores on MMPI-2 scales, $F_{(14,6)} = 5.79$, $p = .02$, as well as for SCL-90, UCL and SIP scores, $F_{(7,13)} = 7.03$, $p = .001$. However, subsequent paired *t*-tests now revealed significant ($p < .05$) differences on MMPI-2 clinical scales 1 (Hs), 2 (D), 3 (Hy), 7 (Pt), 8 (Sc), content scales Anx, Dep, Lse and Wrk, all three UCL and SIP scales, and total score on the SCL-90. These results are displayed in Table 3.

As can be seen in Table 3, the SIP scales showed significant ($p < .05$) differences between assessments on all scales, revealing improvement in all PTSD symptom clusters. UCL scores showed an increase in reported use of active and social coping strategies and a decrease in avoidant coping. A significant decrease ($p < .05$) was achieved in total score on the SCL-90 showing a reduction in overall levels of reported distress. Post-treatment improvement was found in scores on the MMPI-2 scales 1 (Hs), 2 (D), 3 (Hy), 7 (Pt) and 8 (Sc) as well on scales measuring anxiety (Anx), depressive symptoms (Dep), low self-esteem (Lse) and work-related problems (Wrk). Moreover, even though the pre- to post treatment differences were not significant, scores on clinical scale 9 (Ma) shifted towards the clinical range after treatment. As low scores on this scale are often found in combination with high scores on scale 2 (D), measuring depressive features, the slight increase on scale 9 (Ma) after treatment is in agreement with the reduction in depressive symptoms observed on scales 2 (D) en Dep. Raw scores on MMPI-2 scale Pk, which was (also) designed to assess PTSD symptoms, were not significantly lower after treatment. Post-treatment mean scores on the scales 2(D), 3 (Hy), 7 (Pt), 8 (Sc), 9 (Ma), Anx and Wrk were (still) above T = 65, therefore it must be concluded that clinically significant symptoms were still present.

Discussion

This study demonstrated that an exposure-based multimodal outpatient group treatment approach for veterans with PTSD was effective in reducing a number of PTSD and associated symptoms. Pre- to post-treatment comparison showed an overall decrease in reported psychopathology, especially with respect to PTSD, depressive, anxiety and somatic symptoms. Also, the reported levels of work-related problems were reduced post treatment, patients' coping strategies had improved and improvements were made with respect to self-esteem. Inclination to use passive and avoidant coping styles was reduced after treatment whereas active coping increased. This finding is of particular interest as previous research points out that avoidance coping is closely related to levels of distress (Brown, Mulhern, & Joseph, 2002).

The observed differences on MMPI-2 clinical scales 1 (Hs), 2 (D), 3 (Hy), 7(Pt) and 8 (Sc) suggest an overall improvement in personality functioning and a reduction in depressive, somatic and anxiety symptoms as well as diminished somatic concern and fatigue, lower levels of reported (social) alienation and reduced thinking and concentration difficulties. Studies among PTSD patients usually show elevations on many of the clinical scales (Frueh, Hamner, Cahill, Gold, & Hamlin, 2000), therefore, the fact that lower scores on many of the clinical scales were observed after treatment, underlines the notion that these scales measure traits and symptoms that are associated with trauma related psychopathology. Similarly, the observed reduction in scores on scales 2 (D), 7 (Pt) and 8 (Sc), which have previously been reported to be associated with PTSD symptoms (Lyons & Wheeler-Cox, 1999), suggests that these scales indeed represented problems associated with PTSD and that therapy effectively addressed and ameliorated these problems. These results are especially noteworthy because they were obtained in a relatively small sample.

The results obtained with respect to PTSD levels seem to be similar to the results published by Frueh et al. (1996) and somewhat more favourable than the data reported by Schnurr et al. (2003). Like Frueh et al., we observed improvement with respect to anxiety symptoms even though clinical significant levels of anxiety symptoms were still present after treatment. Frueh et al. only found effects on clinician rated instruments however, whereas we used self-report measures exclusively. The present study showed improvement with respect to depressive symptoms as measured by MMPI-2 clinical scale 2 (D) and content scale DEP. This is in contrast to results by Frueh and colleagues who found no significant effects on self-report measures like the Beck Depression Inventory (BDI). Although the MMPI-2 and BDI are dif-

ferent lists, they tap on the same symptoms as is evidenced by previously studies reporting moderate correlations between tests (e.g., Strassberg & Russell, 2000). Therefore, the program described in the present study appears to have yielded better results with respect to depressive symptoms. Schnurr et al. (2003) did not report the effect of treatment on depressive problems, or social functioning and neither of the studies examined self-esteem. As previous studies have found low esteem to be related to PTSD (Adams & Boscarino, 2006; Piotrkowski & Brannen, 2002) the increase in self-esteem in the present sample may represent an increase in personal resilience (Agaibi & Wilson, 2005). This seems to be in accordance with the observed improvement in self-reported coping behaviour.

Table 3: Changes in test-scores from pre- to post-treament assessment.

		T0		T1			
Measure	Scale	Mean	*SD*	Mean	*SD*	*t**	*p*
SIP	Re-experiencing	16.95	4.24	13.25	4.09	3.103	.006
	Avoidance	24.30	4.66	19.50	6.07	3.499	.002
	Hyper-arousal	22.20	3.32	17.85	5.20	3.829	.001
SCL-90	Total score	232.80	55.07	161.45	42.13	5.448	<.001
UCL	Active coping	16.05	5.40	18.10	4.13	-2.115	.048
	Avoidant coping	19.60	4.41	17.45	4.25	3.916	.001
	Social support	9.75	3.43	13.00	3.43	-3.901	.001
MMPI-2	1 (Hs)	68.90	12.52	62.65	13.80	2.285	.034
	2 (D)	79.20	13.84	69.35	16.76	3.238	.004
	3 (Hy)	74.35	13.75	66.30	14.81	2.928	.009
	4 (Pd)	68.65	11.83	64.15	14.58	1.572	.133
	6 (Pa)	66.70	11.26	61.85	12.30	1.757	.095
	7 (Pt)	75.70	13.36	68.50	11.04	2.446	.024
	8 (Sc)	76.25	8.78	69.35	12.09	2.374	.028
	9 (Ma)	61.95	14.85	65.40	15.67	-0.992	.334
	0 (Si)	61.85	12.23	57.55	10.95	1.829	.083
	Anx	74.90	10.76	66.00	13.03	2.768	.012
	Dep	77.30	11.21	62.60	14.04	4.647	<.001
	Lse	62.85	17.63	55.55	12.30	2.458	.024
	Sod	63.75	11.85	60.65	10.58	1.424	.171
	Wrk	71.00	10.04	65.40	12.03	2.126	.047
	Pk (rs)	31.40	16.13	25.75	21.06	1.899	.073

*Note. * DF = 19; T0 = before treatment; T1= after treatment; 1 (Hs) = Hypochondriasis; 2 (D) = Depression; 3 (Hy)= Hysteria; 4 (Pd) = Psychopathic Deviate; 6 (Pa) = Paranoia; 7 (Pt) = Psychastenia; 8 (Sc) = Schizophrenia; 9 (Ma) = Hypomania; 0 (Si) = Social introversion; Anx = Anxiety; Dep = Depression; Lse = Low self-esteem; Sod = Social discomfort; Wrk = Work interference; Pk = Post Traumatic Stress Disorder- Keane; rs = raw score.*

Although the present study indicates that the program was effective in reducing PTSD and associated symptoms for the present sample, it also provides additional evidence that chronic (combat-related) PTSD is an especially difficult disorder to treat (Bradley et al., 2005; Brom et al., 1989; Lyons & Wheeler-Cox, 1999). Despite clinical significant improvement, most veterans still presented with symptom levels that were above those observed in healthy adults. So, even though the program resulted in marked improvement, clinically significant levels of psychopathology were still present after treatment. Although this might have been expected, it is important to note that even an intensive multimodal treatment program, in which veterans participated for close to two years, was not successful in completely resolving post-traumatic morbidity. This raises questions as to whether complete recovery is attainable and whether similar results can be obtained within a shorter time-frame. It is possible that the therapeutic prognosis for (Dutch) UN-peacekeeping veterans with chronic PTSD may be similar to that for Vietnam veterans with PTSD; that improvement can be realized but complete recovery is not likely to be attained. Although this seems to be in accordance with results by Forbes et al. (2005), such a conclusion cannot be made at face value of the present study.

One factor that needs to be mentioned here, as it might have influenced the assessments and thus limits the validity of any inferences on therapeutic efficacy and prognosis, is the possible effect of symptom over-reporting (Frueh et al., 2000). In the US, the phenomenon of symptom over-reporting as a consequence of compensation seeking for instance, has received considerable attention (McNally, 2006; Rosen, 2006). As a result, DSM-IV cautions clinicians to rule out malingering "in situations in which financial remuneration, benefit eligibility, and forensic determinations play a role" (APA, 1994, p.467). Veterans suffering from PTSD following their deployment are eligible for compensation in the Netherlands. Therefore the possible effects of malingering may have influenced (post-treatment) assessments. Even though we excluded two veterans from the analyses because their MMPI-2 scores were indicative of a possible fake-bad response style, we cannot rule out the possibility that post-treatment scores were inflated to some extent as a result of secondary gains.

The design of the present study does not permit inferences or definite conclusions about the therapeutic effects of the various 'ingredients' or the added effect thereof to treatment outcome. However, the observed improvement with respect to overall levels of psychopathology, coping and family and professional functioning, point towards an overall effect which is superior to what would be expected with exposure therapy alone.

Small sample size and lack of a control group limit the generalizability of the present results and make it unclear whether the observed changes were due to therapy, passage of time or random effects. Another limitation to the representativeness is that the reported data pertain to a selected subset of veterans with chronic PTSD. For one thing, the rates of comorbid disorders appear to have been lower in the present sample than usually observed (Kessler et al., 1995). Therefore, the observed effects cannot be generalized to therapeutic prognosis for all veterans with PTSD. On the other hand, Table 2 showed that comorbid axis I disorders were common and that many patients also displayed problems in personality functioning. Therefore the therapeutic effects cannot be extended to a prognosis for veterans with acute (vs. chronic) PTSD and without comorbid disorders. Finally, only self-report measures were used. By definition, these are more vulnerable to response bias. Although we used validated and psychometrically sound questionnaires, additional research is needed to examine whether pre- to post treatment differences would also be present in clinician rated instruments. Additionally, for future studies it would be interesting to combine self-report and clinician rated assessment tools with biological parameters associated with PTSD.

The descriptive nature of the present study does not render it without value. On the con-

trary, scientific study of therapy results is of great importance in evidence based medicine, and to establish whether a certain program 'works', that is, that it will lead to symptom reduction and improved quality of life. Although a randomized controlled trial (RCT) would provide more substantial evidence for the efficacy of the program, RCT's are not the only way of establishing whether treatment works. As Seligman (1995) pointed out in his article describing the differences between effectiveness and efficacy studies, there are a number of issues that may be overlooked in controlled trials, the most salient being that 1) in clinical practice treatment usually is not of fixed duration; 2) treatment may be 'self-correcting, i.e., if one technique fails another may be adopted; 3) patients usually present with multiple complaints, and psychotherapy is usually concerned with improving general functioning rather than specific symptoms. Although an RCT would provide evidence for the efficacy of certain compounds of a treatment and the mechanisms involved, an alternative approach is needed to investigate the effectiveness of treatment as it occurs. The present study was aimed at exactly that purpose: to evaluate the program as a whole and investigate whether it was beneficial for the participants. Moreover, the results showed that improvement can be obtained in a sample of veterans suffering from chronic PTSD. Further research is needed to address the aforementioned limitations, to 'dismantle' the program and to assess the effects of the different therapeutic ingredients on state and trait-related parameters. Additionally, it is recommended to assess the effects of treatment on other relevant parameters e.g., concentration and memory problems, impulsive behaviour, employment and quality of life.

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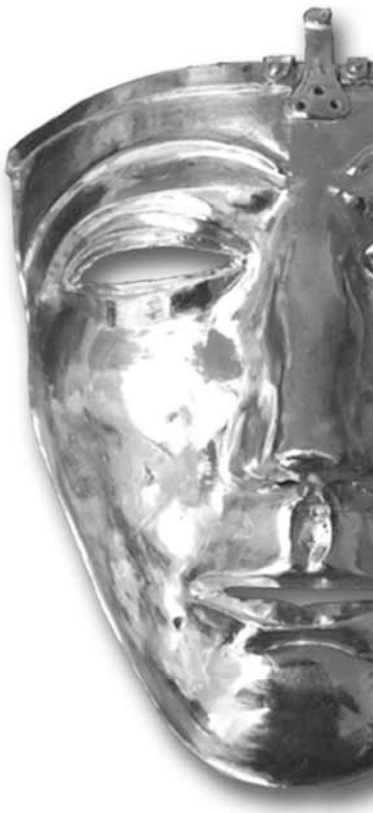
Section 3

Summary & discussion



Chapter 8

Summary, discussion and concluding remarks

Introduction

In the introduction to this dissertation we expressed the need for a more thorough understanding of individual differences in vulnerability and resilience to trauma in military personnel. Insight into individual differences would be particularly relevant for this group of people who – by virtue of their profession - are at increased risk for exposure to prolonged stress and traumatic events. More insight into how personality facilitates or hinders the adaptation to stressful and (potentially) traumatic experiences could be used to improve prevention as well as treatment for this group of professionals.

In the previous chapters we combined different approaches to examine the relationship between personality and PTSD. Specifically, the studies in chapters 2, 3, 4, and 5 examined pathways through which personality moderates risk and resilience to PTSD, whereas in chapters 6 and 7 clinical symptoms and personality profiles were investigated of former peacekeepers with mental problems following deployment. This last chapter summarizes and integrates the findings from the studies described in the previous chapters and highlights the scientific, clinical and military relevance of the results.

Summary of major findings

The aim of this dissertation was to examine individual differences in vulnerability for PTSD from a personality psychology perspective, and to adopt various approaches to examine the relationship between personality and adaptation to stress and trauma in Dutch soldiers and veterans. In other words, the aim was to assert how personality is related to vulnerability or resilience to post-deployment morbidity in the Dutch military.

Chapter 2 reviewed the literature on the relationship between personality, psychotrauma and PTSD, focussing specifically on trait neuroticism. There are three pathways through which personality (neuroticism) can affect the aetiology of PTSD. First, it can act as a risk factor for increased exposure to potential traumatic events (Breslau, Davis, & Andreski, 1995). Second, it reflects an intrinsic vulnerability for PTSD (as well as other mental disorders), and third it may have indirect effects on posttraumatic morbidity as it can affect the way stressors are perceived as well as the way an individual deals or copes with them. At the same time however, exposure to trauma can increase neuroticism levels. Additionally, the predictive value of neuroticism is reduced when controlling for (prior) levels of distress or arousal. This suggests that the relationship between neuroticism and PTSD (as well as other 'distress-disorders') is tautological. The dearth of papers that compare pre- to post-trauma neuroticism ratings warrants further exploration. Research aimed at 'deconstructing' trait neuroticism in order to investigate how specific aspects of this trait relate to psychopathology is recommended.

Chapter 3 focussed on the association between self-reported childhood trauma and adult personality. Previous studies have linked childhood trauma to adult psychopathology, and there is evidence that early trauma is related to (adult) PTSD in military samples. The aim of the study in this chapter was to examine whether the increased risk of adult psychopathology associated with childhood trauma, could be explained from a personality perspective, and whether personality could mediate the relationship between exposure to early trauma and adult psychopathology. Results showed that self-reported exposure to childhood emotional abuse was related to lower scores on character dimensions of cooperativeness and self-directedness. These personality traits are associated with self-motivational and interpersonal difficulties. Moreover, low self-directedness and cooperativeness have previously been associated with increased risk of depressive and anxiety disorders (Matsudaira & Kitamura, 2006), avoidant coping and reduced social support (Ball, Smolin, & Shekhar, 2002; Duijsens & Spinhoven, 2002). Therefore, these results illustrate that early trauma may become ingrained in

adult personality, thereby affecting the ability to effectively deal with stressors and increasing the risk of psychopathology.

In *chapter 4* we explored the relationship between personality and the cortisol awakening response (CAR), a marker of hypothalamic-pituitary-adrenal (HPA) axis functioning and adrenocortical reactivity. The fact that HPA reactivity is dependent on psychological factors including personality is well established. Moreover, both HPA-axis reactivity (Charlton & Ferrier, 1989; De Kloet et al., 2006; Heim, Newport, Bonsall, Miller, & Nemeroff, 2001; Yehuda, 2002) and harm avoidance (Cloninger, Svrakic, & Przybeck, 2006; Gil, 2005) have been implicated in the development of illnesses and in the aetiology of stress-related disorders such as depression and PTSD. Our results showed that low harm avoidance was related to an attenuated awakening cortisol curve and that both harm avoidance and self-directedness were associated with the mean cortisol increase after awakening.

Chapter 5 described the results from a study investigating dispositional risk and resilience factors in relation to posttraumatic stress symptoms in Dutch veterans who participated in peacekeeping operations in Lebanon. The aim of this study was to examine whether previously identified dispositional resilience factors (optimism, hardiness and locus of control) predicted coping, social support and PTSD, *independently* of the effects of neuroticism in a path analysis. Results showed that neuroticism, trauma exposure, optimism and social support were directly related to posttraumatic stress symptoms. Neuroticism accounted for the largest amount of explained variance in posttraumatic stress symptoms. Dispositional optimism had a direct effect on posttraumatic stress symptoms in the opposite direction. Locus of control did not significantly add to the explained variance in any of the outcome measures. Both hardiness and neuroticism were related to coping and social support. Although the results provided evidence for a relationship between personality and coping, this study failed to support a relationship between coping and PTSD.

The aim of *chapter 6* was to examine the association between PTSD and MMPI-2 scores and to provide a description of symptom patterns and personality scores of Dutch veterans who were deployed to Lebanon, the Balkans or other regions of conflict and who applied for treatment. Results showed that veterans who screened positive for PTSD reported diffuse levels of psychopathology. They showed clinically significant scores on seven of ten MMPI-2 clinical scales and produced MMPI-2 profile configurations that were similar to what has been reported in Vietnam veterans with PTSD (Frueh, Hamner, Cahill, Gold, & Hamlin, 2000). Depressive, somatic and anxiety symptoms were present, as well as somatic concern and fatigue, (social) alienation, concentration difficulties, impulsiveness, vigilance and anger. Nonetheless, consistent with the notion that many peacekeeping operations are associated with lower stressor intensity than traditional combat (Dirkzwager, Bramsen, & Van der Ploeg, 2003), the Dutch veterans presented less severe levels of psychopathology compared to Vietnam veterans.

In *chapter 7* we evaluated the outcome of an intensive long-term group treatment program for PTSD on symptom patterns, coping and personality. Veterans participated in a multimodal exposure-based group treatment program that consisted of CBT, case-management, psycho-education, creative arts therapy, psychomotor therapy, psychodrama and socio-therapy, which were provided one day a week for a period of approximately 21 months. Most of the veterans described in this study also met DSM-IV criteria for comorbid disorders including depressive and anxiety disorders, substance or alcohol abuse, or personality problems. Results demonstrated that those veterans who completed treatment showed clinical improvement after treatment. Symptom levels were reduced and an increase in self-esteem and active coping and a decrease in avoidant coping were observed after treatment. As significant improvement

was observed on several parameters, the program may have successfully addressed a broad range of problems frequently reported by veterans with complex and chronic PTSD. Nevertheless, veterans still presented clinically significant symptomatology at the end of treatment. This underscores previous findings that chronic combat-related PTSD is a difficult disorder to treat (Bradley, Greene, Russ, Dutra, & Westen, 2005).

Personality, trauma & psychopathology

The association between personality and trauma related psychopathology is complex. In chapter 2 we presented four different (and potentially not mutually exclusive) conceptual models to describe this association. These included: vulnerability models (e.g., diathesis-stress models), where personality was proposed to play a causal role in the development of the disorder; pathoplasty models, in which personality (also) affected the course and maintenance of the disorder; scar models, which postulated that the presence of a disorder can cause changes in personality functioning; and spectrum models, in which personality and stress-related disorders are viewed as being part of the same continuum and reflecting the same underlying processes, so that neither can be viewed as affecting or causing the other (see also Clark, Watson, & Mineka, 1994).

Lilenfeld et al. (2006) described a number of ways to test the validity of these models. In short, the authors concluded that the best way to test vulnerability models as well as scar and pathoplasty models, would (obviously) be a prospective design, since this would be the only valid way to establish temporal order and causality. Treatment studies could provide some insight into scar effects although they foreclose inferences on the effects of premorbid factors. Cross-sectional studies are of limited use to evaluate the relationship between personality and psychopathology, but they can be used to examine the pathoplasty model, and finally, family studies (e.g., sibling or twin study designs) could be used to examine vulnerability, scar and common-cause models (Lilenfeld et al., 2006, pp. 303, 304).

The studies in this dissertation were not specifically aimed at empirically testing these models, and we are unable to provide any evidence for a spectrum model. Nonetheless, a number of observations can be made with respect to validity of the other models. First, results from chapter 3 provide some support for a vulnerability model concerning character traits self-directedness and cooperativeness. Soldiers who reported more childhood trauma scored lower on these traits. Since results were obtained from a healthy sample, we can assume that they did not meet PTSD criteria. Therefore, these results cannot be explained with the scar hypothesis. Furthermore, since other studies have shown that low scores on self-directedness and cooperativeness are associated with a range of interpersonal problems and mental disorders, including post-traumatic morbidity (e.g., Ghazinour, Richter, & Eisemann, 2003), it would seem safe to assume that these traits reflect a vulnerability factor.

Second, results from chapter 5 highlighted potential pathoplasty effects of the personality traits neuroticism, optimism and hardiness on posttraumatic stress symptoms. The data presented in this study suggest that these traits may affect the course of PTSD as individuals high on neuroticism reported more (severe) PTSD symptoms whereas high optimism was inversely related to symptoms severity. Hardiness was related to PTSD symptoms through social support which suggest that this trait may facilitate coping with stress-related symptoms by increasing the ability to receive support from others.

Finally, the clinical study in chapter 7 showed that MMPI-2 scores of veterans who received group treatment displayed a personality profile that was similar to that of treatment seeking veterans who screened positive for PTSD in chapter 6. It appears unlikely that the MMPI-2 scale elevations in the treatment group were caused by exposure to trauma, because a differ-

ent MMPI-2 profile was observed in veterans that had been deployed but who did not meet PTSD criteria. After treatment, mean MMPI-2 scores were lower, which shows that symptom severity was reduced. However, mean scores remained moderately elevated after treatment. Since moderate elevations on MMPI-2 clinical scales can be interpreted as reflecting personality functioning, these results demonstrate that even an intensive treatment program that continued for close to two years did not substantially alter personality. The only personality changes that could be observed were increases in self-esteem. Therefore, the MMPI-2 scores as observed in chapter 7 either reflected a vulnerable personality profile, or they represented the scar effects of PTSD.

A coping perspective

As noted before, Allport (1937, p. 48) defined personality as the complex and 'dynamic organization within the individual of those psychophysical systems that determine his unique adjustment to his environment'. The coping model that was presented in the first chapter demonstrated that personality can affect (posttraumatic) adjustment at various stages. The coping process is initiated when a stressor is perceived as harmful or threatening. On the one hand, the perception of a threat is usually accompanied by negative emotions, high levels of distress and increased activity of biological systems underlying the stress-response. On the other hand, certain personality traits (like neuroticism and harm avoidance), that are characterized by high baseline levels of distress, increased stress-reactivity and/or prototypical negative emotions, may affect the way potential stressors are appraised. Individuals high on these traits may perceive more situations as threatening or taxing available resources. Indeed, research has demonstrated that soldiers high on neuroticism appraise more situations as stressful or threatening (e.g., Engelhard & Van den Hout, 2007).

After the appraisal of a stressor, the initial coping efforts are often aimed at regulating emotional responses but these efforts may interfere with more instrumental coping behaviours (Folkman & Moskowitz, 2004). As certain personality traits are characterized by high levels of distress and negative emotions (i.e., neuroticism or harm avoidance), individuals high on these traits may be more inclined to regulate these emotions rather than to effectively engage the situation at hand. Accordingly, in chapter 5 we found evidence that neuroticism is positively related to avoidant coping and inversely related to active, problem oriented coping. On the other hand, personality traits that are associated with the appraisal of stressors in terms of challenging and controllable rather than threatening, like hardiness, were shown to be positively related to active coping and negatively to avoidance.

Personality was also demonstrated to be a predictor of available coping resources: both neuroticism and hardiness predicted (perceived) social support. Also, results from the group-treatment study in chapter 7 provided some evidence for the notion that successful coping efforts may lead to positive emotions (Folkman & Moskowitz, 2004) seeing that increases in effective coping style (problem oriented versus avoidance), parallel to a reduction in PTSD symptoms, were related to increases in levels of self-esteem after treatment.

Finally, we demonstrated that personality is directly related to self-reported (mental) health problems and well-being as both optimism and neuroticism were shown to be direct and independent predictors of posttraumatic stress symptoms. Apparently, dispositional optimism can act as a buffer to ameliorate symptom severity whereas neuroticism increases severity of reported symptomatology.

Taken together, the studies in this dissertation illustrate how personality is involved in all the stages of the coping process as depicted in Figure 1 in the first chapter. Although Lazarus and Folkman (1984) emphasized the importance of the context - of stressor characteristics

and situational aspects - in determining subsequent coping efforts and outcome, the previous chapters have shown that personality may at least be equally important in determining post-traumatic adjustment. It is related to appraisal, available resources, coping skills and behavior, and ultimately the outcome of the coping process, where personality itself may be subject to change following exposure to extreme and/ or prolonged stress.

Risk or resilience?

To explain individual differences in susceptibility for stress-related disorders, both risk and resilience factors were examined in this dissertation. According to Aigaibi and Wilson (2005), these are closely related and may reflect twin sides of adaptation to trauma. However, whether they actually are at opposite ends of the same continuum is subject to debate. Additionally, the mechanisms that are involved are not always clear. Our studies showed that vulnerability and resilience are indeed closely related but we conclude that with respect to personality aspects, there is more support for a multi-dimensional model of vulnerability and resilience than for a unidimensional approach.

A unidimensional approach would implicate that the absence of risk or vulnerability factors reflects increased resilience. Low neuroticism would then compose a resilience factor, and as childhood trauma has been shown to increase the risk of adult psychopathology, the absence of childhood trauma could then also be perceived as a marker of increased resilience. Similarly, in a unidimensional approach, the absence of resilience factors would reflect increased vulnerability for PTSD. In this respect, individuals low on hardiness might be at increased risk for posttraumatic morbidity because they perceive more situations as stressful and taxing rather than as challenging. They would be less committed to effectively deal with the situation at hand, and more inclined to use avoidant coping strategies for instance, and may perceive less social support. Also, individuals low on dispositional optimism could then be expected to report more (severe) distress, because they are unable to maintain hope about the future.

We found some evidence in favour of a unidimensional view of risk and resilience associated with personality. As early adverse experiences were related to low self-directedness and cooperativeness, a unidimensional approach to risk and resilience factors would dictate that high self-directedness and high cooperativeness are related to enhanced resilience. This is in agreement with results from Ghazinour et al. (2003) who linked high scores on these character dimensions to enhanced resilience, which they operationally defined as low levels of self-reported depressive symptoms in a high trauma exposure subgroup.

It is unclear how the cortisol awakening response is related to vulnerability or resilience to stress-related disorders. In chapter 3 both harm avoidance and self-directedness were related to a greater cortisol increase after awakening. As high harm avoidance is associated with various distress disorders, low harm avoidance might reflect enhanced resilience. Similarly, high self-directedness has been associated with enhanced resilience (Ghazinour et al., 2003), whereas low self-directedness is indicative of increased vulnerability for mental disorders (Matsudaira & Kitamura, 2006). Thus, personality traits associated with resilience (high self-directedness) as well as increased vulnerability (high harm avoidance) for mental disorders were related to a similar phenomenon. Moreover, whereas an attenuated CAR has previously been associated with disorders like PTSD (e.g., Wessa, Rohleder, Kirschbaum, & Flor, 2006), an increased CAR has been observed in individuals suffering from chronic stress (Schulz, Kirschbaum, Pruessner, & Hellhammer, 1998). Therefore, whether a flattened ACR should be perceived as a risk or resilience factor (or both) for stress-related disorders, and whether the hypothesized buffering effects of low harm avoidance and/ or high self-directedness can be explained through this neurobiological system, is uncertain.

More importantly, although a unidimensional approach can explain the relationship between vulnerability and resilience when the focus is on a specific personality domain, we have demonstrated that resilience cannot be viewed as being synonymous with the absence of risk or vulnerability factors when multiple determinants are examined simultaneously. In chapter five, neuroticism accounted for the largest proportion of explained variance in PTSD scores in peacekeeping veterans deployed to Lebanon, relative to stressor characteristics, coping, social support, dispositional optimism and hardiness. Yet, results also showed that optimism and hardiness had a unique and distinct relationship to coping, social support and PTSD symptoms, independent of neuroticism. Therefore we conclude that resilience is not necessarily the same as the absence of risk factors. Personality may increase resilience through different pathways, parallel to and despite of the presence of risk factors. This corresponds to the notion of resilience as described by Bonanno (2004), who observed that it would seem likely that the absence of previously observed risk factors might be related to enhanced resilience, but that there are multiple ways to 'maintain equilibrium following highly aversive events' (p. 25). He mentioned a number of distinct resilience factors that included hardiness, self-enhancement and positive emotions. As such, the notion that resilience factors may operate parallel to and independent of risk factors also fits well within the view of the 'positive psychology' paradigm where positive traits are proposed to co-exist with negative or maladaptive traits (Seligman, Steen, Park, & Peterson, 2005).

Temperament & character

As noted before, personality can have both direct and indirect effects on trauma-related psychopathology. The vulnerability for stress and trauma related disorders associated with personality may be due to genetic (i.e., temperament) as well as developmental factors (character). Personality may reflect an inborn sensitivity - or diathesis - for stress-related disorders and parallel to this, genetic aspects of personality can increase the risk of exposure to trauma. Stein and colleagues proposed that genetic factors can affect the risk of trauma exposure, and that the genes that have an effect on exposure to trauma may also influence susceptibility to PTSD (Stein, Jang, Taylor, Vernon, & Livesley, 2002). Research has demonstrated that neuroticism is about 50% heritable (Floderus-Myrhed, Pedersen, & Rasmuson, 1980). Similarly, Keller et al. (2005) demonstrated that genetic effects account for about 50% of phenotypic variation in Cloninger's temperament dimensions (e.g., harm avoidance). As harm avoidance and neuroticism have both been demonstrated to increase the risk of PTSD and other 'distress' disorders, the increased vulnerability does indeed appear to be subject to genetic, inborn factors. Furthermore, this dissertation demonstrated a relationship between temperament and a marker of HPA-axis reactivity that was previously shown to be moderately heritable, i.e. the cortisol awakening response (Wust, Federenko, Hellhammer, & Kirschbaum, 2000). Temperamental aspects that are related to harm avoidance and neuroticism may therefore compose a vulnerable endophenotype for stress-related disorders. It would be interesting to assess whether similar genetic factors are implicated in increased risk for exposure as well as for enhanced vulnerability like Stein et al. (2002) proposed.

Parallel to temperamental factors, we illustrated that developmental experiences can affect personality aspects that are associated with increased susceptibility for stress-related psychopathology. We found an association between (self-reported) childhood emotional neglect and the character traits self-directedness and cooperativeness. A previous study in traumatized Iranian refugees demonstrated that self-directedness and cooperatives were inversely related to psychopathological symptoms, and that both traits were moderately to strongly related to effective coping, social integration and social support (Ghazinour et al., 2003). Self-direct-

edness, in particular, was shown to be strongly related to psychopathology (inversely) and coping resources. This suggests that individuals high on self-directedness and cooperativeness may be more proficient at dealing with adverse events and stress. In contrast, low scores may be more vulnerable to the adverse effects of (adult) trauma because they experience more problems in receiving social support and because they may have more problems in effectively coping with the stressors they encounter. Therefore, the association between childhood neglect and adult personality does not only illustrate how early experiences can shape adult personality but it also shows why victims of emotional neglect may be more vulnerable to adult posttraumatic morbidity.

Strengths and limitations

The studies in this dissertation have shown that personality is involved in the aetiology of deployment-related psychopathology through multiple pathways. A strong point of this dissertation was that it simultaneously examined how personality is related to vulnerability and resilience. As such it addressed an important limitation in the extant body of research on risk and resilience factors for stress-related disorders, namely that too often the focus is on risk factors exclusively (Bonanno, 2004; Kleber, 2007). Additionally, we examined personality aspects in both clinical and healthy samples and we used diverse measures i.e., self-report questionnaires and 'biological data'. Therefore the results can be used to further the understanding of adaptation to extreme events in military samples and are not limited to patients suffering from PTSD. Also, the results are not only applicable to PTSD patients or healthy subjects exclusively. Since we examined personality aspects in a rather homogeneous population the results can be readily applied to this particular population at risk for trauma-related psychopathology.

We used multiple scales to assess personality. This facilitates comparison to existing literature and provides a broad scope on personality in relation to trauma. For instance, in the clinical studies in chapters 6 and 7, the MMPI-2 was the main outcome measure. The MMPI-2 remains a commonly used psychological test in mental healthcare practice; thus the results of these chapters provide information that is readily usable for mental healthcare professionals.

Unfortunately however, the cross-sectional nature of the studies does not provide evidence for a causal relationship between personality, trauma exposure and psychopathology. Moreover, in some instances the data may have been biased or flawed due to the study designs and methods that were adopted. Although each chapter also mentions the specific limitations of the particular study at hand, two important sources of bias need to be mentioned here. First of all, the psychological data were based on self-report measures exclusively. Even though we used well validated questionnaires as much as possible, the use of self-report data exclusively may have introduced a response bias. Second, recall bias and state effects may have affected the retrospective ratings of adverse childhood experiences and deployment stressors in chapters 3 and 5 respectively.

The lack of prospective and longitudinal data constitutes an important drawback for the inferences that can be made from the results in the previous chapters. Therefore, additional prospective and longitudinal research is necessary to replicate the findings and test the hypotheses that were formulated. Further research is needed to verify whether the results from the studies in this thesis are representative for other (trauma) populations. For one thing, the results cannot be extended to female samples as all studies focused on exclusively male samples. Second, although it would be expected that the findings can be forwarded to other (male) populations, we cannot be certain.

Nonetheless, the studies in this dissertation add to the existing body of knowledge by fo-

cussing more closely on important aetiological aspects of posttraumatic morbidity. We set out to examine the pathways that may underlie the association between personality and trauma-related psychopathology, and have done just that. We highlighted developmental aspects, neurobiological factors and, most importantly, demonstrated how personality factors may be involved in different stages of the coping process.

Implications for treatment and prevention

For most soldiers, exposure to stress is an inevitable part of military life. Experiences during military deployment can shape individual development and can become deeply ingrained in personality. The relationship between deployment experiences and (mental) health is well documented (e.g., Bramsen, Dirkzwager, & Van der Ploeg, 1997; Dohrenwend et al., 2006; Kleber & Brom, 1989; Richardson, Naifeh, & Elhai, 2007; Stimpson, Thomas, Weightman, Dunstan, & Lewis, 2003). Exposure to specific events, combat and atrocities in particular, though not peacekeeping operations per se, increases the risk of mental disorders (Litz, King, King, Orsillo, & Friedman, 1997; Sareen et al., 2007). Chapters 6 and 7 illustrate that stressful and traumatic events during peacekeeping operations are related to diffuse and, in some cases, chronic psychological problems in Dutch soldiers. Even though the stressors during peace missions are typically different from traditional combat, the symptoms reported by treatment seeking Dutch peacekeepers were not dissimilar from those reported by soldiers after the Vietnam War. Moreover, these symptoms may be difficult to treat and results showed that veterans who received treatment for chronic PTSD symptoms, still reported clinically significant symptoms after treatment had ended. Therefore, the development of effective prevention and treatment programs is of paramount importance.

Selection & screening

The findings reported in this and previous chapters might lead one to conclude that it would not be wise for a soldier high on neuroticism or harm avoidance to be sent out on patrol in Afghanistan, where he or she would run the risk of encountering enemy troops, explosive devices and other threats. If so, would that make individuals high on harm avoidance or neuroticism unfit for military life? Obviously, if we could predict who would develop PTSD, we would be compelled to do so. However, asserting that personality can increase the risk of psychopathology is not the same as stating that individuals with a particular personality make-up will almost certainly develop PTSD after exposure to extreme stress.

Research into personality aspects in posttraumatic adjustment provides screening tools to determine who is at increased risk of developing psychopathological symptoms after military deployment. However, personality tests cannot be used to *determine* who will suffer and who will recover. There are many determinants of posttraumatic morbidity; personality is 'merely' one of them. Brewin et al. (2000) warned against attempting "to build a general vulnerability model for all cases of PTSD" (p. 756) because PTSD is a heterogeneous concept, and because the interplay between pre-trauma factors and trauma responses is unclear. Overall, the relative importance of identified risk factors can vary across trauma type and some risk factors may not be generalizable across situations or individuals. Moreover, PTSD is only one type of disorder than can arise after exposure to prolonged stress or a traumatic event. The influence of personality may be different in the aetiology of other stress-related disorders.

Furthermore, as duly observed by Russell (2000), the military requires both high and low harm avoidant individuals. Although high harm avoidant individuals may not be particularly well suited for combat functions, they can be expected to thrive in a peacetime army (Russell, 2000). So how can assessment of personality improve screening of military applicants?

A professional army will have other attributes and qualities than an army made up out of draftees. Where a conscript army can be expected to represent a more or less representative cross-section of the general (male) population, a professional army may be more affected by 'selection bias'. Nonetheless, the army can be expected to attract both high and low resilient individuals. As reported in chapter 3, the soldiers in that study scored lower on harm avoidance and higher on novelty seeking than males in general population. The pursuit of adventure and other sensation-seeking tendencies may have been part of the motivation for joining the army. As low harm avoidance may reflect enhanced resilience, it is possible that the army attracts people who are more resilient to stress.

Alternatively, it may be that army life is especially appealing to individuals who are looking for a 'sense of direction', i.e., those who are less adept at formulating and pursuing individual goals, as may be the case with low scorers on Cloninger's self-directedness dimension. Similarly, the social aspect and 'team spirit' of military life may be a reason for people to join the military. This may especially be so for individuals who, due to insufficient interpersonal skills for example (e.g., low cooperativeness), feel that they lack a sufficiently satisfying social network. As these characteristics have previously been associated with increased risk for various complaints and disorders, it may be that the military population comprises a (disproportionate) number of people at increased risk for disorder. Surprisingly few studies are available that report on personality traits or types in healthy military samples. So, it is unclear whether the military does indeed attract individuals with a certain personality type. Moreover, it remains uncertain whether the military population as a whole differs from 'the general' civilian population in terms of personality aspects related to risk/ resilience factors for stress-related disorders. Nonetheless, this information is vital in order for selection psychologists to improve assessment procedures. The studies in this dissertation have provided somewhat of a direction, but clearly more work remains to be done

As noted by Hunt et al. in 1944, for screening purposes, excluding soldiers or recruits who are not suitable for the military is as important as effectively selecting those who are. The present situation in the Netherlands is that the armed forces consist of a professional army and the waning number of military recruits highlights the need for assessment procedures that limit false negatives. In this respect, effective screening procedures may be as vital today, as they were during WW II (Hunt, Wittson, & Harris, 1944). Moreover, because military training can improve resilience, it is not necessary to exclude applicants who appear to be at increased risk for post-deployment morbidity at the time of selection assessments. Rather, it would be more fruitful to select individuals who possess particular personality traits for specific functions. For example, neurotic or harm avoidant traits are not necessarily unsuitable for non-combat positions but high hardiness and low harm avoidance might be more 'suitable' in combat units.

Additionally, when psychological screening is applied to identify recruits who display traits associated with increased vulnerability for (stress-related) mental disorders, it could prove more advantageous to try and assert which of those applicants could be expected to benefit from resilience training rather than exclude applicants on the basis of their personality make-up. In this respect, the aim of personality assessment should be twofold: to assert the degree of fit between personality make-up and military requirements on the one hand, and to select soldiers that would benefit most from training programs on the other.

Military training

Military training improves resilience of soldiers. By pre-exposing soldiers to events that resemble deployment stressors during training, they can be taught to maintain a sense of

control, to perceive deployment experiences as challenging rather than as a threat, and to maintain faith and confidence in their ability to effectively deal with the situation at hand. Therefore, if it is possible to mimic deployment experiences in virtual reality setting or in 'the field', exposure and/ or stress inoculation training (Meichenbaum, 1985) can be incorporated into military training and pre-deployment preparation to improve resilience. Obviously, this notion is not new to the military where combat drills and exercises are an important part of day-to-day routine.

Moreover, seeing that soldiers scored in the 'below average' range on temperament scale harm avoidance in chapter 3, which is related to enhanced resilience, it is likely that these personality scores - at least to some extent- reflected the effects of military training. So if soldiers are already effectively being trained to enhance their resilience, what is the added value of this dissertation to military life? The most important conclusion of this dissertation for military training purposes is that vulnerability factors do not necessarily foreclose beneficial effects of resilience training. That is, the fact that neuroticism and hardiness are independent constructs suggests that 'even' highly neurotic soldiers can benefit from resilience training. Hardiness is an important determinant of resilience and post-traumatic growth, and increasing personal hardiness may positively affect perceptions of benefits in military deployment (Britt, Adler, & Bartone, 2001). Studies have shown that hardiness can be improved through training in civilian samples (e.g., Maddi, Kahn, & Maddi, 1998), and it is possible that similar results can be achieved in military samples by developing comparable training programs specifically for soldiers. Additionally, resilience can be improved by improving leadership qualities. Bartone (2006) noted that high hardiness has been associated with improved performance in military leaders. He argued that high hardiness in leaders might improve resilience, unit cohesion and the ability to cope with the stress of military operations in their subordinates.

Second, as the military attracts individuals that differ with respect to their personality make-up and associated risk and resilience aspects, it would seem logical to try and adopt tailor-made training or coaching programs. For instance, stress-inoculation (i.e., combat training) programs would be especially beneficial for neurotic or anxious recruits as they would facilitate habituation to stressful circumstances whereas coaching programs aimed at goal-setting and self-motivational aspects would be especially relevant to recruits low on self-directedness.

Improving treatment programs

Individuals who perceive more situations as threatening, and who are inclined to avoid situations, are at increased risk for developing stress-related symptoms and PTSD. In this respect, neurotic and harm avoidant individuals can be expected to be more vulnerable for stress-related disorders. By contrast, personality traits like hardiness and optimism can improve resilience. The key feature of dispositional optimism is the ability to maintain a positive outlook on the future (Scheier & Carver, 1985) and hardiness reflects a sense of commitment and personal control combined with the ability to adapt to change (Kobasa, 1979). Similar to the recommendation for military training, resilience can be enhanced by cognitive and behavioural psychotherapeutic techniques that affect these (personality) domains.

Although, there are various treatments available for PTSD, the potential effects of these interventions on personality factors need to be assessed. The coping model as described above provides several handles to evaluate treatment programs and to fine-tune interventions aimed at improving and maintaining post-deployment health and well-being in soldiers. Moreover, combining personality research with research into neurobiological determinants of stress- and trauma related illnesses will aid in the evaluation of intervention (and prevention) programs.

For example, if cortisol response to stressors varies as a function of perceived stress controllability (Miller, Chen, & Zhou, 2007), then an intervention that is successful in enhancing sense of control, can be expected to result in changes in adrenocortical reactivity. Assessment of personality could be used to cross-validate any changes in adrenocortical reactivity, and could illustrate whether, for example, an increased cortisol response to awakening is paralleled by increases in self-esteem, hardiness and/ or self-directedness. Similarly, assessment of coping behaviour parallel to neuroendocrine parameters could substantiate the relationship between these factors and posttraumatic adjustment (see also Olff, Langeland, & Gersons, 2005).

Concluding remarks

The topic of this dissertation falls within a large body of data on adaptation to trauma. The detrimental effects of exposure to trauma on (mental) health is well established, and given the amount of research on trauma and PTSD that has accumulated over the years, one would think that few questions remain answered. However, in the course of this dissertation it became more and more clear that research into the relationship between trauma, personality and psychopathology is fragmented and that personality aspects have not received sufficient attention in the field of trauma research. Despite the vast body of literature on psychological trauma that has accrued over the years, only a limited number of studies have focused on personality. The necessity of systematic studies into the influence of personality in the aetiology of posttraumatic morbidity appears to be overlooked by most researchers. It seems as though the majority of studies that include personality measures only do so to control for a source of inter-individual variability rather than to further the knowledge on how personality may contribute to the aetiology of trauma-related psychopathology. Hardly any studies are available that were explicitly aimed at examining the conceptual models to describe the relationship between personality and post-traumatic morbidity and there is only a handful of studies that examined the long-term effects of stress and trauma on personality (e.g., Bramsen, Van der Ploeg, Van der Kamp, & Ader, 2002; Brom, Kleber, & Defares, 1989; Vogt, Rizvi, Shipherd, & Resick, 2008).

Most people would agree on the validity and utility of a person-situation perspective on posttraumatic adjustment. However, the predictive validity of the traumatic stressor is subject to debate (e.g., Rosen & Lilienfeld, 2008). As only a relatively small percentage of people develop PTSD after exposure to traumatic stressors whereas other people report PTSD symptoms after exposure to relatively mild stressors or life-events, individual characteristics may be better predictors of posttraumatic adjustment than stressor characteristics (Bowman, 1999). Moreover, as stated in DSM-IV-TR (APA, 2000) the A2 criterion states that exposure to a stressor must be accompanied by an intense emotional response. This suggests that an individual's emotional reaction is at the very least an important determinant of subsequent symptoms. As many personality traits are proposed to have a strong affective component, it is somewhat surprising that personality factors remain understudied in the field of trauma research.

For example, a Pubmed search that combines search terms 'PTSD' with 'personality' yields approximately 2600 hits, whereas PTSD alone returns almost 14.000 articles (by comparison: combining the search terms personality' and 'depression' will yield almost 10 times as many hits, i.e. little over 24.000 articles). Although a total number of 2600 is considerable, few empirical papers are available that systematically examine: a) the mechanisms that may account for any observed association between trauma and personality, and b) the interaction between trauma, personality, and posttraumatic morbidity in longitudinal designs. Additionally, assessment of personality traits might help to reduce heterogeneity in posttraumatic reactions by identifying subtypes of PTSD (e.g., Miller, 2003).

A possible cause for the relative 'unpopularity' of personality in trauma research is the potentially complex interplay between personality and disorder. Personality may be both cause and effect in adaptation to trauma and this constitutes an important methodological challenge. A second explanation for the lack of adequate personality research might be rooted in sentimental aspects of clinicians and researchers. Possibly, the history of the PTSD concept has given rise to lingering feelings that it would somehow be improper or 'not done' to suggest that PTSD patients are somehow responsible for the presence of the disorder.

Finally, the complexity of the personality construct may be one of reasons that personality has remained relatively understudied in trauma research. As trauma research has the opportunity to examine the development of a disorder like PTSD, clearly defined and causally linked to an identifiable stressor, why would we try to include a notoriously complex construct like personality into aetiological models? The answer should be: because personality is a crucial aspect of daily functioning, and an important determinant of well-being as well as post-traumatic adjustment. Nonetheless, the complexity of the personality construct warrants a clear conceptualization of the domains that are to be examined (e.g., cognition, perception, behaviour, affect) as well as the mechanisms that may be involved in the aetiology of mental disorders.

Without clear operationalization, personality will remain an ill-defined 'container' of individual characteristics and interpersonal variance in research methodology. For instance, in order for trait neuroticism to amount significantly to trauma research, operationalizations need to be forwarded that go beyond descriptions like 'generalized levels of distress'. The relevance of finding an association between increased risk of developing PTSD in individuals high on neuroticism depends entirely on whether or not neuroticism can be linked to known and identified aetiological aspects of PTSD (and other mental disorders), like reduced social support, dysregulation of the HPA-axis, or increased amygdala activation.

An important note in this respect is that the most commonly used method to assess personality, i.e. the self-report measure, appears to be unsuitable for research purposes. One cannot help but feel that questionnaires in which respondents are asked how they 'usually feel' or 'act' have become outdated and that the information they provide are at best a very crude measure of underlying constructs. Therefore, it is not surprising that the association between personality as measured with self-report questionnaires and biological data in this dissertation never amounted to more than 10% of shared variance. If we want to establish how personality is related to the aetiology of stress-related disorders we need to devise research schemes that can validate personality questionnaires by examining the association with (proposed) underlying neurobiological mechanisms on the one hand, and with respect to temporal and intra-individual stability on the other. Potential candidate questionnaires would then need to be examined in healthy individuals first, before we apply them to clinical samples. And if the particular test at hand could not be validated in this way, then we might be better off skipping the administration of that particular questionnaire altogether. Therefore, although much work needs to be done, the studies described in this dissertation have demonstrated that personality psychology has the potential to make an even more substantial contribution to the field of trauma research.

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Chapter 9

Nederlandse samenvatting

Inleiding

Blootstelling aan ernstige gebeurtenissen, zoals oorlog, misbruik, geweld, en natuur- of technologische rampen, kan een diepgaand en blijvend schadelijk effect hebben op de lichamelijke en psychische gezondheid. Doordat militairen vanwege hun beroep vaker blootgesteld worden aan zware en potentieel traumatische omstandigheden lopen ze een verhoogd risico op het ontwikkelen van stress-gerelateerde aandoeningen. Gelukkig zijn de symptomen en klachten die kunnen ontstaan na blootstelling aan stress of trauma, bij de meeste mensen van voorbijgaande aard. In sommige gevallen kunnen deze klachten echter uitgroeien tot stoornissen zoals posttraumatische stress stoornis (PTSS).

Er zijn verschillende verklaringen te vinden voor individuele verschillen in kwetsbaarheid voor stress-gerelateerde aandoeningen. Globaal genomen zijn er drie clusters van factoren aanwijsbaar die in verband gebracht kunnen worden met het ontstaan van posttraumatische klachten: 1) situatie- en contextaspecten; 2) persoonskenmerken, en 3) sociale en culturele determinanten. Het doel van de studies in dit proefschrift was om individuele verschillen in kwetsbaarheid voor PTSD - maar ook in veerkracht en weerbaarheid - vanuit een persoonlijkheidsperspectief te onderzoeken bij Nederlandse militairen en veteranen. Het copingmodel, zoals eerder beschreven door Lazarus en Folkman (1984), vormde daarbij het kader waarbinnen de resultaten geïntegreerd werden. De vragen die ten grondslag lagen aan de studies in dit proefschrift waren:

- *Is er een verband tussen trauma in de kindertijd en persoonlijkheid?*
- *Is er een relatie tussen persoonlijkheid en biologische systemen die eerder in verband werden gebracht met stress-gerelateerde aandoeningen zoals de hypothalamus-hypofyse-bijnier (HHB) as?*
- *Kunnen persoonlijkheidskenmerken die geassocieerd zijn met verhoogd risico op PTSS onderscheiden worden van persoonlijkheidskenmerken die samenhangen met verbeterde weerstand?*
- *Welke zijn de persoonlijkheids- en symptoomprofielen van Nederlandse veteranen die verwezen werden voor behandeling, en wat zijn de effecten van langdurige en intensieve behandeling op persoonlijkheid, klachten en coping?*
- *Wat is het verband tussen persoonlijkheid en coping?*

Om deze vragen te beantwoorden, werden gegevens verzameld van militairen die zich opmaakten voor een uitzending naar Afghanistan, evenals van gezonde veteranen, en veteranen die naar de Militaire Geestelijke Gezondheidszorg werden verwezen voor gespecialiseerde zorg.

Deel 1: niet-klinische studies

Neuroticisme

Neuroticisme is een persoonlijkheidskenmerk dat frequent in verband gebracht wordt met psychopathologie, in het bijzonder angst- en stemmingsstoornissen (zie Bienvenu & Stein, 2003, voor een review). Het is een van de 'Big Five' persoonlijkheidstrekken en wordt soms synoniem gebruikt met negatieve emotionaliteit of negatieve affectiviteit (Watson & Clark, 1984). Een literatuuroverzicht maakte duidelijk dat neuroticisme op verschillende manieren kan bijdragen aan een verhoogd risico op PTSS. Allereerst bleek dat de trek de kans of het risico op blootstelling aan trauma vergroot. Daarnaast werden studies besproken die aantoonden dat neuroticisme een dispositionele kwetsbaarheidsfactor vormt voor stoornissen als PTSS. Ook werd duidelijk dat neuroticisme een indirect effect op het ontstaan van PTSS kan hebben: het kan een rol spelen in de manier waarop het individu potentiële stressoren inschat; het kan de relatie tussen stressor en reactie beïnvloeden; en het kan een effect hebben op coping-

gedrag. Tegelijkertijd is neuroticisme zelf onderhevig aan verandering door blootstelling aan trauma, en ook de aanwezigheid van PTSS kan van invloed zijn op neuroticisme. Al met al wordt er in de literatuur onvoldoende rekening gehouden met deze effecten.

Vroeg trauma

Dit proefschrift toonde een verband aan tussen ervaringen in de kindertijd/ jeugd en persoonlijkheid. Zelf-gerapporteerde blootstelling aan emotionele verwaarlozing in de jeugd ging samen met lage scores op karakter-dimensies Zelfsturend (Zs) en Coöperatief (Co) van de verkorte Temperament en Karakter vragenlijst van Cloninger (Duijsens & Spinhoven, 2002). Lage scores op deze karakterdimensies werden eerder in verband gebracht met vermijdende coping en gereduceerde sociale steun, interpersoonlijke moeilijkheden en identiteitsproblemen (Cloninger, 1999; Duijsens & Spinhoven, 2002). Bovendien blijken mensen met lage scores op deze schalen meer risico te hebben op het ontwikkelen van angst- en depressieve stoornissen (Matsudaira & Kitamura, 2006). Deze resultaten illustreerden hoe trauma in de jeugd kan bijdragen aan een verhoogd risico op PTSS en andere stress-stoornissen in het volwassen leven. Ze toonden aan dat persoonlijkheid een 'mediator' kan zijn tussen vroeg trauma en psychopathologie. Bovendien werd met deze studie geaccentueerd dat ervaringen in de kindertijd verstrekkende invloeden kunnen hebben op de persoonlijkheidsontwikkeling; een thema dat centraal staat in de meeste persoonlijkheidstheorieën.

HHB-as

Dit proefschrift toonde een verband tussen persoonlijkheid en de HHB-as. Het feit dat de activiteit van de HHB-as beïnvloed wordt door psychologische factoren is al lang bekend (Mason, 1968). Ook is de HHB-as betrokken bij diverse stress-gerelateerde aandoeningen (zie bijvoorbeeld Miller, Chen, & Zhou, 2007). De beschikbare literatuur over de relatie tussen persoonlijkheid en de HHB-as wordt echter vertroebeld door tegenstrijdige resultaten. Een recente ontwikkeling in onderzoek naar de HHB-as is het meten van cortisol direct na het ontwaken. Onderzoek heeft aangetoond dat de cortisol concentratie in speekselmonsters die op gezette tijden na het ontwaken verzameld worden - de zgn. Cortisol Awakening Response, CAR -, een betrouwbare indicatie geeft van de reactiviteit van de HHB-as (Pruessner et al., 1997; Wust et al., 2000).

Onze resultaten toonden een verband aan tussen de (absolute) cortisol concentraties in speeksel na het ontwaken en temperament factor Leedvermijdend van de Temperament en Karakter vragenlijst (Cloninger, Svrakic, & Przybeck, 1993; Duijsens & Spinhoven, 2002). De relatieve toename in cortisol concentraties het eerste half uur na het ontwaken was gerelateerd aan scores op karakterschaal Zelfsturend en temperament schaal Leedvermijdend. Mensen die hoog scoren op Leedvermijdend hebben meer risico op het ontwikkelen van aandoeningen als PTSS en depressieve stoornissen (Cloninger, Svrakic, & Przybeck, 2006; Gil, 2005). Hoge scores op Zelfsturend gaan doorgaans samen met gevoelens van zelfvertrouwen en controle en hangen negatief samen met dispositionele angst en neuroticisme (Cloninger et al., 1993; De Fruyt, Van de Wiele, & Van Heeringen, 2000; Jiang et al., 2003). De resultaten toonden daarmee aan dat de CAR beïnvloed kan worden door persoonlijkheidskenmerken die een verhoogd risico op stress-gerelateerde aandoeningen impliceren enerzijds (nl. Leedvermijdend) en persoonlijkheidskenmerken die samengaan met succesvolle aanpassing en mentale gezondheid anderzijds (nl. Zelfsturend).

Dispositionele veerkracht

Met behulp van 'path-analyses' werd in een steekproef van Libanon veteranen onderzocht of, en hoe, persoonlijkheid, coping, sociale steun en PTSS klachten met elkaar samenhingen. Het doel was om te onderzoeken of weerbaarheidfactoren 'hardiness', optimisme en interne 'locus-of-control', onafhankelijk van neuroticisme voorspellers waren voor coping en PTSS klachten. Uit de gerapporteerde resultaten bleek dat naast de stressors tijdens de uitzending, neuroticisme een voorspeller was voor PTSS klachten. Neuroticisme bleek bovendien de grootste proportie variantie in PTSS klachten te verklaren. Hoewel hardiness en optimisme gecorreleerd waren met neuroticisme, bleken deze twee dispositionele eigenschappen op te vatten als unieke en onafhankelijke weerbaarheidfactoren. Optimisme had een direct effect op PTSS klachten, de relatie tussen hardiness en PTSS werd gemedieerd door sociale steun. Zowel hardiness als neuroticisme waren - en onafhankelijk van elkaar - gerelateerd aan coping. Locus-of-control was geen onafhankelijke voorspeller voor coping, sociale steun of posttraumatische stress symptomen en er bleek in deze steekproef geen verband te bestaan tussen copingstijl en PTSS klachten.

Deel 2: Klinische studies

Uitzend-gerelateerde problematiek & behandeling

Veel van de beschikbare literatuur over PTSS bij militairen is gebaseerd op onderzoek onder Vietnam veteranen. Er is aanzienlijk minder onderzoek gedaan naar symptomen en klachten die kunnen ontstaan bij militairen die deelnemen aan vredesoperaties enerzijds en naar de resultaten van behandeling anderzijds. In het tweede deel van het proefschrift werd een tweetal studies gewijd aan deze onderwerpen.

Bij een cohort Nederlandse veteranen die gespecialiseerde behandeling zochten voor hun klachten, bleek er sprake te zijn van diffuse psychopathologie daar zij verhoogde scores vertoonden op zeven van de tien klinische schalen van de MMPI-2. De hoogste scores werden gevonden op MMPI-2 klinische schalen 2 (D), 7 (Pt) en 8 (Sc). De MMPI-2 scores suggereerden daarom dat bij deze groep (ex)militairen sprake was van een gemengd psychiatrisch beeld met inbegrip van depressieve en angstklachten, afgevlakt affect, concentratieproblemen, sociaal teruggetrokken gedrag, somatische klachten, vermoeidheid, interpersoonlijke problemen, impulsiviteit, verhoogde waakzaamheid en boosheid. Als zodanig vertoonden de Nederlandse veteranen veel overeenkomsten met hun Amerikaanse collega's die in Vietnam dienden (Frueh, Hamner, Cahill, Gold, & Hamlin, 2000). Desalniettemin leken de Nederlandse veteranen enigszins minder ernstige klachten te rapporteren dan Vietnam veteranen, aangezien de absolute verhogingen op de MMPI-2 wat lager waren dan scores uit eerder onderzoek (zie bijvoorbeeld Albrecht et al., 1994; Litz et al., 1991) .

Een eerste psychometrische evaluatie van een intensieve en langdurige groepsbehandeling voor Nederlandse veteranen met PTSS en comorbide stoornissen toonde aan dat, hoewel de meeste veteranen klinische verbetering na behandeling toonden, vele van hen nog klinische significante klachtniveaus rapporteerden. De 22 veteranen in deze studie voldeden bij aanvang van de behandeling aan DSM-IV criteria voor PTSS (APA, 2000), en bij de meeste van hen was er sprake van comorbide as-I en / of as-II problematiek. De behandeling was gestoeld op een gefaseerde behandeling van PTSS klachten waarbij 1 dag in de week, voor een periode van gemiddeld 21 maanden diverse behandelmodules groepsgewijs werden aangeboden. De modules bestonden uit cognitieve gedragstherapie, psycho-educatie, case-management, sociotherapie, creatieve therapie, activiteiten therapie en drama- en/ of psychomotore therapie.

Na behandeling was er sprake van een significante afname in PTSS klachten zoals gemeten

met de zelfinventarisatielijst PTSS (ZIL). De SCL-90 toonde een forse afname in gerapporteerde psychoneurotische problemen. Ook waren verschuivingen waarneembaar in gerapporteerde copingstijlen, zoals gemeten met de Utrechtse Coping Lijst (UCL), waarbij meer actieve en minder vermijdende stijlen gerapporteerd werden. Vergelijking van de MMPI-2 scores voor en na behandeling toonde aan dat angst en depressieve klachten waren afgenomen, dat men minder vervreemding en concentratieproblemen rapporteerde, dat de gevoelens van zelfwaardering waren toegenomen en dat men zich minder beperkt voelde in het uitvoeren van professionele werkzaamheden. Toch was er ook na behandeling nog steeds sprake van klinisch significante klachtniveaus. Dit onderstreept het gegeven dat ervaringen tijdens uitzending kunnen leiden tot chronische en moeilijk te behandelen klachten.

Deel 3: Discussie

Het doel van de studies in dit proefschrift was om inzichtelijk te maken hoe persoonlijkheid bij kan dragen aan verhoogd risico op, danwel verbeterde weerstand tegen, PTSS en andere uitzetgerelateerde problematiek. De empirische studies in dit proefschrift hebben enkele mechanismen uitgelicht via welke trauma, persoonlijkheid en PTSS met elkaar in verband gebracht kunnen worden.

Coping

Het (transactionele) coping model van Lazarus en Folkman (1984) kan gebruikt worden om te illustreren *hoe* persoonlijkheid een rol speelt in de omgang met stress en trauma. Allereerst is uit eerder onderzoek gebleken dat bepaalde persoonlijkheidskenmerken de mate van aandacht voor potentiële stressors kunnen beïnvloeden (Engelhard & Van den Hout, 2007). Wanneer een stressor vervolgens als een bedreiging wordt geïnterpreteerd, start het coping-proces. Doordat de *appraisal* van een bedreigende situatie doorgaans gepaard gaat met een negatieve emotionele reactie, zal het individu in eerste plaats proberen om deze emotionele response te reguleren. Daarbij is het aannemelijk dat mensen bij wie sprake is van een hoger basaal onlustniveau, zoals het geval is bij hoog neurotische mensen, meer moeite moeten doen om hun emoties te reguleren. Daardoor kunnen zij minder energie steken in het effectief omgaan met externe stressors. Het is dan ook niet verwonderlijk dat we een omgekeerd verband vonden tussen neuroticisme en probleemgerichte coping.

Persoonlijkheid kan vervolgens van invloed zijn op de inschatting van de middelen die iemand tot zijn of haar beschikking heeft om de stressor het hoofd te bieden zoals de mate van sociale steun die beschikbaar is. Zo is gebleken dat er een positief verband bestaat tussen 'gehardheid' (hardiness) en de mate van sociale steun, terwijl neuroticisme negatief met de mate van sociale steun lijkt samen te hangen. Tot slot is gebleken dat bepaalde persoonlijkheidskenmerken een directe relatie hebben met psychisch welbevinden, waarbij neuroticisme en optimisme een tegengestelde uitwerking lijken te hebben.

Veerkracht versus kwetsbaarheid

Kwetsbaarheid en weerbaarheid zijn nauw met elkaar verweven en er zijn verschillende manieren om deze samenhang te conceptualiseren. Enerzijds moet de mogelijkheid overwogen worden dat weerbaarheid en kwetsbaarheid twee uiterste posities innemen op dezelfde dimensie. Anderzijds zou het ook zo kunnen zijn dat weerbaarheid en kwetsbaarheid multidimensionele constructen zijn. We stellen vast dat er meer bewijs is voor deze laatste opvatting. Er is enige evidentie voor de uni-dimensionaliteit hypothese: zo werden lage scores op Zelfsturend en Coöperatief bijvoorbeeld eerder in verband gebracht met psychopathologie en (inter-persoonlijke) moeilijkheden, terwijl uit een studie van Ghazinour en collega's kan

worden afgeleid dat hoge scores op deze schalen indicatief zijn voor vergrote weerbaarheid (Ghazinour, Richter, & Eisemann, 2003).

Daartegenover staat dat de resultaten in dit proefschrift aantoonden dat er een aantal dispositionele eigenschappen identificeerbaar zijn die onafhankelijk van neuroticisme kunnen bijdragen aan de mate van aanpassing aan ingrijpende gebeurtenissen. Er zijn dus factoren van invloed, die tegelijkertijd naast elkaar en onafhankelijk van elkaar een bijdrage leveren aan het al dan niet ontstaan van klachten. Weerbaarheid is niet synoniem aan het ontbreken van kwetsbaarheid.

Temperament & karakter

De studies toonden aan dat zowel temperament als karakter in verband gebracht kan worden met de etiologie van PTSS. Het verhoogd risico op PTSS (en andere stoornissen) dat met bepaalde persoonlijkheidskenmerken gepaard gaat, lijkt deels aangeboren te zijn (temperament). Uit studies is gebleken dat persoonlijkheidskenmerken als neuroticisme en de dimensie Leedvermijdend uit Cloninger's model, voor ongeveer de helft bepaald worden door genetische aanleg (Floderus-Myrhed, Pedersen, & Rasmuson, 1980; Keller, Coventry, Heath, & Martin, 2005), en juist deze twee persoonlijkheidskenmerken - die overigens ook redelijk sterk met elkaar correleren (De Fruyt et al., 2000) - vergroten het risico op PTSS. Ook (vroege) ervaringen zijn belangrijk voor de persoonlijkheidsontwikkeling, en ingrijpende ervaringen in de jeugd kunnen hun sporen nalaten in de volwassen persoonlijkheid, waarbij ze het vermogen om effectief om te gaan met latere stressors nadelig kunnen beïnvloeden.

Plus- en minpunten

Dit proefschrift toonde aan dat persoonlijkheid op verschillende manieren betrokken is bij het ontstaan van PTSS. Sterke punten aan de studies in voorgaande hoofdstukken zijn dat zowel veerkracht als weerbaarheid werd onderzocht in klinische en gezonde steekproeven, dat gebruik werd gemaakt van verschillende manieren om persoonlijkheid te meten, en dat de focus lag op een vrij specifieke groep mensen. Desalniettemin kunnen een aantal kanttekeningen geplaatst worden bij de onderzoeken in dit proefschrift. In de eerste plaats maakt het cross-sectionele karakter van de studies het onmogelijk om uitspraken te doen over causaliteit. Daarnaast brengt het gegeven dat we uitsluitend gebruik gemaakt hebben van zelfbeoordelingsvragenlijsten met zich mee dat er een zekere mate van 'bias' in de resultaten geslopen kan zijn. Het strekt tot de aanbeveling om de studies die in dit proefschrift beschreven werden, te herhalen in andere zogenaamde 'trauma-populaties'. Daarnaast moeten de resultaten uit dit proefschrift gerepliceerd worden in aanvullend prospectief onderzoek. Toch levert dit proefschrift een belangrijke wetenschappelijke bijdrage aan de kennis over stress-gerelateerde aandoeningen doordat het meer inzicht verschaft in individuele verschillen in de kwetsbaarheid en veerkracht.

Implicaties voor behandeling & preventie

Het feit dat militairen die worden uitgezonden last kunnen krijgen van een scala aan psychische klachten onderstreept de noodzaak van interventies om deze klachten te voorkomen danwel te genezen. Op basis van persoonlijkheid kan worden voorspeld welke militairen meer risico hebben op het ontwikkelen van klachten, maar er zijn meer determinanten (Kleber & Brom, 1992) en in geen van de gevallen is er sprake van een 1-op-1 relatie. Bovendien is PTSS een heterogeen construct en de wisselwerking tussen verschillende factoren in de etiologie van de stoornis is onduidelijk (Brewin, Andrews, & Valentine, 2000). Derhalve kan niet op basis van persoonlijkheid met zekerheid worden vastgesteld wie wel en wie niet last zal krijgen

van klachten na uitzending. Persoonlijkheidsonderzoek kan wel een bijdrage leveren aan het vinden van de optimale afstemming tussen persoon en functie. Ook zou persoonlijkheidsonderzoek in screeningsituaties aangewend kunnen worden om vast te stellen welke rekruten het meest gebaat zijn bij specifieke trainingsprogramma's. Hierbij is het van belang om er rekening mee te houden dat effectieve training 'zelfs' de weerbaarheid van verhoogd kwetsbare individuen kan doen toenemen. Wanneer klachten zijn ontstaan, zou de behandeling voorts gericht kunnen worden op het bevorderen van weerbaarheidsfactoren zoals optimisme en effectieve coping, en het 'stutten' van aanwezige kwetsbaarheid.

Slot beschouwing

Er is de laatste decennia veel onderzoek gedaan naar de schadelijke gevolgen van trauma op de (geestelijke) gezondheid. De invloed van persoonlijkheid in relatie tot PTSS is echter grotendeels onderbelicht gebleven; er is nauwelijks systematisch onderzoek gedaan naar de wisselwerking tussen persoonlijkheid, trauma en PTSS. Dit is op zijn minst opmerkelijk te noemen. Zeker wanneer men er rekenschap van neemt dat de emotionele reactie van het individu een belangrijke voorspeller is voor het al dan niet ontstaan van klachten. Mogelijk draagt de complexiteit van persoonlijkheid als psychologisch construct bij aan de relatieve onderwaardering in onderzoek. Om persoonlijkheidsonderzoek tot volwas te laten komen binnen het veld van trauma-onderzoek is het van belang om te komen tot werkbare operationalisaties van het construct enerzijds en heldere hypothese over de manier waarop PTSS en persoonlijkheid met elkaar samenhangen anderzijds. Daartoe zouden de meest gebruikte zelfbeoordelingsvragenlijsten aan kritische valideringsstudies onderworpen moeten worden waarbij expliciet getoetst wordt of bepaalde kenmerken daadwerkelijk in verband gebracht kunnen worden met stabiele individuele verschillen in aandacht, perceptie, affect en gedrag. Ook zou meer onderzoek gericht moeten worden op het staven van gegevens uit zelfbeoordelingsvragenlijsten met resultaten uit onderzoek naar individuele verschillen in (onderliggende) neurobiologische processen en factoren. Pas dan kan de bijdrage van de persoonlijkheidsleer aan trauma-onderzoek volledig tot volwas komen.

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