Vulnerability to posttraumatic stress disorder

Miriam J.J. Lommen

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Vulnerability to posttraumatic stress disorder

Kwetsbaarheid voor posttraumatische stressstoornis

(met een samenvatting in het Nederlands)

Proefschrift

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Miriam Johanna Judith Lommen

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“I’ve got a great ambition to die of exhaustion rather than boredom.”

-Thomas Carlyle-

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Chapter 1

Introduction

INTRODUCTION

Most individuals will experience a traumatic event during their lives and some will develop subsequent posttraumatic stress disorder (PTSD). The aim of this thesis is to identify individual differences that may increase vulnerability to PTSD and unravel the mechanisms that contribute to PTSD. In this chapter, a short history of trauma and PTSD is provided, followed by information about the prevalence of trauma and PTSD nowadays. Furthermore, a brief summary of predictors of PTSD is presented and influential theories about PTSD are discussed. Then some yet unresolved issues are highlighted that provide the rationale for the studies in this thesis. The introduction concludes with the aims and outline of this thesis.

Trauma and PTSD through history

The effects of traumatic experiences on human mind and body have been described very early, although the view on what may serve as a traumatic experience and the subsequent consequences have changed drastically over time (Jones & Wessely, 2006). Posttraumatic problems as manifested in soldiers with war experience have come under a lot of names, like combat fatigue, soldiers' heart, war neurosis, and shell shock (Jones & Wessely, 2006). In 1980, the negative consequences of traumatic experiences were included as a psychiatric diagnosis in the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; APA, 1980) as Post-Vietnam syndrome or delayed stress syndrome. Traumatic events were seen as severe events outside the range of usual human experience (APA, 1980). However, studies showed that a range of events that were not war-related could evoke similar stress reactions. In the DSM-IV (APA, 1994), the name was revised into posttraumatic stress disorder (PTSD) and the operationalization of traumatic experience was broadened.

As PTSD is caused by the experience of a traumatic event, the DSM-IV-TR diagnosis for PTSD (APA, 2000; Box 1) includes criteria with regard to the traumatic event and the initial reactions to the event. Although the PTSD construct may change again with the arrival of the DSM-V (expected May 2013), the current diagnosis includes three symptom clusters. The first cluster involves re-experiencing symptoms. Individuals with PTSD relive the traumatic event(s) e.g., through sudden or unexpected upsetting memories (intrusions), flashbacks, and nightmares. The second cluster describes avoidance symptoms. Individuals with PTSD try to avoid situations, people or things that remind them of the traumatic event. The third cluster includes hyperarousal. Individuals with PTSD are characterized by hypervigilance and increased emotional arousal, which

may be expressed in sleep difficulties, irritability or outbursts of anger, concentration problems, constant alertness on danger, and heightened startle. Furthermore, the symptoms need to be present for more than one month and cause clinically significant distress or impairment in daily functioning.

Box 1. *DSM-IV-TR diagnosis for PTSD (APA, 2000)*

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

1. The person has experienced, witnessed, or been confronted with an event or events that involve actual or threatened death or serious injury, or a threat to the physical integrity of oneself or others.
2. The person's response involved intense fear, helplessness, or horror. Note: in children, it may be expressed instead by disorganized or agitated behavior.

B: The traumatic event is persistently re-experienced in at least one of the following ways:

1. Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions.
2. Recurrent distressing dreams of the event. Note: in children, there may be frightening dreams without recognizable content
3. Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur upon awakening or when intoxicated). Note: in children, trauma-specific reenactment may occur.
4. Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.
5. Physiologic reactivity upon 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 at least three of the following:

1. Efforts to avoid thoughts, feelings, or conversations associated with the trauma
2. Efforts to avoid activities, places, or people that arouse recollections of the trauma
3. Inability to recall an important aspect of the trauma
4. Markedly diminished interest or participation in significant activities
5. Feeling of detachment or estrangement from others
6. Restricted range of affect (e.g., unable to have loving feelings)
7. Sense of foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)

D: Persistent symptoms of increasing arousal (not present before the trauma), indicated by at least two of the following:

1. Difficulty falling or staying asleep
2. Irritability or outbursts of anger
3. Difficulty concentrating
4. Hyper-vigilance
5. Exaggerated startle response

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

F: The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Prevalence of traumatic events and PTSD

Large cohort-studies have shown that about 90% in U.S. community samples (Breslau, Kessler, Chilcoat, Schultz, Davis, & Andreski, 1998) and 81% of the general population in the Netherlands (de Vries & Olff, 2009) reported a lifetime exposure to one or more traumatic events according to the DSM-IV (APA, 1994). Although PTSD is critically preceded by the experience of a traumatic event, such an experience does not necessarily lead to the development of PTSD. That is, although the majority of people experiences at least one potentially traumatizing event in life, only 7-8 % develops PTSD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; de Vries & Olff, 2009). The relative risk of PTSD varies across types of traumatic events (Kessler et al., 1995), with greater risk associated with interpersonal trauma (e.g., physical or sexual assault; Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993). According to a review of Tolin and Foa (2006), women are two times more likely to develop PTSD, while men have an increased risk of trauma exposure. Although women are more likely to be exposed to traumatic events that are associated with higher PTSD rates, like rape, differences in type of event can only partially explain the sex difference in PTSD (Tolin & Foa, 2006).

Predictors of PTSD

After exposure to a traumatic event, most individuals experience posttraumatic stress reactions (Bryant, 2004). Usually these symptoms subside, however, some individuals keep experiencing symptoms and may develop PTSD. To gain insight in the aetiology of PTSD it is important to understand *why* some, but not others, develop PTSD after a comparable traumatic event. Obviously, individual differences are involved. Many studies identified risk factors for PTSD, which can be divided in factors before, during, and after the traumatic event (pre-, peri-, and posttrauma risk factors, respectively; see Keane, Marshall, & Taft, 2006 for an overview).

Pre-trauma risk factors include demographic variables (e.g., female gender, young age of trauma exposure, race, low education), family psychiatric history, prior trauma (e.g., childhood abuse), prior psychiatric history, and personality (e.g., neuroticism). Peritrauma risk factors like cognitive processing (e.g., dissociation), trauma severity (especially subjective perception of the traumatic event like perceived life threat), and emotional responses have been associated with PTSD. Posttrauma risk factors include lack of social support, additional life stress, and negative appraisals of initial PTSD symptoms. The relative contribution of these predictors has been studied in a few meta-analyses (Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003).

Conditioning theory of PTSD

Contemporary conditioning theories may explain why some individuals develop anxiety disorders, like PTSD (e.g., Craske et al., 2008; Davey, 1997; Engelhard, de Jong, van den Hout, & van Overveld, 2009; Mineka & Oehlberg, 2008). In case of PTSD, the theory states that a neutral stimulus becomes associated with an unconditioned stimulus (US; the traumatic event). Then this previously neutral (now conditioned) stimulus (CS), elicits a conditioned response (CR; e.g., fear) by activating the expectancy of the US (Figure 1). For example, a soldier deployed to Afghanistan is driving when an improvised explosive device explodes beneath the vehicle. At this moment, the soldier fears for his life, but he emerges unscathed. After that experience, stimuli that symbolize or resemble an aspect of this stressful experience activate the expectancy of the stressful experience and the associated fear response. In this example, the explosion represents the US, the CS may be driving a vehicle, and the fear response is the CR. So here the anxiety is a learned response resulting from a true alarm.

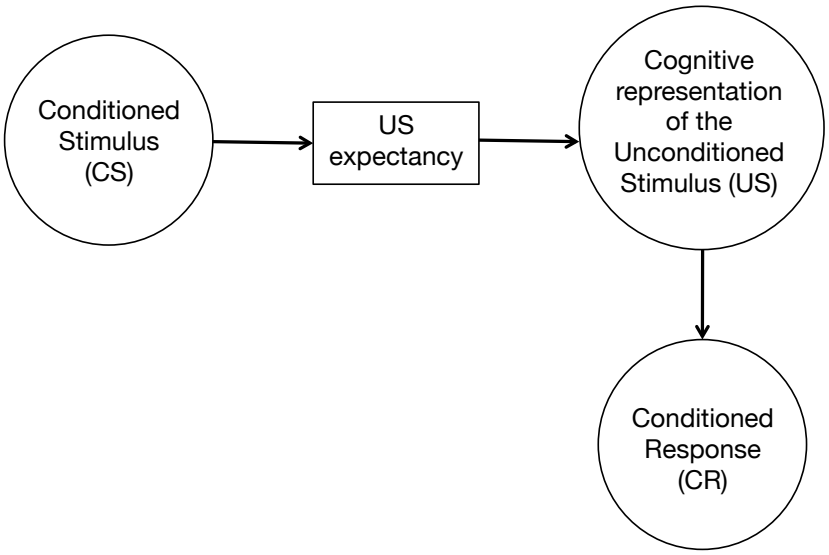

Figure 1. Simplified model of the contemporary conditioning theory (Davey, 1997).

According to the conditioning theory, the learned fear extinguishes when disconfirming information is learned. That is, learning that the CS is no longer associated with the US, so that the CS loses its signaling quality. In the example, when the soldier keeps driving in vehicles and no other explosions occur, the association between driving the vehicle and the explosion weakens (more specifically, the association between driving a vehicle and no explosion strengthens) and the soldier may no longer expect an explosion to occur when driving a vehicle. So due to extinction learning, trauma-related fear would dissipate. In sum, this contemporary conditioning theory explains how fears can be learned and unlearned, and has important clinical implications for exposure therapy (Craske et al., 2008; Craske, Liao, Brown, & Vervliet, 2012).

Cognitive theory of PTSD

Cognitive theories state that external events or internal stimuli are processed in a way that confirms existing schemas that individuals have about themselves, others, and the world. These schemas may lead to biases in the perception, interpretation, and memory of information. With regard to anxiety disorders, cognitive schemas of danger are thought to be involved and anxiety may results from beliefs about impending danger (Beck, 2005; Ehlers & Clark, 2000). According to the cognitive model of PTSD (Ehlers & Clark, 2000), a current sense of threat results from negative appraisals of the traumatic event(s) and/or its consequences, and inadequate processing of the traumatic event(s) into autobiographical memory. Behavioural and cognitive strategies (e.g., avoidance, and rumination) aimed to reduce the sense of current threat maintain negative appraisals and inadequate processing of the traumatic event, and paradoxically enhance PTSD symptoms. Negative appraisals, like “nowhere is safe”, “I will never get over this”, and “nobody is there for me”, have shown to be valuable in predicting PTSD symptomatology (Dunmore, Clark, & Ehlers, 2001; Ehlers, Mayou, & Bryant, 1998; Ehring, Ehlers, & Glucksman, 2006; 2008; Halligan, Michael, Clark, & Ehlers, 2003; van den Hout & Engelhard, 2004). Cognitive (behaviour) therapy, based on the cognitive model of PTSD (Ehlers & Clark, 2000), has shown to be effective in the treatment of PTSD (Bisson & Andrew, 2005; Bisson et al., 2007; Ehlers, Clark, Hackmann, McManus, & Fennell, 2005).

Unresolved issues

Although many predictors of PTSD have been identified, several issues remain unclear. A few are discussed here.

First, there is a lack of prospective studies with a pre-trauma assessment. Because the majority of research includes correlational and longitudinal studies that gathered data after exposure to the traumatic event, it is largely unknown if risk factors reflect a pre-trauma vulnerability factor of PTSD or an acquired PTSD sign that has been developed along with the PTSD symptoms. However, prospective research in which individuals are tested *before* trauma is seldom feasible. A few prospective studies with a pre-trauma assessment have been done, mainly in individuals that are at high risk to experience a potentially traumatic event, like soldiers (e.g., Bonanno et al., 2012; Engelhard, Huijding, van den Hout, & de Jong, 2007; Engelhard, van den Hout, et al., 2007; Rona et al., 2009; van Zuiden et al., 2011), firefighters (e.g., Guthrie & Bryant, 2006; Heinrichs et al., 2005), and police (e.g., Inslicht et al., 2010; Meffert et al., 2008). However, the number of studies with such a design is still limited. Thus there is great need for prospective studies to identify individual differences that increase vulnerability for PTSD (Bomyea, Risbrough, & Lang, 2012).

Second, for the majority of predictors it is unknown *how* the risk of PTSD can be explained. In other words, what mechanism accounts for the association between the predictor and PTSD? As argued by Ingram and Price (2010), risk factors are important in predicting the likelihood of experiencing a disorder, but they are theoretically or empirically uninformative about the mechanisms. In contrast, vulnerability factors are stable traits that are involved in the causal process of psychopathology and may unravel the actual mechanisms that are responsible for the onset or maintenance of a disorder. Knowledge about vulnerability factors may improve theoretical accounts of PTSD. Moreover, this knowledge may add to therapeutic benefits, if the underlying mechanisms that contribute to psychopathology can be addressed in treatment (Bomyea et al., 2012; Ingram & Price, 2010). Although vulnerability factors are resistant to change, they may change with certain corrective experiences (Ingram & Price, 2010). An important next step is to examine how these pathogenic mechanisms, once established, can be changed.

Third, the boundaries of the contemporary influential models are unclear. According to a review of de Girolamo and McFarlane (1996), 94% of the research on PTSD has been done in developed, Western countries. Therefore it is largely unknown if these findings that are mainly based on research in developed countries generalize to non-Western, developing countries. Moreover, it is unclear if findings generalize to individuals with severe mental illness, and co-morbid PTSD. Both trauma and PTSD prevalence are high in samples with severe mental illness, like psychosis (Mueser et al.,

1998; Resnick, Bond, & Mueser, 2003; Seedat, Stein, Oosthuizen, Emsley, & Stein, 2003). However, these groups are often excluded from studies. Since the prevalence of co-morbid PTSD is high in this population, it is important to know if similar underlying mechanisms are involved.

Aims and outline

This thesis focuses on individual differences that may increase vulnerability to PTSD. The first two chapters describe a prospective study among soldiers deployed to Afghanistan, and examine whether individual differences in conditioning and trait anger are pre-trauma vulnerability factors for PTSD. After that, two chapters present experimental studies that focus on pathogenic mechanisms that are involved in PTSD and other anxiety disorders. Finally, two chapters describe studies that test the applicability of the cognitive model of PTSD to different populations.

Chapter 2 includes a prospective study among 249 Dutch soldiers who were tested before and after their deployment to Afghanistan. In this study, it was examined whether reduced extinction learning was a pre-trauma vulnerability factor for PTSD symptom severity, beyond the effects of pre-deployment stress symptoms, neuroticism, and exposure to stressors on deployment.

Chapter 3 includes a study that was conducted in the same sample of soldiers as in Chapter 2. The temporal relationship between trait anger and PTSD symptom severity was tested, when controlling for initial symptoms, neuroticism and exposure to stressors on deployment.

Chapter 4 includes an experiment that examined a proposed underlying mechanism of the association between neuroticism and anxiety. Avoidance behaviour of ambiguous stimuli in students with high neuroticism scores was compared to students with low neuroticism scores.

Chapter 5 includes an experiment that tested if emotional reasoning, a proposed pathogenic mechanism involved in anxiety disorders, could be decreased in a sample of students with fear of spiders, and whether this influenced subsequent fear-related cognitions and behaviour.

Chapter 6 includes a study among 113 tsunami survivors that examined whether psychological factors related to PTSD identified in Western countries generalize to findings in a developing country.

Chapter 7 includes a study involving patients of an outpatient clinical setting with severe mental illness (schizophrenia or schizoaffective disorder). It was tested whether trauma and PTSD were adequately detected in this population, and if negative posttraumatic cognitions were related to PTSD symptom severity.

Chapter 8 includes a general discussion of the studies presented in this thesis.

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Chapter 2

Pre-trauma individual differences in extinction learning predict posttraumatic stress

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ABSTRACT

In the aftermath of a traumatic event, many people suffer from psychological distress, but only a minority develops posttraumatic stress disorder (PTSD). Pre-trauma individual differences in fear conditioning, most notably reduced extinction learning, have been proposed as playing an important role in the etiology of PTSD. However, prospective data are lacking. In this study, we prospectively tested whether reduced extinction was a predictor for later posttraumatic stress. Dutch soldiers (N=249) were administered a conditioning task before their four-month deployment to Afghanistan to asses individual differences in extinction learning. After returning home, posttraumatic stress was measured. Results showed that reduced extinction learning before deployment predicted subsequent PTSD symptom severity, over and beyond degree of pre-deployment stress symptoms, neuroticism, and exposure to stressors on deployment. The findings suggest that reduced extinction learning may play a role in the development of PTSD.

After a traumatic event, many people initially show posttraumatic stress reactions (Bryant, 2004), but these subside with time for most of them. A minority goes on to develop posttraumatic stress disorder (PTSD), with rates varying from 2% after a car accident and 49% after rape (Breslau et al., 1998). PTSD is characterized by symptoms of re-experiencing the traumatic event, avoidance of its reminders, and hyperarousal (American Psychiatric Association, 2000). Over the past years, studies have identified pre-trauma vulnerability factors for PTSD, such as demographic variables (e.g., low education; Parslow et al., 2006), personality (e.g., neuroticism; Engelhard, van den Hout, & Lommen, 2009), prior traumatic experiences, and earlier psychopathology (see Brewin, Andrews, & Valentine, 2000; Ozer, Best, Lipsey, & Weiss, 2003). Since the majority of studies assessed 'pre-trauma' factors retrospectively, contamination with current posttraumatic distress may not be ruled out. However, prospective research in which individuals are tested *before* trauma is seldom feasible (e.g., McNally, 2003).

Contemporary (propositional) conditioning theory may explain why some individuals develop PTSD (e.g., Craske et al., 2008; Engelhard, de Jong, van den Hout, & van Overveld, 2009; Hermans, Vansteenwegen, Crombez, Baeyens & Eelen, 2002; Mineka & Oehlberg, 2008). The theory states that when a neutral stimulus (e.g., car) is associated with an unconditioned stimulus (US; e.g., car accident), then this previously neutral (now conditioned) stimulus (CS), elicits a conditioned response (CR; e.g., fear) by activating the memory representation of the US. To extinguish learned fear, disconfirming information (CS-no US) needs to be learned so that the CS loses its signaling quality. This would imply that, due to extinction learning, trauma-related fear would dissipate. Obviously, this is not the case for individuals with PTSD. May fear extinction have failed in individuals who develop PTSD?

Studies have shown individual differences in extinction learning with a 'de novo' conditioning paradigm (cf. Orr et al., 2000) that consists of an acquisition and extinction phase. In the acquisition phase, a CS+ (e.g., colored circle) is followed by a US (e.g., mild electric shock) and thus becomes a predictor of the US that evokes a US-expectancy (analogous to threat beliefs; Lovibond, Mitchell, Minard, Brady, & Menzies, 2009; p. 716). A CS- (e.g., another colored circle) is never followed by the US. In the extinction phase, the CS+ and CS- are presented without the US, which allows the person to learn that the CS+ no longer predicts the US. A meta-analysis on conditioning (Lissek et al., 2005) showed that, compared to healthy controls, patients with anxiety disorders specifically showed differences with respect to excitatory associations (CS+), but not in discrimination learning (CS+ vs. CS-). Studies using the 'de novo' conditioning paradigm

have found that participants with PTSD show delayed fear extinction, compared to control groups with and without trauma exposure (see Mineka & Oehlberg, 2008). This was shown with physiological measures (e.g., Orr et al., 2000; Peri, Ben-Shakhar, Orr & Shalev, 2000) and US expectancy ratings (Blechert, Michael, Vriends, Margraf & Wilhelm, 2007). These findings suggest that reduced extinction learning may be a pre-trauma vulnerability factor for the development of PTSD. Tentative support for this prediction comes from a prospective study with 45 firefighters showing that reduced extinction before trauma indeed predicted more PTSD symptoms up to two years later (Guthrie & Bryant, 2006). However, this study was limited by the small sample size, long time between the traumatic event and PTSD assessment, and lack of control for baseline symptoms. Furthermore, there was no control for known risk factors, such as neuroticism (Engelhard, van den Hout et al., 2009) and stressor severity (Dohrenwend et al., 2006). Therefore, it remains unknown whether extinction learning has a unique contribution to the existing knowledge on risk factors for PTSD.

Using a larger sample of soldiers deployed to Afghanistan, the current prospective study tested whether reduced extinction of US-expectancies about six weeks before deployment predicts PTSD symptom severity two months after deployment, after controlling for 'baseline' symptoms, neuroticism, and stressor severity.

METHOD

Participants and general procedure

Participants were 249 Dutch Royal Army soldiers who enrolled in this study before a four-month deployment to Uruzgan (Afghanistan) from November 2009 to March 2010. Of the 250 soldiers who were invited to participate, only one refused.

The sample (98% were male) had a mean age of 23.8 years (*SD* = 4.9). The highest attained educational level was primary (2%), secondary (92%), or higher education (6%). About 34% were married or cohabiting, 38% were in a relationship but not cohabiting, and 28% were single. About 43% had not been deployed before. Approximately two months after returning home, 247 soldiers were re-tested, using a diagnostic interview for PTSD (response rate = 99%), and questionnaires measuring stressors on deployment and PTSD symptom severity (response rate = 96%). Non-response was due to undeliverable addresses (e.g., after base transfer) and withdrawal.

Participants were tested individually on military bases across the Netherlands. At various sites, infantry and engineer troops were selected on basis of availability during their preparation program. There were no exclusion criteria in the present study. A few

days after they received general information about the study from their commanding officers, they met the researchers who gave each individual full oral and written information about the study. Participants were told that participation was strictly voluntary without financial compensation, that commanders and participants would be informed only about pooled results, and that refusing to participate would have no negative consequences.

After providing written informed consent, participants completed the pre-deployment assessment, which included the conditioning task, the PTSD symptom scale, and the neuroticism scale (see below). After deployment, deployment stressors were assessed using questionnaires, and PTSD diagnosis and severity were assessed using questionnaires and a diagnostic interview, administered by trained research psychologists. Measures were administered in Dutch.

Because drop-out rates are usually rather high in longitudinal research of military personnel (e.g., due to the high turnover rate; Engelhard, van den Hout, Weerts, Arntz, Hox, & McNally, 2007; Hotopf & Wessely, 2005), strategies for retaining participants were used, such as developing positive relationships (e.g., informing participants about the progress of the study, giving information about names and roles of research personnel), selecting and training suitable research personnel, testing participants at their military base, and obtaining information from participants to facilitate relocating them (cf. Grant, Raper, Kang & Weaver, 2008). This study was part of a larger project and was approved by the Institutional Review Board of Maastricht University.

Measures

De novo conditioning task

CSs were pictures of human faces that were individually evaluated as neutral (cf. Hermans et al., 2002). The US was a mild electric shock (500 ms, 0.2-4.0 mA) on two fingers of the non-dominant hand that was individually set at a 'highly annoying but not painful' level using a work-up procedure prior to the task (cf. Orr et al., 2000). The task consisted of a habituation phase with 4 presentations of the CS+ and CS-, followed by the acquisition phase consisting of 8 presentations of CS+ immediately followed by the US, and 8 presentation of the CS-. During the extinction phase, the CS+ and CS- were presented 16 times without the US. The last 8 trials were included with respect to a subsequent affective priming task in which CSs and an equal number of general negative and positive words were presented, which will not be reported here. Stimuli were presented for 8 s with an intertrial interval (ITI) of 2-5 s in a semi-random order, so that no

more than two successive stimuli (CS+, CS-) or ITIs of the same latency were presented. During the 8-s stimulus presentations, participants were asked to rate their US-expectancy on a 0-100 visual analogue scale (VAS; 0 = *certain no electric stimulation*; 100 = *certain electric stimulation*) on the screen. US-expectancy has sufficient external validity as verbal outcome measure in human fear conditioning (Boddez et al., 2012).

Extinction learning

Extinction learning is the change in conditioned responding over extinction trials (Lissek et al., 2005), and typically follows an asymptotic curve (Myers & Davis 2002). Studies using a differential conditioning task in which each CS+ was followed by a US in the acquisition phase showed the greatest reduction in the first 4 to 5 trials of the extinction phase (e.g., Orr et al., 2000). Therefore, we operationalized individual differences in extinction learning as the difference between US-expectancy to the first and the fourth CS+ extinction trial (CS+ trial 1 minus CS+ trial 4). Higher scores on extinction learning reflect a greater decline in US-expectancy of the CS+ in the extinction phase. Reduced fear extinction learning might be overcome with increases in the number of extinction trials (Norrholm et al., 2011), so US-expectancy may decrease eventually with more extinction trials.

Posttraumatic stress

The Dutch version (van Groenestijn, Akkerhuis, Kupka, Schneider, & Nolen, 1999) of the well-established Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon & Williams, 1996) was used to diagnose PTSD.

The Dutch version (Engelhard, Arntz, & van den Hout, 2007) of the Posttraumatic Symptom Scale – Self Report (PSS; Foa, Riggs, Dancu & Rothbaum, 1993) contains the 17 DSM-IV PTSD symptoms that were each rated on a 0 (*not at all*) to 3 (*almost always*) scale for the prior month. This scale is valid and reliable (Foa et al., 1993; Engelhard, Arntz et al., 2007). Before deployment, participants rated the PSS with respect to the life-event that troubled them the most. This score reflected baseline symptom severity. After deployment, the PSS was completed with respect to deployment-related event(s) that troubled them the most. The sum score was the main outcome.

Stressful events

The Potentially Traumatizing Events Scale (PTES; Maguen, Litz, Wang & Cook, 2004) includes 21 items that assessed frequency of exposure to war-zone related stressors.

For the Dutch version, translation and back-translation was used, one item was omitted ("patrolling areas where there were land mines"), and four were added, including "Having injured civilians due to own action", "being formally told that a colleague got killed" (cf. Engelhard & van den Hout, 2007), "seeing dead or injured Afghan soldiers or police", and "conflict situation with Afghan police". Another item was adjusted to the Afghan context ("patrolling through the zone of separation" was changed to "stand guard during patrol"). For each event, participants indicated whether they had experienced it in Afghanistan. The sum score reflected stressor severity (range 0-24)[1].

Neuroticism

The Dutch version (Sanderman, Arrindell, Ranchor, Eysenck & Eysenck, 1991) of the neuroticism scale of the Eysenck Personality Questionnaire – short version (EPQ-N; Eysenck & Eysenck, 1975) assessed neuroticism. Psychometric properties of this scale are good (Sanderman et al., 1991). This widely used scale consists of 22 items that can be answered with *yes* (= 1) or *no* (= 0). The sum score was used.

RESULTS

Descriptives

There were no casualties in this sample during deployment. On average, participants reportedly experienced 14 war-zone related stressors (*SD* = 4.7), including witnessing an explosion (84.6%), being shot at (60.1%), having to remove human remains (36.5%), and seeing dead or injured Dutch soldiers (23.1%). There were no differences between soldiers who were deployed before and who were not, with respect to PSS scores before deployment, $t = .01$, $p = .99$, PSS scores after deployment, $t = 1.61$, $p = .11$, or extinction learning either, $t = 1.67$, $p = .10$. According to the SCID, 2.8% met the diagnostic criteria for (partial) PTSD related to stressors during deployment. A low PTSD prevalence is typically found in Dutch and British troops (Engelhard, van den Hout et al., 2007; Sundin, Forbes, Fear, Dandeker, & Wessely, 2011).

Extinction learning and posttraumatic stress

For further analyses, we excluded two participants who did not understand the conditioning task instructions, and 31 participants who did not learn the CS-US contingency in the acquisition phase[2]. Figure 1 shows an increasing differential US-expectancy during the acquisition phase. A corresponding ANOVA showed the expected Trial (a1; a8) x CS type (CS-; CS+) interaction, $F(1,208) = 909.60$, $p < .001$, partial η^2

= .81. Paired t-tests showed that US-expectancies increased over time for the CS+, $t(211) = 27.34$, $p < .001$, and decreased for the CS-, $t(208) = 14.69$, $p < .001$.[3] During the extinction phase, a significant interaction between trial (e1; e8) and CS type (CS-; CS+) was observed, $F(1,207) = 148.20$, $p < .001$, partial $\eta^2 = .42$. Paired t-tests showed that US-expectancies decreased over time for the CS+, $t(208) = 26.37$, $p < .001$, and for the CS-, $t(210) = 9.98$, $p < .001$.

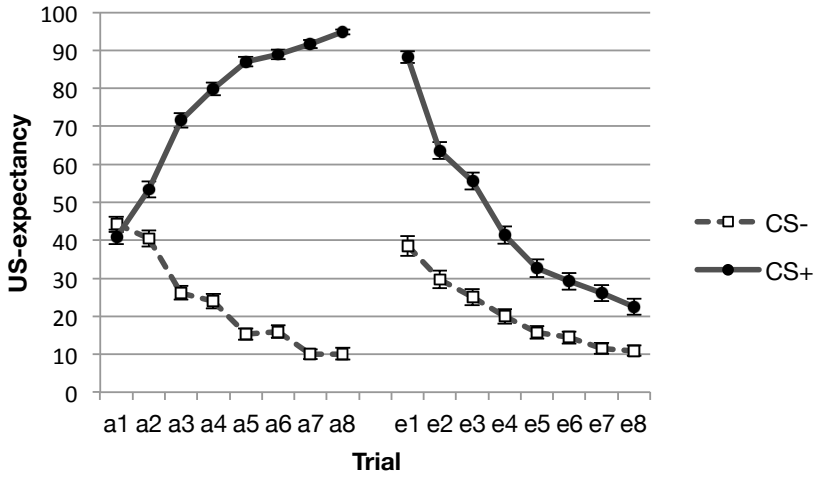

Figure 1. Graphical display of US-expectancies throughout the acquisition (a) and extinction (e) phase. Error bars represent standard errors of the mean.

PSS scores were skewed to the right, and were transformed with square root to a less skewed distribution. Correlations among psychometric variables, resulted in significant, medium effect size associations between all predictors and the post-deployment PSS score (Table 1). Reduced fear extinction was associated with increased PSS scores after deployment.

Table 1

Pearson correlations among psychometric variables

	1.	**2.**	**3.**	**4.**	**5.**
1. PSS post-deployment	1				
2. Extinction learning	-.19**	1			
3. PSS pre-deployment	.32***	-.02	1		
4. EPQ-N pre-deployment	.38***	.05	.48***	1	
5. PTES	.24***	-.13	.04	.07	1

Note. * $p<.05$ ** $p<.01$ *** $p<.001$

To examine the predictive value of pre-trauma fear extinction learning, a regression analysis was conducted with PTSD symptom severity after deployment (PSS score, *M* = 3.1, *SD* = 3.8) as dependent variable and extinction learning as independent variable. Results showed that reduced extinction learning significantly predicted higher PSS scores after deployment, even after controlling for baseline PSS scores, neuroticism and stressor severity, which accounted for 23% of the variance (Table 2). Although PSS scores and neuroticism before deployment, and the number of experienced events during deployment significantly predicted PSS scores after deployment, extinction learning explained unique variance in PSS scores after deployment.

Table 2

Regression analyses predicting PTSD symptom severity after deployment, with corresponding model information and beta weights

	Model		**Beta weights**	
	R^2 (f^2)	*F*	*ß*	*t (p)*
Model 1	.03 (.04)	6.77		
Extinction learning			-.18	-2.60 (.01)
Model 2	.23 (.30)	14.20		
Extinction learning			-.17	-2.69 (.01)
PSS pre-deployment			.18	2.50 (.01)
EPQ-N pre-deployment			.27	3.68 (<.001)
PTES			.17	2.70 (.01)

The predictive value of reduced extinction learning decreased with more extinction trials,[4] but the pattern remained similar: less extinction learning was associated with greater PSS scores at posttest.

To test if non-associative effects might account for the relationship between extinction learning and posttraumatic stress, the analysis was rerun for US-expectancy to the CS- extinction trials (i.e., CS- trial 1 minus CS- trial 4). These scores were not significantly correlated with the PSS at the posttest, r = -.09, p = .21.

DISCUSSION

Results show that reduced fear extinction before deployment predicted subsequent PTSD symptom severity, over and beyond degree of pre-deployment stress symptoms, neuroticism, and exposure to stressors on deployment. To the best of our knowledge, this is the first large, prospective study on extinction learning in PTSD that controls for pre-trauma symptoms and known risk factors. Cross-sectional studies have found that participants with PTSD show reduced fear extinction (Blechert et al., 2007; Orr et al., 2000; Peri et al., 2000) compared to healthy controls with and without trauma exposure. The temporal nature of those findings was unclear: reduced extinction learning might be a result of PTSD, or it may reflect a pre-trauma vulnerability factor for PTSD. Consistent with the preliminary study of Guthrie and Bryant (2006), the current study suggests that reduced extinction learning before trauma puts people at risk of later PTSD symptoms. However, it should be noted that the importance of extinction learning in predicting PTSD symptomatology was modest, and there were other predictors (e.g., neuroticism scores). An interesting area for future research may be to examine why extinction learning fails in some individuals. As extinction learning is mainly an inhibitory process, medial prefrontal cortex functioning may help to explain these individual differences (Quirk, Garcia & González-Lima, 2006).

Before definite conclusions can be drawn, a few considerations should be taken into account. First, the current findings were obtained in a military population that consisted predominantly of healthy young men, with low PSS-scores and low PTSD prevalence, which may raise questions about the generalizability to other populations (e.g., women) and traumatic events. However, reduced extinction learning has been found in various populations (e.g., Blechert et al., 2007; Peri et al., 2000), which suggests that individual differences in extinction learning may be relevant across various traumatic events and populations (Lissek et al., 2005; Mineka & Oehlberg, 2008). Second, only ratings of shock expectancy (as a self-reported measure of learning) were included, rather than also using an objective measure of anxious arousal (e.g., skin conductance response; Lovibond et al., 2009). Although Guthrie and Bryant (2006) found similar results with a physiological measure, and similar patterns of US-expectancies and physiological responses have been observed in some earlier studies (Lovibond, Davis & O'Flaherty, 2000), there is also evidence that the pattern may be different (Blechert et al., 2007). Future studies may address this issue by including multiple indices of fear extinction learning (e.g., physiological and cognitive). Third, there was no PTSD diagnostic interview before deployment, while 57% had been deployed before. However, the relationship

between extinction learning and PTSD symptom severity after deployment remained after controlling for the PTSD symptom severity before deployment, and soldiers who were deployed before did not differ in PTSD symptom severity before deployment from those who had not been deployed before. Moreover, soldiers are screened by the military on mental health before deployment. Fourth, inter-rater reliability of the clinical interview for PTSD was not assessed. Strengths of our study include the prospective design, large sample size, statistical control for relevant PTSD predictors, and low drop-out rate.

The findings are nicely in line with contemporary learning theory that emphasizes the role of CS-US contingencies in the persistence of PTSD and other anxiety disorders (Mineka & Oehlberg 2008). Treatments that are effective for PTSD and other anxiety disorders use exposure-based techniques to enhance the learning of CS-no US contingency (Craske et al., 2008). Our findings support the use of exposure-based techniques that enhance the learning of CS-no US contingency (Craske et al., 2008) and pharmacological interventions that may facilitate extinction (e.g., Davis, Ressler, Rothbaum & Richardson, 2006). Knowledge about strong predictors for PTSD might ultimately be used in primary or secondary prevention in high-risk professions (like firefighters or soldiers) to identify those who are potentially vulnerable to develop PTSD. In conclusion, this prospective study shows that individual differences in fear conditioning before trauma predict later development of PTSD symptom severity, which suggests that reduced fear extinction is a pre-trauma vulnerability factor for PTSD.

FOOTNOTES

[1] For each event that was experienced, participant also rated its negative impact. This subjective severity index correlated strongly with the number of experienced events, $r = .75$, and revealed similar patterns in the analyses.

[2] Participants with a US-expectancy lower than 60 for the last CS+ trial in the acquisition phase were classified as non-learners, and were excluded because acquisition learning is needed for extinction learning to occur (cf. Blechert et al., 2007; Mason & Richardson, 2010). We repeated the analyses with different cut-off scores used by other authors (e.g., cut-off of 70 for the last CS+ acquisition trial, and adding a cut-off for CS- trials; Guthrie & Bryant, 2006), which resulted in similar results. Non-learners did not differ with the learners with respect to posttest PSS scores, t (234) = 1.49, $p = .14$, severity of stressor exposure, t (233) = 2.04, $p = .37$, or demographic variables (age, gender, education, prior deployment).

[3] Acquisition learning seems to be enhanced in patients with anxiety disorders (Lissek et al., 2005), however, enhanced acquisition (defined as the US-expectancy of the fourth CS+ presentation minus the first CS+ presentation in the acquisition phase) was not associated with PSS score at pretest or posttest (p>.05) and did not predict PSS scores at posttest (p > .05) in our sample.

[4] Defining extinction learning as CS+ trial 1 minus CS+ trial 5 also predicted later PTSD symptom severity, β = -.16, t = 2.30, p = .02. Using a definition based on CS+ trial 6, 7 or 8, extinction learning no longer significantly predicted PTSD symptom severity, although the pattern remained similar, with the smallest effect for trial 8, β = -.11, t = 1.51, p = .13.

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Chapter 3

Anger: cause or consequence of posttraumatic stress? A prospective study of soldiers

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ABSTRACT

Many studies have shown that individuals with posttraumatic stress disorder (PTSD) experience more anger over time and across situations (i.e. trait anger) than trauma-exposed individuals without PTSD. However, there is a lack of prospective research that controls for anger levels *before* trauma exposure. The aim of this study was to prospectively assess the relationship between trait anger and PTSD, when controlling for several known risk factors, including baseline symptoms, neuroticism, and stressor severity. Participants were 249 Dutch soldiers tested before (approximately 2 months) and after (approximately 2 months and 9 months) their deployment to Afghanistan. Trait anger and PTSD symptom severity were measured at all assessments. Structural equation modeling including cross-lagged effects showed that higher trait anger *before* deployment predicted higher PTSD symptoms 2 months after deployment, even when controlling for baseline symptoms and stressor severity, but not after controlling for neuroticism. Trait anger at 2 months post-deployment did not predict PTSD symptom severity at 9 months, and PTSD symptom severity about 2 months post-deployment did not predict subsequent trait anger scores. Findings suggest that trait anger is a pretrauma vulnerability factor for PTSD, but does not reach beyond the effect of neuroticism.

There is a growing interest for the role of anger in anxiety disorders, especially posttraumatic stress disorder (PTSD; Olatunji, Ciesielski, & Tolin, 2010). Individuals with PTSD experience more angry feelings currently (state anger), and over time in response to a variety of situations (trait anger) when compared to non-PTSD trauma-exposed controls (Chemtob, Hamada, Roitblad, & Muraoka, 1994; Jakupcak et al, 2007; Lasko, Gurvits, Kuhne, Orr, & Pitman, 1994). The relationship between anger and PTSD is observed in male and female samples, and across types of traumatic events, with the largest effect size in military samples (Orth & Wieland, 2006). Since especially trait anger is related to problematic behavior such as (intimate partner) violent behavior (Norlander & Eckhardt, 2005), it seems clinically relevant to understand the role of trait anger in PTSD.

How can the association between trait anger and PTSD be explained? First, it may be a methodological artifact, because 'irritability or outbursts of anger' is a PTSD symptom (APA, 2000). However, removal of this anger-item from a PTSD instrument does not substantially decrease the association, which excludes a methodological artifact (Novaco & Chemtob, 2002; Orth, Cahill, Foa, & Maercker, 2008). Second, trait anger may be a *vulnerability* factor for the onset of PTSD (cf. a diathesis-stress model, which states that individual differences in personality traits may predispose to a disorder that is triggered by a stressor; McKeever, & Huff, 2003), or the persistence of PTSD (cf. a pathoplasty model, which posits that personality traits may influence the course or expression of symptoms; Clark, Watson, & Mineka, 1994). Third, elevated levels of trait anger may be a *consequence* of PTSD (cf. a 'scar model', which states that a personality trait may be affected by the disorder; Clark et al., 1994). These models are not mutually exclusive, and the relationship between trait anger and PTSD may be reciprocal.

Most studies about the anger-PTSD link are limited by cross-sectional designs. However, some longitudinal studies have been conducted using state anger measures. These studies showed that levels of anger after a traumatic event predicted later posttraumatic stress (Jayasinghe, Giosan, Evans, Spielman, & Difede, 2008; Riggs, Dancu, Gershuny, Greenberg, & Foa, 1992; Ehlers, Mayou, & Bryant, 2003; Feeny, Zoellner, & Foa, 2000). Since level of trait anger reflects level of state anger over time and across situations, these constructs correlate positively (Deffenbacher, 1992). Consequently, the longitudinal data seem to support the diathesis-stress model and pathoplasty model. However, the evidence is not unequivocal. A recent study with a large sample of crime victims found that PTSD symptoms predicted subsequent levels of state anger, but not the reverse (Orth et al., 2008), supporting the scar model. These longitudinal studies with assessments after trauma exposure lack control for the effects

of trauma exposure or baseline ('pretrauma') PTSD symptoms on anger levels. Prospective research in which anger is assessed *before* trauma exposure and baseline symptoms are taken into account is scarce. To our knowledge, only three such studies have been published so far. One study tested 43 firefighters immediately after, and at several time points 6-24 months after their training, showing that initial higher levels of hostility predicted PTSD symptoms at 24 months after training (Heinrichs et al., 2005). Levels of hostility in 470 soldiers before deployment also predicted PTSD symptoms 6 months after deployment (van Zuiden et al., 2011). Another study among 180 police cadets showed that trait anger during training predicted PTSD symptoms 12 months later (Meffert et al., 2008). These data support the diathesis-stress model, but there are several methodological limitations. First, these studies did not all control for initial PTSD symptoms (Heinrichs et al., 2005; Meffert et al., 2008), and trauma exposure (Meffert et al., 2008; van Zuiden et al., 2011). Second, none of these studies tested whether PTSD predicted subsequent trait anger. Thus, the temporal relationship between trait anger and PTSD remains fairly unclear.

Another important question is whether anger predicts PTSD beyond well-known vulnerability factors. Increased anger levels may reflect a specific negative emotion at a lower order level of the more general trait of neuroticism (or 'negative affectivity'; Watson & Clark, 1992), which is related to anxiety disorders and depression (Clark et al., 1994). Prospective research has shown that pretrauma neuroticism scores predict later PTSD symptoms (Engelhard, van den Hout, & Lommen, 2009; van den Hout & Engelhard, 2004). Therefore, it seems important to test whether the relationship between trait anger and PTSD symptoms remains after controlling for pretrauma neuroticism.

In the current study, 249 Dutch soldiers were tested 2 months before their deployment to Uruzgan, Afghanistan, and twice (2 months and approximately 9 months) after returning home. Using structural equation modeling, the temporal relationship between trait anger and PTSD symptom severity was tested, when controlling for baseline symptoms, stressor severity, and neuroticism. Furthermore, it was tested if the relationship could be explained by content overlap by excluding the irritability item from the PTSD measure. We hypothesized that trait anger before deployment predicted PTSD symptom severity 2 months after deployment, and predicted PTSD symptoms at 9 months directly or indirectly through earlier PTSD symptom severity. We expected the predictive value of trait anger to remain after controlling for 'baseline' symptoms, stressor severity, content overlap, and neuroticism. Moreover, a positive relationship between PTSD symptom severity and trait anger after deployment was expected.

METHOD

Participants

Before a 4-month deployment to Afghanistan (November 2009 to March 2010), 250 Dutch Royal Army soldiers were asked to participate in this study. One refused, and the others agreed and were tested about two months before their deployment (Time 1; *N*=249). They completed questionnaires measuring anger, PTSD symptom severity, and neuroticism.

The sample (98% were male) had a mean age of 23.8 (*SD* = 4.9). The highest attained educational level was primary (2%), secondary (92%), or higher education (6%). About 34% were married or cohabiting, 38% were in a relationship but not cohabiting, and 28% were single. About 43% had not been deployed before.

Approximately two months after returning home (Time 2), 247 soldiers were re-tested, using a diagnostic interview for PTSD (response rate = 99%), administered by trained research psychologists, and questionnaires measuring anger, PTSD symptom severity, and deployment stressors (response rate = 96%). At follow-up (Time 3), about 9 months after returning home, 221 soldiers (response rate = 89%) were re-assessed using questionnaires. Testing typically took place on a military base, but for 29% the final assessment was by postal mail. Non-response was partly due to soldiers who were unreachable (e.g., after a transfer; 6%) or withdrew from the study (< 1%).

Soldiers were recruited on military bases across the Netherlands and selection was based on availability during their preparation program. After individual oral and written information about the study by the researcher, written informed consent was obtained. Participation was strictly voluntary without financial compensation. This study was part of a larger project (see Lommen, Engelhard, Sijbrandij, van den Hout, & Hermans, 2013), and was approved by the Institutional Review Board of Maastricht University.

Measures

Trait anger was assessed with the Dutch version (van der Ploeg, Defares, & Spielberger, 1982) of the State-Trait Anger Scale (STAS-T; Spielberger, Jacobs, Russell, & Crane, 1983), which is a former version of the State-Trait Anger Expression Inventory (STAXI; Spielberger, 1988). Trait anger was assessed with 10 items scored on a 4-point scale (1 = *almost never*; 2 = *sometimes*; 3 = *often*; 4 = *almost always*). The mean of the underlying items was computed. Internal consistency was good, with .85, .86, and .88 (Time 1, 2, and 3, respectively).

PTSD symptom severity was assessed with the Dutch version (Engelhard, Arntz, & van den Hout, 2007) of the Posttraumatic Symptom Scale - Self Report (PSS; Foa, Riggs, Dancu, & Rothbaum, 1993). The 17 DSM-IV PTSD symptoms were each rated on a 0 (*not at all*) to 3 (*almost always*) scale for the prior month. Before deployment, participants were instructed to rate the PSS with respect to their most aversive life-event that troubles them most at the time of testing. After deployment, the PSS was completed with respect to deployment-related event(s) that troubled them the most. The sum score was used for descriptive purposes. For the statistical analyses, the mean score of the 3 subscales (5 re-experiencing items, 7 avoidance items, 5 hyperarousal items) was used. The PSS is valid and reliable (Foa et al., 1993; Engelhard et al., 2007). Internal consistency was .53, .81, and .85 (Time 1, 2, and 3, respectively).

PTSD diagnosis was determined using the Dutch version (van Groenestijn, Akkerhuis, Kupka, Schneider, & Nolen, 1999) of the Structured Clinical Interview for DSM-IV Axis-I Disorders (SCID-I; First, Spitzer, Gibbon, & Williams, 1996).

Stressful events were assessed using a Dutch version (Engelhard & van den Hout, 2007) of the Potentially Traumatizing Events Scale (PTES; Maguen, Litz, Wang, & Cook, 2004). Of the original 21 items representing war-zone related stressors, one was omitted ("patrolling areas where there were land mines") and four were added ("having injured civilians due to own action" and "being formally told that a colleague got killed"; Engelhard & van den Hout, 2007) and "seeing dead or injured Afghan soldiers or police" and "conflict situation with Afghan police"). Another item was adjusted to the situation in Afghanistan ("patrolling through the zone of separation" was changed to "stand guard during patrol"). For each of the 24 events, participants indicated whether they had experienced it in Afghanistan. Additionally, for each event that was experienced, participant also rated its negative impact on a 1 (*no negative impact*) to 4 (*very much negative impact*) scale. We calculated the number of endorsed events (as indicator of 'stressor severity'), and the number of events scored at least 3 on the impact scale (as indicator of 'subjective stressor severity'; cf. Engelhard, van den Hout, & McNally, 2008).

Neuroticism was assessed with the Dutch version (Sanderman, Arrindell, Ranchor, Eysenck, & Eysenck, 1991) of the neuroticism scale of the Eysenck Personality Questionnaire – short version (EPQ-N; Eysenck & Eysenck, 1975). The EPQ-N consists of 22 items that were answered with *yes* (= 1) or *no* (= 0). The sum score was used. The scale has good psychometric properties (Sanderman et al., 1991). Internal consistency was .81 (Time 1).

Statistical analysis

Analyses were conducted with Mplus 6.11 (Muthén & Muthén, 2010). Cross-lagged analyses were used to assess the temporal relationship between the latent variables PTSD symptom severity (PSS) and trait anger (STAS-T).

First, subscales of the PSS and item parcels of the STAS-T were used in the structural equation model as indicators of latent variables (cf. Orth et al., 2008), because of the limited sample size in combination with our complex statistical model. Three unidimensional sets of items (item parcels; 2 of 3 items and 1 of 4 items) of the STAS-T were created based on statistical grounds (Kline, 2010; high item intercorrelations).

Second, to ensure that comparisons of the latent variables PSS and STAS-T over time are valid, the underlying structure of the latent variables should be invariant over time. In separate measurement models for PSS and STAS-T, we therefore applied measurement invariance. Only if the model did not show acceptable fit, we tested for measurement invariance (van de Schoot, Lugtig, & Hox, 2012).

Third, a cross-lagged panel model (fig. 1) was used to test the research questions. In this model, the latent factors of the previous step were used, assuming measurement invariance over time. Moreover, stability paths of PSS and STAS-T over time are estimated to control for previous PSS and STAS-T scores. Cross-sectional correlations and partial correlations between PSS and STAS-T were also estimated. The main interests are the cross-lagged paths between the PSS and STAS-T. If, after controlling for stability paths and cross sectional correlations, one or more cross-lagged paths appear to be significant, this indicates a longitudinal relation between the two variables over time. Additional structural models were run that controlled for baseline symptoms (PSS at time 1), stressor severity (PTES), and neuroticism (EPQ-N).

A robust maximum likelihood estimator (MLR) was used, because it is robust to non-normality of the data. Data appeared missing at random (i.e., missingness at time 2 did not depend on age, education, PSS at time 1, or STAS-T at time 1, missingness at time 3 did not depend on age, education, PSS at time 1 and 2, or STAS-T at time 1 or 2, $p > .05$). Therefore, participants with missing data were included in the analyses and full information maximum likelihood (FIML) was used to estimate our model. Model fit was evaluated using root mean square error of approximation (RMSEA, Steiger, 1990), comparative fix index (CFI; Bentler, 1990), and Tucker-Lewis index (TLI; Tucker & Lewis, 1973). Conventional guidelines were followed, considering <0.08 as an adequate fit for the RMSEA value, and >0.90 as adequate fit for CFI and TLI. The chi square (χ^2) is

reported to compare models. Paths in the cross-lagged panel models were tested one-sided (see Kline, 2010 for an overview of fit statistics).

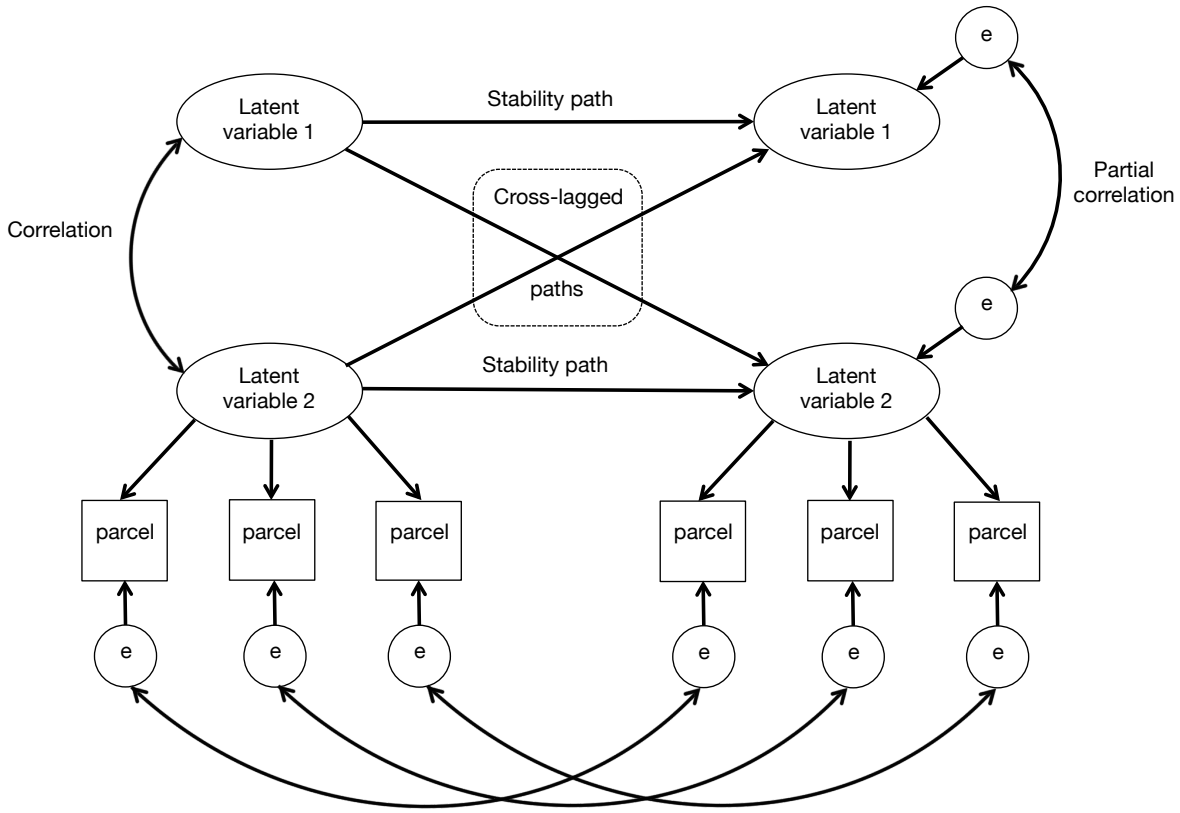

Figure 1. Cross-lagged panel model. For simplicity, the figure only shows parcels of latent variable 2, and a 2-panel instead of 3-panel design.

RESULTS

Descriptives

On average, participants reportedly experienced 14 (*SD* = 4.7) war-zone related stressors, including witnessing an explosion (85%), being shot at (60%), having to remove human remains (37%), and seeing dead or injured Dutch soldiers (23%). According to the SCID (time 2), 2.8 % met the diagnostic criteria for (partial) PTSD[1], compared to 5.0 % according to the PSS (item score of ≥ 1 indicates presence of criterion; Foa & Tolin, 2000). The mean PSS scores were 4.07 (*SD* = 4.79), 3.10 (*SD* = 3.81), and 4.05 (*SD* = 4.73), for the three assessments respectively.

Measurement models

Model fit of the measurement models was evaluated with confirmatory factor analysis (CFA), in which the factor loadings and intercepts of the latent variable were constrained to be equal over time (constrained CFA model) to apply measurement invariance.

For PSS at time 1, 2 and 3, the constrained CFA model revealed an unacceptable fit, χ^2 = 307.51, RMSEA = 0.22, CFI = 0.44, TLI = 0.17. Factor loadings at time 1 appeared to be different from factor loadings at time 2 and 3, as evident from the unconstrained CFA model in which the factor loadings and intercept are freely estimated. The constrained CFA model including only PSS at time 2 and 3 showed acceptable fit, χ^2 = 16.68, RMSEA = 0.06, CFI = 0.97, TLI = 0.96. Since the factor loadings of the PSS at time 1 were different than the factor loadings at time 2 and 3, measurement invariance can be assumed for the PSS only at time 2 and 3. Consequently, PSS at time 1 should be regarded as a different construct, and should be included in the cross-lagged model with factor loadings and intercepts that are not constrained to be equal to PSS factor loadings at time 2 and 3.

For STAS-T at time 1, 2, and 3, the constrained CFA model, χ^2 = 69.23, RMSEA = 0.09, CFI = 0.95, TLI = 0.93, showed acceptable fit[2]. This implies that the construct is invariant over time, thus the constrained CFA model is used in further analyses.

Cross-lagged models

To test the temporal relation between PSS and STAS-T, a cross-lagged panel model including PSS at time 2 and 3, and STAS-T at time 1, 2, and 3 was run (model 1, fig. 2). In model 2, we controlled for the PSS at time 1 (without constraints on factor loadings and intercepts). In model 3 (fig. 3), we controlled for PSS at time 1, and PTES. In model 4 (fig. 4), we controlled for EPQ-N and PTES.

In model 1 (fig. 2), stability paths, correlations and auto-correlations were all significant. The only significant cross-lagged path was trait anger at time 1 as a predictor of PSS at time 2. Further, to make sure that the relation between PSS and trait anger does not depend on content overlap, model 1 was re-analyzed with the irritability item removed from the PSS. Results showed similar patterns and conclusions as the model including the irritability item (trait anger at time 1 predicted PSS at time 2, β = .33, p < .01). Moreover, only a minor decrease in correlation between PSS and STAS-T at time 2 (r = .57) and time 3 (r = .52) was observed, which indicates that the relation between PSS and STAS-T is not a methodological artifact.

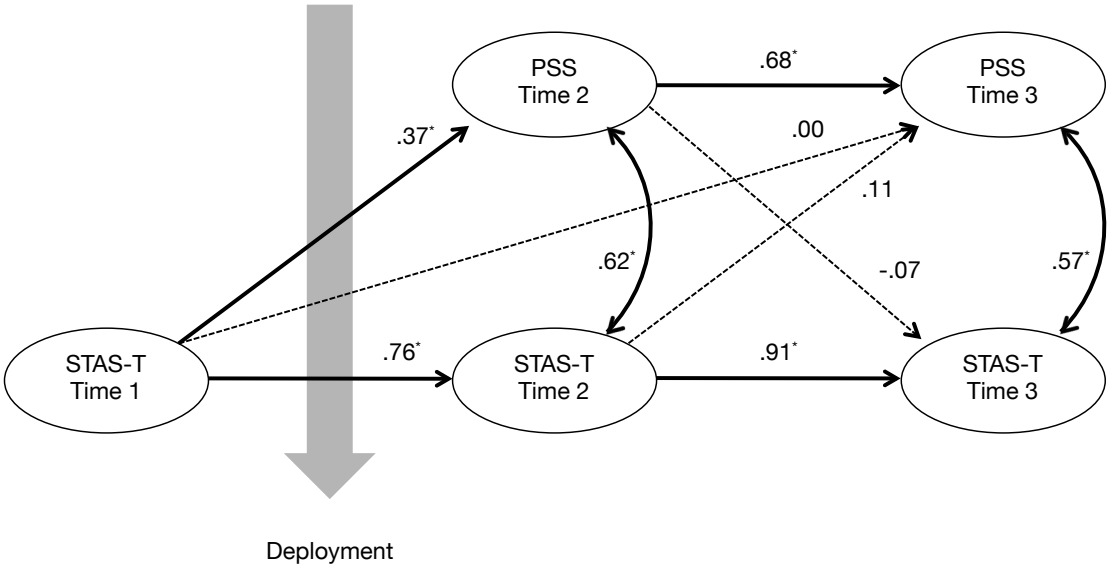

Figure 2. Model 1, including trait anger (STAS-T) at time 1, 2, and 3, PTSD symptom severity (PSS) at time 2, and 3. Values shown are standardized coefficients. Solid lines represent significant relations, dashed lines represent non-significant relations.
*$p < .05$

In model 2, PSS at time 1 was entered in the model with no constraints on factor loadings and intercepts. The similar pattern as in model 1 was observed, with STAS-T at time 1 as a predictor of PSS at time 2 as the only significant cross-lagged path. PSS at time 1 significantly predicted PSS at time 2, but did not predict STAS-T at time 2.

In model 3 (see fig. 3), stressor severity was added to model 2. Also model 3 revealed a similar pattern of results, with STAS-T at time 1 as a predictor of PSS at time 2 as the only significant cross-lagged path. PTES significantly predicted PSS at time 2, but not STAS-T at time 2. Because appraisal of the events may be stronger related to PTSD symptomatology than 'objective' criteria of threat (Bovin & Marx 2011; Bowman, 1999), model 3 was re-analyzed with the subjective stressor severity index, yielding a similar pattern of results.

In model 4 (see fig. 4), model 1 was extended while controlling for EPQ-N and PTES. The stability paths, correlations and auto-correlations were all significant, but none of the cross-lagged paths reached significance. EPQ-N at time 1 significantly predicted PSS at time 2, but not STAS-T at time 2.

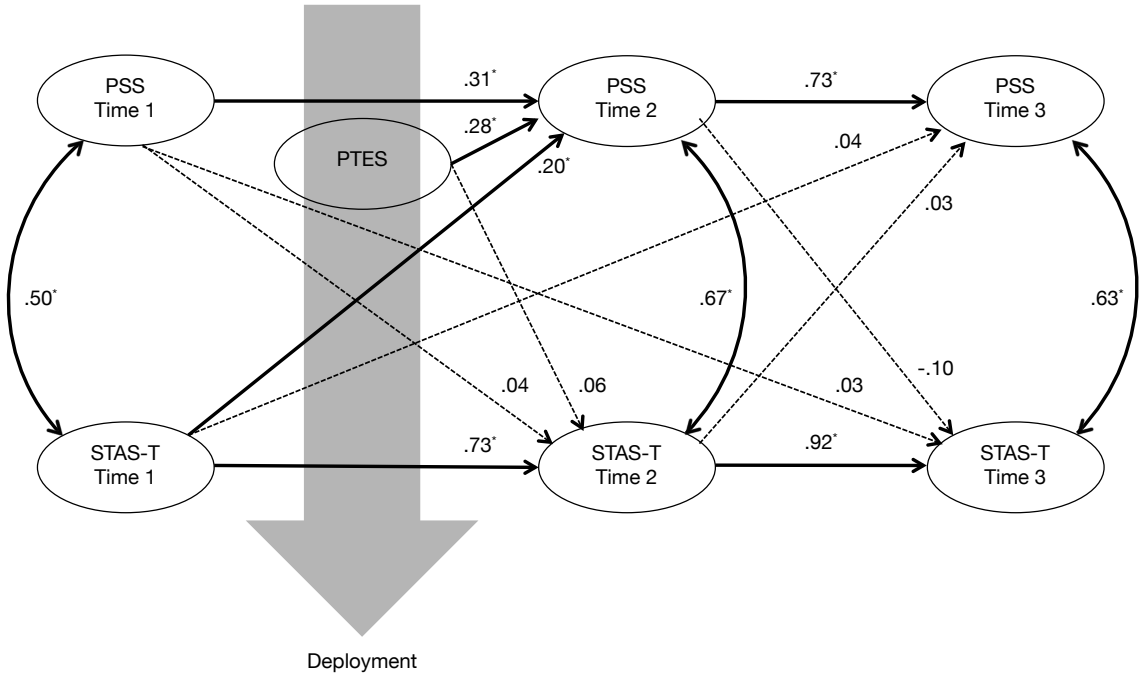

Figure 3. Model 3, including trait anger (STAS-T) at time 1, 2, and 3, PTSD symptom severity (PSS) at time 1, 2, and 3, and stressor severity (PTES). Values shown are standardized coefficients. Solid lines represent significant relations, dashed lines represent non-significant relations. * $p < .05$

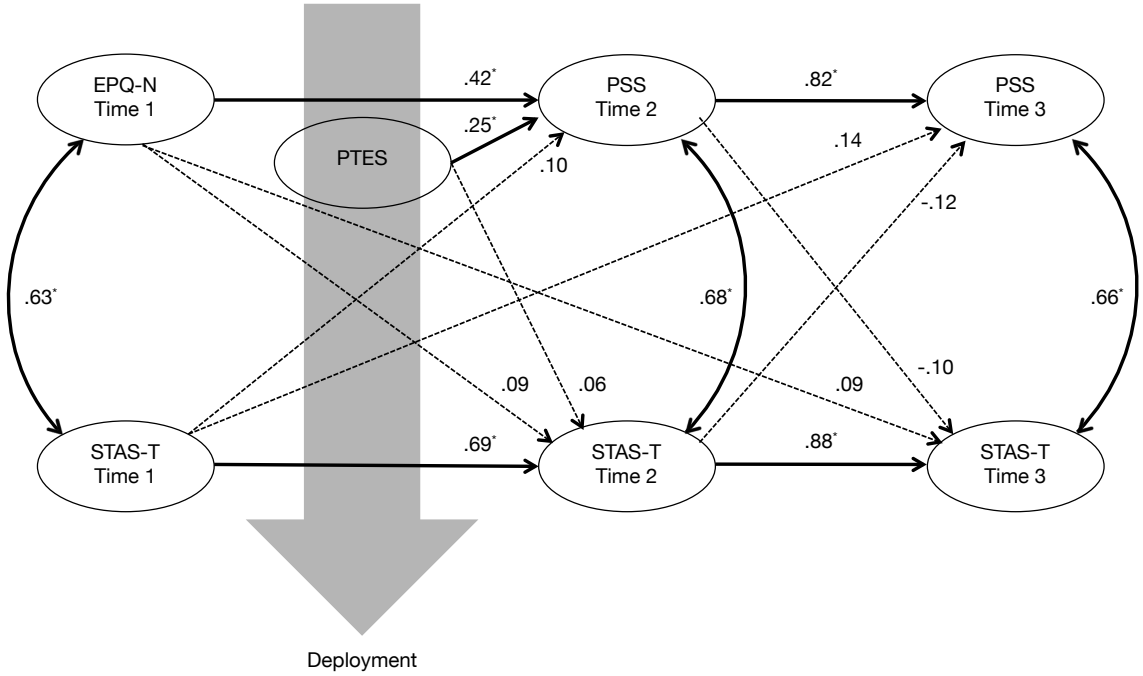

Figure 4. Model 4, including trait anger (STAS-T) at time 1, 2, and 3, PTSD symptom severity (PSS) at time 2, and 3, neuroticism (EPQ-N) at time 1, and stressor severity (PTES). Values shown are standardized coefficients. Solid lines represent significant relations, dashed lines represent non-significant relations. * $p < .05$

In sum, all four models had acceptable model fit (table 1), and resulted in the conclusion that STAS-T at time 1 significantly predicted PSS at time 2 when controlling for PSS at time 1, and PTES, but not after control for EPQ-N. No other cross-lagged paths were significant. Explained variances of the predicted latent variables are shown in Table 2.

Table 1

Model fit information

	χ^2	*df*	*p*	RMSEA	CFI	TLI
Model 1	152.95	54	<.01	.06	.95	.93
Model 2	231.02	67	<.01	.06	.93	.92
Model 3	255.64	69	<.01	.06	.93	.91
Model 4	187.27	61	<.01	.06	.95	.94

Note. Model 1: STAS-T at time 1, 2, and 3, PSS at time 1, and 2
Model 2: STAS-T at time 1, 2, and 3, PSS at time 1, 2, and 3
Model 3: STAS-T at time 1, 2, and 3, PSS at time 1, 2, and 3, and PTES
Model 4: STAS-T at time 1, 2, and 3, PSS at time 2, and 3, EPQ-N at time 1, and PTES

Table 2

Explained variance of trait anger and PTSD symptom severity

Model	Time 2		Time 3	
	STAS-T	PSS	STAS-T	PSS
Model 1	.58	.14	.75	.57
Model 2	.57	.21	.74	.57
Model 3	.57	.28	.75	.59
Model 4	.57	.30	.75	.63

Note. Model 1: STAS-T at time 1, 2, and 3, PSS at time 1, and 2
Model 2: STAS-T at time 1, 2, and 3, PSS at time 1, 2, and 3
Model 3: STAS-T at time 1, 2, and 3, PSS at time 1, 2, and 3, and PTES
Model 4: STAS-T at time 1, 2, and 3, PSS at time 2, and 3, EPQ-N at time 1, and PTES

An additional analysis was run to assess the indirect effect of STAS-T at time 1 at PSS at time 3 through PSS at time 2, which indicated a significant indirect effect in model 1, β = .25, p < .01, and in model 2, β = .14, p = .05. The indirect effect did not reach significance in model 3, β = .15, p = .06, and model 4, β = .08, p = .17.

DISCUSSION

This prospective study examined the temporal relationship between trait anger and PTSD symptom severity in Dutch soldiers who were assessed before, and twice after their deployment to Afghanistan, using a cross-lagged panel analysis in a structural equation modeling framework. The main findings can be summarized as follows. First, in line with our hypothesis, trait anger before deployment predicted PTSD symptom severity 2 months after deployment, and indirectly at 9 months after deployment through PTSD symptom severity shortly after deployment. Second, contrary to our expectations, trait anger 2 months after deployment did not predict PTSD symptom severity at 9 months, and PTSD symptom severity 2 months after deployment did not predict trait anger at 9 months after deployment. Third, the predictive effect of pre-deployment trait anger on subsequent PTSD symptom severity remained after controlling for baseline symptoms, stressor severity, and content overlap. However, the effect disappeared after controlling for neuroticism.

To the best of our knowledge, this is the first prospective study on PTSD that assessed trait anger over time, and controlled for baseline symptoms. The current finding that pre-deployment trait anger predicts subsequent PTSD symptom severity is in line with other prospective studies (Heinrichs et al., 2005; Meffert et al., 2008; van Zuiden et al., 2011), supporting the diathesis-stress model. Trait anger after deployment did not predict later PTSD symptom severity, which contradicts the pathoplasty model, and PTSD symptom severity initially after deployment did not predict trait anger at follow-up. The latter suggests that trait anger is not affected by PTSD symptom severity, which is in contrast with predictions according to the scar model. An earlier longitudinal study that tested the temporal relation between anger and PTSD did not find effects of anger on PTSD symptoms either, but in contrast with our results, PTSD symptoms predicted subsequent anger (Orth et al., 2008). Difference in measures may account for the different findings, since Orth et al. (2008) measured state anger, which might be more sensitive for change than trait anger.

By controlling for neuroticism in the current study, the literature on the relation between trait anger and neuroticism is extended. Our findings replicate earlier findings that neuroticism predicts PTSD (Bramsen, Dirkzwager, & van der Ploeg, 2000; Engelhard, van den Hout, & Lommen, 2009; Parslow, Jorm, & Christensen, 2006; van den Hout & Engelhard, 2004). The results of the current study indicate that neuroticism and trait anger are both vulnerability factor for PTSD symptom severity, with neuroticism as the stronger predictor. Trait anger seems to reflect a lower-order factor of the more general

vulnerability factor neuroticism (cf. Watson & Clark, 1992). A challenging question is through what mechanism pretrauma neuroticism and trait anger predict later PTSD symptom severity. In concordance with the survival-mode theory (Novaco & Chemtob, 2002), anger, similar to neuroticism and trait anxiety, may facilitate threatening interpretations of ambiguous stimuli (Barazzone & Davey, 2009; Wenzel & Lystad, 2005). Furthermore, neuroticism is associated with avoidance of (harmless) ambiguous stimuli (Lommen, Engelhard, & van den Hout, 2010). As negative appraisals of (the sequelae of) a traumatic event and behavioural strategies, like avoidance, intended to control perceived current threat play an important role in the maintenance of PTSD symptoms (e.g., Bovin & Marx, 2011; Ehlers & Clark, 2000; van den Hout & Engelhard, 2004), anger and neuroticism may contribute to PTSD development.

Some limitations of this study should be taken into account. First, we tested a specific sample consisting of mainly healthy, young men. It is unclear to what extent the current findings generalize to other trauma types and samples. However, two earlier studies that tested other occupational samples at risk of trauma exposure have found similar results on the predictive value of pretraumatic anger (Heinrichs et al., 2005; Meffert et al., 2008). Second, PTSD prevalence was low, which is consistent with earlier studies of soldiers deployed to Iraq or Afghanistan, with PTSD rates about 4% in European samples (Engelhard et al., 2007; Sundin, Forbes, Fear, Dandeker, & Wessely, 2011), and 8% in US samples (McNally, 2012). Consequently, there was less variance for predictive variables. Third, baseline symptoms assessed with the PSS as time 1 loaded on different factors than the PSS at time 2 and 3, and internal consistency of the PSS at time 1 was low. Both probably reflect the heterogeneity of the group at time 1. The different factor loadings at time 1 versus time 2 and 3 are not surprising, as these are anchored to different stressful events. Nevertheless, it makes the understanding of the results somehow more complex. Fourth, trait anger reflects the experience of angry feelings over time and does not necessarily lead to (problematic) anger expression (Eckhardt, Barbour, & Stuart, 1996). Although trait anger may predispose to PTSD psychopathology, it might not be problematic in itself. In the current study, mean trait anger scores were relatively low compared to a reference group of randomly selected young men (< 40 years) in the Netherlands (van der Ploeg, Defares, & Spielberger, 1982). Before deployment, only 14.8% of our sample scored above the average score of the reference group. About two months after deployment, this was 11.6%, and about nine months after deployment, it was 18.0%. It is seems unlikely that trait anger was underreported in our study, because the PTSD self-report questionnaire showed that

41.2% reported they “sometimes” have had heightened irritability or anger outbursts during the prior month.

Strengths of this study include the prospective, longitudinal design with a measurement before trauma exposure, the large sample with low attrition rates, inclusion of a trait anger measure over time, control for significant risk factors of PTSD, and the statistical method to analyze the data.

To summarize, our findings suggest that trait anger is a pretrauma vulnerability factor for the development of PTSD, but does not explain unique variance in PTSD symptom severity over and above the higher-order trait variable of neuroticism. Individuals with a high level of trait anger or neuroticism before trauma exposure might be at greater risk to develop later PTSD symptoms. Trait anger did not seem to be a consequence of PTSD symptomatology.

FOOTNOTES

[1] One participant met criteria for PTSD, 6 persons met criteria for partial PTSD (Engelhard, van den Hout, Arntz, & McNally, 2002), mainly due to not meeting avoidance criteria.

[2] The RMSEA value was .087, which is at the boundary of an acceptable fit. Since this cut-off is debatable (e.g., <.10 according to Kline, 2010, p. 206) and other fit indices (CFI and TLI) showed good fit, we evaluated the model fit as acceptable.

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Chapter 4

Neuroticism and avoidance of ambiguous stimuli: Better safe than sorry?

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ABSTRACT

Neuroticism predisposes to anxiety disorders, but the precise pathogenic mechanism is unknown. The aim of this study was to examine whether people with high neuroticism scores use a lower criterion for detecting danger in the face of ambiguous stimuli, and avoid a greater number of ambiguous stimuli than people with low neuroticism scores. Participants high and low in neuroticism were administered a differential conditioning task, in which one conditioned stimulus (CS+; colored circle) was followed by an electric (unconditioned) shock (UCS), whereas another stimulus (CS-; different colored circle) was not. After this acquisition phase, degraded colours on a continuum between CS+ and CS- were presented and could be avoided by the participants, within a latency of 1 or 5 s. Results indicated that the high neuroticism group avoided more ambiguous stimuli than the low neuroticism group, but only at the 5 s. latency trials. The absence of differences at the 1 s. latency trials suggests the involvement of a *strategic* process. Apparently, when confronted with ambiguous threat signals, people high in neuroticism use a “better safe than sorry strategy”. By preventing disconfirmation of irrational fears, this strategy may be involved in the development and maintenance of anxiety disorders.

Neuroticism is a trait sensitivity to negative stimuli: individuals high in neuroticism are more likely to report distress, discomfort, and dissatisfaction over time, regardless of the situation, even in the absence of any overt or objective source of stress (Clark, Watson, & Mineka, 1994; Watson & Clark, 1984). Neuroticism, trait anxiety, and negative affectivity, showing strong intercorrelations (Jorm, 1989; Watson & Clark, 1984), refer to a similar construct, which is associated with affective disorders, including posttraumatic stress disorder (PTSD: Bowman, 1999; Breslau, Davis, Andreski, & Peterson, 1991; Cox, MacPherson, Enns, & McWilliams, 2004). One of the possible explanations for this relation is that high neuroticism individuals are more *reactive* to adverse events than low neuroticism individuals. To test this hypothesis, prospective studies are needed in which pre-event stress symptoms are assessed.

A study by Engelhard, van den Hout, and Kindt (2003) provides prospective data of a sample of pregnant women, who completed questionnaires during their early pregnancy to assess neuroticism and 'baseline' arousal symptoms. A minority of the women (9%) subsequently had a pregnancy loss. Results showed that neuroticism in early pregnancy significantly predicted PTSD symptoms about one month after the loss. Yet, this relationship disappeared when controlling for pre-trauma arousal symptoms: the *increase* in PTSD symptoms from baseline to post-pregnancy loss was similar for high and low neuroticism individuals. Thus, high neuroticism individuals did not show heightened reactivity to adverse events. These results were recently replicated in prospective study of soldiers who were exposed to adverse events during their deployment in Iraq (Engelhard, van den Hout, & Lommen, 2009). Again, high neuroticism individuals reported more PTSD symptoms than low neuroticism individuals after deployment, but this was also the case before deployment; the increase in symptoms was similar for both groups. In conclusion, high neuroticism individuals were not more reactive to stressful events than low neuroticism individuals. The question is what underlying mechanism may be responsible for the heightened level of (baseline) distress symptoms in high neuroticism individuals. As stated by Ormel and colleagues (2004), since neuroticism itself does not seem to have an explanatory role in the aetiology of anxiety disorders, it is important to unravel cognitive and biological mechanisms that produce high neuroticism scores.

Many studies have focused on information processing abnormalities that may play a role in the aetiology of anxiety disorders. High, compared to low, trait anxiety individuals show selective processing of both threatening and ambiguous stimuli (see Eysenck, MacLeod, & Mathews, 1987). One of the selective processes in (clinical) anxiety

includes attentional bias towards threatening stimuli (Bradley, Mogg, Falla, & Hamilton, 1998; Macleod & Rutherford, 1992; Mathews & MacLeod, 1985; van den Hout, Tenney, Huygens, Merckelbach, & Kindt, 1995). Selective attention may lead to excessive processing of minor threats in daily life, resulting in anxiety even in conditions that are innocuous (Watson & Clark, 1984). Furthermore, high (trait) anxiety is associated with interpretation biases, which refers to the tendency to interpret ambiguous stimuli as threatening (Calvo & Castillo, 2001; Eysenck et al., 1987; MacLeod & Cohen, 1993; Mogg et al., 1994), and judgemental bias, the tendency to overestimate the likelihood of negative outcomes and their costs (Butler & Mathews, 1987; Eysenck & Derakshan, 1997; Tomarken, Mineka, & Cook, 1989). These findings suggest that high trait anxious individuals may use a lower decision criterion to detect danger in potentially threatening situations. In terms of the signal detection theory, they may be reluctant to miss a danger-signal, but willing to accept a false alarm. As everyday life involves many uncertain situations, such a “better safe than sorry” strategy should cause many false alarms. Especially if false alarms are followed by avoidance or escape, the inaccuracy of the alarm will not be detected, and this may help to explain why negativity is so persistent. The aim of this study was to investigate whether high compared to low neuroticism individuals avoid a greater number of ambiguous stimuli.

Although several studies have focused on processing of ambiguous stimuli in high trait anxiety, studies that used a behavioural outcome variable such as avoidance of potentially threatening stimuli are scant. Moreover, most studies used stimuli of which ambiguity was assumed, and not established. To withstand these problems, this study uses a de novo conditioning task (based on Orr et al., 2000). In this task, two different neutral, conditioned stimuli (CS; i.e., coloured circles) are presented in random order. One stimulus (CS+) is always followed by an aversive, unconditioned stimulus (UCS; i.e., a mild electric shock), the other stimulus (CS-) is not. After a few presentations, a person generally learns that the CS+ predicts the UCS, and the CS- predicts the absence of the UCS. So in this acquisition phase, both a threat and a non-threat signal are learned. Following the acquisition phase we did not immediately use an extinction phase, but introduced an ‘avoidance’ phase, that included degraded stimuli, with different colour values on a spectrum between CS+ and CS- (see figure 1). In this phase, a potential UCS could be avoided by pushing the spacebar before the offset of the stimulus.

We hypothesized that 1) high compared to low neuroticism individuals would avoid more stimuli, and that 2) the mean colour value of avoided stimuli would be closer to the colour value of CS- for high neuroticism individuals. Moreover, we explored

whether the expected effects (1 and 2) might be partially explained by differences in UCS-expectancy or in subjective aversiveness of the UCS, and whether the expected effects would be more pronounced in short or long latencies. Finally, to gain insight in processes that might be responsible for the effects, state-anxiety, intolerance of uncertainty, anxiety sensitivity, and worry were assessed, because these variables are associated with neuroticism (e.g., De Bruin, Rassin, & Muris, 2007) or individual differences in conditioning (Otto et al., 2007).

METHODS

Participants

A total of 55 students of Utrecht University and University of Applied Sciences enrolled in the study, based on their score on the neuroticism scale of the Eysenck Personality Questionnaire (EPQ-N; Eysenck & Eysenck, 1975). A score of 4 or less was considered to reflect low neuroticism (N-; *n*=24; 12 females), and 11 or higher to reflect high neuroticism (N+; *n*=24; 13 females). Exclusion criteria were 1) past or current psychiatric disorders, 2) visual problems (unless corrected), 3) colour blindness, 4) use of medication or drug that could interfere with attention, reaction time and/or memory, 5) epilepsy, 6) heart condition, and 7) pregnancy. Each exclusion criterion was checked by asking the participant whether he/she met the criterion. Participation was voluntary and written informed consent was obtained prior to participation. Students received course credit or financial compensation for their participation.

Stimulus Materials

A set of 10 coloured circles (Figure 1) was used in the conditioning task, ranging from white (no. 1; RGB 255-255-255) to black (no.10; RGB 0-0-0). To increase ambiguity, two circles were used as CS+ (nos. 1 and 2) and two for CS- (nos. 9 and 10). The grey colours (nos. 2-9) were equally divided over the colour spectrum. CSs with a diameter of 176 mm were presented on a 1280x1024 resolution screen (Eizo flexscan S1911). The UCS consisted of a mild electric shock, delivered via finger electrodes to two fingers of the non-dominant hand. It was adjusted individually to a level that was ‘highly annoying but not painful’ through a work-up procedure prior to the conditioning task (cf. Orr et al., 2000).

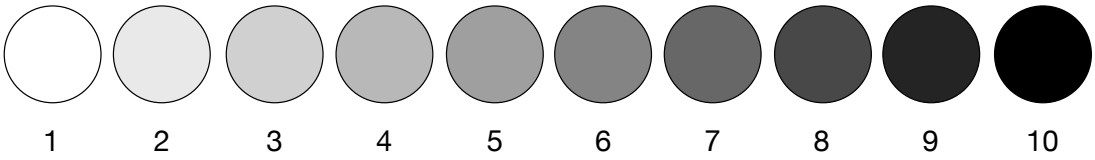

Figure 1. CSs and Corresponding Colour Values

Procedure

The study took place in a humidity- and temperature-controlled, sound-attenuated room. First, exclusion criteria were checked and colour blindness was tested with the Ishihara Test (Ishihara & Ishihara, 1970). If the participant met the inclusion criteria, informed consent was obtained, followed by the work-up procedure to determine the UCS level. Next, participants completed a filler questionnaire and the State scale of the State-Trait Anxiety Inventory (STAI-S; Spielberger, Gorsuch, & Lushene, 1983). Then, the conditioning task started containing the following phases:

Habituation phase. CS+ no. 1 and CS- no.10 were each 5 times presented in a semi-random order, so that no more than two consecutive presentation of the same CS were presented. CS duration was 4 or 5 s., which was determined randomly, with an Inter Trial Interval (ITI) of 7 ± 2.

Acquisition phase. CS+ trials (nos. 1 and 2) and CS- trials (nos. 9 and 10) were each presented 5 times semi-randomly the same way as described before. All CS+ trials were followed by the UCS, whereas none of the CS- trials was combined with the UCS. CS duration was 4 or 5 s., which was determined randomly, with an ITI of 7 ± 2. UCS-expectancy was measured with a 0-100 visual analogue scale (VAS). Participants were asked to rate this scale at least once during the 4/5-s. presence of the CS.

Avoidance phase. CSs more or less similar to the CS- trials (nos. 3-10) were semi-randomly presented as described before. Participants were instructed that if a yellow or white light was shown before the CS onset, they had the opportunity to avoid a potential UCS by pushing the spacebar. The yellow light indicated that the participant had 5 s. to avoid a potential UCS, the white flickering light signified that the person had only 1 s. Nos. 3 to 10 were each combined twice with both lights. Participants did not know that none of the CSs (nos. 3-10) was actually followed by an UCS. To avoid extinction in non-avoiders, 4 presentations of the CS+ trials followed by the UCS were included while both lights were turned off. Consequently, participants received 4 UCSs during the avoidance phase. UCS expectancy was rated after each CS offset.

Extinction phase. In order to invalidate the CS/UCS contingency, all CS+ trials and CS- trials were each presented 5 times in semi random order without the UCS.

After the extinction phase, STAI-S was administered again. Finally, participants filled out the questionnaires described below.

Questionnaires

Neuroticism was assessed with the neuroticism scale of the Eysenck Personality Questionnaire (EPQ-N; Eysenck & Eysenck, 1975). This widely used scale consists of 22 items that can be answered with *yes* (= 1) or *no* (= 0).

State anxiety was measured with the STAI-S (Spielberger et al., 1983). It consists of 20 items that are rated on a 4-point Likert scale ranging from 1 ('not at all') to 4 ('very much so').

Intolerance of uncertainty was assessed with the Intolerance of Uncertainty Scale (IUS, Dutch translation; De Bruin, Rassin, Van der Heiden, & Muris, 2006; Original French version: Freeston, Rhéaume, Letarte, Dugas, & Ladouceur, 1994) This is a self-report scale consisting of 27 items that relate to the idea that uncertainty is unacceptable, reflects badly on a person, and leads to frustration, stress, and the inability to take action. Each item is answered on a 5-point Likert scale, ranging from 1 (*not at all characteristic for me*) to 5 (*entirely characteristic for me*).

Anxiety sensitivity was measured with the Anxiety Sensitivity Index (ASI; Peterson & Reiss, 1992). The ASI assesses the tendency to fear symptoms of anxiety based on the belief that they may have harmful consequences. Answers of the 16 item self-report questionnaire are rated on a 5-point Likert scale ranging from 0 (*very little*) to 4 (*very much*).

Worry was measured with the Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990). This self-report measure consists of 16 items that are rated on a 5-point Likert scale ranging from 1 (*not at all typical of me*) to 5 (*very typical of me*). In this sample, smallest internal consistency of questionnaires estimated with Cronbach's α was .90.

Subjective aversiveness of the UCS was measured with the question 'How unpleasant was the electric shock for you?', which was rated on a 0-100 VAS immediately after the conditioning task.

RESULTS

Group characteristics

Seven participants were excluded from the analyses, because they did not learn the CS/UCS contingency (n=3) or did not evaluate the UCS as aversive (n=4), resulting in a

sample of 48 students[1]. The mean age was 21.65 (*SD*=2.37). Descriptive statistics of the questionnaires (after modifying two outlier scores into M+/- 2.5 *SD*) are presented in Table 1. Gender was equally represented in the N+ and N- groups (χ^2=.08, *p* =.77). Compared to the N- group, the N+ group scored significantly higher on state anxiety (STAI-S) before and after the conditioning task, intolerance of uncertainty (IUS), anxiety sensitivity (ASI), and worry (PSWQ), smallest *t*(46)=2.59, *p*=.01. The groups did not differ in age, *t*(46)=.42, *p*=.67. The mean response time at the 1 s. (638 ms for N-; 619 ms for N+) and 5 s. (1354 ms for N-; 1121 ms for N+) latency trials did not differ significantly between the N- and N+ groups, highest *t*(37)=1.15, *p*=.26.

Table 1

Descriptive Statistics and Internal Consistency Ratings of the Questionnaires (N=48)

	Total sample			N+		N-			
	Mean	SD	Internal consistency	Mean	SD	Mean	SD	*t*	*p*
EPQ-N	8.17	6.57	.93	14.21	3.12	2.12	1.48	17.13	<.001
STAI-S pre-test	34.35	9.38	.94	37.50	8.17	30.67	7.59	3.00	<.01
STAI-S post-test	32.77	9.03	.95	35.96	10.32	29.58	6.24	2.59	.01
IUS	63.94	15.97	.93	74.79	14.63	53.08	7.84	6.41	<.001
ASI	13.06	9.14	.90	16.75	9.54	9.00	4.52	3.60	<.01
PSWQ	44.63	14.51	.95	56.13	10.85	33.13	6.07	9.07	<.001

To examine whether both groups differentiated the CS+ en CS- after acquisition, a three way ANOVA was carried out with UCS expectancy as dependent variable, Group (N+ vs. N-) as between-group factor, Trial (first vs. last) as within-group factor, and Stimulus (CS+ vs. CS-) as within-group factor. The main effect of Group was not significant, *F*(1,35)=.22, *p*=.64, indicating that UCS expectancies, generally, did not differ between the N+ and N- groups.

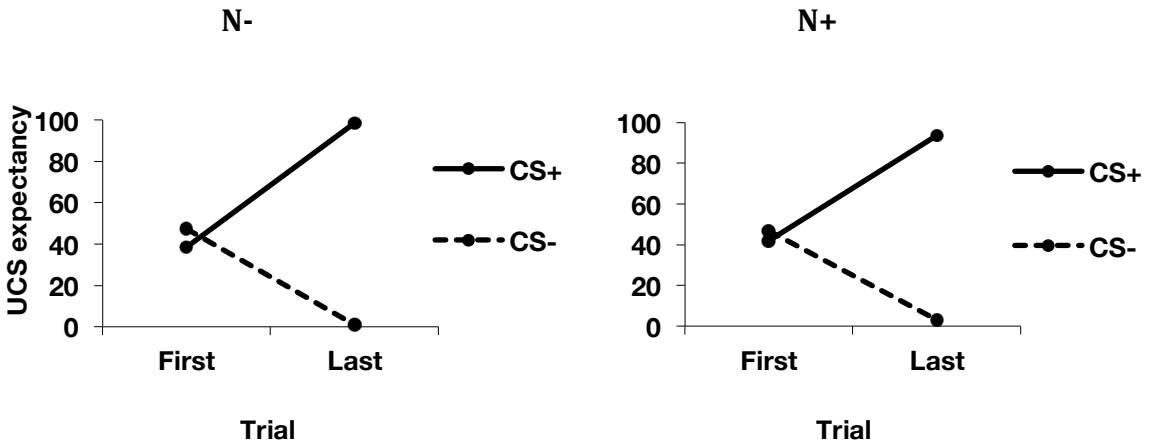

Figure 2. UCS Expectancy at First and Last Trial of Acquisition Phase

Number of avoided CSs at 1 and 5 s. latency

A two way ANOVA with number of avoidance responses as dependent variable, Group (N+ vs. N-) as between-group factor, and Latency (1 vs. 5 s.) as within-group factor revealed a significant main effect of Latency, $F(1,46)=4.59$, $p=.04$, referring to a significant increase in the number of avoided CSs from 1 s. to 5 s. latency. The main effect of Group was in the expected direction, with N+ showing a larger number of avoided CSs, but it was not statistically significant, $F(1,46)=3.04$, $p=.09$ (see figure 3). The crucial Group x Latency interaction was significant, $F(1,46)=4.03$, $p=.05$, indicating that N+, compared to N-, increased in avoidance responses in long compared to short latency. Subsequent simple main effect analyses revealed that N- did not show a significant difference, $M_{diff}=.04$, $F(1,46)=.01$, $p=.93$, in number of avoided CSs between the two latencies, while N+ did, $M_{diff}=1.29$, $F(1,46)=8.61$, $p=.01$. Further, N+ and N- did not differ in number of avoided CSs on 1 s. latency trials, $M_{diff}=.63$, $F(1,46)=.75$, $p=.39$, but they did differ significantly on 5 s. latency trials, $M_{diff}=1.88$, $F(1,46)=4.99$, $p=.03$.

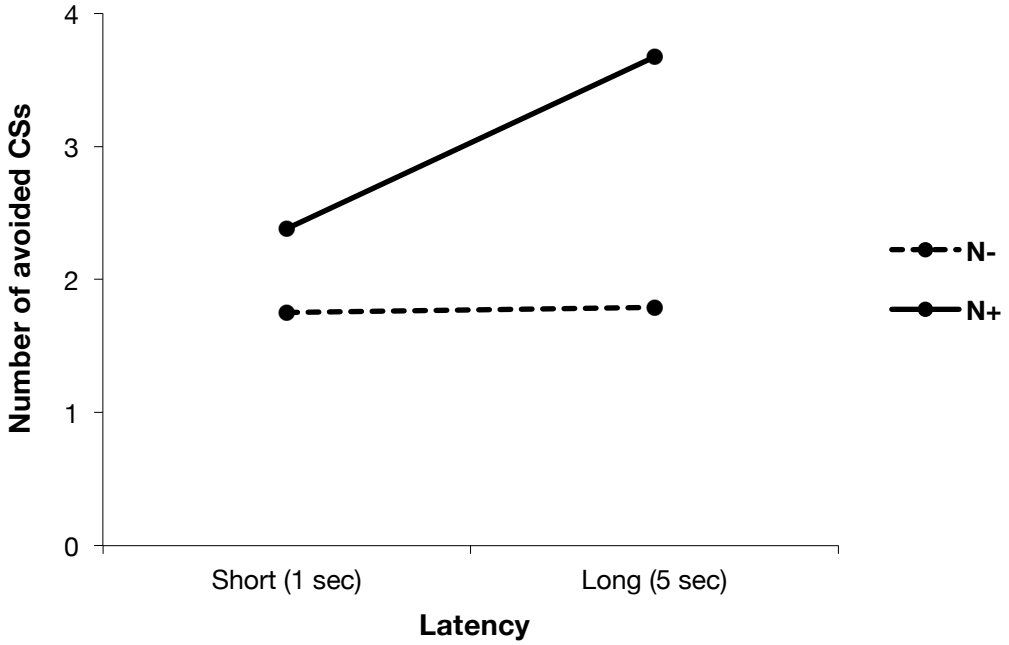

Figure 3. Results of Two-Way ANOVA with Number of Avoided CSs as Dependent Variable

Average colour value of avoided CSs at 1 and 5 s. latency

A similar repeated measures analysis with average colour value of avoided CSs[2] as dependent variable showed no main effect of Latency, $F(1,46)=1.21$, $p=.28$, or Group, $F(1,46)=1.53$, $p=.22$, but, again, the crucial Group x Latency interaction was significant, $F(1,46)=4.33$, $p=.04$ (see Figure 3). Simple main effect analyses yielded results that were similar to the number of avoided CSs: the average colour value of avoided circles did not differ between the short and long latency for N-, $M_{diff}=.17$, $F(1,46)=.48$, $p=.49$, but they did differ significantly for N+, $M_{diff}=.55$, $F(1,46)=5.06$, $p=.03$. N+ and N- did not differ in average colour value of avoided CSs at 1 s. latency trials, $M_{diff}=.07$, $F(1,46)=.03$, $p=.87$, but the N+ showed significantly higher average colour values at the 5 s. latency trials, $M_{diff}=.79$, $F(1,46)=4.35$, $p=.04$.

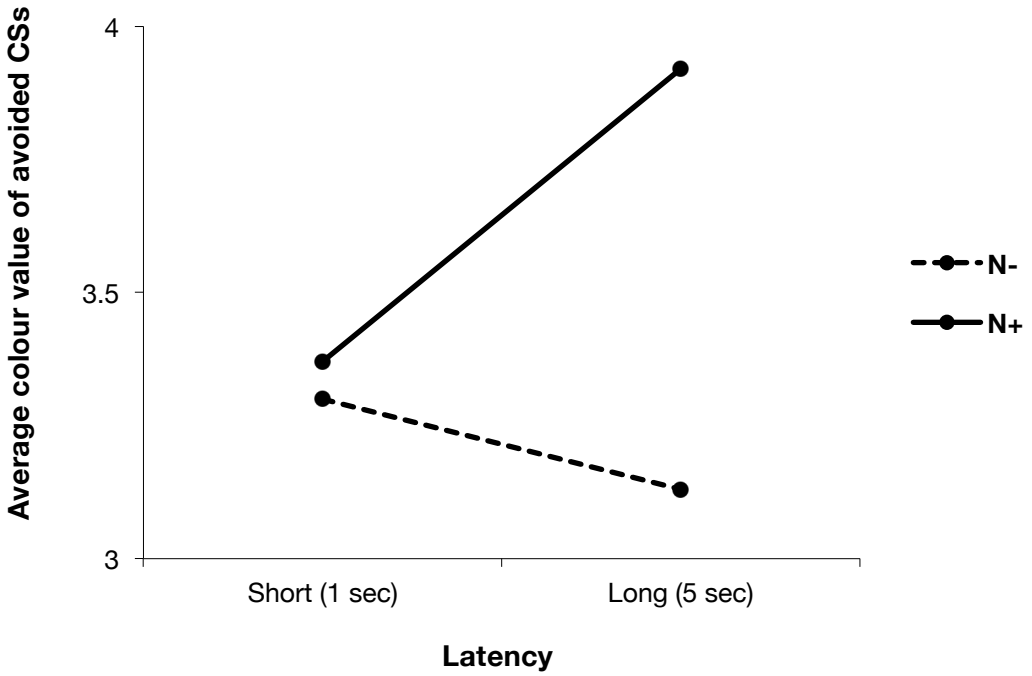

Figure 4. Results of Two-Way ANOVA with Average Colour Value of avoided CSs as Dependent Variable

UCS expectancy and subjective aversiveness of UCS

UCS expectancies tended to be higher for N+ for colour no. 5, $t(45)=1.99$, $p=.05$, but not differ between the N+ and N- groups for the other colours, with t-values ranging from .11 to 1.57, and p-values ranging from .13 to .91[3].

Although N- and N+ had similar UCS levels, $t(46)=1.15$, $p=.26$, the N+ group found the UCS more aversive than the N- group, $t(45)=2.72$, $p=.01$. Nevertheless, inclusion of this UCS evaluation as a covariate did not significantly contribute to number of avoided CSs, $F(1,44)=.35$, $p=.56$, neither to average colour value of avoided CSs, $F(1,44)=1.68$, $p=.20$. Therefore, we did not include it as a covariate in the final analyses.

State anxiety, intolerance of uncertainty, anxiety sensitivity and worry

Compared to the N- group, the N+ group scored higher on state anxiety, intolerance of uncertainty, anxiety sensitivity, and worry, smallest $t(46)=2.59$, $p=.01$. Only inclusion of worry as a potential covariate contributed significantly to the analysis with number of avoided CSs as dependent variable, $F(1,45)=5.50$, $p=.02$. The effects of Group, Latency, and their interaction were no longer significant when worry was taken into account. Further, neuroticism correlated strongly with worry ($r=.80$, $p<.01$). These results suggest

that worry may be an important aspect of neuroticism that may help to explain its relationship with avoidance of ambiguous CSs.

DISCUSSION

Neuroticism is associated with anxiety disorders, but the underlying mechanism of this relation is still unknown. Previous studies have shown that individuals high in neuroticism or trait anxiety show cognitive biases in information processing of ambiguous and threatening stimuli. This suggests that highly anxious individuals may use a "better safe than sorry strategy", that results in 'precautionary avoidance' of potentially threatening stimuli, which prevents disconfirmation of erroneous threat expectations.

In this study, we examined whether high compared to low neuroticism individuals use such a strategy, with behavioural avoidance as the outcome measure. We used a de novo-conditioning task (cf. Orr et al., 2000), in which participants learned that a CS+ predicted the UCS, and a CS- predicted the absence of the UCS. In a subsequent avoidance phase, stimuli that represented a continuum between the CS+ en CS- were presented. Participants were allowed 1 s. or 5 s. to avoid a possible UCS. They rated their UCS expectancy for each stimulus, and their avoidance behaviour was recorded. In line with our first hypothesis, the N+ group avoided a larger number of ambiguous stimuli. This effect was found at 5 s. trials, but not at 1 s. trials, which may indicate that a strategic process that requires more cognitive elaboration is involved. Although neuroimaging studies have shown that brain activation to ambiguous stimuli is higher and more similar to dangerous stimuli in high neuroticism groups than low neuroticism groups (Herwig et al, 2007), and that these responses to ambiguity can already be seen after 250 ms, indicating the involvement of automatic processes (Hirsch & Inzlicht, 2008), the results of the present study suggest that these effects are not pronounced when using a *behavioural* outcome measure. In line with our second hypothesis, the mean colour value of the avoided stimuli was closer to CS- for the N+ group, compared to the N- group. Again, this was shown on 5 s. trials only.

To gain insight in processes that may explain these differences, we explored the role of UCS expectancies, UCS aversiveness, state anxiety, intolerance of uncertainty, anxiety sensitivity, and worry. First, UCS expectancy of avoided stimuli was largely similar for the N+ and N- groups, which makes it unlikely that it was involved in the group effects in avoidance. This is consistent with earlier research that also found that neuroticism was unrelated to the degree of UCS expectancies (Engelhard, de Jong, van den Hout, & van Overveld, 2009). Moreover, also fear conditioning studies using

physiological outcome measures rather than UCS expectancies, failed to establish significant correlations between neuroticism and conditioning indices (Ashcroft, Guimaraes, Wang, & Deakin, 1991; Davidson, Payne, & Sloane, 1964; Fredrikson & Georgiades, 1992). Second, the UCS was more aversive for N+ than N- individuals, even though UCS levels were similar between the groups. This is in line with earlier studies (Butler & Mathews, 1987; Eysenck & Derakshan, 1997; Tomarken et al., 1989), and with studies showing that N+ individuals perceive higher costs of adverse events than N- individuals. Again, this variable did not explain the relationship between neuroticism and the avoidance of ambiguous stimuli. Finally, state anxiety, intolerance of uncertainty, anxiety sensitivity, and worry were taken into account, because these are all related to neuroticism and/or individual differences in conditioning. Only worry was relevant: the group differences for avoidance were no longer significant when worry was taken into account. This suggests that worry is an aspect of neuroticism that is important in processing and avoidance of ambiguous stimuli. Previous findings have shown that a high level of worry predicts greater fear acquisition and a stronger magnitude of the conditioned response (Otto et al., 2007). It also fits nicely with laboratory studies showing that cognitive rehearsal of the UCS may increase its aversiveness (see review Davey, 1992). However, in our task, the time between stimulus presentation and behavioural avoidance was still relatively short, so it is unclear how worry may have contributed to the group differences in avoidance behaviour.

A limitation of the study was the small sample size, which may have resulted in a power problem. Further, recruitment of N+ individuals for a study including a mild electric UCS was far more difficult than N- individuals, which may have resulted in a selective inclusion of N+ individuals who did not avoid the UCS at forehand. It seems plausible that the N+ individuals who were willing to participate would represent the relatively low scorers of the invited N+ group. Nevertheless, the mean score on the neuroticism questionnaire of the tested N+ group (M=14.21, SD=3.12) is comparable with scores of patients in a similar age group from an outpatient clinical setting in the Netherlands (M=12.6, SD=5.2), and higher than scores of a similar age group in the general population (M=7.9, SD=5; Sanderman, Arrindell, Ranchor, Eysenck, & Eysenck, 1995). For future research, it seems advisable to use another aversive UCS that is less likely to elicit avoidance of participation than electric stimuli, for example loud noise. Finally, due to the cross-sectional design, no conclusions can be made about causality. Future research may elucidate the causal relationship.

In conclusion, N+ and N- individuals similarly learned to distinguish a danger (CS+) and safety (CS-) signal, the N+ group subsequently avoided a greater number of ambiguous stimuli that represented the continuum between CS+ and CS-, at the 5 s. latency trials. The absence of differences at the 1 s. latency trials suggests the involvement of a *strategic* process. Apparently, when confronted with ambiguous threat signals, N+ individuals use a "better safe than sorry strategy". By preventing disconfirmation of erroneous expectations, this strategy may be involved in the development and maintenance of anxiety.

FOOTNOTES

[1] Exclusion if the UCS expectancy was lower than 60% for the CS+, or higher than 40% for the CS- at the end of the acquisition phase.

[2] As the dependent variable here is average colour value of avoided CSs, participants who did not avoid any CS in one of the two Latencies would be excluded from the analyses. As this would unfavourably lead to a selective sample possibly excluding the 'real' N-, the average colour value of avoided CSs was changed into 2. The mean colour value of avoided CSs could theoretically range from 3 to 10, however, with the inclusion of the non-avoiders, the range is 2 to 10.

[3] UCS expectancy for avoided CSs was not included in the final analysis, because this would exclude participants who did not avoid at all (n=7 in N- Group; n=1 in N+ Group), and who did not avoid any CSs in one of the two latency trials (n=2 in N- Group; n=5 in N+ Group).

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Chapter 5

Reducing emotional reasoning: an experimental manipulation in individuals with fear of spiders

In revision as:

Lommen, M.J.J., Engelhard, I.M., van den Hout, M.A., & Arntz, A. Reducing emotional reasoning: an experimental manipulation in individuals with fear of spiders

ABSTRACT

Emotional reasoning involves the tendency to use subjective responses to make erroneous inferences about situations (e.g., “if I feel anxious, there must be danger”) and has been implicated in various anxiety disorders. The aim of this study of individuals with fear of spiders was to test whether a computerized experimental training, compared to a control training, would decrease emotional reasoning, reduce fear-related danger beliefs, and increase approach behaviour towards a fear-relevant stimulus. Effects were assessed shortly after the experimental manipulation and one day later. Results showed that the manipulation significantly decreased emotional reasoning in the experimental condition, not in the control condition, and resulted in lower danger estimates of a spider, which was maintained up to one day later. No differences in approach behaviour of the spider were found. Reducing emotional reasoning may ultimately help patients with anxiety disorders attending more to objective situational information to correct erroneous danger beliefs.

Anxiety disorders are characterized by fears that have an irrational element. According to cognitive models (e.g., Clark, 1999), these fears may arise from distorted beliefs about the dangerousness of certain stimuli. For example, a patient with posttraumatic stress disorder (PTSD) related to a robbery may keep expecting another robbery, despite slim chances it will happen again. A patient with panic disorder fears a heart attack when the heart is pounding, despite good health and reassurance from a doctor. A patient with a phobia of flying may be afraid to crash, although chances are very low and the patient never crashed before. Why do such beliefs persist, despite the availability of disconfirming information? Clark (1999) lists several cognitive factors that may prevent patients to self-correct their negative thinking. One of these is emotional (or 'ex-consequentia') reasoning, which involves using feelings as a validation of thoughts, like "If I feel anxious, there must be danger" (Arntz, Rauner, & van den Hout, 1995; Beck & Emery, 1985; Engelhard & Arntz, 2005). This conclusion further evokes anxious feelings and avoidance of the anxiety-evoking stimulus, resulting in a vicious circle in which irrational beliefs are maintained (Clark, 1999).

The role of affect in beliefs about danger has also been emphasized by affect-as-information theories. These theories state that people misattribute their anxiety to a target of judgement (Curtis & Locke, 2007; Gasper & Clore 2000). That is, judgement of a situation will be influenced by feelings at time of judgement in an affect-congruent way. With regard to risk estimates for instance, both cognitive evaluations, including objective information about the probability of the outcome, and internal feelings, including emotions, are consulted (Clore & Storbeck, 2006). However, when potential outcomes are highly emotionally undesirable, like exposure to a fear-evoking stimulus, objective information about the probability of the outcome may become marginalized (Slovic, Finucane, Peters, & MacGregor, 2002). In the context of anxiety disorders, individuals may misinterpret anxious feelings as a sign for impending danger, while there is objective information that the emotion is a false alarm. Individual differences in this 'emotional reasoning' have been linked to anxiety disorders.

Arntz et al. (1995) examined whether patients with anxiety disorders engaged in emotional reasoning compared to healthy controls, and if it reflects a general or situation-specific (i.e., related to the specific anxiety disorder) tendency. Four groups of patients (suffering from spider phobia, panic disorder, social phobia, and other anxiety disorders) and a healthy control group were presented 16 brief scenarios and asked to rate each on degree of danger. There were four types that related to spider phobia, panic disorder, social phobia, and general anxiety, and each had four versions, with 1) objective safety

information or 2) objective danger information, combined with 3) an anxious response or 4) a non-anxious response. Results showed that the patient groups inferred danger not only from objective information, but also from subjective anxiety response information, irrespectively of the type of situation, whereas healthy controls inferred danger from objective information. Similar findings have been shown in Vietnam combat veterans with PTSD, compared to combat veterans without the disorder (Engelhard, Macklin, McNally, van den Hout, & Arntz, 2001). Furthermore, emotional reasoning has been related to anxiety symptoms in healthy children[1] (Morren, Muris, & Kindt, 2004).

There is some evidence that emotional reasoning may be involved in the maintenance of anxiety disorders. A longitudinal study among witnesses of a train crash showed that emotional reasoning (based on intrusions) predicted chronic PTSD symptoms (Engelhard, van den Hout, Arntz, & McNally, 2002). Moreover, unpublished data indicate that emotional reasoning after cognitive behavioural therapy for anxiety disorders predicts relapse beyond residual post-treatment symptoms (Arntz, 2001).

To examine whether reducing emotional reasoning may contribute to the treatment of anxiety, the aim of this study was to experimentally attenuate emotional reasoning, and examine the influence on cognitive beliefs of a fear-evoking stimulus and behaviour towards the stimulus. In a sample of students with fear of spiders, we tested whether 1) an experimental manipulation would decrease emotional reasoning, and 2) decreases in emotional reasoning would reduce danger beliefs and increase approach behaviour towards a fear-relevant stimulus in a behavioural approach task.

METHOD

Participants

Inclusion criteria were fear of spiders (initially based on a general question), age of 18 years or older, and an above-average score (>4.2; based on a pilot study among a random sample of students) on a screener for emotional reasoning (i.e., 8 scenarios; see measures below). Participants were recruited with flyers and posters, which asked for individuals with fear of spiders. A total of 183 students of Utrecht University and the Utrecht University of Applied Sciences completed the screener and 90 of them were invited to participate in the study. A total of 61 participants (9 males) with a mean age of 22 years (*SD* = 2.56) were tested. Three participants (1 male) with low spider fear scores (i.e., FSQ score < 11; Huijding & de Jong, 2006) were excluded from the analyses, resulting in inclusion of 58 participants (8 males). They received course credit or financial compensation for participating. Participants signed informed consent before participating.

With *N* = 58 the study was powered to detect a large between group difference of Cohen's *d* = .75 with a power of 80%.

Procedure

To diminish potential demand bias, participants were told a cover story. They were led to believe that they participated in two different studies: the first about their knowledge of spiders, the second about the relationship between reading behaviour and imagination. Participants were given written and oral information about the study. After providing written informed consent, participants completed a questionnaire (FSQ; see measures below) that assessed spider fear, followed by a quiz including seven questions about spider-related facts (e.g., "How long do spiders live?"). After that, they answered questions about their reading behaviour (e.g., "How many books do you read a year?") and rated their state anxiety (STAI-1; see measures below). Note that the spider quiz and reading questionnaire were included to increase plausibility of the cover story. Then, they were alternately assigned to the control (*n* = 29; 5 males) or experimental (*n* = 29; 3 males) condition. The manipulation, aimed to reduce emotional reasoning in the experimental condition and maintain emotional reasoning in the control condition, consisted of a computerized training of approximately 30 minutes, after which state anxiety (STAI-2) was re-assessed. Furthermore, participants rated eight scenarios on dangerousness to assess the level of emotional reasoning. Finally, participants carried out a Behavioural Approach Task (BAT) and re-rated state anxiety (STAI-3). They were asked to return one day later for the follow-up assessment, which consisted of the BAT.

Measures

Emotional reasoning

Emotional reasoning was measured with 16 brief scenarios: 12 were disorder-specific scenarios used by Arntz et al. (1995), and four were general anxiety scenarios, taken from Engelhard et al. (2002). Half of them (panic and spider scenarios; eight scenarios) were used as screener (ER-1) and baseline measure of emotional reasoning. The other half (social and control; eight scenarios) were used as manipulation-check (ER-2).

As described earlier, there were four versions of each scenario type, in which objective danger information and subjective anxiety information were crossed. For example, the spider scenarios began with:

"You just came back from the supermarket."

After that, the scenario included:

(1) objective danger information:

"You bought a cluster of bananas. You know that poisonous spiders are imported with bananas. You have just seen a documentary on television about this, where those spiders were shown. At home you notice a 1-inch spider in your shopping bag, the kind of spider you have seen on television. You think: "A tropical spider!"

or (2) objective safe information:

" At home you see a big house spider in your shopping bag.".

The objective information was followed by:

(3) an anxious response:

"You are scared to death…"

or (4) a non-anxious emotional response:

"That seems a nice present for your friend who collects spiders. For a good price you have bought the bananas and a nice present! You are looking forward to tell your friend about it…".

Participants evaluated the scenarios on degree of danger, safety, uncontrollability, anxiety, and positive of negative outcome on 0-100 mm visual analogue scales (VASs). The danger score (0 = absolutely not dangerous; 100 = extremely dangerous) was used to calculate the degree of emotional reasoning, which was operationalized as the mean difference in scores of the objective danger/safe information scenario with an anxious response minus the objective danger/safe information scenario with a non-anxious response (cf. Engelhard et al., 2001). In this study, the other VASs were included as filler items.

Manipulation

A computerized training was developed for this study and piloted, in which 60 brief scenarios (15 types of scenarios; e.g., skiing the advanced track, sea swimming, turbulence during a flight) were presented. Scenarios included objective safe or danger information, with an anxious or non-anxious response, and an unfinished sentence. Participants were instructed to imagine the described scenario as if it happened to them, and to choose one out of two options (a positive or negative outcome) to complete the sentence.

For example, one type of scenario described a car ride back home after a party. The objective danger information included: "*Your friend who just obtained his driving licence is driving too fast and tells you he had 5 beers at the party*". The objective safe

information stated: "*Your father is driving the car and speeds up to pass a truck.*" The objective information was followed by an anxious response "*You start sweating and cling to the grip*" or a non-anxious response "*You start evaluating the party and you realise how quick you will be home".* Each of these car ride scenarios ended with: "*Shortly after that, the car*…", followed by the options "*1) crashes into a tree"*, and "*2) drives into the garage at home".* Participants received feedback on their choice ("*correct*" or "*incorrect, try again*") and the training continued to the following scenario as soon as the participant chose the correct answer. Feedback crucially differed across conditions. To reduce emotional reasoning in the experimental condition, a scenario that included objectively safe information had a positive outcome, while a scenario with objective danger information had a negative outcome. Participants could learn that the objective information was the only relevant information that predicted the outcome, irrespectively of the emotional response. In contrast, to maintain the above-average level of emotional reasoning in the control condition, a scenario that included non-anxious response information had a positive outcome, while a scenario with anxious response information had a negative outcome. Thus, the subjective response information was the only relevant information that predicted the outcome, irrespectively of objective situational information.

Behavioural Approach Task (BAT)

After positioning the participant's chair at a fixed point close to the table, a closed jar with a spider (young tarantula) was put on the table. Participants rated the dangerousness of the spider, their anxious feelings, and their physical tension on a 0 (*absolutely no…)* to 100 (*ultimately…)* mm VAS. After that, they were instructed to pull the rope attached to the jar towards them, as far as they dared to within 30 seconds. The distance the jar had been moved reflected approach behaviour (minimal approach = 0 cm; maximal approach = 69.5 cm). Then participants indicated if they would dare to do the following: 1) touch the jar, 2) take up the jar, 3) open the jar, 4) put your hand in the jar, 5) touch the spider, and 6) let the spider walk over your hand. Each question was answered with "*yes"* (1) or "*no"* (0).

Fear of Spiders

The Dutch version (Muris & Merckelbach, 1996) of the Fear of Spiders Questionnaire (FSQ; Szymanski & O'Donohue, 1995) includes 18 items to assess self-reported fear of spiders. Each item was rated on a 0 ('*completely disagree')* to 7 ('*completely agree'*) scale (range 0-126). The FSQ has shown good reliability in spider phobics and non-clinical

levels of spider fear (Muris & Merckelbach, 1996). The Cronbach's alpha in this study was .93.

State anxiety

State anxiety was measured with the State subscale of the State-Trait Anxiety Inventory (STAI-S; Spielberger, Gorsuch, & Lushene, 1983). It consists of 20 items that are rated on a 4-point Likert scale ranging from 1 ('not at all') to 4 ('very much so'). The Cronbach's alphas in this study were .91, .91, and .96 (STAI-1, STAI-2, and STAI-3, respectively).

Statistical analyses

The scores of 4 outliers (1 on ER-1; 3 on VASs) in the experimental condition and 2 outliers (STAI-1; dare exposure steps) in the control condition were replaced by $M \pm 2.5 \times SD$ to obtain a normal distribution. A repeated measures analysis with Condition (control; experimental) as between-subjects variable and Time (ER-1; ER-2) as within-subjects variable was run to assess the effect of the experimental manipulation on emotional reasoning. ANOVAs were run to test the differences between conditions on the BAT. To control for baseline differences in spider phobia and reduce measurement error, FSQ was included as a covariate in the analyses of the experimental effect on danger beliefs and approach behaviour.

RESULTS

Fear of spiders and state anxiety

FSQ scores of the experimental condition, $n = 29$, $M = 56.00$, $SD = 23.57$, did not differ from the control, $n = 29$, $M = 60.97$, $SD = 25.16$, $t(56) = .78$, $p = .44$, $d = 0.21$, 95% CI [-7.86, 17.79]. To test whether the training itself affected state anxiety differently in the two conditions, a repeated measures analysis with Time (STAI-1; STAI-2; STAI-3) as within-subjects variable and Condition (control; experimental) as between-subjects variable revealed a main effect of Time, $F(1.43,112) = 41.78$, $p < .01$, $d = 1.73$, with increased state anxiety after exposure to the spider, but no main effect of Condition, $F(1,56) = 0.14$, $p = .71$, $d = 0.10$, and no Time x Condition interaction, $F(1.43,112) = 1.52$, $p = .23$, $d = 0.33$. Since the assumption of sphericity was violated, χ^2 (2) = 27.76, $p < .01$, results of this analysis represent Greenhouse-Geisser corrected values. Thus STAI-scores and STAI-score changes over time were similar for the conditions.

Emotional reasoning manipulation

A repeated measures analysis with Time (ER-1; ER-2) as within-subjects variable and Condition (control; experimental) as between-subjects variable revealed a main effect of Time, $F(1,56) = 5.95$, $p = .02$, $d = 0.65$, and a main effect of Condition, $F(1,56) = 9.03$, $p < .01$, $d = 0.80$. The crucial Time x Condition interaction was significant, $F(1,56) = 5.68$, $p = .02$, $d = 0.64$ (Figure 1). Simple main effect analysis showed a decrease in emotional reasoning in the experimental condition, $M_{diff} = 12.65$, $p < .01$, 95% CI: [5.22, 20.08], but no change in the control condition, $M_{diff} = .15$, $p = .97$, 95% CI: [-7.28, 7.58].

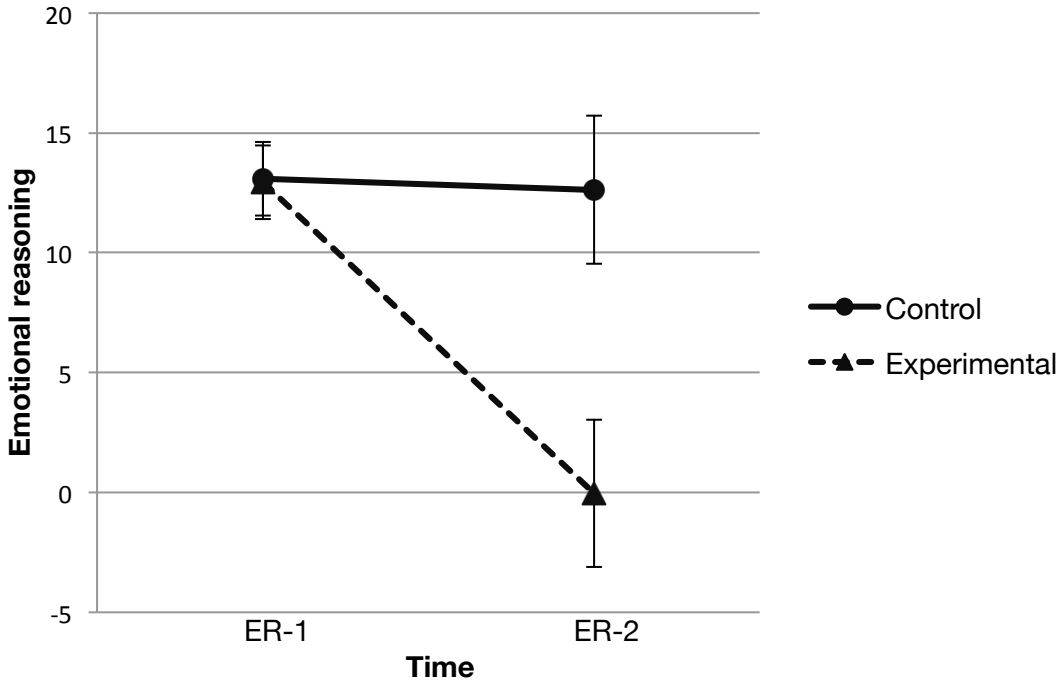

Figure 1. Emotional reasoning scores of the two conditions before (ER-1) and after (ER-2) the manipulation. Error bars represent standard errors.

Behavioural Approach Task (BAT)

First, the VAS score that represented anxious feelings was compared, when controlling for FSQ scores. FSQ corrected means were 53.74 (*SD* = 3.52) and 37.94 (*SD* = 3.59) for the experimental and control condition, respectively. An ANOVA revealed that the experimental condition scored higher on anxious feelings than the control condition, $F(1,54) = 9.82$, $p < .01$, $d = 0.85$, after controlling for FSQ, $F(1,54) = 43.19$, $p < .01$, $d = 1.77$.

FSQ corrected means of physical tension were 57.39 (*SD* = 3.21) and 44.07 (*SD* = 3.27) for the experimental and control condition, respectively. Physical tension was

higher in the experimental condition, $F(1,54) = 8.40$, $p = .01$, $d = 0.78$, after controlling for FSQ, $F(1,54) = 49.30$, $p < .01$, $d = 1.89$.

Second, differences in danger ratings and approach behaviour between conditions were tested. Because the level of anxious feelings would influence the level of emotional reasoning (e.g., it would be far more difficult to ignore overwhelming anxious feelings than a very low level of anxious feelings), the level of anxious feelings should be similar in both groups to compare their dangerousness ratings, as we are interested in differences in dangerousness due to emotional reasoning and not due to differences in anxious feelings itself. Since the anxious feelings ratings (VAS) were different for the conditions, these ratings, in addition to FSQ, were included as covariate in the following analyses. Table 1 shows means and standard deviations of the BAT outcome measures, corrected for FSQ and anxious feelings scores. The experimental condition showed lower danger ratings than the control condition, $F(1,53) = 6.79$, $p = .01$, $d = 0.70$, after controlling for anxious feelings, $F(1,53) = 26.75$, $p < .01$, $d = 1.39$, and FSQ, $F(1,53) = 0.96$, $p = .33$, $d = 0.26$.

The moved distance of the jar also did not differ between the conditions, $F(1,53) = 0.51$, $p = .48$, $d = 0.19$, after controlling for anxious feelings, $F(1,53) = 9.57$, $p < .01$, $d = 0.83$, and FSQ, $F(1,53) = 3.11$, $p = .08$, $d = 0.48$.

The average of reported dare steps did not differ between the conditions either, $F(1,53) = 1.11$, $p = .30$, $d = 0.28$, after controlling for anxious feelings, $F(1,53) = 1.21$, $p = .28$, $d = 0.30$, and FSQ, $F(1,53) = 5.29$, $p = .03$, $d = 0.62$.[2]

Table 1

Descriptive statistics of the behavioural approach task outcome variables after correction for FSQ and anxious feelings VAS score.

	Immediately after manipulation					*One day after manipulation*				
	Condition					*Condition*				
	Experimental		*Control*			*Experimental*		*Control*		
BAT outcome variable	*M*	*SD*	*M*	*SD*	*N*	*M*	*SD*	*M*	*SD*	*N*
Dangerousness	20.54	3.07	32.45	3.13	57	17.49	4.01	30.80	4.46	31
Moved distance of jar	45.72	3.08	49.01	3.14	58	46.58	3.90	54.62	4.34	31
Dare steps	1.83	0.21	2.15	0.21	58	2.37	0.31	2.62	0.35	31

Follow-up

Seventeen participants of the experimental condition, and 14 of the control condition completed the BAT again about one day later (*M* = 29.21 hours). Analyses revealed patterns of results similar to the earlier BAT results. FSQ corrected means of anxious feelings were 38.23 (*SD* = 5.73) and 25.30 (*SD* = 6.35) for the experimental and control condition, respectively. FSQ corrected means of physical tension were 40.82 (*SD* = 5.53) and 28.23 (*SD* = 6.23) for the experimental and control condition, respectively. Table 1 shows means and standard deviations of the BAT outcome measures, corrected for FSQ and anxious feelings scores. Although there were no significant differences between the conditions in anxious feelings or physical tension, largest $F(1,28) = 2.18$, $p = .15$, the experimental condition showed lower ratings on dangerousness, $F(1,27) = 4.53$, $p = .04$, $d = 0.79$, when controlling for anxious feelings, $F(1,27) = 24.31$, $p < .01$, $d = 1.84$, and FSQ, $F(1,27) = 1.00$, $p = .33$, $d = 0.37$. No significant differences between conditions were found for behaviour outcome variables, that is moved distance of the jar or average of reported dare steps, largest $F(1,27) = 1.75$, $p = .20$.

DISCUSSION

This study tested if an experimental manipulation could decrease emotional reasoning in a student sample with spider fear, and whether this would reduce danger beliefs and increase approach behaviour towards a fear-relevant stimulus in a behavioural approach task. Results showed that the experimental manipulation led to a significant decrease in emotional reasoning in the experimental condition, whereas emotional reasoning did not change in the control condition. Immediately after the manipulation, the experimental condition showed lower danger estimates of a spider, when controlling for anxious feelings and fear of spiders. This effect was maintained up to one day later. This was consistent with our predictions. However, contrary to our expectations, no differences were found between conditions in approach behaviour of a spider immediately after the manipulation or one day later.

To the best of our knowledge, this is the first study showing that emotional reasoning can be reduced using a computerized training. Moreover, the results indicated that declines in emotional reasoning influenced fear-related cognitions. However, fear-related behaviour was not affected. There are several possible explanations for the lack of differences between conditions on approach behaviour in the Behavioural Approach Task (BAT). First, the training in this study may have been too short (and indirect) to affect behaviour. Cognitive bias modification techniques including computerized training

targeting attention bias and interpretation bias have shown beneficial effects in clinical and non-clinical populations (Hertel & Mathews, 2011; MacLeod, 2012). These studies often involved multiple trainings over several weeks, which may optimize learning benefits and retention (Cepeda, Pashler, Vul, Wixted, & Rohrer, 2006; Hallion & Ruscio, 2011). An interesting future direction might be to provide participants with a rationale of the experimental training to promote conscious inhibition of the tendency to engage in emotional reasoning. Second, the BAT may have been too challenging because it included a stimulus specifically related to participants' fear. Nevertheless, the relatively short training did decrease danger beliefs about a fear-relevant stimulus, which maintained up to one day later, providing evidence for the effectiveness of the manipulation.

Third, the training included situations that were not specifically related to spider fear. Although individual differences in emotional reasoning did not appear to be situation-specific (Arntz et al., 1995), a training targeting idiosyncratic fears may be even more effective in tackling problematic engagement in emotional reasoning and subsequent behaviour. Fourth, participants were not actually asked to carry out the BAT dare steps, which could have led to an overestimation of the reported number of dare steps. However, this seems unlikely since the mean number of reported dare steps (0 to 6) was only 2. Fifth, the study was powered to detect only large effects. Our training had a slightly stronger than medium effect on the emotional reasoning test (d = 0.64). It seems unlikely that an effect of this training to decrease emotional reasoning would have stronger effects (that is, a strong effect of d > 0.80) on approach behaviour in another area (spider fear) than the training was directed at. Thus future studies should either increase sample size to allow detection of weak effects of about d = 0.30, or increase the training effects. Note that firm conclusions about the causal influence of the training on fear-related cognitions and behaviour cannot be drawn, as a BAT before the training was not included. Lastly, the notable difference in anxious feelings ratings between the conditions is unlikely to be a result of the training itself. It seems more likely and plausible that it reflects pre-training differences in anxious feelings towards a spider. Future studies may anticipate this by including a pre-training BAT.

We found evidence that a decrease in emotional reasoning was related to a change in fear-related cognition, but not behaviour. The latter may be due to the methodological issues described earlier. Future studies may further identify optimal conditions for generalization of the experimental manipulation to real-life. The emotional reasoning training in this study promotes the reliance on objective information about

dangerousness instead of subjective feelings of threat. Nevertheless, commonly used therapeutic techniques may also target emotional reasoning. Clinical samples that are characterized by emotional reasoning (Arntz et al, 1995) may benefit from interventions in which they learn to attend to more objective information, like the probability of the outcome (Slovic et al., 2002). It remains to be seen whether reduced emotional reasoning may add to the efficacy of cognitive behavioural therapy (CBT), by allowing the disconfirmation of erroneous beliefs about danger that maintain anxiety disorders.

FOOTNOTES

[1] Although several studies used a different outcome variable to measure emotional reasoning that was more similar to anxiety (e.g., Muris, Merckelbach, & Spauwen, 2003).

[2] Because the representation of males as not equal for the conditions, we repeated the analyses including females only, which resulted in similar results.

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Chapter 6

Psychosocial predictors of chronic posttraumatic stress disorder in Sri Lankan tsunami survivors

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ABSTRACT

This study aimed to determine whether psychological factors associated with Post-Traumatic Stress Disorder (PTSD) identified in Western samples generalize to low Social-Economical-Status (SES) populations in an underdeveloped Asian country. The study included 113 survivors of the 2004-tsunami on the south coast of Sri Lanka, recruited from 4 preschools and 10 villages for displaced persons. With logistic regressions the relations between interview-based PTSD-diagnosis and psychological factors were assessed, controlling for putative confounders. Fifteen months post-trauma the prevalence of PTSD was 52.2%. Multivariate analyses indicated that negative interpretation of tsunami-memories was significantly ($p<.005$) related to PTSD. Of the putative confounders, gender and (non-replaced) lost work equipment were related to current PTSD ($p<.05$). The results indicate that the relation between negative interpretation of trauma-memories and PTSD is quite universal, suggesting that interventions focusing on this factor may be important in treatment of tsunami survivors who are suffering from chronic PTSD.

On the 26th of December 2004, an earthquake with a magnitude of 9.3 on the Richter scale took place under the Indian Ocean. In Sri Lanka, the tsunami affected about a million mainly poor people living at the seashore. More than 30 000 people lost their life, over a million were left homeless or were injured, and many are still missing (BBC News, n.d.). Post-Traumatic Stress Disorder (PTSD) is the psychological condition most often assessed and observed in samples of disaster victims (Norris *et al.,* 2002). It can therefore be expected that PTSD is a highly prevalent mental health problem in tsunami survivors. The criteria for PTSD as defined in DSM-IV include exposure to a traumatic event and at least one month of experiencing distressing symptoms of re-experiencing the trauma, avoidance and numbing, and increased arousal (American Psychiatric Association, 1994).

Studies among Western samples have identified psychological factors associated with PTSD and with the maintenance of PTSD symptoms. However, it is unclear whether these findings can be generalized to low SES-samples in underdeveloped Asian countries. This study aimed to determine the generalizability of psychological factors associated with PTSD in Western samples to a sample of Sri Lankan tsunami survivors fifteen months post-tsunami. The following factors were included:

Negative interpretation of tsunami-memories. Negative interpretations of intrusive memory symptoms may generate a sense of current threat, motivating cognitive and behavioural strategies that prevent recovery and are associated with the maintenance of PTSD (Dunmore, Clark & Ehlers, 1999; 2001; Halligan, Michael, Clark & Ehlers, 2003; Steil & Ehlers, 2000).

Rumination about the tsunami. Rumination serves a cognitive avoidance function, inhibits the ability to deploy adaptive coping resources (Fresco *et al.,* 2002) and predicted persistent PTSD in several studies (Clohessy & Ehlers, 1999; Ehlers, Mayou & Bryant, 1998; Michael, Halligan, Clark & Ehlers, 2007; Steil & Ehlers, 2000).

Satisfaction with social support. According to Bal, Crombez, Van Oost and Debourdeaudhuij (2003) perceived high availability of social support is directly associated with fewer trauma-related symptoms. Not the size of the social network, but the satisfaction with social support matters (McCormick, 1999).

Suppression of tsunami-related intrusions. Thought suppression is an attempt to avoid or end particular thoughts, but paradoxically increases the occurrence of intrusions and it prevents emotional processing of the traumatic event (Clohessy & Ehlers, 1999; Steil & Ehlers, 2000).

Causal attribution of the tsunami. Joseph and co-workers (1991; 1993) showed that more internal attributions for negative disaster-related events were associated with greater posttraumatic symptomatology.

Avoidance of the sea and *avoidance of talking about the tsunami.* Avoidance of trauma reminders prevents adequate emotional processing of traumatic experiences as well as habituation to traumatic memory cues (Dunmore *et al.*, 2001; Steil & Ehlers, 2000).

Negative self-cognitions, negative world-cognitions and self-blame. Traumatic events shatter individual's beliefs and assumptions, and negative beliefs about the self, others and the world and trauma-related self-blame are related to PTSD (Dunmore *et al.*, 1999; Foa, Ehlers, Clark, Tolin & Orsillo, 1999).

Therapeutic approaches like prolonged exposure therapy and cognitive behavioural therapy (CBT) are based on these psychological maintenance-factors. Individual CBT is a common and effective treatment for PTSD in Western samples (Van Etten & Taylor, 1998; Bradley, Greene, Russ, Dutra & Westen, 2005*;* Schnurr *et al.,* 2007; Bisson *et al.,* 2007). But the effectiveness of such an intervention in underdeveloped Asian countries is unknown. Interventions provided to reduce traumatic stress in large populations affected by disaster in resource-poor countries are psychological debriefing and benzodiazepine medication. These interventions have little evidence of effectiveness and their indiscriminate application can be harmful (Van Ommeren, Saxena & Saraceno, 2005). Further, there is no evidence for effects of psychosocial services in non-Western settings on mental health problems like PTSD (Igreja, Kleijn, Schreuder,Van Dijk & Verschuur, 2004; Neuner, Schauer, Klaschik, Karunakara, & Elbert, 2004; Tol *et al.,* in press). Thus it is of theoretical importance to determine whether psychological factors that are identified through Western studies generalize to lower SES samples in underdeveloped Asian countries. Additionally, results are potentially useful in the development of effective interventions for these populations.

Because natural disasters like the tsunami can change life conditions dramatically, these changes were included in the analyses to control for possible confounding effects. The included changes were: death of people who were part of the participants household, death of relatives or friends who were not part of the participants household, people missing, (non-replaced) loss of house, physical consequences of the tsunami nowadays, injuries, illnesses and scars after tsunami, (non-replaced) loss of work equipment and (non-replaced) loss of earned income. It is well known that factors like gender, age and education may affect an individual's chance to develop PTSD after a trauma (Brewin, Andres & Valentine, 2000; Ozer, Best, Lipsey & Weiss; 2003). We therefore also included

these factors, adding use of medication and whether the participant received counselling after the trauma or not as putative confounders in the analyses.

METHODS

Study Population

This study is a population-based survey among 113 tsunami survivors (32 men and 81 women) living in the southern part of Sri Lanka. Figure 1 shows the recruitment stages.

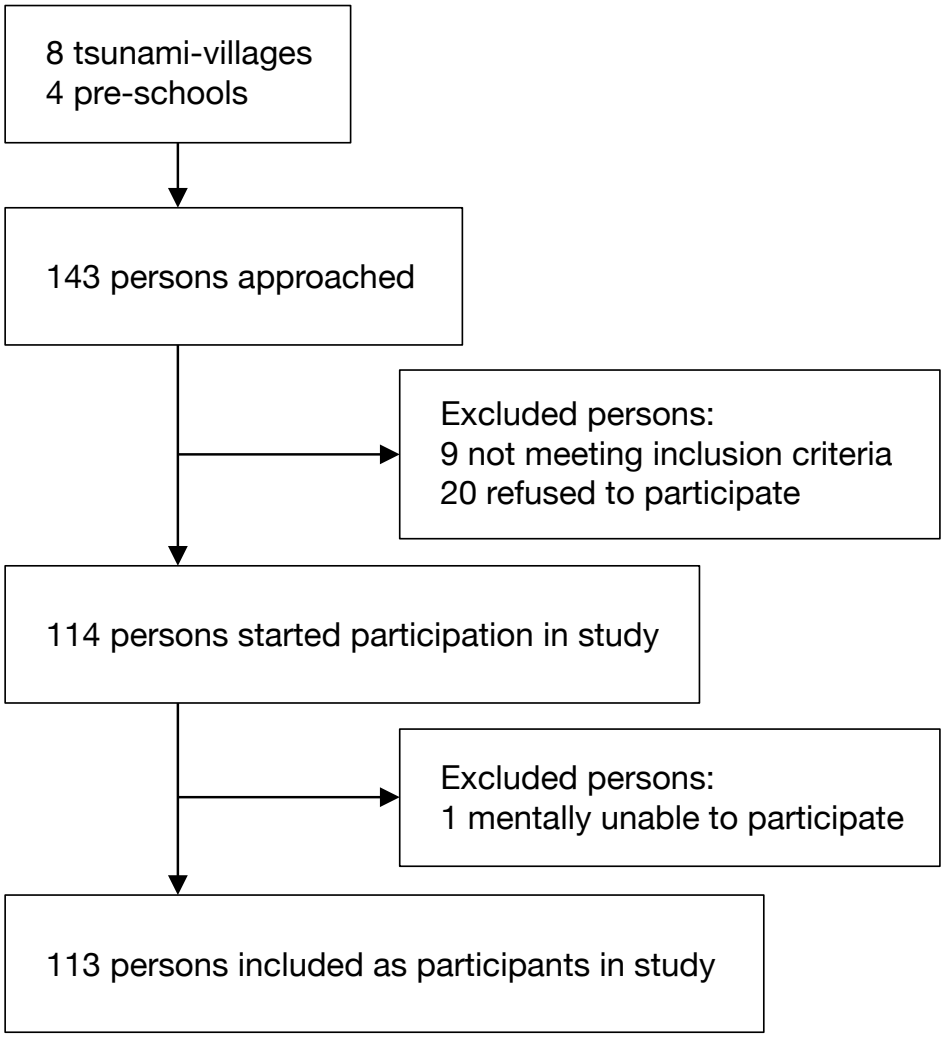

Figure 1. Sampling Stages for Tsunami Survivors in Southern Sri Lanka.

Participants had an average age of 35.9 years (SD=10.1 years, range 18-68). Fifty-six participants had a low educational level (lower than O-level, i.e. did not finish secondary education [O-level is comparable to the British General Certificate of Education]). All were affected by the tsunami because they experienced or witnessed the tsunami themselves, because someone in their household was affected by the tsunami or because their properties were devastated by the tsunami. Eighty-five participants (75.2%) lost their house, 12 of them (10.6%) still had no permanent house fifteen months after the tsunami. Seventy people (61.9%) lost their work equipment, 41 people (36.3%) did not have their equipment replaced more than one year post-tsunami. About half of the participants (n=56 [49.6%]) reported that they or other household members still had the same job as compared to a month before the tsunami. Forty-seven participants (41.6%) lost one or more jobs within their household. Most found a new source of income, but earned less than before. A small minority (n=10 [8.8%]) lost their job and did not find another one within fifteen months after the tsunami. This study only included people who experienced the tsunami of 2004 as the most traumatic event in their life. People younger than 18 years or older than 70 years were excluded. According to the DSM-IV (APA, 1994) children and youngsters might show other types of responses than adults. We therefore excluded participants younger than 18 years. People of 70 years and older were excluded because of possible problems with cognitive functioning due to aging (e.g. dementia). Everyone participated in this study voluntarily.

Procedures

The study was conducted between March 6 and April 20, 2006 in preschools and several villages for displaced tsunami survivors. At preschools the teacher invited mothers and fathers who were affected by the tsunami. In the villages the interpreter approached inhabitants indiscriminately. Because many participants had reading problems, the interpreters administered all questionnaires as an interview in Sinhalese to all participants. Scorings were written in Singhalese for those participants who were able to read, but were also often read to the participants. All questionnaires were originally in English and before the start of the interviews translated by the interpreters into Sinhalese. Informed consent was obtained from participants prior to completion of the questionnaires and the interview. All the questionnaires posed question about the last month, apart from the PDS assessing symptomatology in the first month after the tsunami and the PDS assessing PTSD symptoms during the last three months. The assessments took 1-3 hours.

PTSD was assessed with the Posttraumatic Stress Diagnostic Scale (PDS; Foa, 1995). The PDS asked participants to rate how often they experienced each of the PTSD symptoms specified in DSM-IV during the last three months (APA, 1994), ranging from 0 (not at all / only one time) to 3 (5 or more times a week / almost always). A symptom was scored as present if it was rated as 1 (once a week or less/ once in a while) or more (Foa, 1995; Foa et al., 1997). The individual gave a description of the traumatic experience to assess the DSM-IV criteria for exposure to a traumatic event. Duration of symptoms and the impairments caused by the symptoms were investigated to assess DSM-IV PTSD criteria E-F (APA, 1994). Participants completed two PDS versions: (1) PTSD at the time of the interview; (2) PTSD in the first month after the tsunami. Internal consistency of present PDS was .83 and of PDS one month post-trauma was .73 (all internal consistencies estimated with Cronbach's α).

Negative appraisals of posttraumatic intrusive memories were measured with Halligan's et al. (2003) version of the Interpretation of PTSD Symptoms Inventory (IPSI; Clohessy & Ehlers, 1999; Dunmore *et al.*, 2001; Steil & Ehlers, 2000). This version comprised seven items (e.g. "My memories of the tsunami mean that I must be losing my mind"; "My memories of the tsunami mean that I will never have normal emotions again"). In each item 'assault' was replaced by 'tsunami'. Ratings ranged from 0 ("not at all") to 4 ("very strongly"). In this sample, internal consistency was .73.

A questionnaire created for this study was used to assess rumination. The questionnaire consisted of eight items and answers were rated on a five-point scale ranging from "never" to "always". Six out of eight questions were from the six-item short form of the Rumination-Reflection Questionnaire (RRQ), originally consisting of a 12-item rumination scale and a 12-item reflection scale (Trapnell, 2005; Trapnell & Campbell, 1999). Two questions that included a negative (e.g. "I never ruminate or dwell on myself for very long") were changed on advice of the interpreters into a question without a negative to make it less confusing for low-educated participants. Two additional questions assessed trait worry and rumination about intrusive recollections of the tsunami ("When you have recollections of the tsunami, how often do you think: 'Why did it happen to me?'"; "How often do you dwell on memories of the tsunami?")(Ehlers *et al.*, 1998). The word 'trauma' was replaced by 'tsunami'. Cronbach's α of the eight items in this sample was .71.

The Social Support Questionnaire (Short Form) (SSQSR) consisted of twelve items and was used to determine the size of the social support network and the satisfaction with the social support (Sarason, Sarason, Shaerin & Pierce, 1987). Odd numbered

questions assessed total support network size ranging from 0 (“nobody”) to 9 (“nine persons or more”). Even numbered questions assessed satisfaction with the social support ranging from 1 (“totally dissatisfied”) to 6 (“totally satisfied”). The sum of the even numbered questions represented the satisfaction with the perceived social support. The Cronbach’s α for the whole questionnaire, the odd and even numbered questions were respectively .88, .93 and .92.

To assess avoidance of the sea, the question “Do you try to avoid the sea nowadays?” was rated on a scale ranging from 0 (“never”) to 4 (“always”). Two questions about talking about the tsunami were posed: “Do you talk with other people about your experiences and feelings about the tsunami?” and “Do you avoid talking about the tsunami?”. A scale ranging from 0 (“never”) to 4 (“always”) was used. The score on the first question was converted to a new value of 4 minus the original score. The sum of the converted score and the score on the second question represented the factor avoidance of talking about the tsunami. The Cronbach’s α for these questions was .54. Suppression of tsunami-intrusions was rated on the same 5-point scale, asking how often the participant tried to push thoughts or images of the tsunami out of his/her mind when they occurred. Causal attribution of the tsunami was investigated by two questions: “What was the cause of the tsunami?” and “Why did it happen to you?”. Participants’ responses were analyzed and coded by the two researchers as to whether they represented internal, external or a combination of internal and external causal attributions. If it was not clear how an answer should be coded, the researchers discussed the answer with each other. Two independent raters listened to taped interviews and classified attributions. The agreement with the interviewer was for rater 1 81.8% (Cohen’s Kappa .60) and for rater 2 100% (Kappa 1.00). One-rater ICC was .75.

Negative self-cognitions (21 items), negative cognitions about the world (7 items) and self-blame (5 items) were assessed by the Posttraumatic Cognitions Inventory (PTCI; Foa et al., 1999). Answers were rated on a 7-point scale ranging from 1 (“totally disagree“) to 7 (“totally agree“). The Cronbach’s α for the whole questionnaire, the subscale negative self-cognitions, negative cognitions about the world and self-blame were respectively .88, .90, .53 and .50.

From the structured interview the following putative confounders were derived: gender, age, educational level, use of medication nowadays (only medication for somatic complaints and illnesses was reported), received counselling, death of people due to the tsunami within the participants household, death of relatives or friends who were not part of the participants household, people missing, (non-replaced) lost house, physical

consequences of the tsunami nowadays, injuries, illnesses and scars after tsunami, (non-replaced) lost work equipment and (non-replaced) lost earned income.

Statistical Analysis

Analyses were done with SPSS version 14.0. First of all, descriptive statistics (proportions, means, standard deviations) were calculated. Skewness and Kurtosis were calculated in order to check if the variables were normally distributed. Extremely skewed putative confounders were dichotomized (educational level, number of died household members, number of people still missing) or log-transformed (number of died people outside household). Educational level was dichotomized as less than O-level (did not finish secondary school) vs. O-level or A-level (secondary school). Logistic regression analyses were conducted to determine the relationship between the investigated factors and the dependent variable PTSD diagnosis. First, univariate logistic regression analyses followed by backward and forward multivariate analyses were used to determine which putative confounders were associated with PTSD. Bonferroni correction was not applied to this analysis to prevent type-II error: we did not want to risk to exclude a control variable on the basis of a *p*-level chosen to minimize type-I error. Next, univariate logistic regression analyses were run to determine which proportion each psychological factor explained in PTSD. After that, all significant confounders, and all psychological factors were put together in one logistic regression analysis. Backward and forward stepwise multivariate logistic regression analyses with significant confounders forced into the equation and with $p<.005$ criterion were used to determine the final model. In the final multivariate analysis we used a Bonferroni corrected *p*-level of .005 (.05/10) for the psychological factors. Pearson correlations were calculated to identify the relationships among the independent and dependent variables.

RESULTS

Retrospectively, one month post-tsunami 72 participants (63.7%) met the criteria for PTSD. The number of people meeting the criteria for PTSD at the time of the study decreased to 59 (52.2%), including 1 person (0.9%) diagnosed as PTSD with delayed onset.

Univariate logistic regression analyses on the putative confounders revealed that only (non-replaced) lost work equipment was significantly ($p=.03$) related to PTSD (Table 1). Forward and backward multivariate analyses, yielding the same result, revealed that

gender and (non-replaced) lost work equipment were significantly related to PTSD (Table 1).

Table 1

Univariate and Multivariate Analyses of Effects of Putative Confounders on PTSD

		OR (95% CI)	***P* Value**
Univariate Results			
Gender	Female	1.92 (0.84-4.41)	.12
Age		0.98 (0.94-1.02)	.26
Level of education	≥ O-level	1.38 (0.66-2.88)	.40
Medication nowadays	Yes	0.80 (0.38-1.70)	.56
Received psychological treatment	Yes	1.10 (0.51-2.36)	.82
Death of people within the participants household due to the tsunami	Yes	1.00 (0.40-2.50)	>.99
Death of relatives or friends who were not part of the participants household	Number of deaths	1.13 (0.75-1.69)	.56
People missing nowadays	Yes	.59 (0.28-1.23)	.16
House	Replaced loss	1.25 (0.52-3.00)	.61
	No replaced loss	2.31 (0.56-9.47)	.25
Physical consequences of the tsunami nowadays	Yes	1.72 (0.72-4.07)	.22
Injuries after tsunami	Yes	1.43 (0.68-3.05)	.35
Illnesses after tsunami	Yes	1.04 (0.47-2.33)	.92
Scars after tsunami	Yes	1.15 (0.43-3.02	.78
Work equipment	Lost and not replaced	2.41 (1.09-5.34)	.03
Earned income	Lost and not replaced	2.29 (0.56-9.34)	.25
Multivariate Results			
Gender	Female	2.49 (1.02-6.08)	.046
Work Equipment	Lost and not replaced	2.93 (1.26-6.83)	.013

Abbreviations: CI, confidence interval; OR, odds ratio; PTSD, posttraumatic stress disorder

Univariate logistic regression analyses on the psychological factors using the Bonferroni-correction (p=.005) revealed that negative interpretation of tsunami-memories and

negative self-cognitions were significantly related to PTSD (Table 2). For the final analyses gender and (non-replaced) lost work equipment were entered in the first block. In the second block, all psychological factors were analyzed with backward and forward procedures, which yielded the same result. The final model included negative interpretation of tsunami-memories (OR=1.24; $p<.001$), next to gender (OR=2.82; $p=.051$) and (non-replaced) lost work equipment (OR=3.58; $p=.009$).

Table 2

Univariate and Multivariate Analyses of Effects of Psychological Factors on PTSD

	OR (95% CI)	***P* Value**
Univariate Results		
Negative interpretation of tsunami-memories	1.23 (1.12-1.34)	<.001†
Rumination about the tsunami	1.10 (1.01-1.19)	.02
Satisfaction with social support	0.99 (0.92-1.07)	.76
Suppression of tsunami-related intrusions	1.05 (0.74-1.49)	.78
Causal attribution of the tsunami	1.37 (0.55-3.41)	.50
Avoidance of the sea	1.37 (1.05-1.78)	.02
Avoidance of talking about the tsunami	1.53 (1.09-2.15)	.01
Negative self cognitions	1.05 (1.03-1.07)	<.001†
Negative world cognitions	1.05 (0.98-1.13)	.17
Self-blame	1.12 (1.02-1.22)	.02
Multivariate Results		
Gender (Female)	2.82 (0.998-7.94)	.051
Work equipment (lost and not replaced)	3.58 (1.38-9.33)	.009
Negative interpretation of tsunami-memories	1.24 (1.13-1.36)	<.001†

Abbreviations: CI, confidence interval; OR, odds ratio; PTSD, posttraumatic stress disorder

† Statistically significant at the Bonferroni-corrected level of .005

Pearson correlations for all putative psychological factors and PTSD diagnosis were calculated to investigate the associations between them (Table 3). Note that negative interpretation of tsunami-memories is associated with all psychological factors that correlate with PTSD, except for avoidance of talking about the tsunami. Nevertheless, the intercorrelations indicate that different concepts were measured.

Table 3

Pearson Correlations Among Putative Predictors and PTSD Diagnosis

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
1. PTSD diagnosis	1										
2. Negative interpretation of tsunami-memories	.456†	1									
3. Rumination about the tsunami	.220‡	.371†	1								
4. Satisfaction with social support	-.029	-.172	-.044	1							
5. Suppression of tsunami-related intrusions	.026	.011	.092	.176	1						
6. Causal attribution of the tsunami	.064	.066	.037	.019	.002	1					
7. Avoidance of the sea	.224‡	.245†	.333†	-.013	.205‡	-.012	1				
8. Avoidance of talking about the tsunami	.239‡	.175	.211‡	-.046	.360†	.085	.279†	1			
9. Negative self cognitions	.450†	.742†	.473†	-.186‡	.062	.018	.251†	.093	1		
10. Negative world cognitions	.130	.238‡	.104	-.088	-.072	.125	.127	-.039	.136	1	
11. Self-blame	.237‡	.391†	-.034	-.026	.029	.069	.196‡	.135	.402†	-.142	1

†Statistically significant at $p = .01$

‡Statistically significant at $p = .05$

DISCUSSION

This study aimed to determine psychological factors related to PTSD, validated in Western countries, in a low SES-population in an underdeveloped Asian country. The study included survivors of the tsunami in December 2004 and was conducted in Sri Lanka more than one year post-trauma. Possible confounding variables were included in the study to control for non-psychological factors and changed life conditions.

In order to know the extent of the PTSD problems, the prevalence of PTSD among the tsunami survivors was determined in this study. Fifteen months after the tsunami, PTSD was diagnosed in 52.2 percent of the participants, including 0.9 percent diagnosed as PTSD with delayed onset (APA, 1994). Taken into consideration that 63.7 percent had met the criteria for PTSD one month post-trauma, the recovery rate within the group including people with PTSD diagnosis fifteen months post-trauma was 21.5 percent. The PTSD prevalence found in this study is much higher than found in two other studies of PTSD in survivors of the 2004-tsunami. These studies, conducted in Sri Lanka and Thailand during January and February 2005, found that 14-39% of the Sri Lankan children and 3-12% of the Thai adults fulfilled the criteria for tsunami-related PTSD (Neuner, Schauer, Catani, Ruf & Elbert, 2006; Van Griensven et al., 2006). However, a study by Ranasinghe and Levy (2007) among Sri Lankan people living in temporary shelters housing tsunami survivors found a similar PTSD prevalence to our findings; 56% at 6 months post-trauma. As the studied population and results of the last study are comparable to this study, difference in PTSD prevalence with the other two studies is probably due to the different population. Moreover, differences might be partially due to differences in instruments, scoring rules, sampling strategies, socio-economic factors, received support after the tsunami and cultural differences.

Logistic regression analyses showed that the relationship between PTSD and the psychological factors negative interpretation of tsunami-memories and negative self-cognitions were significant at Bonferroni-corrected level ($p<.005$) when these factors were considered separately. Of the confounders, gender and (non-replaced) lost work equipment were related to PTSD, showing an expected higher risk of PTSD for females (Brewin *et al.*, 2000; Olff, Langeland, Fraijer & Gersons, 2007), and for people who did not have their lost work equipment replaced compared to people who still had their work equipment or had it replaced. These factors would not have a significant confounding effect at Bonferroni-corrected level, but as the Bonferroni-correction in analyses of confounders increases the risk of a type-II failure, this correction was not applied here. The results are nevertheless interesting, suggesting that replacement of lost work

equipment has a beneficial effect on the development of chronic PTSD. This finding suggests that early intervention programmes should focus more on interventions that help recover individual's future perspective and economic safety than on psychosocial interventions like debriefing and counselling. An explanation for the effect of lost work equipment on PTSD is that it reinforces negative cognitions about permanent change caused by the trauma and that it remains a sense of current threat, as income is insecure. Both are associated with PTSD (Dunmore *et al*., 1999; 2001). Probably, replacing lost work equipment helps people to direct their attention to the future and helps them to recreate a safe base for their life. When controlled for gender and (non-replaced) lost work equipment in a backward logistic regression analysis, only negative interpretation of tsunami-memories was significantly ($P<.005$) related to PTSD. Most correlations between excluded psychological factors and negative interpretation of tsunami-memories were significant, but not excessively strong, so it is unlikely that collinearity explains the findings. There is one possible exception, as negative interpretation of tsunami-memories and PTCI negative self-cognitions correlated quite highly. On the other hand, both were (nearly) significant predictors in the backward regression analysis in the last steps, suggesting that they had independent contributions to the explanation of PTSD.

Our present findings support Ehlers and Clark's cognitive model of PTSD (2000). They argue that trauma victims who suffer from persistent PTSD process the trauma in a way that leads to a sense of current threat. Negative interpretation of the initial post-trauma symptomatology can be a source of this sense of current threat. Negative interpretations motivate dysfunctional cognitive and behavioural strategies. For example, survivors who believe intrusive memories mean that they lose their mind will make efforts to suppress tsunami-related memories, which paradoxically leads to more intrusions. The present results suggest that people may use a variety of strategies, but that negative interpretation of trauma memories is the most important underlying factor, so that only this factor was retained in the analyses.

To the best of the present authors' knowledge, this is the first study investigating psychological factors related to PTSD, validated in Western countries, in a poor population in an underdeveloped Asian country. The results indicate that the association between PTSD and negative interpretation of trauma-related memories is quite universal. The results also suggest that interventions to treat tsunami survivors suffering from chronic PTSD should focus on this factor, and also on the replacement of lost work equipment.

Limitations of this study include the following. First, variables regarding avoidance could be symptoms of PTSD instead of predictors of PTSD. This could be true for avoidance of the sea and rumination. In the present study this is not a problem, as both factors were not statistically significant in the analysis.

Second, conclusions about the effectiveness of counselling received by a part of the participants in this study cannot be made, as this study has a cross-sectional design. For example, the lack of relation between counselling and PTSD does not prove its ineffectiveness, as it may have reduced PTSD in the most severe cases. Moreover, the content, duration and period of counselling was not investigated. Therefore, no definite conclusions regarding the effectiveness of counselling can be drawn from this study. Nevertheless, our clinical impression of treatments delivered is that they lacked well-known active CBT ingredients.

Third, there was a language-barrier. Some translated Singhalese words were difficult to understand for the participants and they needed more explanation about the meaning of the questions. Fourth, the circumstances of the interview varied in accommodation (either in a special room, in the participants' houses or outdoors) and in people who were present (e.g. children). Fifth, cultural differences may have led to under- or over-reporting. Sixth, because this study was a cross-sectional study, no definitive conclusions can be made about causal relationships. A longitudinal study needs to be done to investigate this. Finally, it is unclear to what degree the findings of the present study can be generalized to survivors of other traumas or to tsunami survivors who live in other cultures.

Despite these limitations, this study shows clear evidence of a strong association between PTSD and negative interpretation of tsunami-memories. Further, female gender and non-replaced lost work equipment were associated with PTSD, suggesting that early intervention programmes should focus on replacement of lost work equipment. Future research should focus on the development, testing and implementation of psychosocial programs focusing on negative interpretations of tsunami-related memories, to help the large numbers of tsunami survivors who are still suffering from chronic PTSD.

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Chapter 7

Trauma and posttraumatic stress disorder (PTSD) in patients with schizophrenia and schizoaffective disorder

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ABSTRACT

Aims: Trauma and posttraumatic stress disorder (PTSD) have high prevalence among individuals with severe mental illness, such as schizophrenia or schizoaffective disorder. This study examined whether trauma and PTSD are under-detected in this population, and whether the cognitive theory of PTSD is applicable to these individuals. *Method:* Traumatic experiences, PTSD symptoms and negative posttraumatic cognitions were directly measured with questionnaires, and compared to information obtained via chart-review. *Results:* Results showed clear evidence of under-report of trauma and under-diagnosis of PTSD in patients' charts. Furthermore, negative posttraumatic cognitions were positively related to PTSD symptom severity, supporting the cognitive model of PTSD. *Conclusion:* These findings underscore the importance of assessing trauma history as well as PTSD in the routine evaluation of patients with schizophrenia or schizoaffective disorder in outpatient clinical settings. Furthermore, the finding of negative posttraumatic cognitions suggests that the cognitive model of PTSD may be applicable to patients with schizophrenia or schizoaffective disorder.

INTRODUCTION

Trauma and PTSD in severe mental illness

Trauma and posttraumatic stress disorder (PTSD) have high prevalence among individuals with severe mental illness, such as schizophrenia. Between 69.5 and 98 % of this population experienced a traumatic event in their life (Gearon, Kaltman, Brown & Bellack, 2003; Mueser et al., 1998; Neria, Bromet, Sievers, Lavelle & Fochtmann, 2002; Resnick, Bond & Mueser, 2003), compared to 39-56 % in the general population (Breslau, Davis, Andreski & Peterson, 1991; Kessler, Sonnega, Bromet, Hughes & Nelson, 1995). Rates of current PTSD in individuals with a severe mental illness range from 29-48% (Cascardi, Mueser, DeGiralomo & Murrin, 1996; Craine, Henson, Colliver & MacLean, 1988; Mueser et al., 1998; Switzer et al, 1999). Rates of current PTSD in studies focusing on schizophrenia specifically found 13%, 28% and 29% (Mueser et al., 1998; 2004; Resnick et al., 2003). These rates are much higher than the prevalence of lifetime PTSD in the general population, which ranges from 7.8 to 9.2% and the point prevalence of 2% (Breslau et al, 1991; Kessler et al., 1995; Stein, Walker, Hazen & Forde, 1997).

Although trauma and PTSD are common among individuals with a severe mental illness, clinicians frequently overlook trauma and PTSD. A recent study by Howgego et al. (2005) found that 33% of their sample of patients of a community mental health setting with a severe mental illness met the diagnostic criteria for PTSD. Only 4% of the sample had a formal diagnosis of PTSD in their medical record. The general lack of recognition and documentation of trauma and PTSD has been demonstrated in a number of studies (Callcott, Standart & Turkington, 2004; Craine et al., 1988; Davidson, 2001; Davidson & Smith, 1990; McFarlane, Bookless & Air, 2001; Mueser et al., 1998; Rose, Peabody & Stratigeas, 1991; Zimmerman & Mattia, 1999).

The need for recognition of trauma and PTSD is important among patients with a severe mental illness, as previous studies show that trauma among these patients is related to higher rates of substance abuse, higher risk of relapse, and higher costs of psychiatric services, such as hospitalizations (Beck & van der Kolk, 1987; Briere, Woo, McRae, Foltz & Sitzman, 1997; Bryer, Nelson, Miller & Krol, 1987; Goff, Brotman, Kindlon, Waites & Amico, 2001; McFarlane et al., 2001; Mueser, Rosenberg, Goodman & Trumbetta, 2002; Mueser et al., 2004; Rose et al., 1991). The experience of a traumatic event can be considered as a psychosocial stressor, which increases the level of schizophrenic symptoms (Mueser et al., 2002). Furthermore, a study by Vogel, Spitzer, Barnow, Freyberger and Grabe (2006) of inpatients with schizophrenia found that posttraumatic symptomatology rather than trauma itself, increased the risk of greater

current psychopathological distress. PTSD may be expected to worsen the course of the severe mental illness and may contribute to substance abuse, psychiatric and medical co-morbidity, and psychiatric and health service utilization. Finally, as long as PTSD is not recognized, it cannot be treated and may continue its negative effect on both physical as well as mental health outcome (Hidalgo & Davidson, 2000; Mueser et al., 2004).

Relationships between trauma, PTSD and psychotic disorders

Although trauma and PTSD are more prevalent in people with a psychosis compared to the general population, the precise relationship between trauma, PTSD and psychosis is still unclear. Three different kinds of possible relationships seem most likely. First, psychosis can cause PTSD. According to the traditional view, a traumatic event must include actual or threatened death or serious injury, or threat to physical integrity. However, this threat may be subjectively as well as objectively experienced: for example, a psychosis can have the ability to threaten one's own or others' life. A study by Kilpatrick et al. (1989) supports the validity of subjective interpretations of threat, showing that the way assault was perceived by their victims was predictive of the development of PTSD, regardless of the objective threat like use of weapons. Moreover, people may display the full PTSD symptoms without the experience of a direct experience of acute precipitating trauma similar to those experiencing more catastrophic trauma (Morrison, Frame & Larkin, 2003). A growing number of research studies suggests that the experience of a first psychosis and its treatment can be traumatic and leading to PTSD-like problems (McGorry et al., 1991; Mueser & Rosenberg, 2003; Shaner & Eth, 1989; Williams-Keeler et al., 1994). Further, a review of Morrison and colleagues (2003) shows that many studies using different methodologies found consistent high rates of PTSD in response to psychosis. These findings make it reasonable to conclude that some people do develop PTSD as a response to psychotic experiences (Morrison et al., 2003).

Secondly, trauma can cause psychosis. As many people with psychotic symptoms may have endured experiences of trauma prior to the onset of their psychosis, especially childhood sexual and physical abuse, traumatic life events may contribute to the development of psychosis (Morrison et al., 2003). A review on childhood trauma and psychotic disorders showed that the prevalence of reported childhood traumas ranged between 28% and 73%, with childhood sexual abuse ranging from 13% to 61% and childhood physical abuse ranging from 10% to 61% (Bendall, Jackson, Hulbert &

McGorry, 2007). Although the exact role of trauma is still unknown, childhood abuse seems to be implicated in the development of psychosis for a substantial portion of patients. However, more research is required to draw definite conclusions (Bendall et al., 2007; Friedman & Tin, 2007; Morrison et al., 2003; Morgan & Fisher, 2007).

Thirdly, psychosis and PTSD can both be part of a spectrum of responses to a traumatic event. There is some evidence for a common developmental process, as factors such as dissociation or attribution style may mediate the development of psychosis as well as PTSD following a traumatic event (Morrison et al., 2003). Moreover, the symptom similarity of these two disorders suggests the possibility that they may be similar entities, which may be part of a spectrum of responses to a traumatic event (Morrison et al., 2003). Nevertheless, more research studies are required for a more detailed understanding of the relationships between trauma, PTSD and psychosis.

Treatment of PTSD in schizophrenia or schizoaffective disorder

Since both trauma and PTSD are overrepresented among individuals with a psychotic disorder, treatment of PTSD in this population may be an important but often overlooked component. Recently, Mueser et al. (2007) studied the effectiveness of a cognitive-behavioural program for PTSD in persons with a severe mental illness, including schizophrenia and schizoaffective disorder. Results showed that this treatment contributed to clinical improvement in PTSD as well as improvement in other symptoms. The cognitive-behavioural treatment is based on the cognitive model of PTSD, in which PTSD is seen as a normal reaction to a traumatic event (Ehlers & Clark, 2000). Ehlers and Clark (2000) hypothesized that PTSD becomes persistent when individuals process the trauma in a way that leads to a sense of current threat. This sense of threat arises as a consequence of excessively negative appraisals of the trauma and/or its sequelae on the one hand and a disturbance of autobiographical memory characterized by poor elaboration and contextualization, strong associative memory and strong perceptual priming on the other hand. Those negative appraisals maintain PTSD by directly producing negative emotions, and by encouraging individuals to engage in dysfunctional coping strategies that have the paradoxical effect of enhancing PTSD symptoms. The negative appraisals about traumatic events and how they are responded to can have major effects on individuals' beliefs or underlying cognitive schemas about themselves, other people, or the world in general. Changes in the negative appraisals and the trauma memory are prevented by a series of problematic behavioural and cognitive strategies. According to this theory, a wide range of cognitive-behavioural interventions are useful to

modify these factors that have contributed to the maintenance of PTSD. Many studies in non-psychiatric populations support this cognitive model of PTSD (Clohessy & Ehlers, 1999; Dunmore, Clark & Ehlers, 1999; Ehlers, Clark, Hackmann, McManus & Fennel, 2005; Ehlers, Mayou & Bryant, 1998; Halligan, Michael, Clark & Ehlers, 2003; Steil & Ehlers, 2000). Although the study of Mueser et al. (2007) showed that changes in trauma-related cognitions may mediate changes in PTSD symptoms in people with a severe mental illness, it is questionable if these results can be generalized to specifically the group with schizophrenia or schizoaffective disorder which was represented by only 10 participants (12%). Therefore, it is still unclear whether the cognitive model of PTSD can be applied to people with schizophrenia or schizoaffective disorder.

Aims of the present study

The aim of this study is to examine whether trauma and PTSD are under-reported in a population of psychotic patients. A second aim is to investigate whether the cognitive theory of PTSD can be partially generalized to people with schizophrenia or schizoaffective disorder. The first hypothesis of this study is that lifetime traumatic events are more prevalent among people with schizophrenia or schizoaffective disorder in an outpatient clinical setting than in the general population, as measured in other studies. As we expect to find that trauma is under-reported in this population, the second hypothesis supposes that lifetime trauma experiences are higher when measured directly with questionnaires than when measured with information obtained with chart-review. Thirdly, we expect to find a higher prevalence of PTSD in people with schizophrenia or schizoaffective disorder than in the general population, as measured in other studies. Fourthly, we expect to find that PTSD is under-diagnosed in this population, as manifested by a higher rate of PTSD using a self-report questionnaire than using chart-review. Finally, we expect that PTSD symptom severity will be positively related to negative posttraumatic cognitions in this sample, supporting the applicability of the cognitive model of PTSD to people with schizophrenia or schizoaffective disorder. If our hypotheses are confirmed, there will be implications for both routine assessments of trauma and PTSD in individuals with schizophrenia or schizoaffective disorder. Confirmation of our last hypothesis would support the extension of the cognitive model of PTSD to this population, and would suggest that treatment based on this model might be beneficial in this population.

METHODS

Participants

Thirty-three patients recruited from an outpatient clinical setting in Sittard, the Netherlands, participated in this study. This sample consisted of twenty-three men and ten women, who received the primary diagnosis of schizophrenia (*N* = 23) or schizoaffective disorder (*N* = 10) according to their charts. Participants were in the age of 21 to 63 (*M* = 42.3, *SD* = 10.6).

Instruments

Prior research suggests that assessment tools for trauma and PTSD developed for the general population are appropriate for use among people with schizophrenia (Goodman et al., 1999; Mueser et al., 2001, 2004; Resnick et al., 2003). Based on these findings, the following measures were used.

The Trauma History Questionnaire – Revised (THQ-R) was used to assess experiences of traumatic events in childhood as well as over lifetime (Mueser et al., 1998). This self-report questionnaire consists of 16 items, which are rated as 0 (no) or 1 (yes). All items met the DSM-IV criteria A_1 about the objective threat of the traumatic experience. This questionnaire was successfully used in previous studies of trauma and severe mental illness (Mueser et al., 1998, 2007; Resnick et al., 2003). Total score ranges from 0 to 16. The internal consistency in this sample was Cronbach's $\alpha = .65$.

The severity of posttraumatic stress disorder was measured by the PTSD Symptom Scale, self-report (PSS-SR; Foa, Riggs, Dancu, & Rothbaum, 1993). The PSS-SR asked participants to rate how often they experienced each of the 17 PTSD symptoms specified in DSM-IV (American Psychiatric Association, 1994; Table 1) during the last month, ranging from 0 (not at all / only one time) to 3 (5 or more times a week / almost always). The sum of these scores represented the severity of PTSD symptoms. Total scores ranges from 0 to 51. Foa et al. (1993) reported satisfactory internal consistency, high test-retest reliability, and good concurrent validity with other PTSD measures. The internal consistency in this sample was Cronbach's $\alpha = .88$. The individual was also asked to give a short description of their worst traumatic experience in order to determine if the participant met the A_1 criteria for exposure to a traumatic event specified in DSM-IV (APA, 1994; Table 1). To investigate the patient's response to the traumatic event, three self-report questions assessing intense fear, helplessness, and horror were added (criteria A_2). Further questions were posed about the duration of the symptoms and the impairments these symptoms caused in order to investigate whether the person

met the E and F criteria for PTSD specified in DSM-IV (APA, 1994; Table 1). To assess whether the participant met the criteria for PTSD, two different scoring rules were used. One scoring a symptom as present if it was rated as at least 1 (once a week or less/ once in a while), and another more conservative scoring rule scoring a symptom as present if it was rated at least 2 (2 to 4 times a week / half the time).

To assess participants' thoughts and beliefs the Post Traumatic Cognitions Inventory (PTCI) was used (Foa, Ehlers, Clark, Tolin & Orsillo, 1999). This 33-itemed self-report questionnaire contained three subscales, namely negative cognitions about self (21 items), negative cognitions about the world (7 items) and self-blame for the trauma (5 items). Answers were rated on a 7-point scale ranging from 1 ("totally disagree") to 7 ("totally agree"). Total score ranges from 33 to 231. The PTCI was successfully used in a previous study of trauma and severe mental illness (Mueser et al., 2007). In the present sample, the Cronbach's alphas for the whole questionnaire, the subscale negative self-cognitions, negative cognitions about the world and self-blame were respectively .92, .92, .74 and .68.

Demographic information was collected using information in the patients' charts, including age, gender, primary diagnosis and medication.

Procedure

The study was conducted between July 23, 2007 and November 6, 2007. The target group consisted of patients who were in care of an outpatient clinical setting in Sittard, the Netherlands, who had schizophrenia or schizoaffective disorder as primary diagnosis. According to their charts, 173 patients satisfied this criterion (Figure 1). All primary therapists of these patients were approached. They were informed about the study and the procedure. After that, they were requested to make a selection of patients who met the inclusion criteria and to ask those patients for permission to report their names to the researcher. The inclusion criteria were age of at least 18 years, Dutch speaking and able to understand the questions. Exclusion criteria were severe medical problems preventing participation, insufficient mastery of the Dutch language, too florid psychotic condition or too chaotic speech preventing sufficient communication, and exclusion of the patients by the primary therapists themselves. Selected patients who gave their permission were contacted by telephone or during their visit at the setting. If they were interested in participation, an appointment was made. The majority of the appointments took place in the outpatient clinical setting, but when necessary, the researcher visited patients at their homes. In all situations, the patient and researcher were alone in one room during the

research. After the study was described to the participants, written informed consent was obtained from all patients preceding participation in the study.

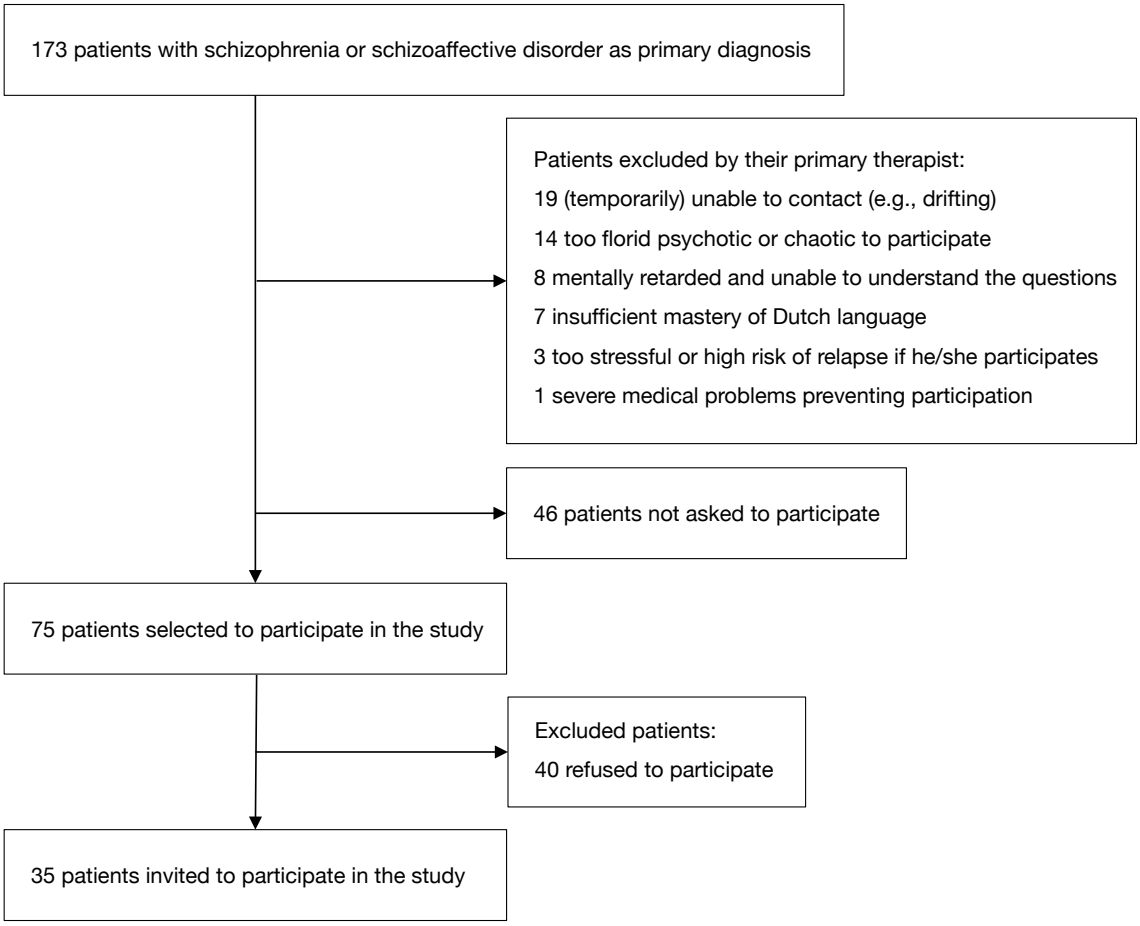

Figure 1. Recruitment stages

The participants completed the three self-report questionnaires in the presence of the researcher and answered the three open questions, which were added to the PSS-SR to assess the DSM-IV criteria for PTSD (APA, 1994; Table 1). All questions of the self-report questionnaires were read out loud. After each question, participants rated their answer. In this way, the researcher had the opportunity to detect concentration problems and to offer participants a short break. In addition, this manner of testing made it easier for the participants to ask for explanation if there was something unclear to them. The duration of the questionnaires varied between thirty and ninety minutes.

Chart review included all available information in the electronic charts of patients. Old paper charts were also included, as these were already scanned into the electronic charts. The present diagnosis of the patient was also available in the chart. To determine if a specific traumatic event was reported in the chart, parts of the chart that normally report information about traumatic events were read, furthermore all information available was searched for words including 'trauma', and other words specifically related to the event itself.

Data-analysis

Analyses were done with SPSS version 15.0. First of all, descriptive statistics (proportions, mean, and standard deviation) were calculated. Skewness and Kurtosis were calculated in order to check if the variables were normally distributed. Those deviating from normality were transformed using the square root (scores on the PTCI subscale negative self and whole PTCI). Independent sample t-test determined the difference in THQ-R total score between men and women. Chi-Square tests were used to determine the differences in gender on the individual THQ-R items. Linear regression analyses were conducted to determine the relationship between posttraumatic cognitions and PTSD symptom severity. In order to determine which proportion each subscale of the PTCI explained in PTSD symptom severity, backward regression analyses with $p<.05$ criterion including the PTCI subscales were run and Pearson correlations were calculated among the PTCI scales and PTSD symptom severity. Additional linear regression analyses were run to identify confounding and interactive effects of gender and diagnosis.

The authors certify responsibility for this study and have no known conflicts of interest.

Table 1

Diagnostic criteria posttraumatic stress disorder (PTSD) from DSM-IV

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

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

(2) the person's response involved intense fear, helplessness, or horror.

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

(1) recurrent and intrusive distressing recollections of the event, including images, thoughts, and/or perceptions.

(2) recurrent distressing dreams of the event.

(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and/or dissociative flashback episodes, including those that occur on awakening or when intoxicated).

(4) intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

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

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

(1) efforts to avoid thoughts, feelings, and/or conversations associated with the trauma

(2) efforts to avoid activities, places, and/or people that arouse recollections of the trauma.

(3) inability to recall an important aspect of the trauma

(4) markedly diminished interest or participation in significant activities

(5) feeling of detachment or estrangement from others

(6) restricted range of affect (*e.g.*, inability to have loving feelings)

(7) 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 at least two of the following:

(1) difficulty falling or staying asleep

(2) irritability or outbursts of anger

(3) difficulty concentrating

(4) hypervigilance

(5) exaggerated startle response

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

F. The disturbance causes clinically significant distress and/or impairment in social, occupational, and/or other important areas of functioning.

RESULTS

Prevalence of lifetime traumatic experiences

To test the hypothesis that lifetime traumatic events are more common among people with schizophrenia or schizoaffective disorder than in the general population, the Trauma History Questionnaire – Revised (THQ-R) was used. Results are presented in Table 2. 97.0% of the participants reported having experienced at least one traumatic event during their lifetime, 81.8% at least two, and 60.6% at least three. The most common traumatic experience in this sample was the sudden and unexpected death of a close friend or loved one due to an accident, illness, suicide or murder (*N* = 23, 69.7%), followed by the threat of death or serious body harm (*N* = 13, 39.4%) and stalking causing fear or worries about own safety (*N* = 12, 36.4%).

Table 2

Traumatic experiences in the sample assessed by the THQ-R

	Males (*N* = 23)	*Females* (*N* = 10)	*Total* (*N* = 33)
	N (%)	*N (%)*	*N (%)*
Motor vehicle accident	6 (26.1)	3 (30.0)	9 (27.3)
Other accidents	3 (13.0)	1 (10.0)	4 (12.1)
Warfare or combat	1 (4.3)	0	1 (3.0)
Sudden and unexpected death of a close friend or loved one	16 (69.6)	7 (70.0)	23 (69.7)
Robbery involving a weapon	4 (17.4)	0	4 (12.1)
Been hit or beaten up and badly hurt by a stranger	6 (26.1)	3 (30.0)	9 (27.3)
Witnessed someone seriously injured or killed	5 (21.7)	2 (20.0)	7 (21.2)
Threat of death or serious body harm	9 (39.1)	4 (40.0)	13 (39.4)
Childhood physical abuse	2 (8.7)	0	2 (6.1)
Witness to family violence	4 (17.4)	0	4 (12.1)
Intimate partner abuse	3 (13.0)	3 (30.0)	6 (18.2)
Before 16th birthday, unwanted sexual contact with someone at least 5 years older	3 (13.0)	1 (10.0)	4 (12.1)
Before 16th birthday, unwanted sexual contact with someone about the same age	4 (17.4)	2 (20.0)	6 (18.2)
After 16th birthday, unwanted sexual contact	1 (4.3)	3 (30.0)	4 (12.1)
Stalking	7 (30.4)	5 (50.0)	12 (36.4)
Any other events that were life threatening, highly disturbing or distressing, or caused serious injury	12 (52.2)	4 (40.0)	16 (48.5)

Independent sample t-test showed no significant difference in the number of lifetime traumatic events between women and men (t = -.06, df = 31, p = .759), although Chi-Square tests showed that women reported significantly more unwanted sexual contact after 16th birthday than men (χ^2 = 4,320, df = 1, p = .038).

Trauma according to chart-review

In order to test whether the rate of lifetime trauma experiences were higher when measured with questionnaires than measured with information obtained with chart-review as stated by the second hypothesis, the description of the worst event that ever happened, assessed with the additional question to the PTSD Symptom Scale - self-report (PSS-SR), was used (see methods). Of these worst events, 69.7% were documented in the participants' charts.

Prevalence of posttraumatic stress disorder

To test the third hypothesis that the PTSD prevalence in people with schizophrenia or schizoaffective disorder is higher than in the general population, the PTSD Symptom Scale – self-report was used. There are several ways to determine the rate of current PTSD in this sample and two different scoring rules were used. Because the DSM-IV criterion A_1 of an objective threat may be less relevant in this population (as will be further discussed), rates including and excluding this criterion were determined. Including only the A_2 to F criteria according to the DSM-IV (APA, 1994; Table 1), using the scoring rule that a symptom is present if it is rated at least as 1 (once a week or less/ once in a while), 39.4% of the participants met the criteria for current PTSD. When DSM-IV criterion A_1 was also included, so only traumas with an objective threat were included, the prevalence decreased to 18.2 %. Using the conservative scoring rule scoring a symptom as present if it was rated at least 2 (2 to 4 times a week / half the time) and excluding criterion A_1, the number of participants meeting the criteria for current PTSD at the time of the study was 21.2%. The inclusion of criterion A_1 led to a decrease to 9.1%. A chart-review tested the fourth hypothesis supposing PTSD rates to be higher using a self-report questionnaire than using a chart-review. Results showed that none of the participants had PTSD diagnosis in their charts.

Posttraumatic cognitions related to PTSD symptom severity

To test the hypothesis that PTSD symptom severity is positively related to negative posttraumatic cognitions in people with schizophrenia or schizoaffective disorder, linear

regression analysis was used. The total score on the Post Traumatic Cognitions Inventory (PTCI) was significantly related to PTSD symptom severity as assessed with the PSS-SR (r = .74, p < .001). Additional linear regression analyses were conducted for the separate PTCI subscale scores to investigate if all three kinds of negative cognitions were related to PTSD symptom severity. Negative cognitions about self, negative cognitions about the world as well as self-blame were significantly positive related to PTSD symptom severity. Results are presented in Table 3.

Table 3

Pearson correlations among PTSD symptom severity and the posttraumatic cognitions

	1	2	3	4	5
1. PTSD symptom severity	1				
2. Total score on PTCI	.74 **	1			
3. Score on PTCI subscale negative cognitions about self	.67 **	.94 **	1		
4. Score on PTCI subscale negative cognitions about the world	.57 **	.81 **	.63 **	1	
5. Score on PTCI subscale self-blame	.46 **	.52 **	.28	.41 *	1

*** Correlation is significant at the .01 level (2-tailed)*

** Correlation is significant at the .05 level (2-tailed)*

To control for content overlap of the predictor variables, backward regression analysis including the PTCI subscales were run and the Pearson correlations among the subscales were calculated. The backward regression analysis led to the exclusion of the subscale negative cognitions about the world ($ß$ = .14, p = .415). Although this subscale (r = .57, p < .01) correlated higher with PTSD symptom severity than the subscale self-blame (r = .46, p < .01), exclusion may be the consequence of the content overlap with the subscale negative cognitions about self (r = .63, p < .01) (Table 3).

Compared to the individual subscales of the PTCI, the total score on the PTCI correlated stronger to PTSD symptom severity. Moreover, the explained variation of the model including PTCI total score was higher than that of the model including the PTCI subscales, which remained after backward regression (55.0%; 52.9%). Hence, the PTCI total score was used in further analyses.

To identify possible confounding effects of gender (1 = male; 2 = female) and primary diagnosis (1 = schizophrenia; 2 = schizoaffective disorder), these variables were added to the linear regression analyses. The inclusion of one of these variables did not

lead to obvious differences in the value β, so no confounding effects of these variables were found (Table 4).

Table 4

Linear regressions with PTSD symptom severity as dependent variable to identify possible confounding effects

	β_1	β_2
Model 1	.74 ($p < .001$)	
Model 2	.74 ($p < .001$)	.04 ($p = .773$)
Model 3	.75 ($p < .001$)	.06 ($p = .631$)

Model 1: PTSD symptom severity = $\beta_0 + \beta_1 \cdot$ PTCI total score + ε

Model 2: PTSD symptom severity = $\beta_0 + \beta_1 \cdot$ PTCI total score + $\beta_2 \cdot$ gender + ε

Model 3: PTSD symptom severity = $\beta_0 + \beta_1 \cdot$ PTCI total score + $\beta_2 \cdot$ primary diagnosis + ε

Linear regression analyses including the interaction variables gender (ß = .43, p = .648) and primary diagnosis (ß = .46, p = .608) with PTCI total score, showed no significant interactions of these variables, considering the relationship between posttraumatic cognitions and PTSD symptom severity.

DISCUSSION

This study examined the prevalence of trauma and PTSD in patients with schizophrenia or schizoaffective disorder in an outpatient clinical setting as assessed by questionnaires and compared these with rates found using chart-review and rates found in the general population. Furthermore, we examined the applicability of the cognitive model of PTSD in this population by investigating the association between posttraumatic cognitions and PTSD symptom severity.

The results showed that 97.0% of the patients reported having experienced at least one traumatic event in lifetime, confirming our hypothesis that the prevalence of lifetime traumatic events in this sample of people with schizophrenia or schizoaffective disorder is higher than in the general population (39-56%; Breslau et al., 1991; Kessler et al., 1995). The presence of gender differences in the general trauma literature were not found in this study, as the number of experienced traumatic events was not significantly higher for women. However, significant gender differences were found for one specific trauma: women reported experiencing more unwanted sexual contact after age 16 than men. These findings correspond partially with previous studies reporting gender

differences in traumatic experiences among severe mental illness samples. However, this study did not show that women experienced more sexual abuse as a child or that men were more likely attacked with a weapon or witnessed a killing or serious injury more frequently, as was found in previous studies (Kessler et al., 1995; Mueser et al., 1998, 2007). Our findings may differ from results of prior research due to the small sample size.

The results also confirm our second hypothesis that lifetime trauma experiences are higher when measured with a questionnaire than measured with information obtained with chart-review. Chart-review determined that only 69.7% of the events that were experienced by the patients as worst event in their life were reported in their charts. In conclusion, these findings indicate that lifetime traumatic events are under-detected in this sample. Note that the chart-review included charts, which were put into the archives. The primary therapists can retrieve these, but it is unclear whether primary therapists are well acquainted about all information in the archived charts. Hence it is possible that more than 30.3% of traumas are not known by the therapist.

The results also support the third hypothesis that PTSD is more prevalent among people with schizophrenia or schizoaffective disorder than in the general population, as measured in previous studies. In this sample, 9.1% to 39.4% of the patients had current PTSD, depending on the scoring rule used and the inclusion or exclusion of DSM-IV criterion $A_{1,}$ while the rates of lifetime PTSD in the general population range from 7.8 to 9.2% (Breslau et al, 1991; Kessler et al., 1995) and the point prevalence which was determined at 2% (Stein et al., 1997). By definition, DSM-IV excludes psychotic experiences as meeting the A_1 criterion for a traumatic event. However, many studies suggest that a substantial proportion of people with psychosis develop PTSD in response to their psychotic experiences and/or their hospital treatment (Morrison et al., 2003; Mueser & Rosenberg, 2003; Shaner & Eth, 1989; Williams-Keeler et al., 1994). In a sample of participants who had been hospitalized following a psychotic episode, 52.3% met the criteria for post-psychotic PTSD (Shaw, McFarlane, Bookless & Air, 2002). The development of PTSD was associated with the psychological distress of the experience, supporting the view that subjective instead of solely objective threat determines the impact of the traumatic event. The way an event is perceived seems to play a major role in the development of PTSD (Kilpatrick et al., 1989). Therefore, the A_2 criterion (participant's response to the event involving intense fear, helplessness, or horror) may be of importance in the determination of PTSD diagnosis, regardless of the (objective) nature of the precipitating traumatic event (A_1 criterion). According to this argument, the prevalence of current PTSD in the studied sample ranges from 21.2% to 39.4%,

depending on the scoring rule. This prevalence is consistent with prior research on current PTSD in patients with schizophrenia or schizoaffective disorder (Mueser et al., 1998; 2004; Resnick et al., 2003). Although Mueser et al. (1998) found a higher PTSD rate in people with schizoaffective disorder than in schizophrenia, these differences in primary diagnosis were not found in this study. Neither were any gender differences found. Although gender difference is one of the most robust findings in the general PTSD literature, results of earlier studies among men and women with a severe mental illness found no differences (Brewin, Andrews & Valentine, 2000; Cascardi et al., 1996; Davidson & Smith, 1990; McFarlane et al., 2001; Mueser et al., 1998, 2001; Ollf, Langeland, Draijer & Gersons, 2007; Switzer et al., 1999). The lack of gender differences in PTSD among patients with schizophrenia or schizoaffective disorder may be due to the overall high rate of trauma exposure in this population.

A reason to believe that the actual prevalence of current PTSD is closer to the 21.2% than the 39.4% is the symptom overlap between schizophrenia and PTSD. Participants could have scored higher on certain items of the PSS-SR due to symptoms of their primary diagnosis. For example difficulty with concentrating can be symptom of schizophrenia as well as PTSD. Nevertheless, the diagnosis of PTSD depends on several criteria, so the influence of the symptom overlap on PTSD diagnosis will be limited. Despite the limitation in our ability to draw definite conclusions about the precise prevalence of PTSD in people with schizophrenia or schizoaffective disorder due to the above difficulties differentiating psychotic and post-traumatic symptoms in this population, even the most conservative estimate of the prevalence of current PTSD supported the hypothesis that PTSD prevalence in this group is higher than in the general population.

A chart-review confirmed the fourth hypothesis that rates of PTSD are higher when using a self-report questionnaire than when using a chart-review, because none of the participants in this study received this diagnosis in their charts. In accordance with previous studies, these findings suggest that PTSD is under-diagnosed in this population (McFarlane et al., 2001; Mueser et al., 1998; Zimmerman & Mattia, 1999). Several explanations are suggested to explain this phenomenon of under-detection and under-diagnosis. One of these explanations assumes that PTSD symptoms are often not the presenting complaint, as patients generally will not volunteer this information either because of reluctance to revisit the trauma, or because they are afraid of the clinician's response, or because they simply do not recognize the relevance of any prior trauma to their current problem (Howgego et al., 2005; Jacobsen & Richardson, 1987). Moreover,

physicians and psychiatrists working with schizophrenic patients may be so focused on assessing psychotic symptoms and the patient's functional level that they do not think about the issue of trauma. There may also be an underlying assumption that psychosis is the main problem to focus on in this population. Further, the results may represent poor documentation of assessments and findings. However, they may also be a consequence of change in PTSD symptoms after intake or initial assessment, or patients being more forthcoming, or more comfortable sharing in the study context as opposed to during the clinical consultations with their therapist. Another explanation is the high degree of symptom overlap between PTSD and other diagnoses, contributing to diagnostic confusion when trauma histories are not specifically obtained (Brady, Killeen, Brewerton & Lucerini, 2000). Symptom overlap is especially evident in schizophrenia, as symptoms of schizophrenia may be confused with or contribute to symptoms of PTSD. For example, hallucinations may be confused with flashbacks or other re-experiencing symptoms, and negative symptoms may be confused with avoidant symptoms of PTSD. However, there is evidence that PTSD can be diagnosed reliably among patients who have a severe mental illness (Rosenberg et al., 2001).

The results of this study also support the last hypothesis about the association between posttraumatic cognitions and PTSD symptom severity. Negative posttraumatic cognitions were positively related to PTSD symptom severity. No confounding effects were found of gender or diagnosis, nor significant interaction of these variables. This suggests that there are no significant differences in the relationship between posttraumatic cognitions and PTSD symptom severity according to gender or primary diagnosis in this sample.

Each of the three subscales of the PTCI containing negative cognitions about self, negative cognitions about the world and self-blame for the trauma, correlated positively with PTSD symptom severity. These results support the cognitive model of PTSD of Ehlers and Clark (2000), which suggests that negative appraisals about traumatic events and how they are responded to can have major effects on individuals' beliefs or underlying cognitive schemas about themselves, other people, or the world in general and play a role in the maintenance of PTSD. This relationship has been demonstrated in previous studies, but to the best of the author's knowledge, this was the first study identifying this relationship in a sample of exclusively patients with schizophrenia or schizoaffective disorder, for whom cognitive deficits are part of the disorder. A recent study among people with a severe mental illness showed that changes in trauma-related

cognitions may mediate changes in PTSD symptoms in this group as well (Mueser et al., 2007). Together with our findings, these results suggest that the cognitive model of PTSD may be applied to this group, despite the known cognitive deficits among these individuals. These cognitive deficits do not appear to significantly interfere with cognitive therapy, as suggested by the several studies showing positive results about the effectiveness of cognitive-behavioural therapy for the treatment of schizophrenia (Turkington et al., 2008). As the effectiveness of cognitive-behavioural therapy (CBT) in treating PTSD has been shown in many studies among non-psychotic samples and recently in a sample including schizophrenia and schizoaffective disorder (Bisson et al, 2007; Bradley et al, 2005; Mueser et al., 2007; Schnurr et al, 2007; Van Etten & Taylor, 1998), this would be an important adjunct treatment for individuals with schizophrenia or schizoaffective disorder and co-morbid PTSD.

Relation between trauma, PTSD and psychosis

Looking more closely at the seven cases identified with PTSD using the conservative scoring rule and excluding the A_1 criterion, three PTSD-related traumas were absolutely non-psychotic and involved objective threat as mentioned in the A_1 criterion of the DSM-IV definition of PTSD. In two of the seven cases the PTSD-related trauma happened before the onset of their illness, two after the onset of their illness and in the other cases the trauma was experienced in the same period as the onset of the illness and it remained unclear what happened first. These findings seem to correspond to the three relationships found in previous studies: psychosis can cause PTSD, trauma can cause psychosis, and psychosis and PTSD can both be part of a spectrum of responses to a traumatic event (Morrison et al., 2003).

Limitations

There are several limitations of this study. First of all, the recruitment procedure could have caused sampling bias, as the approach of patients meeting the inclusion criteria relied upon referrals from the primary therapists of these patients. Therapists may have been reluctant to recommend participation to individuals with severe symptoms or known trauma histories, due to concerns about potential stress of the study. As a consequence, the prevalence of current PTSD may be higher than found in this study.

Another limitation was the use of the primary diagnosis as documented in the charts of the participants, instead of using a structured clinical interview to determine the psychiatric diagnosis. Furthermore, using the self-report questionnaires was a less

rigorous way of assessing trauma history, current PTSD and posttraumatic cognitions than using a structured clinical interview. However, in this study the self-report questionnaires were also read aloud to the participants, thus decreasing any possible comprehension problems associated with attention or reading problems; in addition, many other studies have successfully used self-report questionnaires in comparable samples (Goodman et al., 1999; Mueser et al., 2001; 2007; Resnick et al., 2003).

Not all internal consistencies of the questionnaires used were satisfying. Firstly, the internal consistency of the THQ-R was with a Cronbach's alpha of .65 weak. The exclusion of the item asking about the experience of a sudden and unexpected death of a close friend or loved one would lead to a higher reliability. However, as such an event can be very traumatic and was experienced by many people is our sample, we decided to maintain this item in the scale. Secondly, the PTCI subscale self-blame had an internal consistency of .68, which can probably be attributed to the small number of items this scale consisted of. Despite of these two weak reliabilities, all measures that were included in the analyses in order to determine the relationship between the posttraumatic cognitions and PTSD symptom severity, had satisfying to good internal consistencies.

Although participants were asked to report one single traumatic event prior to the questionnaire-part of the PSS, many found it hard to choose one event and reported multiple events. It would have been preferable to assess the PTSD symptoms for every qualifying traumatic event separately. Moreover, chart-review included only the event reported as worst event by the patient. It is unclear whether the findings about under-report can be generalized to other events.

Another limitation is the relatively small sample size of the study, which make the results less reliable. Caution is required when generalizing the conclusions of this study to other groups. It should be noted that nearly all participants were taking a variety of medications, including antipsychotics, anxiolytics, antidepressives and sleep medication. However, medication use will probably not influence the results in a systematic way. Furthermore, it is not feasible to conduct a study of schizophrenia without having the patients who are on multiple medications. Because medications as well as co-morbid disorders were not excluded in this study, the results are more generalizable to other patients with schizophrenia or schizoaffective disorder in outpatient community settings. Finally, because this study was a cross-sectional study, no conclusions can be made about causal relationships between trauma and psychosis. A longitudinal study needs to be done to investigate this.

Clinical implications

This study provides evidence of under-report of trauma and under-diagnosis of PTSD in charts of patients with schizophrenia or schizoaffective disorder in an outpatient clinical setting. At the same time, both trauma and PTSD are more prevalent in this group than in the general population. These findings underscore the importance of assessing trauma history as well as PTSD in the routine evaluation of patients with schizophrenia or schizoaffective disorder in outpatient clinical settings, preferably with help of valid assessment techniques. It is important that therapists recognize the prevalence and impact of traumatic experiences in the lives of their patients, considering their negative consequences on mental health outcomes. Improvements in training of physicians, psychiatrists and therapists working with patients with schizophrenia on recognition and detection of trauma and PTSD is recommended to help remedy this problem.

The results concerning the posttraumatic cognitions in relation to PTSD symptom severity support the cognitive model of PTSD, suggesting that treatment based on this model may be effective in treating PTSD in patients with schizophrenia or schizoaffective disorder. Cognitive-behavioural interventions may be able to modify the factors that are contributing to the maintenance of PTSD. Accurate detection and treatment of PTSD may be critical to reducing distress and improving the psychiatric and health functioning of these patients.

Future research directions

More research is needed to develop definite treatment guidelines for PTSD in people with schizophrenia or schizoaffective disorder. Longitudinal studies are needed to prove that posttraumatic negative cognitions play an important role in the maintenance of PTSD in this specific sample. Randomized controlled trials of cognitive-behavioural therapy in this specific group are recommended to determine the effectiveness of the treatment and the improvement on both PTSD as other psychiatric symptoms. Moreover, future studies should focus on the differences between psychotic-related and non-psychotic-related PTSD and the relation between trauma, PTSD and psychosis. Finally, longitudinal studies are needed to draw more definite conclusions about the causal relationships between trauma and psychosis.

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Chapter 8

General discussion

GENERAL DISCUSSION

The main results will be presented and discussed with respect to the three unresolved issues that were described in the introduction. Studies on pre-trauma vulnerability factors for posttraumatic stress disorder (PTSD) will be discussed first (chapter 2 and 3), followed by studies about pathogenic mechanisms that are involved in PTSD (chapter 4 and 5), and studies addressing generalizability of findings from trauma research to other populations (chapter 6 and 7).

1. Pre-trauma vulnerability factors for PTSD

Chapter 2 describes a prospective study that examined whether pre-trauma individual differences in extinction learning predict PTSD symptom severity among soldiers deployed to Afghanistan. Earlier cross-sectional studies have found an association between reduced extinction learning and PTSD (Blechert, Michael, Vriends, Margraf, & Wilhelm, 2007; Orr, et al., 2000; Norrholm et al., 2011), and one longitudinal study that took place shortly after trauma exposure suggests that reduced extinction learning predicts later PTSD symptoms (Engelhard, de Jong, van den Hout, & van Overveld, 2009). However, it remains unclear whether reduced extinction learning reflects a "pre-trauma" vulnerability factor for PTSD. One small prospective study with a pre-trauma assessment of extinction learning has been conducted, but did not control for pre-trauma symptoms (Guthrie & Bryant, 2006). It can therefore not be excluded that reduced extinction learning is a consequence of existing symptoms that actually put individuals at risk for posttraumatic stress.

In the current study, Dutch soldiers were tested about two months before they were deployed to Afghanistan, and, again, two months after their return home. Results showed that reduced extinction learning in a de novo conditioning task, before deployment, predicted PTSD symptom severity after deployment, even after controlling for pre-deployment stress symptoms, pre-deployment neuroticism scores, and number of stressors on deployment.

This study showed that reduced extinction learning is a pre-trauma vulnerability factor for PTSD, but several further issues deserve studying. There is general consensus that extinction learning does not erase the original fear association, and involves (corrective) inhibitory learning (i.e., inhibition of fear to danger cues due to secondary inhibitory associations; for overview, see Lissek et al., 2005; Myers & Davis, 2007). Compared to a low-symptom PTSD group, a high-symptom PTSD group showed impaired inhibition of a fear response in the presence of a safety signal (Jovanovic et al.,

2009). In the participants described in chapter 2, impaired inhibition learning after deployment predicted the persistence of PTSD symptoms over time (Sijbrandij, Engelhard, Lommen, Leer, & Baas, 2012). However, it remains unclear why some individuals show reduced extinction/inhibitory learning.

In the current study, extinction learning was operationalized as the decrease in US expectancy over the first 4 extinction trials. Its predictive value decreased with more extinction trials (cf. Norrholm et al., 2011), which suggests that *slow* extinction learning increases vulnerability to PTSD symptoms.

It is possible that patients who suffer from PTSD who show reduced extinction learning in a de novo conditioning task before treatment need more extensive extinction training, but this is an empirical issue. It is also important to examine interventions that may accelerate extinction learning in "slow learners". Recent clinical research found that the addition of D-cycloserine to exposure therapy resulted in heightened treatment effects specifically in patients with high levels of PTSD who needed longer treatment (de Kleine, Hendriks, Kusters, Broekman, & van Minnen, 2012). Such strategies for enhancing inhibitory learning may have important clinical implications for exposure therapy (Craske et al., 2008; Craske, Liao, Brown, & Vervliet, 2012).

Chapter 3 describes a prospective study of participants of study 2 to explore the temporal relationships between trait anger and PTSD symptoms. Participants were tested once about two months before deployment and about two and nine months after deployment. Results indicated that trait anger before deployment predicted PTSD symptom severity two months after deployment. This remained after controlling for pre-deployment stress symptoms and number of stressors on deployment, but not after controlling for pre-deployment neuroticism scores. Furthermore, PTSD symptoms severity and anger correlated strongly after deployment, but had no predictive value (i.e., PTSD symptoms at two months did not predict anger levels at nine months, or vice versa). Findings suggest that both trait anger and neuroticism are pre-trauma vulnerability factors that increase risk of PTSD (symptoms), with neuroticism being the stronger predictor.

An interesting question is how neuroticism and trait anger may contribute to PTSD. The former question (neuroticism) is addressed in chapter 4, but the latter (anger) is discussed here. Some experimental studies have shown that anger, similar to neuroticism and trait anxiety, facilitates threatening interpretations of ambiguous stimuli (Barazzone & Davey, 2009; Wenzel & Lystad, 2005). Since negative appraisals of (sequelae of) the traumatic event are involved in the maintenance of PTSD symptoms

(e.g., Bovin & Marx, 2011; Ehlers & Clark, 2000; van den Hout & Engelhard, 2004), this may explain the relationship between anger and PTSD development.

With respect to clinical implications, it is important to note that although neuroticism scores predicted PTSD symptoms, this relationship is partly based on symptom overlap: both constructs involve hyperarousal (see Engelhard, van den Hout, & Lommen, 2009). If patients show improvement after treatment, but remain symptomatic, this may be explained by trait-like symptoms (e.g., irritability, sleeping problems). The same may hold for trait anger. This seems relevant with respect to setting realistic treatment goals.

Several general issues related to **chapter 2 and 3** will be discussed below. These relate to the nature of the outcome variable, different trajectories related to psychological adjustment after trauma, and the issue of screening for vulnerability to PTSD.

An important issue related to prediction of symptoms relates to the nature of the outcome variable. The studies in chapter 2 and 3 used “PTSD symptom severity” as an outcome measure. However, PTSD is a heterogeneous construct, and the variety of clinical presentations is large. In fact, there are no less than 79,794 possible combinations of symptoms that meet DSM-IV criteria for PTSD. Different mechanisms may be involved in the development of specific symptoms, like re-experiencing symptoms (which is predicted by low working memory capacity; Brewin & Smart, 2005) and hyperarousal (which is predicted by emotional reasoning; Engelhard & Arntz, 2005). Future research using large samples to provide sufficient statistical power may elucidate such specific processes related to similar symptoms across psychopathology (e.g., Brewin, Gregory, Lipton, & Burgess, 2010).

Another promising way of examining risk factors for PTSD is by using statistical methods (e.g., latent growth modelling) that take different trajectories of symptoms into account. A recent large prospective, longitudinal cohort study among US military service members (Bonanno et al., 2012) showed that the majority of participants reported low PTSD symptoms before deployment to Iraq while symptom level remained low until several years later. A minority reported stable high PTSD symptoms across time, some showed reductions in PTSD symptoms from pre- to post-deployment, and some (6.7%) showed increases in PTSD symptoms after deployment (i.e., deployment-related PTSD). Studies taking place post-trauma cannot take such trajectories into account, but it seems important to examine various vulnerability and resilience factors for different trajectories.

Furthermore, studies on vulnerability factors for PTSD typically focus on a few variables. The field would benefit from studies that take into account multiple vulnerability factors, which enables testing the relative contribution of factors (Elwood, Hahn, Olatunji, & Williams, 2009; Lommen, Engelhard, & van den Hout, in prep).

A final issue discussed here relates to the role of pre-trauma screening in the prevention of PTSD arising after traumatic events. The fact that pre-trauma vulnerability factors predicted later PTSD symptoms in a military sample does not imply that these factors can be used for screening. The effects were small to modest, and there are currently no strong predictors that can be used for screening purposes before trauma exposure (i.e., that predict later PTSD or other psychopathology) (for discussion, see Wessely, 2005; Rona et al., 2006). In fact, psychological screening may even have negative consequences due to false positives. For intervention purposes, it seems more beneficial to identify those with high symptom-levels as soon as possible after trauma exposure, for example, individuals who develop acute stress disorder (e.g., Harvey & Bryant, 1998). Brief cognitive behavioural therapy in individuals with acute stress disorder is very effective in preventing chronic PTSD (Bryant, 2007).

2. Pathogenic mechanisms involved in PTSD

Chapter 4 describes an experimental study that examined a potential pathogenic mechanism that may account for the well-established association between neuroticism and anxiety disorders, including PTSD. Based on information processing research, we hypothesized that individuals with high neuroticism levels use a lower decision criterion to detect danger, which would lead to the detection of true alarms, at the expense of false alarms. Such a 'better safe than sorry' strategy may maintain fear: if false alarms are followed by avoidance, then irrational fear beliefs will not be disconfirmed. The study described in chapter 4 indeed found that, relative to healthy participants with low neuroticism scores, healthy participants with high neuroticism scores show increased avoidance of ambiguous stimuli (that are on a continuum between a safety and danger signals).

These results show an interesting parallel with findings of a recent study (Haddad, Pritchett, Lissek, & Lau, 2012) that showed increased fear potentiated startle responses to ambiguous stimuli in individuals with high trait anxiety (which is similar to neuroticism; Jorm, 1989), compared to low trait anxiety. These increased fear reactions were explained by exaggerated stimulus generalization (i.e., increased anxious reactivity to cues that show similarity to a danger cue), and not by sensitization (i.e., generally

increased anxious reactivity to cues). Exaggerated stimulus generalization may also explain increased avoidance of ambiguous stimuli in our study. This issue awaits future research.

Future research may test whether this better safe than sorry strategy differentiates patients with anxiety disorders from healthy controls, and predicts the development of anxiety in longitudinal studies. If this is the case, then meta-cognitions about thresholds of risk acceptance may be an interesting target for clinical interventions.

Chapter 5 describes an experimental study that tested whether computerized training would decrease emotional reasoning in individuals with fear of spiders, and if this would reduce fear-related cognition and behaviour. Emotional reasoning involves the tendency to use subjective feelings as a validation of thoughts and has been implicated in various anxiety disorders (Arntz, Rauner, & van den Hout, 1995; Beck & Emery, 1985; Engelhard & Arntz, 2005). In this study, individuals with fear of spiders were either assigned to experimental training aimed to decrease emotional reasoning or to control training. Results showed that the experimental manipulation decreased emotional reasoning in the experimental condition, compared to the control condition. After the training, danger ratings of a spider were lower in the experimental condition than in the control condition, after controlling for degree of spider fear and anxious feelings. This difference was maintained up to one day later. There were no differences between conditions in approach behaviour.

The experimental manipulation may have been too short (and indirect) to affect fear-related behaviour. Studies that have shown beneficial effects of computerized training targeting other cognitive biases in non-clinical and clinical populations (e.g., attention bias or interpretation bias; Hertel & Mathews, 2011; MacLeod, 2012) often involved multiple trainings over several weeks, which may optimize learning benefits and retention (Cepeda, Pashler, Vul, Wixted, & Rohrer, 2006; Hallion & Ruscio, 2011). To affect emotional reasoning outside of the laboratory, it seems likely that multiple sessions are needed. It may also be more effective to reduce emotional reasoning by cognitive strategies, in which people learn to detect emotional reasoning, and replace it by factual reasoning, but this issue awaits future research.

If emotional reasoning is a key factor in maintaining anxiety disorders (see Engelhard & Arntz, 2005), then training that directly reduces it should improve the disorder. As emotional reasoning training teaches participants to attend to more objective situational information (e.g., outcome probability; Slovic et al., 2002), one would expect them to process more disconfirmatory information about erroneous danger

beliefs. Thus, reducing emotional reasoning before exposure therapy may be particularly fruitful. This is an important area for future research.

3. Generalizability of findings from trauma research to other populations

Chapter 6 presents a study that aimed to test whether psychological factors that have been associated with PTSD in Western samples were also related to PTSD symptoms in Sri Lankan survivors of the tsunami that took place on December 26, 2004, which was 15 months before the study was conducted. Results showed that 52% of the participants met criteria for current PTSD, based on a questionnaire and DSM-IV criteria for PTSD. Symptoms of PTSD were higher for participants who had negative interpretations of intrusive memories, were female, and for whom work equipment was lost and not replaced.

Negative interpretations of PTSD symptoms and female sex are also related to PTSD in Western developed countries (e.g., Brewin et al., 2000; Ehlers & Clark, 2000). These findings suggest that trauma-exposed individuals in developing countries may also benefit from cognitive behavioural interventions that have shown to be effective for individuals with PTSD in Western countries. A recent study in individuals exposed to terrorist attacks in Thailand confirmed that cognitive behavioural therapy (CBT) was effective for PTSD, depression, and complicated grief (Bryant et al., 2011). It was delivered by Thai counsellors who had received a relatively short training on CBT, which suggests that implementation of psychological programs to help large numbers of PTSD sufferers in non-Western countries seems feasible. CBT may be particularly effective for refugees and individuals from non-Western developed countries if ethno-cultural differences in expression of traumatic stress are incorporated (Nicholl & Thompson, 2004).

Furthermore, the results suggest that in addition to psychological interventions, practical and basic needs should be taken into consideration after a disaster like the tsunami. In this Sri Lankan sample, replacement of lost work equipment may have remediated negative cognitions about permanent change and may have helped creating a safe base for their life. In addition, replacement of work equipment may have encouraged exposure to trauma-related stimuli, as many participants were fisherman or traders on a market situated near the sea.

Chapter 7 describes a study that explored the prevalence of traumatic events and PTSD in patients with schizophrenia and schizoaffective disorder, and tested applicability of the cognitive model of PTSD. In a study of 33 outpatients with

schizophrenia or schizoaffective disorder, we found that nearly all participants (97%) reported at least one prior traumatic experience. Based on a questionnaire and DSM-IV criteria for PTSD, 18% of the participants screened positive for current PTSD. However, trauma exposure was underreported, and PTSD was undiagnosed in these patients. Furthermore, negative posttraumatic cognitions were positively related to PTSD symptom severity, which suggests that the cognitive model of PTSD may be applicable to patients with schizophrenia or schizoaffective disorder who suffer from co-morbid PTSD.

With regard to clinical implications, it seems important to assess past traumatic events and PTSD in the routine evaluation of patients with schizophrenia or schizoaffective disorder, because PTSD may adversely affect health and treatment outcome, and effective treatments are available. The current and other results (Mueser et al., 2007) further suggest that the cognitive model of PTSD may be applied to this group. Moreover, the first studies that have applied prolonged exposure therapy for PTSD to patients with present psychotic disorders are promising (van Minnen, Harned, Zoellner, & Mills, 2012), and suggest that standard PTSD treatment may also be effective to more complex patient populations. Also, Eye Movement Desensitization and Reprocessing (EMDR) therapy for PTSD has been used in a recent pilot study in patients with psychotic disorders and co-morbid PTSD (van den Berg & van der Gaag, 2012). As argued by van Minnen et al. (2012), clinicians are often hesitant to provide PTSD treatment in patients with co-morbid and complex problems, while empirical evidence supports the use of trauma-focused treatment in these populations. Future research may test the applicability of the cognitive theory of PTSD and its treatment in other populations that are usually excluded in research, such as elderly patients and individuals with mental disabilities.

In sum, **Chapter 6 and 7** found that negative posttraumatic cognitions are associated with PTSD symptomatology in a traumatized population in a non-Western developing country and in a sample of outpatients with a psychotic disorder. This is consistent with influential cognitive theory of PTSD (Ehlers & Clark, 2000) and many empirical studies that took place in Western developed countries among people without severe mental illnesses. These findings suggest that the contribution of cognitive factors to PTSD may be quite universal.

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Nederlandse samenvatting

Dutch summary

Na het meemaken van een stressvolle gebeurtenis hebben veel mensen last van psychische klachten. Bij de meeste mensen verdwijnen deze klachten weer geleidelijk, maar bij sommige blijven ze bestaan. Wanneer de klachten langer dan een maand aanwezig zijn en leiden tot een beperking in het dagelijks leven, zoals relatieproblemen of niet meer goed kunnen functioneren op het werk, is er sprake van posttraumatische stressstoornis (PTSS). PTSS wordt gekenmerkt door herbelevingen van de gebeurtenis, vermijding van dingen die herinneringen aan de gebeurtenis oproepen en verhoogde prikkelbaarheid. Om inzicht in de etiologie van PTSS te vergroten, is het belangrijk om te begrijpen hoe het kan dat de één blijvende klachten ontwikkelt en de ander niet. Individuele verschillen lijken dus een rol te spelen in de kwetsbaarheid voor PTSS. Veel studies hebben onderzoek gedaan naar risicofactoren voor PTSS. Zo blijken factoren vóór de traumatische gebeurtenis (zoals geslacht, opleidingsniveau, persoonlijkheidstrekken), tijdens deze gebeurtenis (zoals dissociatie, ervaren levensbedreiging), en erna (zoals sociale steun, betekenisgeving aan aanvankelijke PTSS symptomen) samen te hangen met PTSS.

Conditioneringstheorie over PTSS

De huidige conditioneringstheorie (Davey, 1997) biedt een mogelijke verklaring waarom sommige mensen wel een angststoornis zoals PTSS ontwikkelen en andere niet. In het geval van PTSS raakt volgens deze theorie een neutrale stimulus geassocieerd met een ongeconditioneerde stimulus (de traumatische gebeurtenis), die op zichzelf een ongeconditioneerde respons (bijvoorbeeld angst) activeert. De oorspronkelijk neutrale stimulus wordt dan een geconditioneerde stimulus en roept de verwachting van de ongeconditioneerde stimulus op met de daarbij horende geconditioneerde respons. Bijvoorbeeld: een soldaat rijdt tijdens een uitzending in Afghanistan op een bermbom. Op het moment van de ontploffing vreest de soldaat voor zijn leven, maar hij weet ongeschonden uit het voertuig te komen. Na de ervaring roepen stimuli die op een bepaalde manier op (een aspect van) deze ervaring lijken de verwachting van de stressvolle ervaring op en de daarbij horende angstreactie. Zo kan na deze ervaring rijden in een voertuig op zichzelf bij deze soldaat een angstreactie oproepen, doordat het rijden geassocieerd is geraakt met de ontploffing.

Volgens deze theorie kan een angst ook weer afgeleerd worden door te leren dat de geconditioneerde stimulus niet langer geassocieerd is met de ongeconditioneerde stimulus. Met andere woorden: de geconditioneerde stimulus roept niet langer de verwachting van de ongeconditioneerde stimulus op. In het voorbeeld van de soldaat

betekent dit dat wanneer de soldaat blijft rijden in voertuigen en er geen explosies meer zijn, de associatie tussen het rijden in een voertuig en de explosie verzwakt. Ofwel: de associatie tussen rijden in een voertuig en *geen* explosie versterkt. Als gevolg hiervan daalt de verwachting van een explosie tijdens het rijden weer en vermindert of verdwijnt de angstreactie. De huidige conditioneringstheorie verklaart dus hoe een angst aangeleerd en ook weer afgeleerd kan worden. Deze kennis heeft daarmee belangrijke implicaties voor de behandeling van angststoornissen.

Cognitieve theorie van PTSS

Volgens het cognitieve model van PTSS (Ehlers & Clark, 2000) komt angst voort uit een gevoel van dreiging in het hier en nu. Deze huidige dreiging wordt veroorzaakt door een negatieve interpretatie van de traumatische gebeurtenis zelf en de nasleep ervan, alsmede door het ontoereikend verwerken van de traumatische gebeurtenis in het autobiografische geheugen. Om het gevoel van huidige dreiging te verminderen, worden verscheidene gedrags- en cognitieve strategieën gebruikt, zoals vermijding van dingen die doen denken aan de gebeurtenis, of rumineren over de gebeurtenis. Op de lange termijn houden deze strategieën echter de PTSS-symptomen in stand en kunnen ze er zelfs door verergeren. Uit onderzoek is gebleken dat cognitieve factoren uit dit model, waaronder negatieve interpretatie van de traumatische gebeurtenis zelf en de consequenties, voorspellend zijn voor de ontwikkeling van PTSS-symptomen. Daarnaast is cognitieve (gedrags)therapie gebaseerd op dit model effectief gebleken voor de behandeling van PTSS.

Onopgeloste zaken

Ook al is er uit onderzoek al veel bekend over risicofactoren voor PTSS, er zijn altijd nog vragen die tot nu toe onbeantwoord zijn. De onderzoeken in dit proefschrift hebben zich op drie van deze vragen gericht, die hieronder worden toegelicht.

Ten eerste betreft veel onderzoek naar risicofactoren voor PTSS correlationeel en longitudinaal onderzoek, waarbij de risicofactoren en de PTSS beide na de traumatische gebeurtenis zijn gemeten. Hierdoor is het onduidelijk of de risicofactor al voor de gebeurtenis aanwezig was, of een teken van PTSS-symptomatologie is dat zich tegelijkertijd ontwikkeld heeft met de PTSS-symptomen. Om antwoord te geven op deze vraag is prospectief onderzoek nodig, waarbij wordt getest of individuele verschillen die reeds vóór de traumatische gebeurtenis aanwezig zijn invloed hebben op de ontwikkeling van PTSS symptomen.

Ten tweede geldt voor veel voorspellers van PTSS dat het onduidelijk is *hoe* ze het risico op PTSS vergroten. Met andere woorden: welk mechanisme kan de relatie tussen de voorspeller en PTSS verklaren? Deze kennis is belangrijk om het ontstaan en de instandhouding van PTSS te begrijpen, maar kan ook van therapeutisch belang zijn als de onderliggende mechanismen die bijdragen aan symptomatologie, aangepakt kunnen worden in therapie.

Ten derde zijn de grenzen van hedendaagse, invloedrijke modellen voor PTSS nog onduidelijk. Het grootste deel van het onderzoek naar PTSS is uitgevoerd in ontwikkelde, westerse landen. Het is de vraag in hoeverre conclusies op basis van deze onderzoeken generaliseren naar onderontwikkelde, niet-westerse landen. Hetzelfde geldt voor een andere groep die vaak wordt uitgesloten van onderzoek, namelijk mensen met een ernstige psychische stoornis, zoals psychose. Aangezien traumatische gebeurtenissen en co-morbide PTSS relatief vaak voorkomen in deze groep, is het van groot belang om te weten of dezelfde onderliggende mechanismen een rol spelen.

Bevindingen in dit proefschrift

In **hoofdstuk 2** is een prospectieve studie beschreven waarin bij 249 militairen werd onderzocht of individuele verschillen in het afleren van een angst vóór een uitzending naar Afghanistan voorspellend waren voor posttraumatische klachten na de uitzending. Uit de resultaten bleek dat het moeilijk afleren van angst vóór uitzending een voorspeller was voor de ernst van PTSS-symptomen twee maanden na uitzending, zelfs na controle voor andere risicofactoren (reeds bestaande klachten vóór uitzending, neuroticisme vóór uitzending en aantal stressoren tijdens uitzending). Het moeilijk afleren van een angst, ofwel verminderd extinctieleren, draagt dus bij aan de kwetsbaarheid voor de ontwikkeling van PTSS-symptomen. Deze resultaten sluiten aan bij hedendaagse theorieën die het belang benadrukken van extinctieleren en de focus richten op leren dat de geconditioneerde stimulus niet langer de ongeconditioneerde stimulus voorspelt in de behandeling van angststoornissen.

In **hoofdstuk 3** is de relatie tussen boosheidsdispositie (boosheid als een karaktertrek) en de ernst van PTSS-symptomen over de tijd getest in dezelfde groep militairen als in het voorgaande hoofdstuk. In deze studie werden data verzameld twee maanden vóór uitzending, twee maanden na uitzending en negen maanden na uitzending. De resultaten laten zien dat boosheidsdispositie vóór uitzending de ernst van PTSS klachten voorspelde na uitzending. De voorspellende waarde bleef bestaan na controle voor reeds bestaande klachten vóór uitzending en aantal stressoren tijdens uitzending,

maar verdween na controle voor neuroticisme. Verder bleken boosheid en de ernst van PTSS-symptomen na uitzending hoog te correleren, maar boosheid twee maanden na uitzending was geen voorspeller voor PTSS-symptoomernst op negen maanden en andersom ook niet. Zowel neuroticisme en boosheidsdispositie zijn dus kwetsbaarheidsfactoren voor de ontwikkeling van PTSS-symptomen, waarbij neuroticisme een betere voorspeller is. Uit eerder onderzoek (Engelhard, van den Hout, & Lommen, 2009) weten we dat de samenhang tussen neuroticisme en PTSS-symptomen gedeeltelijk verklaard kan worden door symptoomoverlap (beide constructen worden gekenmerkt door schrikachtigheid). Als patiënten na behandeling symptomen blijven ervaren, is het mogelijk dat deze symptomen voortkomen uit een dispositie en dus vóór het begin van de stoornis ook al aanwezig waren. Hetzelfde geldt mogelijk voor boosheidsdispositie. Met het oog op klinische implicaties is het van belang om rekening te houden met karaktertrekken bij het stellen van realistische therapiedoelen.

In **hoofdstuk 4** is in een experimentele studie onderzocht via welk mechanisme neuroticisme mogelijk het risico op angststoornissen vergroot. Er is getest of mensen die hoog of laag scoorden op neuroticisme anders reageerden op een reeks ambigue stimuli die op een continuüm lagen tussen een veiligheidsstimulus en een gevaarsstimulus. De resultaten laten zien dat de groep met hoge neuroticismescores meer ambigue stimuli vermeden en de gemiddelde stimulus die vermeden was op het continuüm dichterbij het veiligheidssignaal lag dan de groep met lage neuroticismescores. Dit verschil was echter alleen aanwezig als de groep 5 seconden kreeg om te kiezen of ze de stimulus wilde vermijden, niet als ze maar 1 seconde de tijd kreeg voor deze keuze. De groep met hoge neuroticismescores leek dus een strategie te hanteren die omschreven kan worden als “het zekere voor het onzekere nemen” en vermijdingsgedrag in de hand werkt. Vermijding van ambigue stimuli voorkomt mogelijke negatieve uitkomsten, maar voorkomt ook dat irreële cognities over negatieve uitkomsten, zoals voorkomt bij mensen met angststoornissen, worden gecorrigeerd. Deze strategie kan dus verklaren hoe neuroticisme bijdraagt aan het ontstaan en de instandhouding van angststoornissen.

In **hoofdstuk 5** is in een experimentele studie onderzocht of emotioneel redeneren verminderd kan worden door middel van een experimentele computertraining en wat de eventuele gevolgen hiervan op angstgerelateerde cognities en vermijdingsgedrag zijn. Emotioneel redeneren betreft de neiging om gevaar in te schatten op basis van gevoelens van gevaar in plaats van objectieve informatie over gevaar en is kenmerkend voor mensen met een angststoornis. Studenten met (enige mate van) spinnenangst en een hogere score dan gemiddeld op emotioneel redeneren werden

ingedeeld in een experimentele of een controlegroep. In de experimentele groep leerden participanten om gevaar in te schatten op basis van objectieve informatie in plaats van gevoelens en dus om niet langer emotioneel te redeneren, terwijl emotioneel redeneren in stand werd gehouden in de controlegroep. Na deze training werden cognities en toenaderingsgedrag ten opzichte van een spin getest. Uit de resultaten blijkt dat de training heeft geleid tot een vermindering in emotioneel redeneren in de experimentele groep en, zoals verwacht, geen verandering teweeg heeft gebracht in de controlegroep. Na controle voor spinnenangst en de mate van angstgevoelens rapporteerde de experimentele groep lagere gevaarscognities dan de controlegroep. Er was echter geen verschil tussen de groepen in toenaderingsgedrag van de spin. Het verlagen van emotioneel redeneren lijkt bij te kunnen dragen aan het aanpakken van irrationele gevaarscognities die kenmerkend zijn voor angststoornissen.

In **hoofdstuk 6** is onderzocht of dezelfde psychologische factoren die gerelateerd zijn aan PTSS op basis van onderzoek in westerse landen, gerelateerd zijn aan PTSS in een onderontwikkeld, niet-westers land. Honderddertien slachtoffers van de tsunami in Sri Lanka, die plaatsvond op 26 december 2004, zijn 15 maanden na de ramp geïnterviewd. Ongeveer de helft voldeed aan criteria voor PTSS. In overeenstemming met onderzoek in westerse landen was de ernst van de PTSS-symptomen hoger onder vrouwen dan onder mannen. Daarnaast bleken slachtoffers van wie verloren arbeidsmiddelen niet vervangen waren meer PTSS-symptomen te hebben dan slachtoffers van wie de spullen vervangen waren. In overeenstemming met het cognitieve model van PTSS waren negatieve interpretaties van PTSS-symptomen geassocieerd met de ernst van PTSS-symptomen. Deze resultaten impliceren dat traumaslachtoffers in ontwikkelingslanden mogelijk baat hebben bij cognitieve gedragsinterventies die effectief zijn gebleken voor de behandeling van PTSS in ontwikkelde landen. Dit sluit aan bij recente bevindingen over de effectiviteit van cognitieve gedragstherapie bij slachtoffers van terroristische aanslagen in Thailand (Bryant et al., 2011). Verder laten de resultaten zien dat bij een ramp zoals de tsunami niet alleen psychologische interventies belangrijk zijn, maar ook interventies gericht op praktische behoeften en basisbehoeften.

In **hoofdstuk 7** is onderzocht hoe vaak traumatische ervaringen en PTSS voorkwamen en gerapporteerd werden bij cliënten met een hoofddiagnose van schizofrenie of schizoaffectieve stoornis. Bovendien is getest of posttraumatische cognities, die volgens het cognitieve model een belangrijke rol spelen in de instandhouding van PTSS, ook in deze populatie samenhingen met PTSS-symptomen. Uit gegevens van 33 cliënten van een ambulante zorginstelling bleek 97% minstens één

traumatische gebeurtenis te hebben meegemaakt. Ongeveer 18% van de cliënten rapporteerden klachten die voldeed aan criteria voor PTSS. Uit dossieronderzoek bleek dat deze traumatische ervaringen vaak niet gerapporteerd waren en dat in geen van de gevallen PTSS was gediagnosticeerd. Verder hingen ook in deze groep negatieve posttraumatische cognities samen met de ernst van PTSS-symptomen. Gelet op de klinische implicaties lijkt het dus belangrijk om routinematig te screenen op traumatische ervaringen en PTSS-symptomen bij deze doelgroep, mede omdat co-morbide PTSS een negatief effect heeft op gezondheid en op behandeluitkomst. Daarnaast dragen deze resultaten bij aan het bewijs dat het cognitieve model voor PTSS ook toepasbaar is op deze doelgroep.

Curriculum Vitae

CURRICULUM VITAE

Miriam J.J. Lommen was born on July 9, 1984 in Tilburg, the Netherlands. She obtained her Bachelor's (2007) and Master's (2008) degree in Mental Health Science at Maastricht University, the Netherlands. In 2008, she started with her PhD project "Learning, reasoning, and trauma" at Utrecht University, the Netherlands, under supervision of Prof. dr. Iris Engelhard and Prof. dr. Marcel van den Hout. During her PhD, she worked as a Clinical Research Fellow at the Ambulatorium of the Faculty of Social Sciences, Utrecht University, and obtained her VGCt registration as a cognitive behavioural therapist in 2012. Since 2013 she works as a Postdoctoral Researcher at the Oxford Centre for Anxiety Disorders and Trauma, University of Oxford, in the United Kingdom.

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Poster Award at the 2010 Annual Congress of Vereniging voor Gedragstherapie en Cognitieve Therapie (VGCT najaarscongres) [Dutch Association of Behavioral therapy and Cognitive Therapy] with poster Lommen, Engelhard, Sijbrandij, and van den Hout, Do individual differences in fear conditioning before trauma predict later posttraumatic stress?

Honourable mention for poster presentation at the 2009 Annual Congress of Vereniging voor Gedragstherapie en Cognitieve Therapie (VGCT najaarscongres) [Dutch Association of Behavioral therapy and Cognitive Therapy] with poster Lommen and Restifo, Trauma and posttraumatic stress disorder: overlooked in schizophrenic patients?

Poster Award at the 2009 annual meeting of the European Association for Behavioural and Cognitive Therapies in Dubrovnik, Croatia, for the poster-presentation Lommen and Restifo, Trauma and posttraumatic stress disorder: overlooked in schizophrenic patients?