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Narrative Exposure Therapy
as Treatment for Posttraumatic Stress Disorder:
An Intervention Study

HOVEDOPPGAVE

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Bakgrunn: Narrativ Eksponeringsterapi (NET) er en behandlingsmetode som blir definert som en standardisert, korttids- intervensjon for behandling av posttraumatisk stressforstyrrelse (PTSD). Den har hovedsakelig blitt anvendt på individer utsatt for organisert vold i form av krigshandlinger og som lever under truende og usikre forhold. Det er foreløpig ingen vitenskapelige studier gjennomført på PTSD pasienter bosatt i Norge med annen traumatisk historie enn krigstraumer. **Mål for studiet:** Å undersøke effekten av NET på diagnostiserte PTSD symptomer, generell psykologisk tilstand og depresjon i et utvalg på 10 voksne pasienter rekruttert fra poliklinikker i region Vest. **Metode:** Pasientene ble kartlagt med Klinisk-Administrert PTSD-skala for DSM-IV (KAPS), Symptom Sjekkliste-90-Revidert (SCL-90-R) og Becks Depresjonsinventorie-II (BDI-II) før behandling og 1 måned etter behandling. **Resultater:** T-test for avhengig utvalg viste en signifikant reduksjon på 5 av 6 subskalaer på KAPS (n=10) etter behandling og 50 % av pasientene oppfylte ikke lenger PTSD-kriteriene. På SCL-90-R (n=8) viste subskalaen somatisering signifikant reduksjon. Reduksjon i total skåre på BDI-II (n=9) var tilnærmet signifikant. **Konklusjon:** Resultatene indikerer at NET kan være en lovende behandlingsmetode for PTSD pasienter som lever under ikke-truende, sikre forhold i Norge.

Abstract

Background: Narrative Exposure Therapy (NET) is a treatment method defined as a standardized, short-term intervention for treating posttraumatic stress disorder (PTSD). NET has mainly been applied on victims of organized violence and whose life conditions are threatening and unsafe. There are no published studies of NET applied on PTSD patients living in Norway with another trauma history than war-traumas. **Aim of the study:** To investigate the effect of NET on diagnosed PTSD symptoms, general psychological status and depression symptoms in a sample of 10 adult patients recruited from outpatient clinics in Health-region West. **Method:** The patients were assessed with the Clinical Administered PTSD-scale for DSM-IV (CAPS), the Symptom Checklist -90-Revised (SCL-90-R) and the Beck Depression Inventory (BDI-II) before and one month after NET-treatment. **Results:** T-tests for dependent samples showed a significant reduction on 5 of 6 subscales on CAPS (n=10) post-treatment and 50% of the patients did no longer fulfil the PTSD criteria. On SCL-90-R (n=8) there was a significant reduction of the somatisation subscale. Reduction in the total depression score on BDI-II (n=9) were borderline significant. **Conclusion:** The results indicate that NET can be a promising treatment method for PTSD patients living in non-threatening and safe life-conditions in Norway.

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Introduction

Outline

This thesis considers Narrative Exposure Therapy (NET) as a treatment method for PTSD, based on an intervention study of a Norwegian sample. It begins with an overview of the PTSD diagnosis in both a historical and contemporary context. Different aspects of the PTSD diagnosis, such as recent definitions, epidemiology, simple versus complex PTSD and etiological aspects will be presented. Relevant method elements such as cognitive behavioral treatments, exposure therapy and testimony therapy will be described to give the reader some background information of the basic principles and content of NET. NET is then described in further detail. Other treatment methods as EMDR will not be elaborated, but mentioned when suitable. KIDNET will also be mentioned, but not further described. A short presentation of ethical considerations and reaserch questions is followed by methods, procedures and results. The discussion will be introduced with a summary of the main findings, continued with a discussion of the findings related to CAPS according to the DSM-IV criteria for PTSD. Findings from the self-reported questionnaires SCL-90-R and BDI-II are presented, before a general discussion. Methodological considerations and some future directions will end the thesis.

There are a significant number of people suffering from post-traumatic stress disorder (PTSD), both in industrialized and developing countries. Population based studies have shown that between 5-15% of the population suffer from PTSD (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Yehuda, 2002). A PTSD diagnosis is characterized by being the cause of specific traumatic events. The diagnosis is often associated with severe functional and emotional impairment, and is a disorder characterized by high chronicity and comorbidity (Bichescu, Neuner, Schauer, & Elbert, 2007; Bradley, Greene, Russ, Dutra, & Westen, 2005; Lasiuk & Hegadoren, 2006). The knowledge remains scarce on why some people are more

vulnerable than others with regard to developing PTSD and concerning the underlying environmental and individual mechanisms.

Efforts have been made to develop psychotherapeutic treatment approaches for PTSD, and several proven effective. Among these are exposure therapies (Foa, Hembree, & Rothbaum, 2007; Foa, Rothbaum, Riggs, & Murdock, 1991), eye movement desensitization (EMDR) (Shapiro, 1989, 1996), cognitive behaviour treatments and cognitive therapy (Bradley et al., 2005). Although these methods have shown promising results, there is a need for further research on treatment approaches to PTSD. NET is a recently developed treatment method that is defined in literature as a standardized, short-term intervention for posttraumatic stress disorders resulting from organized violence (Bichescu et al., 2007; E. Schauer et al., 2004; M. Schauer, Neuner, & Elbert, 2005). This method has shown promising results cross-culturally. Studies have investigated different groups of refugees (Neuner, Schauer, Klaschik, Karunakara, & Elbert, 2004), political detainees (Bichescu et al., 2007) and torture victims (M. Schauer et al., 2005), but evidence based studies from Norway are yet to be done. Our study will investigate this further. NET incorporates elements from both exposure therapy (Foa et al., 2007) and testimony therapy (Cienfuegos & Monelli, 1983) (see p. 19). The theoretical foundation is based on psychological and biological knowledge about PTSD (M. Schauer et al., 2005).

Historical Background of PTSD

PTSD was recognized as a diagnosis and included in the DSM-III manual by the American Psychiatric Association (APA) in 1980. This created a new framework for the investigation and treatment of PTSD. It also represented a shift of focus regarding the perceived cause of the illness, from residing within the individual to be located outside the individual in the form of a traumatic event (Lasiuk & Hegadoren, 2006).

The first clinical reports of symptoms resembling PTSD can be traced back to the early 19th century. Dr. Waller Lewis described in 1861 a consistent group of symptoms in some travelling employees of railway post-offices who had been involved in collision and accidents but where no injury was apparently present prior to the accident. He called the syndrome *post-concussion syndrome* or *railway spine*. This was later renamed *traumatic neurosis* by Hermann Oppenheim in 1889 (Weisaeth & Eitinger, 1991). Apparently this was the first time the word "trauma" was mentioned in relation to the symptoms. The early debate around *railway spine* gave rise to the more general debate about the nature of psychological trauma. In the late 19th century there were also discussions about the relation between *railway spine* and *hysteria* as many of the symptoms were similar or overlapping. A student of the French neurologist Jean-Martin Charcot, Pierre Janet (1859-1947), observed that some of his patients experienced altered states of consciousness when reminded of distressing experiences from their past. He hypothesized that this was caused by a failure to integrate the memories of painful events, and the strong emotions associated with them, into their narrative memory. He concluded that distressing memories and emotions remained dissociated from consciousness, and Janet made a link between hysteria and dissociation (Van der Kolk & Van der Hart, 1989).

A vast amount of early clinical literature about psychological trauma is related to war. Observations of soldiers revealed that they often displayed symptoms of aroused cardiovascular system, extreme fatigue, dyspnea, palpitations, sweating, tremors and occasionally extreme fatigue after they were dismissed from duty. Arthur B.R. Meyers (1838-1921) termed the condition *soldiers heart* in 1870. Jacob Mendez Da Costa (1871) adopted parts of Meyers work and launched the term *irritable heart*, *effort syndrome* and *Da Costa syndrome* to describe the symptoms he observed among the victims of the American Civil War (Lasiuk & Hegadoren, 2006; Saigh & Bremner, 1999). World War I brought new

actuality to the question of how psychological trauma affects the human mind. The British military psychiatrist Charles Samuel Myers (1873-1946) used the term *shell shock* in an article published in the Lancet in 1915 where he described the psychological suffering of soldiers. At first he thought the condition was a neurological damage caused by the proximity of the soldiers to exploding shells. When the symptoms appeared in soldiers who had not been near exploding shells he had to reconsider the concept. He constructed two sub-groups, *shell concussion* (neurological) and *shell shock* (psychological) (Lamprecht & Sack, 2002).

Abram Kardiner's (1891-1981) work published during World War II (1941), created the foundation for the official diagnosis of PTSD in the DSM-III. Having studied under Sigmund Freud he explained the soldiers' symptoms within a neo-Freudian framework. He named the observed symptoms *war neurosis.* In his view the observed amnesia and physiological arousal were caused by soldiers attempt to preserve their ego integrity. Even though he saw similarities between *hysteria* and *war neurosis* he was hesitant to compare them, because of the negative connotations of *hysteria* (Kardiner & Spiegel, 1947). After World War II the various symptoms observed after witnessing or experiencing traumatic events were grouped under the term *psychic trauma.* The condition was linked to a nonspecific damage to the nervous system (Herman, 1997).

At the end of World War I, the US military developed a variety of screening instruments to select military personnel. The purpose was to select out service personnel that was unfit to stand the psychological demands of war. However, during World War II it became clear that despite this selection, a high number of psychiatric causalities among soldiers where still salient. This indicated that although some soldiers were more vulnerable to suffer from psychiatric illness in the aftermath of war, all soldiers were at risk thus leading to a shift from the ideas of symptom formation as neurological or personality inadequacies

towards the importance of environmental factors (Lasiuk & Hegadoren, 2006; Marlowe, 2000).

During the 1960s there was a paradigmatic shift within psychology and psychiatry. The focus shifted from psychoanalytic theory and research towards an emphasis on social and behavioural psychology. In particular, behaviourism was the most important approach in psychology where researchers primarily were interested in observable behaviour. The recognition of the complex interaction between man and environment resulted in the conceptualization of stress. The psychological health problems experienced by the Vietnam Veterans made the questions on psychological trauma of current interest. Their battle for economical compensation finally led to the inclusion of PTSD in the third revision of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III), including both civilian victims of trauma and victims of military trauma (Lasiuk & Hegadoren, 2006).

Recent Definitions of PTSD

The biological perspective on PTSD, and the research in this field, has made significant contributions to the understanding of the diagnosis. This research has focused on the neuroendocrinology of PTSD (Simeon et al., 2007; Yehuda, 2002; Yehuda, McFarlane, & Shalev, 1998), the autonomous nervous system (Yehuda et al., 1998), the genetic basis for vulnerability to PTSD (Radant, Tsuang, Peskind, McFall, & Raskind, 2001) and neuroanatomical changes in patients with PTSD (Bremner, 2007; Bremner et al., 1997; McEwen, 1999, 2002)

Today, there exist two main diagnostic systems classifying mental disorders; the DSM fourth edition revised (DSM-IV-R) developed by the American Psychiatric Association (2000), and the International Statistical Classification of Diseases and Related Health Problems 10th revision (ICD-10) developed by the World Health Organization (1992). These

systems classify PTSD somewhat differently. In ICD-10, PTSD is defined as a stress disorder, while in DSM-IV-R it is defined as an anxiety condition.

In the DSM-IV-R, PTSD is defined by the development of characteristic symptoms following exposure to an extreme traumatic stressor. The principal symptoms of PTSD include re-experiencing phenomena, avoidance of stimuli associated with the trauma and autonomic hyperarousal (see Table 1 in Appendix A). The DSM-IV-R has more extensive criteria for diagnosing PTSD. There is a specification of how intense the fear was experienced by the individual (criterion A), which is not specified in the ICD-10. There are further differences regarding the onset of the symptoms. According to the DSM-IV-R the duration of the symptoms must be present more than one month prior to the diagnosis, whereas ICD-10 does not state such a criterion. The DSM-IV-R also makes a distinction between acute, chronic and delayed onset of the symptoms. The disorder is considered acute if the duration of symptoms is less than three months, chronic if the duration of the symptoms is three months or more. The condition is considered having a delayed onset if the symptoms develop at least six months after the stressor. Importantly, it has a distinct diagnosis if the onset of the symptoms occurs *before* one month after the traumatic event. This is termed *Acute Stress Disorder* (ASD). ICD-10 does not include such distinctions. In our study, the DSM-IV-R criteria have been applied.

Epidemiology

The reported prevalence of PTSD in the literature varies. The National Comorbidity Survey, NCS, in the United States showed that 61% of men and 51% of women had experienced one or more traumatic events in their life as defined by the DSM-III-R criteria. Nearly 90% of the respondents in the NCS reported about exposure to a traumatic event (Kessler et al., 1995). The conditional risk of developing PTSD varies according to type of experienced trauma. For instance, there is seen a higher risk if the trauma involved assaultive

violence (Kessler, 2000). The results from the NCS indicate that the prevalence of experiencing a traumatic event is much higher than the probability of developing PTSD (see Figure 1 in Appendix B). The prevalence of PTSD depends on the presence of potential traumatic events in the population. According to DSM-IV-R, the prevalence in United States is approximately 8%. Among at-risk individuals, such as rape victims, military combat, captivity, ethnically or politically motivated trauma or genocide, the occurrence of PTSD is known to be as high as one third to half of the population (American Psychiatric Association, 2000).

There is also a gender difference concerning who develops PTSD, with women being twice at risk (Kessler et al., 1995). Both Yehuda (2002) and Kessler with coworkers (1995) find support for the presence of significant gender differences.

The occurrence of PTSD in less economically developed countries tends to be higher than in more developed countries (Keane, Marshall, & Taft, 2006). Studies have shown variations in prevalence from 0 to 10.4 % across Western industrialised countries (Gabbay, Oatis, Silva, & Hirsh, 2004; Kessler et al., 1995; Lindal & Stéfansson, 1993; Perkonigg, Kessler, Storz, & Wittchen, 2000). Substantially higher rates of PTSD are found in Non-Western, developing countries. One study showed prevalence rates ranging from 15.8 % to 37.4 % (de Jong et al., 2001). However, PTSD is a psychiatric diagnosis with culture bound aspects. What is experienced by the individual and the society as a traumatic event varies between cultures (Bowman & Yehuda, 2004).

The prevalence of PTSD between children exposed to different kinds of traumas varies, although it is known to be high among those who have experienced maltreatment (Gabbay et al., 2004).

PTSD is known to have high comorbidity rates with other psychiatric disorders, such as major depression, anxiety disorders, and drug abuse (Breslau, Davis, Andreski, & Peterson,

1991; Kar & Bastia, 2006; Kessler et al., 1995). Hence, it is important to assess possible differential diagnoses before diagnosing PTSD. There can be different explanations for high comorbidity rates. Either that a prior history of a psychiatric disorder contributes to an increased risk of developing PTSD or that PTSD is associated with an increased risk of developing a psychiatric disorder after developing PTSD (Kessler, 2000).

Malik and colleagues (1999) found that PTSD was associated with impaired quality of life. However, they found an improvement among those patients who received treatment. There is also an economic burden associated with having PTSD, both on an individual and on a societal level where for example the degree of work impairment is high (Kessler, 2000).

Simple versus Complex PTSD

In recent years, attempts have been made to differentiate the PTSD diagnosis into simple and complex PTSD. This is based on characteristics of the trauma and the pathological consequences for the individual (Herman, 1992, 1997; Taylor, Asmundson, & Carleton, 2006). The trauma involved in simple PTSD is characterized by being an isolated traumatic event in an otherwise safe life. This means that the trauma is an isolated event that happens once in a person's life, such as a natural disaster, a car accident, or experiencing a robbery or assault. It is not an ongoing event and the risk for prolonged exposure is low. The symptomatology of simple PTSD is known to be different from complex PTSD. Simple PTSD is less associated with severe personality disturbances, persistent somatisation, depression and dissociative symptoms (Taylor et al., 2006). Complex PTSD is more characterized by prolonged and repeated type of trauma as when the victim is held in captivity or is unable to flee from the perpetrator. Example of groups exposed to these kinds of multiple traumas include victims of repeated sexual abuse, domestic violence or neglect, refugees, political captives and victims of torture (Herman, 1992). These were the target groups for whom NET was developed. The symptomatology of complex PTSD is more

extensive and diffuse than simple PTSD. There is seen alterations in the personality, and vulnerability to repeated harm (Herman, 1992; Taylor et al., 2006) A study by Taylor et al. (2006) showed no differences in response to treatment between patients suffering from simple and complex PTSD. Research indicates that there is a heightened risk for developing PTSD among people with previous psychopathology and adverse life experiences such as previous traumas (Keane et al., 2006; Kolassa & Elbert, 2007).

Simple PTSD is what is being described as PTSD in the DSM-IV and ICD-10, while complex PTSD is not yet defined as a distinct diagnosis in these diagnostic manuals. Therefore, patients suffering from complex PTSD are not diagnosed differently from people suffering from simple PTSD.

Etiological Aspects of PTSD

Many aspects of the etiology of PTSD are yet to be discovered. As mentioned earlier, many people experience one or more traumatic events during their lifespan without developing PTSD. This is still an enigma which provokes a vast amount of investigations.

Several studies have shown that people who have been exposed to previous multiple traumatic events have an increased risk of developing PTSD (Bowman & Yehuda, 2004; Kolassa & Elbert, 2007; Neuner, Schauer, Karunakara et al., 2004). Victims of adverse childhood experiences or sexual abuse seem to have an increased risk of developing PTSD as adults (Brewin, Andrews, & Valentine, 2000). Elbert and Kolassa (2007) explains this as resulting from a "building block" effect, where different types of traumatic events add up to produce PTSD. Each traumatic experience contributes to an incremental enlargement of a neural fear network (about fear network, see p. 16). Neuner and colleagues (2004) propose that this could cause nearly anyone to develop PTSD if exposed to a sufficiently high number of traumatic experiences. Presence of psycopathology prior to trauma exposure have also been suggested as being a contributional factor to the development of PTSD, but a meta-analysis

found only a small association between psychiatric history and development of PTSD both in studies of military and civilians (Brewin et al., 2000). However, the presence of certain psychiatric conditions such as addictive disorder or conduct disorder have been proposed to increase the risk for exposure to traumatic events (Breslau et al., 1991). Other factors found to be associated with increased risk of developing PTSD are trauma severity, lack of social support and overall life stress (Brewin et al., 2000). Inexpedient coping styles and certain personality traits as neuroticism (Bowman & Yehuda, 2004) have also been suggested to play a significant role in the risk of developing PTSD.

There is evidence for neuroendocrinological changes and changes in brain function and structure in patients with PTSD. The main question is whether underlying abnormal neurophysiology contributes to a susceptibility of developing PTSD or how such vulnerability develops as a result of individual life experiences. This is still an ongoing debate.

Stress Hormones

PTSD is characterized by decreased levels of circulating cortisol in the body (Boscarino, 1996; Neylan et al., 2005; Song, Zhou, & Wang, 2008; Yehuda et al., 1995). There is an increased concentration and responsiveness in glucocorticoid receptors and an increased sensitivity of the HPA (hypothalamic-pituitary-adrenal) –axis' negative feedback system in individuals with PTSD (Yehuda, 2002; Yehuda et al., 1998) (see Figure 2 in Appendix B).This differs from others psychiatric conditions such as depression which is characterized by elevated levels of basal cortisol due to a *decreased* sensitivity of the HPA-axis' negative feedback system. Studies have found an association between decreased adrenocortical responsiveness and the exposure to traumatic events which may indicate that multiple traumatic events contributes to a sensitization of the arousal systems capacity to respond adequately (Bowman & Yehuda, 2004). Another explanation may be the presence of *initially* low levels of cortisol among those individuals who develops PTSD in the aftermath

of a traumatic event (Bowman & Yehuda, 2004). Low levels of cortisol measured immediately after trauma exposure (motor vehicle accident) predicted later development of PTSD (Yehuda et al., 1998). Animal models have supported these findings where rats with initial low levels of corticosterone (rodent parallel to human cortisol) were significantly more prone to display elevated startle responses in addition to develop increased vulnerability to chemically induced colonic pathology after a short series of foot shocks (Milde, Sundeberg, Grøseth, & Murison, 2003).

Brain Structure and Function in PTSD

Neuroanatomical research has shown that there is several brain structures involved in the development of PTSD. Brain imaging studies have shown changes in both the amygdala and the hippocampus in patients suffering from PTSD. Hippocampus is known to play an important role in learning and memory. Amygdala is a key structure in the mediation of fear (Breedlove, Rosenzweig, & Watson, 2007), and plays an important role in conditioned learning to stimuli associated with fear (LeDoux, 1996). The medial frontal cortex is known to be central in the mediation of extinction of such conditioned fear responses (Morgan & LeDoux, 1995; Rauch et al., 2000). Psychophysiological studies have shown that subjects diagnosed with PTSD are more likely to develop conditioned fear responses to stimuli than people exposed to traumatic event without PTSD (Orr et al., 2000). Functional Magnetic Resonance Studies (fMRI) studies have shown increased reactivity in the amygdala towards trauma-related stimuli. Interconnected areas associated with fear inhibition do not seem to provide sufficient inhibition of the fear response. A study by Rauch and coworkers (2000) using fMRI demonstrated that patients with PTSD had an increased activation of the amygdala in response to a general negative stimuli (masked facial stimuli) compared to a group of subjects who had been exposed to trauma, but who had not developed PTSD. Other studies using neuroimaging techniques found alterations in brain regions associated with

memory functions in PTSD patients and other patients suffering from other stress-related psychiatric disorders. These brain regions were the amygdala, the hippocampus and prefrontal cortex (Bremner, 2007; Bremner et al., 1997). Neuroimaging studies have also shown that the insular cortex plays an important role in many different neuropsychiatric disorders (Nagai, Kishi, & Kato, 2007). Amongst these is PTSD. The insular cortex is associated with functions such as motor and sensory functions, pain, eating, olfactory, tactile, and auditory information. It is supposed that the insular cortex has an integrational function for this information (Nagai et al., 2007). None of the mentioned studies differentiates between simple and complex PTSD.

The development and use of animal models have made significant contributions to the understanding of the biological basis of PTSD. Animal models have shown that stress hormones released from the adrenal cortex in the face of traumatic events affects the structure and the function of the hippocampus (McEwen, 1999).

Normally exposure to glucocorticoids increases the activity of the hippocampus, but when experiencing extreme stress the functioning is severely impaired. As a consequence the hippocampus and the medial frontal cortex does not mediate the intense signals of distress and arousal that is sent from the amygdala (Nutt & Malizia, 2004). Studies have also suggested that high exposure to adrenal steroids may cause permanent atrophy in the hippocampus (McEwen, 2002). These alterations have been suggested to be a neuroanatomical substrate for memory related symptoms in PTSD, such as intrusive memories (Yehuda, 2002). Even though several studies have shown alterations in certain brain regions among patients diagnosed with PTSD compared to healthy controls, one should be careful to draw conclusions about the causality. The observed alterations could be a predisposing factor for PTSD, which would implicate that the above mentioned brain regions in PTSD patients have been divergent from controls prior to the development of PTSD.

Elements of NET

Different short-term treatments have been developed for treating PTSD. This paper will focus on Cognitive Behavioural Therapies (CBT) including Exposure Therapies and Testimony Therapy as being particular important for the development of NET. NET includes elements from all of these.

Several studies have proved that CBT, and especially exposure and cognitive techniques, are powerful methods for treating PTSD in those who have experienced sexual assault (Foa et al., 1999; Foa et al., 1991) or physical assault or accidents (Marks, Lovell, Noshirvani, Livanou, & Thrasher, 1998; Tarrier et al., 1999).

Cognitive Behavioural Treatments

CBT is a method which combines both cognitive and behavioural principles. The clients are helped to recognize patterns of distorted thinking and dysfunctional behaviour, and through systematic discussion and carefully structured behavioural assignments helped to evaluate and modify both their distorted thoughts and their dysfunctional behaviours (Clark, 1989). In this thesis, CBT will be considered a superior concept for many different kinds of treatments (Rothbaum, Meadows, Resick, & Foy, 2000).

CBT for PTSD includes a wide variety of techniques, such as systematic desensitization, relaxation training, EMDR, prolonged exposure and cognitive therapy (Rothbaum et al., 2000). The different techniques have shown different results on treatment of PTSD, and in the following some will be considered.

Contemporary learning theory attempts to account for much of the development and maintenance of the PTSD symptoms (Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). Re-experiencing and arousal symptoms are considered as conditioned emotional responses that result from classical conditioning and are elicited by environmental stimuli. According to behavioural theory, although initial symptoms may be caused by trauma, many current

symptoms may represent attempts to manage trauma-induced distress (Rothbaum et al., 2000). Exposure to conditioned stimuli in the absence of the negative consequences is hypothesized to extinguish conditioned emotional reactions (Passer & Smith, 2001; Rothbaum et al., 2000). Therefore, exposure is assumed to be the appropriate treatment for PTSD (Rothbaum et al., 2000).

Exposure Therapy

Rothbaum and colleagues (2000) have compared numbers and types of studies supporting each type of CBT, and found that exposure therapy (flooding/in vivo/prolonged/directed) has the greatest number of well-controlled studies to support its use. They also found that exposure therapy was effective for a mixed variety of trauma survivors, compared to other treatments that were limited to single populations, like female assault survivors.

Exposure therapy is mainly focused on the behavioural part of CBT. This part is based on the assumption that maladaptive behaviours are learned the same way as normal behaviours, and that they can be unlearned through application of principles derived from research on classical and operant conditioning (Passer & Smith, 2001). This unlearning can be done in different ways and exposure with and without relaxation will be presented here.

One way of treating anxiety disorders based on the classical conditioning principles was introduced by Joseph Wolpe (1915-1997) in 1958, and called systematic desensitization (Passer & Smith, 2001). His goal was to eliminate the anxiety by using a procedure called counterconditioning, in which a new response that is incompatible with anxiety is conditioned to the anxiety-arousing conditioned stimulus. The patient was taught relaxation, then encouraged to progress step-by-step through a hierarchy of feared situations while maintaining the relaxation in order to reciprocally inhibit the fear response. Initially, Wolpe used *in vivo* (real life) exposure, but later changed to imaginal presentation because of the

greater controllability and ease of presentation this offered (Clark, 1989). Both approaches are highly effective in reducing anxiety (Passer & Smith, 2001). In treatment of PTSD in particular, exposure therapy without relaxation has been favoured over systematic desensitization (Rothbaum et al., 2000). Therefore elements from this kind of exposure therapy are included in NET, and prolonged exposure is most prominent.

The process of habituation. Habituation is the process where repeated exposure to the anxiety-producing situation results in an eventual decrease in anxiety (Foa & Rothbaum, 1998) (see Figure 3 in Appendix B). Dishabituation is the process where a habituated response temporarily returns. This may take place if the environment in which the habituated response was presented is changed, if a new stimulus is presented together with the habituated stimulus, or by changing the habituated stimulus (Schwartz, Wasserman, & Robbins, 2002) The process of habituation and dishabituation is important for successful treatment of PTSD. In treatment of anxiety disorders the principle of habituation is used through repeatedly exposing the client to the feared stimulus until the physiological anxiety associated with that stimulus is reduced (Jaycox, Foa, & Morral, 1998). In treatment of PTSD, the patient is requested to repeatedly talk about the worst traumatic event in detail while re-experiencing all emotions associated with the event (Neuner, Schauer, Klaschik et al., 2004). Re-tellings of the trauma story in a safe environment seem to be a standard component of most current therapeutic interventions with trauma survivors. This may facilitate psychological recovery by habituating trauma survivors to the anxiety associated with traumatic memories (Kaminer, 2006). For trauma survivors, it is the trauma memory itself that is the feared stimulus, because it has come to be associated with danger. The survivor equates remembering and re-telling with re-traumatisation and therefore attempts to avoid this (criterion C, see Table 1 in Appendix A). Due to this avoidance, the survivor never has the opportunity to learn that remembering will not place him back in danger (Foa & Rothbaum, 1998). Therefore,

exposure therapy in treatment of PTSD involves exposing the trauma survivor to his or her memories of the traumatic event (imaginal exposure) and to traumatic reminders in the environment (in vivo exposure) if that is possible (Foa & Rothbaum, 1998). In order to ensure long lasting effects of PTSD treatment it is important to be aware of the possible occurrence of dishabituation. One may reduce the risk of dishabituation through the exposure to general stimuli in the environment that may be fear- inducing.

Emotional processing theory. As mentioned at the beginning of this paper, Janet hypothesized already in the early 1900s that the reason why some of his patients experienced altered states of consciousness when reminded of distressing events was a failure to integrate these memories into their narrative memory (Van der Kolk & Van der Hart, 1989). Foa and Kozak (1986) have developed the theoretical approach called "emotional processing theory" (EPT). This theory integrates learning, cognitive and personality theories of PTSD, and explains why some individuals recover satisfactorily from a traumatic experience while others develop chronic disturbances (Foa & Rothbaum, 1998). Emotions are viewed as represented by information structures, or networks, in memory, and anxiety is thought to occur when an information structure that serves as program to escape or avoid danger is activated. Emotional processing is defined as the modification of memory structures that underlie emotions (Foa & Kozak, 1986). EPT holds that PTSD emerges because a pathological fear structure concerning the traumatic event develops (Foa, Steketee, & Rothbaum, 1989). Like other fear structures, this structure, or network, includes representations about stimuli, responses, and their meaning. That means that the fear structure of the traumatic event includes both sensory, cognitive, emotional and physiological elements (M. Schauer et al., 2005) (see Figure 4 in Appendix B). Any information associated with the trauma activates the fear structure which is mediated by the limbic structure. It is thought that the fear structure in people with PTSD includes a particularly large number of stimuli elements and therefore is easily activated. To

prevent this activation, people with PTSD must avoid many kinds of situations which may be associated with the trauma and consequently activate this structure. This results in the avoidance symptoms of PTSD (Rothbaum et al., 2000) (see Table 1 in Appendix A). According to EPT, successful therapy involves correcting the pathological elements of the fear structure, and that this corrective process is the essence of emotional processing. For the fear to reduce it is proposed that two conditions are required. The fear structure must be activated, and new information that includes elements which are incompatible with the existing pathological elements must be provided to be corrected. Through exposure therapy, the patient is assisted to get the trauma memory activated by confronting the trauma-related material. This activation gives an opportunity to integrate corrective information, and thus modify the pathological elements of trauma memory (Rothbaum et al., 2000). The assumption that emotional engagement is necessary for successful processing of the traumatic event and resultant recovery was first suggested by Janet and Freud (Breuer & Freud, 1957) so these theoretical concepts can easily be traced back in history.

It has been proposed that the representation in memory of traumatic events is different in individuals who remains traumatised after a severe experience, than in those who recovers (Foa & Rothbaum, 1998). Foa and Kozak (1986) proposed that persistent emotional disturbances following a highly distressing event indicate an inadequate processing of that event. Foa and Riggs (1993) proposed that traumatic memories are often disorganized and fragmented because they are encoded when the individual experiences extreme anxiety. Fragmentation can be defined as lack of flow in the narrative, with repetitions, unfinished thoughts and speech (Foa, Molnar, & Cashman, 1995). Foa and Riggs (1993) suggested that the natural recovery of trauma involves the organizing and streamlining of the memories. According to this assumption, successful trauma therapy should help victims to increase the traumatic memory organization towards getting more coherent narratives. Foa and colleagues

(1995) found that during the process of narrative organization in treatment, the narratives were reported to change in the direction of more organized thoughts.

Flooding versus prolonged exposure. Imaginal exposure is a technique that often makes use of re-tellings of the trauma narrative (Kaminer, 2006). Direct and prolonged exposure is examples of imaginal exposure techniques. They make use of re-tellings with and without relaxation respectively.

The technique of direct exposure, also called flooding, has frequently been employed in treatment of PTSD among Vietnam veterans (Boudewyns, Hyer, Woods, Harrison, & McCranie, 1990). This method starts with the patient learning to manage his or her anxiety through relaxation exercises and visualisation. In the following, the therapist and patient together develop a written description of the traumatic event, including facts, emotions and meaning constructions. The patient narrates the script to the therapist, and the therapist encourages the patient to give expressions to his or her feelings. This process is repeated for an average of 12 to 14 sessions (Keane, Fairbank, Caddell, & Zimering, 1989).

The method of prolonged exposure was developed by Foa & Rothbaum (1998) during their work with rape survivors. During this method, the trauma survivor undergoes continual exposure to traumatic memories through repeated re-tellings of the trauma story, over several sessions with each session lasting 60 minutes. The exposure is *not* paired with relaxation in order to activate and work with the pathological fear structure that has resulted from the trauma (Foa & Rothbaum, 1998). During treatment, the client creates a verbal narrative of what is being remembered from the traumatic event, while the therapist is encouraging him/her to verbalise all sensory, emotional and cognitive responses to the trauma. The client is also required to recount the events. Prolonged exposure has been demonstrated to significantly reduce PTSD symptoms (Foa et al., 1999; Foa et al., 1991).

Testimony Therapy

Testimony Therapy (TT) is a therapeutic method developed by Lira and Weinstein (published under the pseudonyms Cienfuegos and Monelli, 1983) for the treatment of traumatized survivors of the Pinochet regime in Chile. It has also been successfully applied in an uncontrolled trial to Bosnian refugees in the United States (Weine, Kulenovic, Pavkovic, & Gibbons, 1998). Testimony therapy is a brief individual psychotherapeutic method for working with survivors of state-sponsored violence. In Weine and colleagues' (1998) study with Bosnian refugees, they received in average six sessions, approximately 90 minutes, weekly or biweekly. With this method the patients construct a narration about their life from birth up to the present situation while focusing on details of the traumatic experiences. As the name suggests, TT can also serve social and political purposes by the documentation of human rights violations (Bichescu et al., 2007; Neuner, Schauer, Klaschik et al., 2004; M. Schauer et al., 2005). Together, the patient and the therapist make it possible to reveal the patient's trauma story. Several writers have also noted that testimony therapy offers survivors some therapeutic benefits in their trauma recovery. Testimony is based on theories that consider collective traumatisation to be at least as significant as individual traumatisation. It is assumed that testimony has effect through narration of the individual's personal experience of collective traumatisation. This usually takes place in a new social context where their story can be used to develop new collective understandings of history and communal identity. Through the testimony, the survivor will explicitly understand that their stories are becoming parts of a collective inquiry. The testimony seems to reduce individual suffering, even though the survivors have not sought trauma treatment (Weine et al., 1998). Despite these promising results for treatment, testimony therapy has not been widely used in mainstream psychosocial organizations, which primarily favour non-political approaches, such as supportive counselling (M. Schauer et al., 2005).

Narrative Exposure Therapy

Most of the studies which have proved that CBT, especially exposure and cognitive techniques, are effective methods for treating PTSD have been done in western, industrialized countries where the patients' life conditions are safe. Studies with NET have shown promising results in populations of civilians affected by war who continue to live in unsafe conditions (Bichescu et al., 2007; Neuner, Schauer, Klaschik et al., 2004; M. Schauer et al., 2005). NET has also been adapted for treatment of traumatized children and adolescent war survivors (KIDNET), with promising results (Onyut et al., 2005; E. Schauer et al., 2004).

NET was developed by the non-governmental organization Vivo (see p. 28) to meet the needs of traumatized survivors of war and torture (Bichescu et al., 2007; Neuner, Schauer, Klaschik et al., 2004; M. Schauer et al., 2005). With the successful results from both Testimony Therapy and Exposure Therapy, these techniques were included in NET on the assumption of being able to treat multiple traumas in a short time. Pure exposure methods (prolonged exposure therapy, direct exposure) usually work with the worst traumatic event in a person's life. People who have experienced organized violence, war and torture, usually have experienced several traumatic events and it is often impossible to identify the worst event before treatment starts (Neuner, Schauer, Klaschik et al., 2004; M. Schauer et al., 2005). To overcome this, exposure therapy was combined with the chronicity of testimony therapy in NET (Bichescu et al., 2007; Neuner, Schauer, Klaschik et al., 2004; M. Schauer et al., 2005). Instead of defining a single event as target for therapy, the patient constructs a narration of his/her whole life, following the timeline from birth to present, while focusing on the detailed report of the traumatic experiences (M. Schauer et al., 2005). The positive emotional events will also be included in the narrative, but in less detail than the traumatic events.

During NET, the patient is supposed to construct a detailed and consistent narration of his/her biography from birth to present in cooperation with the therapist. The therapist writes

the testimony down in past tense, and in first person. It can be used for documentary purposes if wanted by the patient. This procedure has been adapted to the special needs of refugees (Onyut et al., 2005). During the therapy, the narrative is being re-read at the beginning of every session, for the patients to do modifications and for the patient to be exposed to his/her story.

Little is known about treatment of PTSD in populations of civilians affected by war who continue to live under unsafe conditions. NET was developed to treat traumatized civilians in crisis regions where longer-term interventions are not possible, and designed for treating civilians who were unable to escape their countries safely, but were forced to stay in insecure places either in their own country, or in other unsafe regions also affected by war. The conditions for these people might also be affected by malnourishment and poverty, which make them dependent on humanitarian aid. According to Maslow's hierarchy of needs, treatment for psychological problems can not be conducted as long as the basic needs of nutrition and safety are not fulfilled (Neuner, Schauer, Klaschik et al., 2004; M. Schauer et al., 2005). However, Neuner and colleagues' (2004) have experienced that survivors see their mental health as being of their highest priority, and that mental functioning is the prerequisite for self-efficacy and meeting one's basic needs. They have also experienced that NET has been successful even for people who continue to live in unsafe conditions (Bichescu et al., 2007; Neuner, Schauer, Klaschik et al., 2004).

In settlements like refugee camps, the monetary resources are limited and there exist large groups of people who need treatment. Accordingly, any psychotherapeutic intervention in such settlements must be brief. It is important that the treatment programs are pragmatic and easy for local personnel to learn, even without much access to medical or psychological education or additional training. As a consequence, it is also important that the method is adaptable to multiple cultural environments and can be implemented easily. Narrative

approaches such as NET seem ideally suited for cross-cultural applications as the oral tradition is a common element among many cultures (Onyut et al., 2005). Support exists in many populations for the use of written disclosure to express thoughts and emotions about a traumatic event (Burke & Bradley, 2006). Literature on neurobiology suggests that developing a coherent trauma narrative can help to organise split-off sensory memories into narrative linguistic memories, thus reducing the intrusive and involuntary memories that characterise PTSD (Kaminer, 2006).

The inclusion of these two therapeutic techniques, Testimony Therapy and Exposure Therapy, makes NET serve two different purposes; as a method for facilitating the emotional processing of the patient's traumatic event, and as a document which can be used directly for political purposes (Onyut et al., 2005). This integral approach is what makes NET different from most cognitive-behavioural techniques, as it looks at the whole life history of the patient with the purpose of revealing all the positive and negative emotional moments (Bichescu et al., 2007). The detailed description of personal experience in NET facilitates imaginary exposure to the trauma through the emotional memories that are put into words and contrasted with the emotional experience elicited during the recall (Neuner, Schauer, Klaschik et al., 2004; M. Schauer et al., 2005). Through reliving one's own life by seeing, hearing and smelling scenes from the past, the patient becomes able to understand the meaning and the development of their life. The patient also becomes able to recognize coherences, life-patterns, coincidences and to understand who he/she was when experiencing the different traumas. In this way, the emotional structure, or network, can get assigned to a specific time and place in the life-story (Bichescu et al., 2007) and make a consistent narrative through the reconstruction of autobiographical (contextual) memory. The sensory-perceptual-emotional representations of the traumatic event have also been called *hot memory* or *situationally accessible memory*, whereas the autobiographical memory has been called *cold memory* or

verbally accessible memory (M. Schauer et al., 2005). This procedure is in line with the emotional engagement hypothesis of traumatic memories as mentioned above (Breuer & Freud, 1957; Foa & Kozak, 1986) and the principles of habituation and prolonged exposure (Foa & Rothbaum, 1998). The main procedure of emotional exposure within NET consists of two processes that must be present at the same time; the fear structure (the hot memory) must be activated, and the elements of the fear structure must be put into words and inserted in the narration about the traumatic event. As the emotional intensity about the trauma is reduced after exposure to the hot memory, habituation has taken place, and the hot memory is transformed into cool memory. The length of the exposure must be long enough for the trauma to loose its emotional intensity. This length may vary between patients and kinds of traumas. It is also stressed that the arousal peak, or the highest level of emotional intensity around telling the traumatic event ("the hottest spot") is reached during the exposure, and that a notable reduction in fear and excitement must have been present before a session should be ended (M. Schauer et al., 2005). Full habituation can not be expected after one single session, but some fear reduction will occur. When the habituation process takes place the patient's high level of arousal as initially experienced when exposed to the hot memory, will be lower after every session (see Figure 3 in Appendix B). It is assumed that the habituation process will take place also between the sessions, as the patient most likely will continue to think about the event in a different manner than before treatment started. It is assumed that this process leads to a cognitive restructuring of the traumatic memory (see Figure 5 in Appendix B). In addition, the detailed narration created during NET, often leads to a more in-depth understanding of the patient's behaviour during the event. This may help to modify feelings of guilt and shame as the patient gets to realise that he/she did not have any other choice at that time. The goal is to achieve the maximum level of habituation possible by the final session of NET (M. Schauer et al., 2005) (see Figure 6, in Appendix B).

Cognitive restructuring as a treatment method was first developed by Aaron Beck treating depression (Beck, Rush, Shaw, & Emery, 1979). In the context of PTSD, cognitive restructuring is based on the assumption that a patient will experience symptom reduction if he/she gets to identify and modify his/her catastrophic and unrealistic interpretations of the traumatic experience and well-being of the future. This symptom reduction is thought to occur because the cognitive schema resulting from the identification and modification of the misinterpreted traumatic events will not result in psychopathological states (Bryant et al., 2008; Bryant, Moulds, Guthrie, Dang, & Nixon, 2003).

The numbers of sessions necessary in NET depend on the setting of therapy, and on the severity of the patient's PTSD. Neuner and colleagues (2004) found that the minimum number of sessions needed in an African refugee settlement was 4. For the treatment of torture victims, more sessions may be necessary. In general, 8-12 sessions are proposed, but the number of sessions should be determined in consistency with the patient before treatment starts. This to prevent a possible increase in avoidance as a result of an undefined length of treatment (M. Schauer et al., 2005) (for a general guideline of organization of sessions, see Table 2 in Appendix A. For a detailed description of the sessions, see p. 33).

Ethical Considerations

This study is approved by the Committee for Medical Research Ethics (Regional Etisk Komité) (see Appendix C). However, there are some particular ethical concerns that need consideration. This is a treatment study and the participants were all severely traumatised as they fulfilled the PTSD criteria in DSM-IV. NET is quite a new and unexplored treatment method, and has yet limited clinical evidence for its effectiveness in treatment of Western, non-refugee adult subjects. There is a high level of discomfort experienced by traumatised patients while talking about the traumatic event in detail. In our study, the participants had to do that repeatedly during the treatment, but not during the assessments. The therapist always

made sure that the patient felt safe. Trauma exposure is essential for reducing the symptoms. To ensure that the patient understands the rationale of the procedure and the discomfort, there is an extensive component of psychoeducation built in to NET (M. Schauer et al., 2005). All the participants gave informed consent to participate, but one should always consider the aspect of autonomy of this (Cozby, 2007). Patients with a high symptom load might be more uncritical towards an enquiry. They might also be desperate to receive treatment and therefore give their informed consent without the necessary understanding of the rationale. The exclusion criteria in our study contribute to ensure that the patients are well suited for the project.

In this study there exist 8 fixed treatment sessions, each session lasting 90 minutes. As mentioned, the number of sessions required depends on the setting and severity of the patient's PTSD symptoms. This may have the implication that some of the patients in this study would have been given more sessions if they were considered individually.

Overall Research Question and Hypothesis

The present study was conducted on one group of patients in outpatient clinics. All patients fulfilled the criteria for PTSD. The overall aim was to investigate if NET would contribute to a significant relief in various symptoms assessed through the patients' reports and questionnaires administered immediately prior to treatment, and 1 month after completed treatment. The various symptoms of interest included symptoms of PTSD, general psychopathology, and level of depression. Our null hypothesis was that NET will not have any effect. The following hypotheses were addressed:

1. There is a significant symptom relief in PTSD symptoms on all subscales in CAPS measured one month after the final treatment session of NET. This includes a reduction of the frequency and intensity of re-experience symptoms (criterion B), avoidance and numbness

symptoms (criterion C) and hyperarousal symptoms (criterion D) respectively. In addition, there will be a significant reduction in total frequency and intensity of criterion B, C and D.

2. A significant number of patients will not fulfil the PTSD diagnosis one month after NET.

3. For PTSD symptoms, there will be significantly stronger positive correlations between the different subscales of CAPS one month after treatment. Then all the subscales show lower scores than prior to treatment which strengthens the evidence of NET being an effective method in treating PTSD.

4. There will be a significant reduction in general psychopathology one month after NET measured by subscales on SCL-90-R. Here scores on the subscales somatisation, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, psychoticism, other symptoms and global severity index will be lower than prior to treatment.

5. Overall, there will be a significant reduction of depressive symptoms one month after completed NET, measured by the total score of BDI-II.

Methods and Materials

Subjects

Subjects included in this study were 10 adults, 6 females and 4 males (> 18 years) living in Norway. All the subjects met the criteria for a PTSD diagnosis. They were within the age range of 19-55 (mean=37,2). All subjects received NET-treatment. 2 subjects were refugees and 8 subjects were ethnic Norwegians (see Table 3 in Appendix A). All potential participants were recruited by clinicians working in outpatient clinics in the western part of Norway through their usual intake referrals. The included patients in this study are recruited and treated at the following outpatient clinics: 1 at VOP Åsane, 3 at VOP Bergenhus, 2 at VOP Fjell, 2 at Center for Crisis Psychology, 1 at RVTS-West and 1 at a private clinic.

Initially, 19 subjects were evaluated for participation, but 9 were excluded for different reasons. 2 were excluded after started treatment; 1 because of pregnancy and 1 because of suicidality and extensive psychosocial problems. 1 was excluded because of drug abuse. 4 patients choose to withdraw from the study after the pre-treatment assessment, and 2 patients were excluded because they did not meet the PTSD criteria.

Exclusion criteria were severe mental illness including; schizophrenia, severe dissociation, ongoing substance abuse, risk of suicidal behaviour, and self mutilation. In addition, diseases in the central nervous system, head injury, loss of consciousness for more than 30 minutes, pregnancy, or endocrinological diseases, the latter was due to analysis of cortisol levels (see Table 4 in Appendix A). All participants had to speak fluent Norwegian as there were no translators engaged in the project. All assessment instruments were in Norwegian. The types and duration of traumas that the subjects had been exposed to varied. For example are war related traumas often more prolonged than traumas from a car accident in an otherwise safe environment (see Table 5 in Appendix A). None of the subjects received any compensation for their participation in the project.

Clinicians Performing Treatment

According to the main protocol, a total number of 20 clinicians were willing to receive training in NET and to treat two PTSD patients each. One patient is set on a waiting-list, and one patient in a treatment group. As mentioned, the study presented in this thesis includes 10 patients from the treatment group only. RVTS-West has been responsible for the training of the therapists in close collaboration with representatives from the VIVO group at the University of Konstanz, Germany. VIVO is an alliance of professionals experienced in research and service provision in the fields of psychotraumatology, public health, human rights advocacy, humanitarian aid, behavioural neuroscience and sustainable development (Onyut et al., 2005; M. Schauer et al., 2005). The initial training consisted of a three day course (24 hours) free of charge in which several sessions of role play was performed under close supervision by experienced NET therapists in addition to theoretical lessons. Supervision by telephone was offered regarding planning and completion of the therapy.

The therapists performing therapy in the present study were 9 females and 1 male, and each has treated 1 patient. None of the therapists have received any economic compensation for participating in the project.

Clinical Measures

Clinician-Administered PTSD Scale for DSM-IV

The Clinician-Administered PTSD Scale (CAPS) for DSM-IV is a structured interview for Posttraumatic Stress Disorder (Blake et al., 1998) developed by the National Centre of PTSD in 1989. The Psychosocial Refugee Team at the Resource centre on violence, trauma, and suicide prevention, Mid-Norway (RVTS-Mid), is responsible for the Norwegian translation (2005). The interview is widely used both for clinical and research purposes.

It consists of 30 items where 17 items assess core PTSD symptoms that are related to re-experiencing, avoidance, numbing and hyperarousal. Each item asks both for the frequency

of a specific symptom, and the intensity. 5 items assess symptoms known to be associated with PTSD such as dissociation and trauma-related guilt. 5 items assess variables such as onset of trauma, subjective distress and functional impairment. 3 items assess an overall response validity, symptom severity and symptom improvement (Blake et al., 1998; Weathers et al., 2004).

CAPS also includes a life event checklist with 17 items assessing self-reported potentially traumatic events. This checklist was administered prior to the interview – procedure. In general, if a patient has experienced multiple events the interviewer must investigate symptoms regarding two or three events, and in mutual agreement with the patient, decide which one of these events are considered to be the most severe causing the majority of symptoms at the present time. Hence, his or her experience serves as the reference when answering further questions.

CAPS' psychometric values are known to be solid, both with regard to validity and reliability. Six different studies examined the internal consistency of the instrument. The internal consistency ranged from .25 - .95. The interrate reliability ranged from 1.0 - .81 across nine studies. Regarding content validity, CAPS is directly based on the DSM-IV criteria's for PTSD. Several factor analysis studies have confirmed the construct validity of the instrument. One study that investigated the criterion validity of CAPS compared the results with the PTSD module of SCID (Structured Clinical Interview for DSM disorders) revealing a 83 % sensitivity, 94% specificity, 93% efficiency, and a kappa value of .74 for current PTSD diagnosis (Weathers et al., 2004).

The scoring rule applied in this study is the original F1 / I2 rule which defines a symptom as present if the Frequency rating is >1 and the Intensity rating is >2 thus being more sensitive to the presence of PTSD and therefore reduces the risk of false negatives

(Weathers, 2004). Here, a 15 point change in the Total Severity Score indicates that a clinically significant change has occurred in a single case (Weathers et al., 2004).

Symptom Checklist -90-Revised

The Symptom Checklist -90-Revised (SCL-90-R) was developed by Leonard R. Derogatis (1975, 1994) and is a self-report inventory that helps evaluate a broad range of psychological problems and symptoms of psychopathology. It is also useful in measuring patient progress or treatment outcomes. It consists of 90 items to assess psychological symptoms and distress of the respondent in the 7 previous days. Each item is rated on a five point Likert scale (0-4), ranging from "not at all" to "extremely". These items are divided into 9 primary symptom dimensions; Somatisation (SOM), Obsessive Compulsive (OBS), Interpersonal Sensitivity (INT), Depression (DEP), Anxiety (ANX), Hostility (HOS), Phobic Anxiety (PHOB), Paranoid Ideation (PAR), and Psychoticism (PSY). In addition, there are 3 global indices; the Global Severity Index (GSI), the Positive Symptom Distress Index (PSDI), and the Positive Symptom Total (PST). In this study, the analysis includes all of these scales except PSDI and PST, but includes an additional "Other symptoms" scale.

The psychometric properties of the SCL-90-R are known to be good. The internal consistency reliability between the different subscales is 0.70-0.90 where a test-retest reliability after one week is shown to be 0.80-0.90. The validity of the SCL-90-R is also known to be good with regard to convergent-discriminative, predictive and construct validity (Derogatis, 2000). The Norwegian translation used in this study is performed by Vassend & Nielsen (1991).

Beck Depression Inventory-II

The Beck Depression Inventory-II (BDI-II) is a self-report inventory that is widely used to measure depressive symptoms among adolescents' and adults, and also in monitoring symptom changes during and after treatment (Beck, Steer, & Brown, 1996). BDI-II consists

of 21 items and uses a four point Likert scale ranging from 0-3. One of the purposes of the inventory is to reflect the severity of depression. The total score range from 0- 63. 0-13 equals minimal depression, 14-19 equals mild depression, 20-29 equals moderate depression and 29-63 equals severe depression. The items evaluate mood, pessimism, sense of failure, self-dissatisfaction, guilt, punishment, self dislike, self-accusations, suicidal ideas, crying, irritability, social withdrawal, indecisiveness, body image, work difficulty, insomnia, fatigability, loss of appetite, weight loss, somatic preoccupation and loss of libido (Lambert & Stephenson, 2000). BDI-II can be used both for clinical and research purposes, for detecting depressive symptoms, suicidal thoughts and to assess improvement after treatment. BDI-II was translated to Norwegian by Harcourt Assessment (2005).

A review of 25 years of research where BDI was applied found a high internal consistency across 25 studies (Beck, Steer, & Garbin, 1988). The test-retest reliability of BDI-I shows good reliability over short time intervals. Retesting after 1-6 hours gives a reliability coefficient on r=0.83 and after 4-6 hours r=0.81 in a student population (Yonkers & Samson, 2000). Validity data gives correlations from 0.55-0.96 (mean=0,72) between clinical evaluations and BDI for submitted patients (Yonkers & Samson, 2000). The reliability based on test-retest administration among psychiatric patients has been reported to range between a Pearson Product-moment correlation of .48-.86. For non-psychiatric patients the test-retest coefficients ranged from .60. Studies of discriminant validity have indicated that the test is able to discriminate between psychiatric patients and healthy individuals (Lambert & Stephenson, 2000).

Procedure

All outpatient clinics in the “Health-region West” (Hordaland, Rogaland and Sogn og Fjordane) were contacted by RVTS-West by mail addressed to the head of the clinic. They

were given written and oral information about the project. The head of each clinic distributed the information to their clinicians. The project was carried out in three different phases:

In phase one, each clinician who decided to take part in the project evaluated the referrals at the respective outpatient clinic with respect to any trauma history, and decided whether the subject could fulfil the inclusion criteria for further evaluation. The patients were contacted by written letter and given an appointment with the clinician. In a face-to-face session, they were verbally informed about the project. If verbal consent to participate were given, written information was distributed and written, informed consent was collected.

In phase two, an experienced psychologist from the project (from RVTS-West) contacted each participant by mail or phone for appointments in which the psychiatric screening was performed to either confirm or reject a PTSD diagnosis. All patients were assessed with the clinical interviews Clinical Administered PTSD scale for DSM-IV (CAPS) and MINI International Neuropsychiatric Interview Version 5.0.0 (MINI), and the self-reporting questionnaires Symptom Checklist 90 Revised (SCL-90-R), Beck Depression Inventory-II (BDI-II), Utrecht coping list, Impact of Event Scale Revised (IES-R) and Dissociation Experience Scale (DES) pre-treatment. All patients were assessed with the same assessment instruments post-treatment, except MINI. CAPS was performed by a trained clinician at RVTS-West. The patients filled out the self-reported questionnaires at home and returned them to RVTS-West by mail. When all the subjects had gone through this first clinical screening procedure, and if they fulfilled the criteria for participation in the project, they were randomized into either the treatment group or the waiting-list group. The patients who did not fulfil the criteria for participation, were offered another kind of treatment at the outpatient clinic where they had been recruited.

In phase three, the treatment for the treatment-group began immediately after they had received information about the project and given their consent. It consisted of one or two 90

minutes' sessions of NET a week, for a total of 8 sessions. One month after the treatment was completed the patients came back to be assessed again with all assessment instruments except MINI. The patients will also come back for a 6 month follow-up assessment, but that time has still not come as the main project is still running (see Table 6 in Appendix A).

10 subjects in the treatment group have gone through pre-treatment assessment, NET-treatment, and post-treatment assessment. In this study, it will be focused upon these participants' measures on CAPS, SCL-90-R and BDI-II in the pre-treatment assessment and in the 1 month post-treatment assessment (see Table 7 in Appendix A).

The Procedure of NET

The therapists in this study have followed the procedure of NET as described in the manual (M. Schauer et al., 2005). It will therefore be presented here in general terms.

In the first session, the therapist starts with introducing him or herself (name, profession), explains his or her interests (the purpose of the project/mission) and explains the ethical stance. After the PTSD diagnosis is established the most advisable is to immediately start psychoeducation. The psychoeducation must be done thoroughly and include the following elements; normalization (reactions after a trauma is understandable and normal), legitimization (the "symptoms" experienced today are the result of responses from the traumatic situation), description of trauma reactions (including related symptoms) and explanation of the therapeutic procedure (imaginative exposure, habituation, narration and a step-by-step explanation of the therapeutic process) (M. Schauer et al., 2005).

In the second session, the narration can usually begin. To assess both the traumatic and positive events in the patient's life, "The Life-Line Exercise" can be used. Through the use of a rope (the beginning of the rope illustrating birth, the rest of the rope illustrating the future), flowers (positive events) and stones (negative events) put chronologically by the patient, both the therapist and the patient get a visual impression of the patient's life that can be used as a

guide for subsequent sessions. The life-line is pictured and written down for this purpose, and can be compared to how the patient puts his/her lifeline at the end of therapy (M. Schauer et al., 2005) (for illustration, see Figure 7 in Appendix B). Sometimes the life-line exercise is done during the first session. Through the second session, the narration from birth and through the first traumatic event is done. The therapist writes a first draft of the patient's life narration up to the point at which the narration stopped after every session.

During the third and subsequent sessions, the narrative collected in previous sessions is re-read, and the narration of subsequent life and traumatic events are continued.

In the final and eighth session, the report is reread in its entirety and the final corrections are made. Then the patient and the therapist sign the written testimony, one copy is kept by the therapist, and the original is handed to the patient (M. Schauer et al., 2005).

Data Analysis and Statistics

The data were analysed with the statistical software package Statistica version 8.0. Descriptive statistics were applied for the variables education and age (n=10), a parametric one-way ANOVA were used for assessing possible gender differences in education. All data are expressed as mean and standard deviation (SD).

Within-group effect-sizes (pooled SD) were calculated using Cohen's *d* – formula. In behavioral sciences, an effect-size of $d = 0.2$, is considered small; $d = 0.5$ is considered moderate, and $d = 0.8$" is considered large (Cohen, 1988).

Pearson Chi-Square with Fisher's Exact Test was used to explore if the number of patients with a PTSD-diagnosis was significantly reduced from pre- to post-treatment assessment.

All 10 subjects in the sample completed the PTSD screening instrument CAPS pre- and post-treatment, but 1 had to be excluded from the dataset on the questionnaire BDI-II (n=9) and 2 from the dataset on the questionnaire SCL-90-R (n=8) due to missing data.

Due to the low number of participants, further analysis does not consider a gender aspect since a minimum of n=5 are required for a variance analysis (e.g. non-parametric analysis of variance) to be statistically valid. Additionally, two replicated measurements for each participant as shown here, requires within-subject differences (paired data).

With respect to the hypothesis where changes in symptoms are reported to be reduced after NET treatment, a t-test for dependent samples was applied since all patients are treated as one group within a pre-post test design.

The results are considered significant at P level <.05.

Correlation analyses were performed for the pre-and post-assessment of the different subscales on CAPS. This was done by using a Spearman rank analysis for all the CAPS variables for pre-and post-assessment respectively.

Results

Descriptive Statistics

The sample comprised 4 males and 6 females. The overall mean age was 37,2 (SD=12,6), and the were no gender differences in either age or education (see Table 8). No further analyses were performed with regard to gender differences due to the small sample size.

Table 8: Sample characteristics

	Males	Females	Total
n	4	6	10
Age, mean (SD)	34.5 (12.1)	39 (13.8)	37.2 (12.8)
Education, mean (SD)	15.8 (3.1)	12.8 (3.7)	14.0 (3.7)

*<.05; **<.01 (significance level refer to gender differences)

Treatment effects

CAPS

There was a significant treatment effect as measured by the CAPS on all subscales when comparing pre-treatment scores with one month post-treatment scores, except for the frequency and intensity of hyperarousal symptoms (CAPS-D-FI) (see Table 9). All effects-sizes ranged from moderate (0.59) to large (0.83).

Table 9: Treatment effect measures by the CAPS subscales (n=10).

	Pre-treatment Mean (SD)	Post-treatment Mean (SD)	t	*P*-level	ES*
CAPS-B-FI	21.90 (7.52)	14.50 (10.20)	3.70	0.004**	0.83
CAPS-C-FI	26.40 (9.78)	17.30 (15.11)	2.67	0.03*	0.72
CAPS-D-FI	22.90 (5.40)	18.60 (8.82)	2.16	0.06	0.59
CAPS-BCD-F	37.10 (9.53)	26.80 (18.16)	2.84	0.02*	0.71
CAPS-BCD-I	34.10 (9.63)	23.60 (14.97)	3.46	0.007**	0.83
CAPS-BCD-FI	71.20 (18.55)	50.40 (32.98)	3.23	0.01*	0.78

* ES = Cohen's *d* effect size (pooled SD)

There was a significant reduction in the CAPS subscale for measuring frequency and intensity of re-experience symptoms (CAPS-B-FI) (P=.004) (see Figure 8).

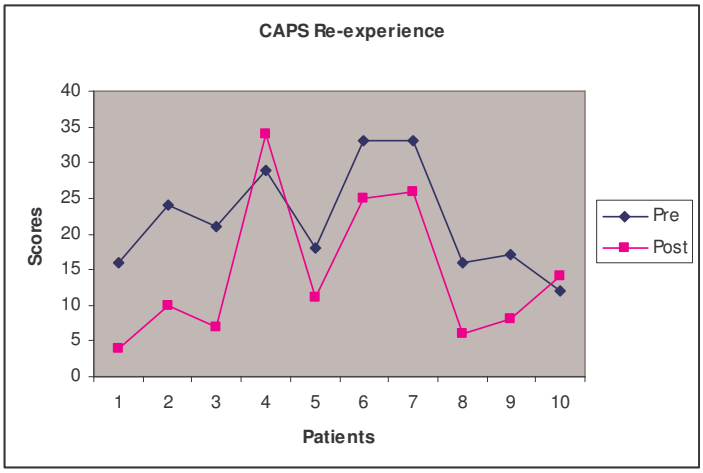


Figure 8: The figure illustrates the level of re-experiencing symptoms in the sample as measured by the CAPS subscale for re-experience in each of the patients in the sample both in the pre-test and post-test assessment.

There was a significant reduction in CAPS subscale for measuring frequency and intensity of avoidance and numbness symptoms (CAPS-C-FI) (P=.02). The CAPS subscale for measuring frequency and intensity of hyperarousal symptoms (CAPS-D-FI) was only borderline significant (P=.058), although the effect-size was moderate. There was a significant symptom reduction in CAPS subtotal for the frequency of criteria B+C+D (CAPS-BCD-F) (P=.02) and in CAPS subtotal for the intensity of criteria B+C+D (CAPS-BCD-I) (P=.007) (see Figure 9).

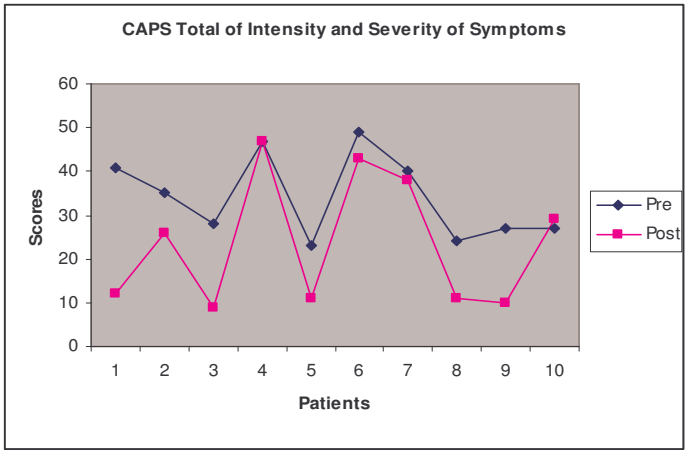


Figure 9: The figure illustrates the intensity and severity of re-experiencing, avoidance/numbness and hyperarousal/activation symptoms in the sample as measured by the CAPS subscale for frequency of these symptoms in each of the patients in the sample both in the pre-test and post-test assessment.

The total severity score on CAPS (CAPS-BCD-FI) was significantly reduced (P=.01).

There was a significant reduction in number of patients fulfilling the DSM-IV-R criteria for a PTSD-diagnosis, from 10 patients before treatment to 5 patients (4 females and 1 male) at treatment completion (χ^2=6.67, df=1, P=.033).

Correlation analysis of the association between the different subscales of CAPS, revealed that there in general was a stronger correlation between the subscales in the post-assessment compared to the pre-assessment (see Table 10 and 11). These correlations were in accordance with the reduction in the mean scores on the subscales of CAPS (see Table 9).

TABLE 10: Correlations between subscales of CAPS pre-treatment.

	CAPS-B-FI	**CAPS-C-FI**	**CAPS-D-FI**	**CAPS-BCD-F**	**CAPS-BCD-I**	**CAPS-BCD-FI**
CAPS-B-FI	1,00	0,56	0,16	0,75*	0,62	0,71*
CAPS-C-FI	0,56	1,00	0,28	0,84**	0,92***	0,87**
CAPS-D-FI	0,16	0,28	1,00	0,49	0,48	0,54
CAPS-BCD-F	0,75*	0,84**	0,49	1,00	0,79**	0,96***
CAPS-BCD-I	0,62	0,92***	0,48	0,79**	1,00	0,86**
CAPS-BCD-FI	0,71*	0,87**	0,54	0,96***	0,86**	1,00

*p<.05; **p<.01; ***p<.001

TABLE 11. Correlations between subscales of CAPS post-treatment.

	CAPS-B-FI	**CAPS-C-FI**	**CAPS-D-FI**	**CAPS-BCD-F**	**CAPS-BCD-I**	**CAPS-BCD-FI**
CAPS-B-FI	1,00	0,79**	0,71*	0,95***	0,78**	0,91***
CAPS-C-FI	0,79**	1,00	0,84**	0,86**	0,93***	0,90***
CAPS-D-FI	0,71*	0,84**	1,00	0,86**	0,94***	0,89***
CAPS-BCD-F	0,95***	0,86**	0,86**	1,00	0,90***	0,98***
CAPS-BCD-I	0,78**	0,93***	0,94***	0,90***	1,00	0,95***
CAPS-BCD-FI	0,91***	0,90***	0,89***	0,98***	0,95***	1,00

*p<.05; **p<.01; ***p<.001

SCL-90-R

One of the subscales yields significant results, the subscale measuring somatisation. Here, the patients scoring was significantly lower 1 month after treatment than prior to treatment, P=.02. However, as measured by the Cohen's d effect-sizes, the majority of the subscales had moderate to large reductions. (See Table 12.)

Table 12. Treatment effect measures by the SCL-90-R (n=8).

	Pre-treatment Mean (SD)	Post-treatment Mean (SD)	t	*P*-level	ES*
Somatisation	26.25 (9.83)	18.25 (12.63)	2.77	0.02	0.71
Obsessive-Compulsive	20.75 (8.36)	15.63 (9.65)	2.13	0.07	0.57
Interpersonal Sensitivity	19.00 (6.97)	12.88 (8.98)	1.67	0.14	0.76
Depression	25.88 (19.96)	18.13(11.38)	1.86	0.10	0.48
Anxiety	20.88 (8.00)	15.25 (12.04)	2.09	0.07	0.55
Hostility	6.88 (3.14)	4.50 (3.78)	1.60	0.15	0.68
Phobic Anxiety	13.00 (4.87)	10.50 (8.47)	1.53	0.17	0.36
Paranoid Ideation	10.00 (5.45)	5.88 (3.64)	1.72	0.13	0.89
Psychoticism	8.25 (2.82)	4.88 (4.88)	2.17	0.07	0.85
Other Symptoms	14.25 (5.04)	11.38 (8.14)	1.24	0.26	0.42
Global Severity Index	165.13 (45.92)	117.25 (77.65)	2.22	0.06	0.75

* ES = Cohen's *d* effect size (pooled SD)

BDI-II

The reduction in depressive symptoms from pre-treatment (25.6 [SD=10.4])) to post-treatment (20.1 [SD=13.6])) as measured by the BDI-II, reached close to statistical significance (t=2.22, P=.056), with an estimated Cohen's d effect size of 0.45. 2 patients improved over two severity degree cut-offs, as they improved from moderate to minimal

depression. 3 patients improved over one cut-off; 1 from severe to moderate depression, 1 from moderate to mild, and 1 from mild to minimal depression (see Figure 10).

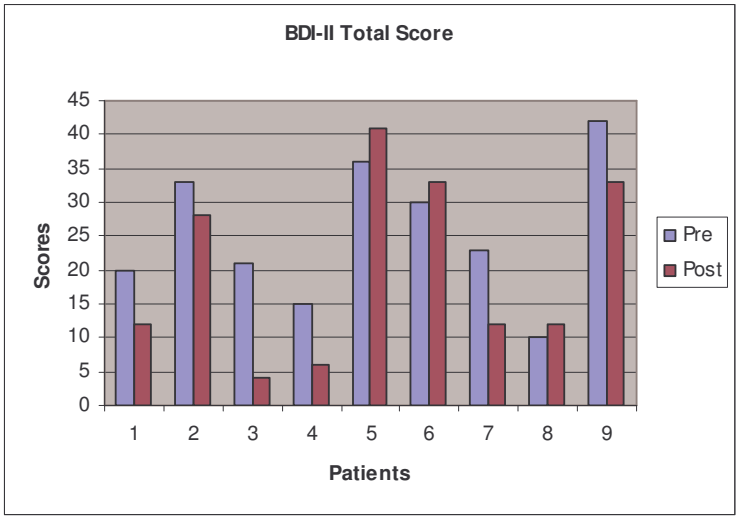


Figure 10: Level of depressive symptoms as measured by the BDI-II total score in each of the patients at pre- and post treatment (n=9).

Discussion

Summary of Main Findings

We have compared pre- and post- treatment scores on CAPS, SCL-90-R and BDI-II of one group of PTSD patients treated with Narrative Exposure Therapy. The overall aim was to investigate whether Narrative Exposure Therapy is an effective treatment for PTSD assessed by the patients' reports on these clinical measures. The main findings showed a significant reduction in PTSD symptoms on 5 out of 6 subscales on CAPS before and after treatment. All the subscales had effect sizes that ranged from moderate to large. 50% of the sample (1 male and 4 females) no longer fulfilled the DSM-IV criteria for PTSD one month after treatment. There were also strong correlations between all the subscales on CAPS in the post-treatment assessment compared to the pre-treatment assessment. There was a significant reduction in symptoms on the somatisation subscale of SCL-90-R in the post-treatment assessment compared to the pre-treatment assessment. None of the remaining subscales including the global severity index were significant, although the latter was close to significance. Effect sizes on a majority of the subscales showed moderate to large reductions. There was a close to significant reduction in depression symptoms after treatment; overall, the patients were still in the clinical range of moderate depression. However, the effect size almost reached moderate level.

Symptom Relief as Measured by CAPS

There was a significant reduction in five of six subscales on CAPS and the effect sizes ranged from moderate to strong on all subscales. The symptom reduction measured by CAPS' total severity score was significant. This indicates a general relief of PTSD symptoms in our sample. In the following the subscales measuring re-experience symptoms, avoidance and numbing symptoms, and hyperarousal symptoms will be discussed more specifically.

The re-experiencing symptoms (criterion B; intrusive thoughts, nightmares, flashbacks, cued distress, and cued physiological arousal) is the subscale of CAPS that showed the most significant reduction. This might be interpreted as an indication that NET is effective in treating these kinds of symptoms. It may also mean that there has been a successful exposure during therapy, and as a consequence, an integration of trauma related memory material into the autobiographical memory has taken place (the hot memory has been transmuted into cool memory). Our finding is in contrast to Bichescu and colleagues (2007) findings of no significant reduction of re-experience symptoms after NET treatment. Possible explanations for this might be differences related to the research design, such as different time intervals for follow-up and different samples.

The avoidance and numbing symptoms (criterion C; avoidance of thoughts or feelings related to the trauma, avoidance of reminders of the trauma, amnesia for some part of the trauma, loss of interest in usual activities, feelings of detachment or estrangement from others, restricted range of affect and sense of foreshortened future) also showed a significant reduction. As mentioned in the introduction, exposure is known to cause habituation or extinction, which in turn can reduce anxiety associated with the trauma. Habituation or extinction can lead to reduced avoidance of the situations, thoughts or feelings that used to evoke the anxiety. Our finding indicates that this is the case.

The only subscale that did not show significant reduction is the subscale measuring hyperarousal symptoms (criterion D; difficulty sleeping, irritability or anger, difficulty concentrating, hypervigilance and exaggerated startle response). Although not significant this subscale had a moderate effect size. The lack of significant results on this particular subscale might be due to the small sample. The moderate effect size supports this explanation. The cognitive restructuring that is assumed to be in progress *between* sessions might not be sufficient to reduce these particular symptoms. This might suggest a need for more explicit

focus on management of hyperarousal symptoms through e.g. relaxation exercises and cognitive strategies, as in different cognitive behavioural therapies. Such strategies and techniques could be used by the patient in the situation were the hyperarousal- symptoms occur. Several studies that have compared the effectiveness of exposure therapy and cognitive restructuring, have found that the combination of the two is superior to either one administered alone (Bryant et al., 2008; Bryant et al., 2003). The suggested mechanism for this superior effect has been that cognitive restructuring adds a mastery dimension to the patients' behavioural repertoire. Following this, one could speculate that insufficient cognitive restructuring through NET could be contributing to the lack of significant results on the particular subscale of hyperarousal in our study. In contrast to such an explanation, Bichescu and colleagues (2007) found a significant reduction in the subscale of hyperarousal among former political detainees in a 6 months follow-up after completed NET-treatment. Although several aspects differentiates our study from Bichescu and colleagues' (2007) it is interesting to notice variations concerning which PTSD symptoms that were reduced after applying NET. These differences may be explained by several factors, such as characteristics of the sample, or the point of time for the follow-up assessment. It is also possible that processes as habituation and cognitive restructuring takes time, and that a significant symptom reduction will be found in the 6 month follow-up assessment scheduled in the main project.

Our results confirmed the hypothesis that a significant number of patients would not fulfil the DSM-IV criteria for a PTSD diagnosis one month after completing NET-treatment. This is in line with other treatment studies using NET (Bichescu et al., 2007; Bradley et al., 2005; Neuner, Schauer, Klaschik et al., 2004). 50 % of our sample (5 out of 10) did not meet the criteria for PTSD in the post-treatment assessment.

A meta analysis by Bradley and colleagues (2005), investigating the effectiveness of PTSD-treatment across various treatment methods, showed that, 67 % of the subjects that

completed any of the given treatments for PTSD did not meet the criteria for PTSD post-treatment. This number is somewhat higher than the results reported in our study. However our sample is not comparable with the sample of studies in the meta-analysis, due to sample size, lack of waiting-list/control group, and use of a different treatment method.

One could argue that the DSM-IV criteria for fulfilling a PTSD diagnosis are somewhat rigid, in the sense that the patient has to present with PTSD symptoms above threshold on all the criteria dimensions (experience of trauma, avoidance/numbing, hyperarousal and re-experience) in order to fulfil the PTSD diagnosis. This may cause patients with high symptom load on some of the criteria dimensions, but no symptom load on one of them, to be treated as "recovered" from PTSD in the evaluation of treatment methods. Bradley and colleagues (2005) point to this problem and argue that the way one measures improvement from other psychiatric disorders differs from the way one measures improvement from PTSD. With other psychiatric disorders it is more common to measure improvement than recovery. The ICD-10 criteria for PTSD are less extensive and more flexible, and one would assume that more of the patients with PTSD would still fulfil the PTSD diagnosis post-treatment if these criteria were applied. This is an important point to be aware of in the evaluation of effectiveness of treatment methods for PTSD, also when assessing the results in the present study.

Possible effects caused by same-sex or different-sex relations between patients and therapists were not controlled for in this study. Here, all male patients had a female therapist and one female had a male therapist (see Table 13 in Appendix A). Traumas, especially those involving sexual assaults, might be more difficult to expose depending on the therapists sex, especially if the patient is from a culture or religion where exposing these kind of issues to a person of opposite sex is considered shameful and unacceptable. Such factors might interfere with full exposure to the "hot spots". The patient might find it too difficult and shameful to

describe the most traumatic event he/she has experienced, and instead choose to tell about the second worst trauma experience. This might cause insufficient exposure which in turn reduces the treatment effect. It can be assumed that substantial age differences between therapists and patients could create similar effects. One could therefore speculate that for the treatment effect to increase even further, one should be more aware of these factors when assigning patients to NET-treatment.

The results in the study confirmed our hypothesis that for PTSD symptoms, there will be significantly stronger positive correlations between the different subscales of CAPS one month after treatment. The strengthening of the correlations between the subscales post-treatment, in combination with the findings that all the subscales showed lower scores, may indicate a general symptom relief in the sample.

Psychological Symptom Status Assessed by SCL-90-R

On this questionnaire, we only found a significant reduction of symptoms on the subscale somatisation 1 month after treatment. The remaining subscales (obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, psychoticism, other symptoms and global severity index) were not significant; hence our hypothesis that all subscales should be reduced after treatment was not fulfilled. However, since the effect-sizes on the majority of the subscales showed moderate to large reductions, these results will also be discussed. The global severity index was close to significant, which may indicate that the level of psychological symptoms and intensity of perceived stress post-treatment was lower than it was pre-treatment. The GSI is known to be the best single indicator of the patient's current symptom level or depth of the disorder. It should be used when a single summary measure is required (Derogatis, 1992). However, as the reduction on GSI in our study was not significant, this can be an indication that NET is not effective in reducing the level of general psychological symptoms. Another explanation for the non-

significant results could be the small sample size (n=8) or that the follow-up was only one month after treatment. One can expect that there will be a significant reduction in the 6 month follow-up assessment (in the main project), as it is assumed that the effects of NET are strengthened over time.

The subscales on paranoid ideation and psychoticism showed the highest effect-sizes. The subscale on paranoid ideation includes characteristics as hostility, suspiciousness, fear of loss of autonomy and delusions. The subscale of psychotisism includes symptoms as hallucinations, withdrawal and isolation (Derogatis, 1992). The high effect sizes on these subscales might indicate that NET did have some effect in reducing these symptoms, even though they were not significant.

The somatisation subscale reflects the distress people experience through perceptions of bodily dysfunction which is not found to be reflections of any physical disease (Derogatis, 1992; Escalona, Achilles, Waitzkin, & Yager, 2004). These perceptions can be headaches, faintness and dizziness, nausea and upset stomach, pains in heart or chest, and/or backaches, among others. According to our study, NET seems to be an effective treatment method for reducing somatisation. Somatisation is assumed to be related to PTSD (Andreski, Chilcoat, & Breslau, 1998; Escalona et al., 2004). Many different physical symptoms have been associated with PTSD. Higher rates of cardiovascular, respiratory, musculoskelatal and neurological symptoms were found among fire-fighters suffering from PTSD, compared to fire-fighters not suffering from PTSD. Other studies have reported increased rates of cardiopulmonary symptoms, audiological symptoms, headache and back pain among people with PTSD (Andreski et al., 1998). Many of these symptoms are included in the somatisation subscale in SCL-90-R. The results from these studies indicate that high rates of somatisation symptoms will be found among people with PTSD, and it can be assumed that as the symptoms of PTSD are reduced, so will the somatisation symptoms. This is shown by our

findings, and may explain why somatisation is the only subscale that showed a significant reduction after treatment. This also supports NET as an effective treatment of PTSD as somatisation is a subscale specifically associated with PTSD. The results could also be a consequence of the fact that females are twice as likely as males to suffer from both PTSD and somatisation (Escalona et al., 2004). Our sample on SCL-90- R included more females than males (5 females, 3 males). Due to the small sample size, this could have made an impact on the results. However, the females did not seem to show more somatisation symptoms than males pre-treatment.

Level of Depression Assessed by BDI-II

The results on the post-treatment did not confirm our hypothesis that there would be a significant reduction of depressive symptoms one month after NET measured by the total score of BDI. However, there was seen a change in the total score of BDI as the reduction was close to significance and the effect size was close to moderate level.

It is well known that many patients suffering from PTSD also suffer from comorbid psychiatric disorders such as anxiety and depression (Breslau, Davis, Peterson, & Schultz, 2000; Kar & Bastia, 2006; M. Schauer et al., 2005). Both the pre- and post-treatment assessment of the total score on BDI-II revealed a moderate depression level in the sample. Although not significant, the general tendency among most of the patients in the sample was a reduction in depressive symptoms after NET treatment. 5 out of 9 patients improved across cut-offs on the BDI-II from the pre- to the post-treatment. This means they improved from either severe to moderate, moderate to mild, or mild to minimal depression. 3 patients (all females) improved across two cut-offs. 2 patients (1male and 1 female) improved across 1 cut-off. 3 out of the 9 (2 females and 1 male) reported more symptoms post-treatment than pre-treatment. 1 male improved, but not over cut-offs.

NET is known to sometimes reduce the symptoms of associated disorders like depression (M. Schauer et al., 2005). A study of former political detainees found significant improvement in depression symptoms in the NET treatment group compared to the psychoeducation group (Bichescu et al., 2007). This was thought to result from the cognitive restructuring that is assumed to take place during NET. The narration of the traumatic event might also contribute to such changes, as it may change the person's self-interpretation and sense of mastery in a positive way.

The reason why 3 of the patients in this study reported more depressive symptoms after treatment is unclear. The increase of depressive symptoms among some patients might point to a weakness in NET in addressing such symptoms sufficiently. This may be an indication of a need to combine NET with other treatment methods. It also points to the fact that patients have individual needs when it comes to treatment of PTSD and comorbid psychiatric conditions. It is important to be sensitive to this, especially if a certain treatment method increases the patients' depression symptoms while decreasing the PTSD symptoms. Another possible explanation for the lack of significant results is the small sample size in this study. The results could also be caused by individual factors or characteristics of these 3 patients' trauma experiences and current life circumstances. 2 of these patients were severely depressed also in the pre-treatment assessment, and were still severely depressed post-treatment. The 3rd patient was minimally depressed both pre- and post-treatment. It could be that the patients, who are already severely depressed before receiving NET, will not benefit from NET regarding their depression symptoms. One could also speculate that an effect of NET will appear in the 6 month follow-up assessment even on the depression symptoms.

Neuner and colleagues (2004) reports high prevalence of comorbid symptoms post-NET treatment. The high prevalence of comorbid symptoms was also the case for groups treated with psychoeducation and supportive counselling. It is noteworthy that the sample in

the this study consisted of refugees living in unsafe conditions, which may have contributed to the negative results. Supplemental therapies such as cognitive restructuring or other cognitive techniques might be needed to address depression symptoms among PTSD patients. E. Schauer (personal communication, November 4, 2008) recommends that this is done after NET treatment has been completed. Depression symptoms can be caused by the PTSD and thereby be reduced when sufficient treatment is given. If depression symptoms remain after treatment, additional treatment can be offered.

A study that compared the relative effects of exposure therapy, cognitive restructuring and a combination of the two, with relaxation as a control condition for reducing depression revealed higher effect sizes for exposure therapy than any of the other treatment conditions at 1 month follow-up (Marks et al., 1998). This might indicate that the process initiated by exposure therapy goes beyond the core PTSD symptoms.

Bryant and colleagues (2008; 2003) found that the combination of two exposure methods (imaginal exposure and in-vivo exposure) together with cognitive restructuring techniques was most efficient in reducing both PTSD and depression symptoms among PTSD patients. They offer different explanations for these findings. Either the reduction of depression symptoms was secondary to the reduction of PTSD symptoms, or it was a result of this particular combination of treatments being more suited for treating depression. Cognitive restructuring was initially developed to treat depression (Beck et al., 1979). It might therefore be a more powerful method when it comes to treating depression symptoms specifically also among PTSD patients. This supports the assumption that NET could contribute to a reduction in depression symptoms indirectly, through cognitive restructuring.

General Discussion

The results of the present study confirmed our hypothesis that NET is an effective treatment for PTSD, as we found significant symptom relief measured by CAPS.

As NET was initially developed for refugees (living in refugee settlements) suffering from PTSD (M. Schauer et al., 2005), and as there are few published studies of the method, the present study contributes to a further understanding of the treatment of PTSD and the effectiveness of NET. Previous samples (mostly refugees or people who have suffered from war-related traumas) have been studied with different research designs (case-studies, randomized-controlled studies) (Bichescu et al., 2007; Neuner, Schauer, Klaschik et al., 2004; Neuner, Schauer, Roth, & Elbert, 2002; Onyut et al., 2004; Onyut et al., 2005). Our findings support the above mentioned studies regarding the effectiveness and usefulness of NET. The sample in our study consists of mainly ethnical Norwegians (8), but also some refugees (2), all living in Norway. The results indicate that NET is also effective for people suffering from PTSD in a safe country, and that have experienced other traumas than war-related. Even if the therapists in this study were not familiar with NET, our results indicate that the method can be learned by therapists outside the Vivo-group. Onyut and colleagues (2004) found that local paramedic personnel inside refugee settlements also can administer NET successfully after receiving training, thus supporting this assumption.

Therapeutic Mechanisms in NET that can Account for the Results

As mentioned in the introduction, most trauma interventions use the principle of re-telling or reconstruction of the survivor's trauma story (Kaminer, 2006). Kaminer (2006) points out through her review that by producing a trauma narrative as part of the therapy process, six relatively distinct, but inter-connected therapeutic processes, seem to facilitate recovery from post-trauma pathology. In the following some of these will be discussed in relation to NET.

Cognitive processing has shown to be an important process for improvement from PTSD (Kaminer, 2006; Resick, Nishith, Weaver, Astin, & Feuer, 2002). Studies have shown that exposure and cognitive restructuring in combination are more effective in reducing PTSD

symptoms than each of them alone (Bryant et al., 2008; Bryant et al., 2003). These results suggest that optimal treatment outcome for PTSD may be achieved by combining cognitive restructuring with exposure therapy. This gives support to the method of NET, as it is assumed that a cognitive restructuring is going on between NET-sessions, after some habituation to the traumatic experiences has taken place (M. Schauer et al., 2005). After exposure to the trauma, the patients might get new insights about the meaning of the event for his/her life, and they often reach an understanding of how unhealthy emotions and behaviour can be related to the traumatic event. The effects gained through NET can thus be a result of an indirect influence of the narrative-, exposure- and habituation-processes on the cognitive construction of the traumatic experiences. If that occurs, one can assume that the PTSD symptoms will be reduced. This is supported by Resick and colleagues (2002) who found that cognitive processing therapy (CPT), in treatment of sexual assault victims, overall was as effective in reducing PTSD symptoms as prolonged exposure (except that CPT showed better scores on 2 of 4 guilt subscales). CPT includes the integrated components of cognitive therapy and exposure in the form of writing and reading about the traumatic event (Resick et al., 2002). Resick et al (2002) study further supports the effects of NET, as it is supposed that feelings of guilt are modified as a part of the habituation and cognitive restructuring processes going on during NET (M. Schauer et al., 2005).

As a consequence of the growing literature on the conceptualization of post-traumatic pathology to be a failure of memory, Kaminer (2006) found that the creation of linguistic representation seems to be a process that helps patients to recover from PTSD. As the developers of NET also point out, the patient's trauma memories are thought to be fragmented and non-linguistic, and retrieved in a different way than other emotionally charged memories. The developing of a coherent verbal trauma narrative that names and organises the affects, cognitions, behaviours and sensory experiences associated with the trauma, is thought to be

the central process of recovery for trauma survivors. The split-off sensory memories from the trauma are thought to be integrated and organized into narrative linguistic memories through linguistically representing them. Consequently the intrusive and involuntary memories that characterise PTSD (as flashbacks) are reduced (Foa et al., 1995; Foa & Riggs, 1993; Foa et al., 1989; Kaminer, 2006; M. Schauer et al., 2005; Van der Kolk & Van der Hart, 1989). This assumption is in line with our findings of a significant reduction in the re-experience subscale.

As earlier pointed out, habituation of anxiety is an essential process in exposure therapy. NET is likely to have therapeutic effect through this process, as habituation is thought to happen when re-reading the narrative in therapy-sessions (Foa & Rothbaum, 1998; Neuner, Schauer, Klaschik et al., 2004). This is supported by the review done by Kaminer (2006).

Through NET, the patient gets to tell his/her story to a therapist who presumably is empathic, appreciative and non-judgemental. Kaminer (2006) has pointed out the importance of the relation that arises through the course of treatment for a successful healing process and treatment outcome. This is supported by the successful effect of reducing PTSD symptoms through testimony therapy (Cienfuegos & Monelli, 1983), on which the NET principles are based (Neuner, Schauer, Klaschik et al., 2004; M. Schauer et al., 2005). The fact that the patient develops an explanatory account through the making of the narrative has also been pointed out as important for a successful treatment outcome. This helps the patients to work out a meaningful coherence in their narrative (Foa & Rothbaum, 1998; M. Schauer et al., 2005). Patients often develop explanatory models of themselves, others and/or the world to make sense of their traumatic experiences. Sometimes they fail to do so, other times the explanatory narrative is not adaptive and helpful. This is thought to be corrected through systematically working through the narrative as it is assumed that a cognitive restructuring process is taking place (Kaminer, 2006; M. Schauer et al., 2005). In NET, the narrative

includes both the positive and the traumatic events from birth to present (although the positive events are in less detail). One could speculate that this contributes to an even stronger coherence and meaning in the narratives. The life line-exercise (see p. 33) is used as a tool for visualising all these events in the person's life, and used as a guideline for the subsequent sessions, and in creating the narrative (M. Schauer et al., 2005). In addition to integrating the traumatic experiences as a part of their life history, the positive events may help the patients to realize that they also had good times, which in turn may increase hope for the future. Through the narrative it is also thought that the identification of purpose, or value in the traumatic stories, could be a way of making cognitive meaning of the trauma, and to see the trauma as a source of personal strength. This process can be seen in a positive psychological perspective, where patients suffering from PTSD may find value and purpose in their trauma experiences through using the trauma as an opportunity to re-evaluate their life in a more positive way (Kaminer, 2006).

Developing narratives thereby seem to have several healing effects. However, as Kaminer (2006) points out, the re-telling of the trauma narrative is not the only component in recovery of trauma. Psychoeducation about post-traumatic symptoms, and establishing the patient's physical safety are other critical aspects of the treatment process (Kaminer, 2006). NET has also proven effective even if the patient's life-situations are not safe (Bichescu et al., 2007; M. Schauer et al., 2005). Psychoeducation in itself may have some treatment effect, but a comparison study of NET, supportive counselling and psychoeducation for treating PTSD in a sample of Sudanees refugees, showed that NET was more effective than the two other treatment conditions 1 year after treatment (Neuner, Schauer, Klaschik et al., 2004). In the NET procedure, psychoeducation is included as an important part of the treatment.

NET as Treatment for Multiple Traumas

Several studies have shown that NET has been successful in treating individuals who have experienced multiple traumas. According to Kolassa and Elbert's (2007) "building block effect" (see p. 9), PTSD seldom develops as a result of one single traumatic event. This is supported by several other studies (Bowman & Yehuda, 2004; Brewin et al., 2000; Keane et al., 2006). The building block effect has been seen as a direct result of the development of a neural fear network, which is strengthened and extended in response to each new traumatic event. Hence, the individual increases his/her vulnerability for developing PTSD after each experienced trauma (Kolassa & Elbert, 2007). In our sample, all patients reported having experienced more than one traumatic event, in line with the assumption of a building block effect. According to this NET seems to be suited to treat people who suffer from what has been defined as complex PTSD (Herman, 1992, 1997). In line with research on the etiology of PTSD, such as the building block effect, one could argue that the concept of simple PTSD needs modification, since PTSD with only one single traumatic experience does not seem to exist. If this assumption is correct, there will be an increasing demand for treatment methods applicable for multiple traumas, such as NET (about simple and complex PTSD, see p. 8).

Drop-Out in NET

In the sample of our study there was no dropout after treatment was initiated. This has also been the case in other treatment studies using NET (Bichescu et al., 2007; Neuner, Schauer, Klaschik et al., 2004). In treatment studies using exposure therapy it is common with high drop-out rates due to avoidance (Foa et al., 1991). The fact that there was no drop-out during treatment in our study may be an indication that certain aspects of NET keep the patients motivated to finish therapy, even when it is experienced as demanding and unpleasant. Bichescu and colleagues (2007) have suggested that the limited duration of NET

in combination with the emphasis on the whole personal story of the patients, contributes to motivation to complete treatment.

Clinical versus Research Applications of this Study

Treatment methods based on manuals are well suited for research applications, as they give a standardized instruction on how to apply treatment. In this study we investigated the effectiveness of NET by the clinical measures CAPS, SCL-90-R and BDI-II in the sample as a whole, not at an individual level. To increase the clinical value of this study, one could look at each participant's scores on all three measures and then compare the results across the sample. In this study the small sample size would make it difficult to see any tendencies in any direction, as we only have 8 subjects to compare across all three clinical measures.

Methodological Considerations

One of the issues that need consideration is the lack of a control group, such as a waiting-list group. However, in the main project, the design includes such a control factor although we were not able to implement these data here.

The small sample of 10 patients has restricted our choice of statistical analyses. Due to some missing data, the sample varied on SCL-90-R and BDI-II. Clearly, a small sample is less able to give generalised implications, but may still give relevant information restricted to the patients involved, and to some extent, be comparable to other studies using exposure techniques on traumatised individuals.

The present study only has one post-treatment assessment, which makes it difficult to draw conclusions about the persistence of symptom change over time. A study that compared NET-treatment to supportive counselling and psychoeducation in a sample of Sudanese refugees found an increased effect on PTSD symptoms at a one year follow-up, compared to a 4-months follow-up (Neuner, Schauer, Klaschik et al., 2004). This underlines the importance

of several follow-up assessments in research on sustained improvement. The main project includes a 6 months follow-up assessment, from which data is not yet available.

None of the treatment sessions were videotaped. Hence, the design does not control for interreliability using independent NET trained professionals to evaluate the therapy sessions. This would have increased the interreliability of the study, but it might have caused trouble with both the recruitment to the study and the effectiveness of exposure. Successful exposure demands that patients talk about, and re-experience, highly sensitive memory material in detail. Videotaping might interfere with the exposure and thereby prevent habituation.

The exclusion criteria (see p. 27) was, among others, ongoing substance abuse and risk of suicide behaviour. The main reason for this selection was due to the therapists' novelty with NET. Patients suffering from PTSD are known to have several comorbid psychiatric conditions including the ones mentioned here. Bradley and colleagues (2005) argue that treatment studies for PTSD in general are too strict in setting exclusion criteria. This is an important factor for the generalisation of treatment outcome. Many patients requiring treatment for PTSD also have clinically relevant comorbid symptoms or problems. In the present study we are not able to draw any conclusions on the effectiveness of NET beyond the selected patient sample.

Future Directions

Future research on NET should take the following aspects into consideration: To include a control or waiting-list group in the design, increase the sample to give more statistical power to the study and increase the likelihood of significant results. A further consideration should be the time of follow-up. A 1 month follow-up, as in the present study, is a short time-interval when assessing effectiveness of PTSD treatment. Previous studies of NET have shown an increased effect over time. Future studies should include follow-ups with

more extended time-intervals, up to a year or more (Bradley et al., 2005). In order to increase the potential for generalisation of the results to patients suffering from PTSD with common comorbid psychological problems, exclusion criteria should also be carefully reconsidered.

Conclusions

This study has found, through the use of the clinical interview CAPS, and the self-reported questionnaires SCL-90-R and BDI-II, that patients who suffered from PTSD before treatment with NET, experienced symptom relief after treatment. Symptoms of PTSD and somatisation, as assessed by CAPS and SCL-90-R, were significantly reduced, and 5 of the 10 patients in the sample no longer fulfilled the PTSD-criteria 1 month after treatment ended. The reduction of depression symptoms was borderline significant. The sample in this study is small, but the results are promising with regard to the usefulness and effectiveness of NET, especially as other studies have found increased treatment effects in later follow-up assessments. The results also indicate that NET can be a promising treatment method for PTSD patients living in non-threatening and safe life-conditions in Norway.

References

American Psychiatric Association. (2000). *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)* (Fourth Edition ed.). Washington DC: American Psychiatric Association.

Andreski, P., Chilcoat, H., & Breslau, N. (1998). Post-traumatic stress disorder and somatization symptoms: A prospective study. *Psychiatry Research, 79*, 131-138.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression*. New York: Guilford Press.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *Manual for the Beck depression inventory-II*. San Antonio, TX: Psychological Corporation.

Beck, A. T., Steer, R. A., & Garbin, M. G. (1988). Psychometric properties of the Beck depression inventory: Twenty five years of research. *Clinical Psychology Review, 8*, 77-100.

Bichescu, D., Neuner, F., Schauer, M., & Elbert, T. (2007). Narrative exposure therapy of political imprisonment-related chronic posttraumatic stress disorder and depression. *Behaviour Research and Therapy*

Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Charney, D. S., & Keane, T. M. (1998). *Clinician-administred PTSD scale for DSM-IV*. Los Angeles: Wps Publishers and Distributors.

Boscarino, J. A. (1996). Posttraumatic stress disorder, exposure to combat, and lower plasma cortisol among vietnam veterans: Findings and clinical implications. *Journal of Consult Clinical Psychology, 64*, 191-201.

Boudewyns, P. A., Hyer, L., Woods, M. G., Harrison, W. R., & McCranie, E. (1990). PTSD among vietnam veterans: An early look at treatment outcome using direct therapeutic exposure. *Journal of Traumatic Stress, 3*(3), 359-368.

Bowman, M. L., & Yehuda, R. (2004). Risk factors and the adversity-stress model. In G. M. Rosen (Ed.), *Posttraumatic stress disorder issues and controversies*. West Sussex: John Wiley & Sons, Ltd.

Bradley, R., Greene, J., Russ, E., Dutra, L., & Westen, D. (2005). A multidimensional meta-analysis of psychotherapy for PTSD. *Am J Psychiatry, 162*(2), 214-227.

Breedlove, S. M., Rosenzweig, M. R., & Watson, N. V. (2007). *Biological psychology. An introduction to behavioral, cognitive and clinical neuroscience* (Fifth ed.). Sunderland, Massachusetts: Sinauer Associates, Inc.

Bremner, J. D. (2007). Neuroimaging in posttraumatic stress disorder and other stress-related disorders. *Neuroimaging Clinics of North America, 17*(4), 523-538.

Bremner, J. D., Randall, R., Vermetten, E., Staib, L., Bronen, R. A., Mazure, C. S., et al. (1997). Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse. *Biological Psychiatry, 41*(23-32).

Breslau, N., Davis, G. C., Andreski, P., & Peterson, E. (1991). Traumatic events and posttraumatic stress disorder in an urban population of young adults. *Arch Gen Psychiatry, 48*, 216-222.

Breslau, N., Davis, G. C., Peterson, E. L., & Schultz, L. R. (2000). A second look at comorbidity in victims of trauma: The posttraumatic stress disorder-major depression connection. *Biological Psychiatry, 48*(9), 902-909.

Breuer, J., & Freud, S. (1957). *Studies on hysteria*. New York: Basic Books.

Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma exposed adults. *Journal of Consulting and Clinical Psychology, 68*(5), 748-766.

Bryant, R. A., Moulds, M. L., Guthrie, R. M., Dang, S. T., Mastrodomenico, J., Nixon, R. D. V., et al. (2008). A randomized controlled trial of exposure therapy and cognitive restructuring for posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology., 76*(4), 695-703.

Bryant, R. A., Moulds, M. L., Guthrie, R. M., Dang, S. T., & Nixon, R. D. V. (2003). Imaginal exposure alone and imaginal exposure therapy with cognitive restructuring in treatment of posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 71*(4), 706-712.

Burke, P. A., & Bradley, R. G. (2006). Language use in imagined dialogue and narrative disclosures of trauma. *Journal of Traumatic Stress, 19*(1), 141-146.

Cienfuegos, J., & Monelli, C. (1983). The testimony of political repression as a therapeutic instrument. . *American Journal of Orthopsychiatry, 53*, 43-51.

Clark, D. (1989). Anxiety states. Panic and generalized anxiety. In K. Hawton, P. M. Salkovskis, J. Kirk & D. M. Clark (Eds.), *Cognitive behavior therapy for psychiatric problems. A practical guide.* New York: Oxford University Press.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, N. J.: Laurence Erlbaum.

Cozby, P. C. (2007). *Methods in behavioral reasearch* (Ninth Edition ed.). Crawfordsville: Mc Graw Hill Higher Education.

de Jong, J. T. V. M., Komproe, I. H., Van Ommeren, M., El Masri, M., Araya, M., Khaled, N., et al. (2001). Lifetime events and posttraumatic stress disorder in 4 postconflict settings. *JAMA, 286*(5), 555-562.

Derogatis, L. R. (1975). *The SCL-90-R.* Baltimore: Clinical Psychometric Research.

Derogatis, L. R. (1992). *SCL-90-R. Administration, scoring & procedures. Manual-II.* (2nd ed.). Towson: Clinical Psychometric Research.

Derogatis, L. R. (1994). *SCL-90-R: Administration, scoring and procedures manual.* (3 ed.). Minneapolis: National Computers System.

Derogatis, L. R. (2000). SCL-90-R. In A. E. Kazdin (Ed.), *Encyclopedia of Psychology* (Vol. 7, pp. 192-193). Washington D.C.: American Psychological Association.

Escalona, R., Achilles, G., Waitzkin, H., & Yager, J. (2004). PTSD and somatization in women treated at a VA primary care clinic. *Psychosomatics, 45*(4), 291-296.

Foa, E. B., Dancu, C. V., Hembree, E. A., Jaycox, L. H., Meadows, E. A., & Street, G. P. (1999). A comparison of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault victims. *Journal of Consulting and Clinical Psychology, 67*(2), 194-200.

Foa, E. B., Hembree, E. A., & Rothbaum, B. O. (2007). *Prolonged exposure therapy for PTSD: Emotional processing of traumatic experiences: Therapist guide*. New York, NY: Oxford University Press.

Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99*(1), 20-35.

Foa, E. B., Molnar, C., & Cashman, L. (1995). Change in rape narratives during exposure therapy for posttraumatic stress disorder. *Journal of Traumatic Stress, 8*(4).

Foa, E. B., & Riggs, D. S. (1993). Posttraumatic stress disorder in rape victims. In J. Oldham, M. B. Riba & A. Tasman (Eds.), *American psychiatric press review of psychiatry.* (Vol. 12, pp. 273-303). Washington D.C.: American Psychiatric Press.

Foa, E. B., & Rothbaum, B. O. (1998). *Treating the trauma of rape. Cognitive-behavioral therapy for PTSD.* New York: The Guilford Press.

Foa, E. B., Rothbaum, B. O., Riggs, D. S., & Murdock, T. B. (1991). Treatment of posttraumatic stress disorder in rape victims: A comparison between cognitive-

behavioral procedures and counseling. *Journal of Consulting and Clinical Psychology, 59*(5), 715-723.

Foa, E. B., Steketee, G., & Rothbaum, B. O. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behavior Therapy, 20*, 155-176.

Gabbay, V., Oatis, M. D., Silva, R. R., & Hirsh, G. (2004). Epidemiological aspects of PTSD in children and adolescents. In R. R. Silva (Ed.), *Posttraumatic stress disorder in children and adolescents. Handbook.* (First ed.). New York: W.W . Norton & Company, Inc.

Hayes, S. C., Wilson, K. G., Gifford, E., Follette, V. M., & Strosahl, K. D. (1996). Emotional avoidance and behavioral disorders: A functional dimensional approach to diagnosis and treatment. *Journal of Consulting and Clinical Psychology, 64*, 1152-1168.

Herman, J. L. (1992). Complex PTSD: A syndrome in survivors of prolonged and repeated trauma. *Journal of Traumatic Stress, 5*(3), 377-391.

Herman, J. L. (1997). *Trauma and recovery: The aftermath of violence - from domestic abuse to political terror.* New York: Basic Rooks.

Jaycox, L. H., Foa, E. B., & Morral, A. R. (1998). Influence of emotional engagement and habituation on exposure therapy for PTSD. *Journal of Consulting and Clinical Psychology, 66*(1), 185-192.

Kaminer, D. (2006). Healing processes in trauma narratives: A review. *South African Journal of Psychology, 36*(3), 481-499.

Kar, N., & Bastia, B. (2006). Post-traumatic stress disorder, depression and generalised anxiety disorder in adolescents after a natural disaster: a study of comorbidity. *Clinical Practice and Epidemiology in Mental Health, 2*(1), 17.

Kardiner, A. (1941). *The traumatic neuroses of war*. New York: Hoeber.

Kardiner, A., & Spiegel, H. (1947). *War, stress and neurotic illness* (Rev. ed. ed.). New York: Hoeber.

Keane, T. M., Fairbank, J. A., Caddell, J. M., & Zimering, R. T. (1989). Implosive (flooding) therapy reduces symptoms of PTSD in Vietnam combat veterans. *Behavior Therapy, 20*(2), 245-260.

Keane, T. M., Marshall, A. D., & Taft, C. T. (2006). Posttraumatic stress disorder: Etiology, epidemiology, and treatment outcome. *Annual Review of Clinical Psychology, 2*, 161-197.

Kessler, R. C. (2000). Posttraumatic stress disorder:The burden to the Individual and to society. *Journal of Clinical Psychiatry, 61 (Suppl 5)*, 4-12.

Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry., 52*, 1048-1060.

Kolassa, I.-T., & Elbert, T. (2007). Structural and functional neuroplasticity in relation to traumatic stress. *Current Directions in Psychological Science, 16*(6), 321-325.

Lambert, M., & Stephenson, A. (2000). Beck depression inventory. In A. E. Kazdin (Ed.), *Encyclopedia of Psychology* (Vol. 1). Washington D.C.: Oxford University Press/American Psychological Asspciation.

Lamprecht, F., & Sack, M. (2002). Posttraumatic stress disorder revisited. *Psychosomatic Medicine, 64*(2), 222-237.

Lasiuk, C. G., & Hegadoren, K. M. (2006). Posttraumatic stress disorder part I: Historical development of the concept. *Perspectives i Psychiatric Care, 42*(1), 13-19.

LeDoux, J. E. (1996). *The emotional brain*. New York: Simeon & Shuster.

Lindal, E., & Stéfansson, J. G. (1993). The lifetime prevalence of PTSD of anxiety disorders in Iceland as estimated by the National institute of mental health diagnostic interview schedule. *Acta Psychiatrica Scandinavica, 88*, 29-34.

Malik, M. L., Connor, C. M., Sutherland, S. M., Smith, R. D., Davison, R. M., & Davidson, J. R. T. (1999). Quality of life and posttraumatic stress disorder: A pilot study assessing changes in SF-36 scores before and after treatment in a placebo-controlled trial of fluoxetine. *Journal of Traumatic Stress, 12*(2), 387-393.

Marks, I., Lovell, K., Noshirvani, H., Livanou, M., & Thrasher, S. (1998). Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring. *Arch Gen Psychiatry, 55*, 317-325.

Marlowe, D. H. (2000). Psychological and physiological consequences of combat and deployment with special emphasis of the Gulf War. . Retrieved 26.04 from http://gulflink.osd.mil/library/randrep/marlowe_paper/index.html, 2008

McEwen, B. S. (1999). Stress and hippocampal plasticity. *Annual Review of Neoroscience, 22*, 105-122.

McEwen, B. S. (2002). *The end of stress as we know it*. Washington DC: Joseph Henry Press/Dana Press.

Milde, A. M., Sundeberg, H., Grøseth, A., & Murison, R. (2003). Proactive sensitizing effects of acute stress on acoustic startle response and experimentally induced colitis in rats: Relationship to corticosterone. *Stress, 6*, 49-67.

Morgan, M. A., & LeDoux, J. E. (1995). Differential contribution of dorsal and ventral medial prefrontal cortex to the aqusition and extinction of conditioned fear in rats. *Behavioral Neuroscience, 109*, 681-688.

Nagai, M., Kishi, K., & Kato, S. (2007). Insular cortex and neuropsychiatric disorders: A recent review of the literature. *European Psychiatry, 22*(6), 387-394.

Neuner, F., Schauer, M., Karunakara, U., Klaschik, C., Robert, C., & Elbert, T. (2004). Psychological trauma and evidence for enhanced vulnerability for posttraumatic stress disorder through previous trauma among West Nile refugees. *Journal, 4*(34). Retrieved from http://www.biomedcentral.com/1471-244X/4/34. doi:10.1186/1471-244X-4-34

Neuner, F., Schauer, M., Klaschik, C., Karunakara, U., & Elbert, T. (2004). A comparison of narrative exposure therapy, supportive counseling, and psychoeducation for treating posttraumatic stress disorder in an african refugee settlement. *Journal of Consulting and Clinical Psychology, 72*(4), 579-587.

Neuner, F., Schauer, M., Roth, W. R., & Elbert, T. (2002). A narrative exposure treatment as intervention in refugee camp. A case report. . *Journal of Behavioural and Cognitive Psychotherapy, 30*, 205-209.

Neylan, T. C., Brunet, A., Pole, N., Best, S. R., Metzler, T. J., Yehuda, R., et al. (2005). PTSD symptoms predict waking salivary cortisol levels in police officers. *Psychoneuroendocrinology, 30*(4), 373-381.

Nutt, D. J., & Malizia, A. L. (2004). Structural and functonal brain changes in post traumatic stress disorder. *Journal of Clinical Psychiatry*(65), 11-17.

Onyut, L. P., Neuner, F., Schauer, E., Ertl, V., Odenwald, M., Schauer, M., et al. (2004). The Nakivale Camp mental health project: Building local competency for psychological assistance to traumatised refugees *Intervention: International Journal of Mental Health, Psychosocial Work & Counseling in Areas of Armed Conflict, 2*(2), 90-107.

Onyut, L. P., Neuner, F., Schauer, E., Ertl, V., Odenwald, M., Schauer, M., et al. (2005). Narrative exposure therapy as a treatment for child war survivors with posttraumatic stress disorder: Two case reports and a pilot study in an African refugee settlement. *BMC Psychiatry, 5*(7).

Orr, S. P., Metzger, L. J., Lasko, N. B., Macklin, M. L., Michael, L., Peri, T., et al. (2000). De novo conditioning in trauma- exposed individuals with and without posttraumatic stress disorder. *Journal of Abnormal Psychology, 109*(2), 290-298.

Passer, M. W., & Smith, R. E. (2001). *Psychology. Frontiers and applications.* (1st ed.). New York: McGraw-Hill.

Perkonigg, A., Kessler, R. C., Storz, S., & Wittchen, H. U. (2000). Traumatic events and posttraumatic stress disorder in the community: prevalence, risk factors and comorbidity. *Acta Psychiatrica Scandinavica, 101*(1), 46-59.

Radant, A., Tsuang, D., Peskind, E. R., McFall, M., & Raskind, W. (2001). Biological markers and diagnostic accuracy in the genetics of posttramatic stress disorder. *Psychiatry Research 102*, 203-204.

Rauch, S. L., Whalen, P. J., Shin, L. M., McInerney, S. C., Macklin, M. L., Lasko, N. B., et al. (2000). Exaggerated amygdala response to masked facial stimuli in posttraumatic stress disorder: A functional MRI study. *Biological Psychiatry, 47*(9), 769-776.

Resick, P. A., Nishith, P., Weaver, T. L., Astin, M. C., & Feuer, C. A. (2002). A comparison of cognitive-processing therapy with prolonged exposure and a waiting condition for the treatment of chronic posttraumatic stress disorder in female rape victims. *Journal of Consulting and Clinical Psychology, 70*(4), 867-879.

Rothbaum, B. O., Meadows, E. A., Resick, P., & Foy, D. W. (2000). Cognitive-behavioral therapy. In E. B. Foa, T. M. Keane & M. J. Friedman (Eds.), *Effective treatments for PTSD. Practice guidelines from the international society for traumatic stress studies.* (pp. 60-83). New York: The Guilford Press.

RVTS-Mid., P. R. T. (2005). CAPS, Unofficial Norwegian Translation.

Saigh, P. A., & Bremner, J. D. (1999). The history of posttraumatic stress disorder. In P. A. Saigh & J. D. Bremner (Eds.), *Posttraumatic stress disorder. A comprehensive text*. Needham Heights: Allyn & Bacon.

Schauer, E., Neuner, F., Elbert, T., Ertl, V., Onyut, L. P., Odenwald, M., et al. (2004). Narrative exposure therapy in children: A case study. *Intervention: International Journal of Mental Health, Psychosocial Work & Counseling in Areas of Armed Conflict, 2*(1), 18-32.

Schauer, M., Neuner, F., & Elbert, T. (2005). *Narrative exposure therapy. A short term intervention for traumatic stress disorders after war, terror, or torture.* Gõttingen: Hogrefe & Huber Publishers.

Schwartz, B., Wasserman, E. A., & Robbins, S. J. (2002). *Psychology of learning and behavior* (5th ed.). New York: W. W. Norton & Company, Inc.

Shapiro, F. (1989). Eye movement desensitization: A new treatment for posttraumatic stress disorder. *J. Behav. Ther. & Exp. Psychiat., 20*(3), 211-217.

Shapiro, F. (1996). Eye movement desensitization and reprosessing (EMDR): Evaluation of controlled PTSD research. *Journal of Behavior therapy and experimental psychiatry, 27*(3), 209-218.

Simeon, D., Knutelska, M., Yehuda, R., Putnam, F., Schmedler, J., & Smith, L. M. (2007). Hypothalamic-pituitary-adrenal axis function in dissoscitive disorders, post-traumatic stress disorder, and healthy volunteers. *Biological Psychiatry, 61*, 966-973.

Song, Y., Zhou, D., & Wang, X. (2008). Increased serum cortisol and growth hormone levels in earthquake survivors with PTSD or subclinical PTSD. *Psychoneuroendocrinology, 33*(8), 1155-1159.

Tarrier, N., Pilgrim, H., Sommerfield, C., Faragher, B., Reynolds, M., Graham, E., et al. (1999). A randomized trial of cognitive therapy and imaginal exposure in the

treatment of chronic posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology, 67*(1), 13-18.

Taylor, S., Asmundson, G. J. G., & Carleton, R. N. (2006). Simple versus complex PTSD: A cluster analytic investigation. *Journal of Anxiety Disorders, 20*, 459-472.

Van der Kolk, B. A., & Van der Hart, O. (1989). Pierre Janet and the breakdown of adaptation in psychological trauma. *Am J Psychiatry, 146*(12), 1530-1540.

Vassend, O., & Nielsen, G. H. (1991). SCL-90-R Uoffisiell norsk oversettelse.

Weathers, F. W. (2004). *Clinician-Administered PTSD Scale Technical Manual*. Los Angeles: Wps Publishers and Distributors.

Weathers, F. W., Newman, E., Blake, D. D., Nagy, L. M., Schnurr, P. P., Kaloupek, D. G., et al. (2004). *Clinician-Administered PTSD scale. Interviewer's guide*. Los Angeles: Wps Publishers and Distributors.

Weine, S. M., Kulenovic, A. D., Pavkovic, I., & Gibbons, R. (1998). Testimony psychotherapy in Bosnian refugees: A pilot study. *Am J Psychiatry, 155*(12), 1720-1726.

Weisaeth, L., & Eitinger, L. (1991). Reasearch on PTSD and other post-traumatic reactions I: European literature. *PTSD Reasearch Quarterly, 2*(2), 1-7.

World Health Organization. (1992). *The ICD-10 Classification of mental and behavioral disorders. Clinical descriptions and diagnostic guidelines.* Geneva: Universitetsforlaget.

Yehuda, R. (2002). Post-traumatic stress disorder. *New England Journal of Medicine, 346*(2), 108-114.

Yehuda, R., Kahana, B., Binder-Brynes, K., Southwick, S. M., Mason, J. W., & Giller, E. L. (1995). Low urinary cortisol excretion in Holocaust survivors with posttraumatic stress disorder. *American Journal of Psychiatry, 152*(7), 982-986.

Yehuda, R., McFarlane, A. C., & Shalev, A. Y. (1998). Predicting the development of posttraumatic stress disorder from the acute response to a traumatic event. . *Biological Psychiatry, 44*, 1305-1313.

Yonkers, K. A., & Samson, J. (2000). Mood disorders measures. In A. P. Asssociation (Ed.), *Handbook of psychiatric measures* (First ed.). Washington D.C.: American Psychiatric Association.

Appendix A

Tables

Table 1. Diagnostic Criteria for Post Traumatic Stress Disorder according to 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 persons response involved intense fear, helplessness or horror. Note: In children, this may be expressed instead by disorganized or agitated behaviour.
B.	**The traumatic event is persistently reexperienced in one (or more) of the following ways**
1)	recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. Note: In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.
2)	recurrent and distressing dreams of the event. Note: In children there may be frightening without recognizable content.
3)	acting or feeling as if the traumatic event was recurring)including a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). Note: In young children, trauma-specific re-enactment may occur.
4)	intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
5)	psychological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event
C.	**Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:**
1)	efforts to avoid thoughts, feelings, or conversations associated with the trauma
2)	efforts to avoid activities, places, or people associated 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 form 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 normal life span)
D.	**Persistent symptoms of increased arousal (not present before the trauma), as indicated by two or more of the following:**
1)	difficulty falling or staying asleep
2)	irritability or outbursts of anger
3)	difficulty concentrating
4)	hypervigilance
5)	exaggerated startle response
E	**Duration of the disturbance (symptoms in Criteria B, C and D) is more than 1 month.**
F.	**The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.**

Table 2. A general guideline of the organization of sessions in NET.

Session 1: Diagnosis and psychoeducation. **Session 2:** Start of the narration beginning at birt continuing through to the first traumatic event. **Session 3:** and subsequent sessions: Rereading of the narrative collected in previous sessions. Continuing the narration of subsequent life and traumatic events. **Final session:** Reareading and signing of the whole document.

Table 3. Age, gender and ethnicity in the sample.

	Age	Gender	Ethnicity
1	21	Male	Norwegian
2	42	Male	Norwegian
3	47	Male	Refugee
4	28	Male	Norwegian
5	55	Female	Norwegian
6	30	Female	Norwegian
7	19	Female	Norwegian
8	53	Female	Norwegian
9	41	Female	Refugee
10	36	Female	Norwegian

Table 4. Inclusion and exclusion criteria.

Inclusion criteria	Exclusion criteria
>18 years old Both sexes Fluent in Norwegian PTSD diagnosis	Mental Illness Shizofrenia Severe dissociation Ongoing substance abuse Suicidality Self mutilation Disease in the central nervous system Head injury Loss of consciousness >30 minutes Pregnancy Endocrinological diseases

Table 5. Types of traumas.

Traumatic experience	Numbers of patients reported having experienced this trauma
War (torture, imprisonment, physical injury and/or witnessed severe violence)	4
Rape	2
Sexual abuse	3
Assault	2
Accidents (car-accidents or accidents at work)	3
Serious physical illness/injury	1
Parental substance abuse	1
Natural disaster	1
Fire	2

Table 6. Research design of the main project.

PTSD-treatment group	Assessment I	Treatment	Assessment II		Assessment III
PTSD-waiting-list group	Assessment I		Assessment II	Treatment	Assessment III

Table 7. Research design of our study.

PTSD-treatment group	Assessment I	Treatment	Assessment II

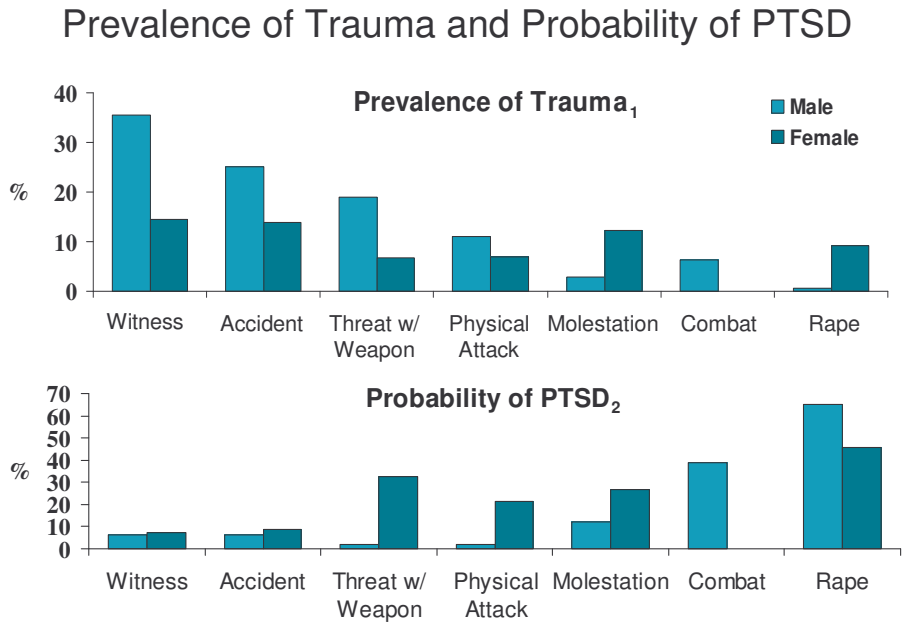
Table 13. Sex of the patients and the therapists.

Patient	Sex of the patient	Sex of the therapist
1	Female	Female
2	Female	Female
3	Female	Female
4	Male	Female
5	Male	Female
6	Male	Female
7	Female	Female
8	Female	Male
9	Female	Female
10	Male	Female

Appendix B

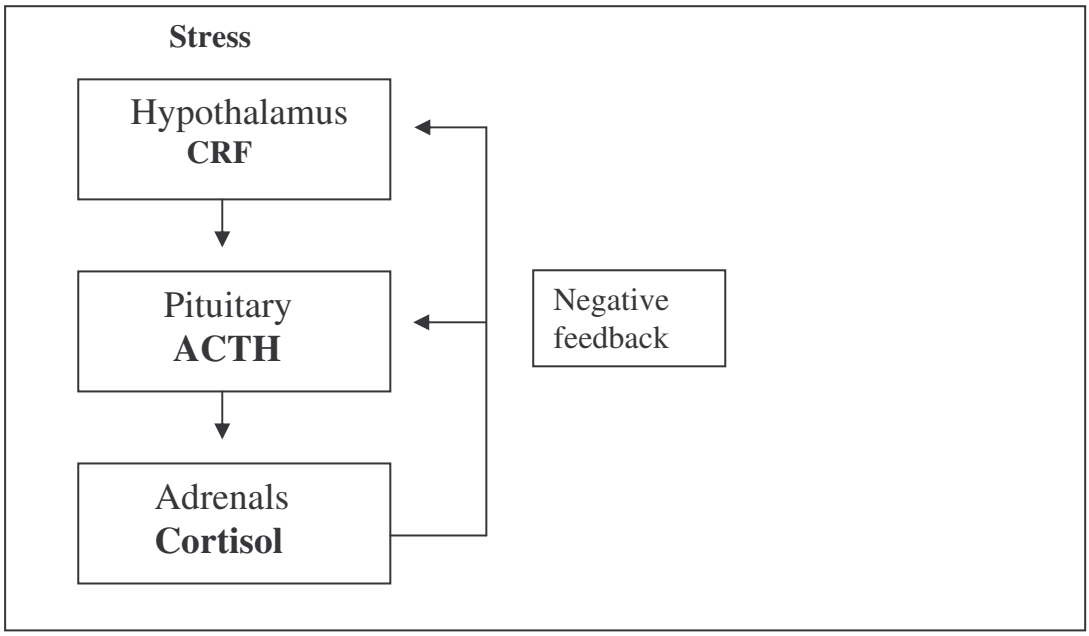
Figures

Figure 1. Prevalence of Trauma and Probability of PTSD.



1 Kessler R et al. J Clin Psychiatry. 2000;61(Suppl 5):4-14.
2 Kessler R et al. Arch Gen Psychiatry. 1995;52:1048-1060.

Figure 2. The HPA-axis.



CRF- Corticotropin realising factor
ACTH- Adrenocorticotropic hormone

Figure 3. The habituation process.

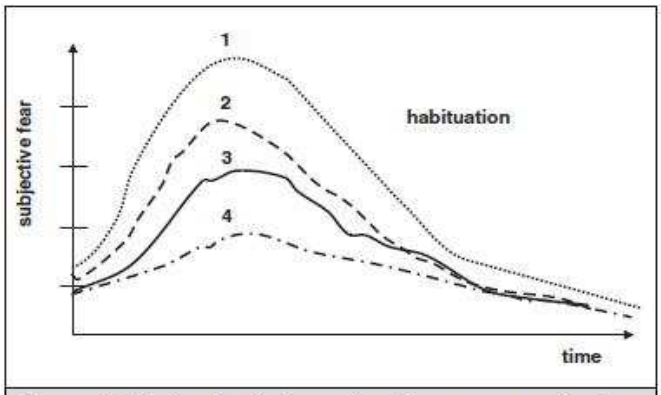


Figure 7. During the first emotional exposure, patients might have the perception that fear will increase infinitely. Initially (1), fear will go up until it reaches a peak and decreases naturally. After a second (2), third (3), and fourth (4) emotional exposure, fear will decrease significantly and habituation settles in. When exposure is stopped while fear increases, e.g., by avoidance, before habituation can take place, a negative pattern of increased anxiety is likely.

Schauer, M., Neuner, F., & Elbert, T. (2005). *Narrative Exposure Therapy. A Short Term Intervention for Traumatic Stress Disorders after War, Terror, or Torture.* Gõttingen: Hogrefe & Huber Publishers, p. 38.

Figure 4. Schematic presentation of a fear-structure/fear-network.

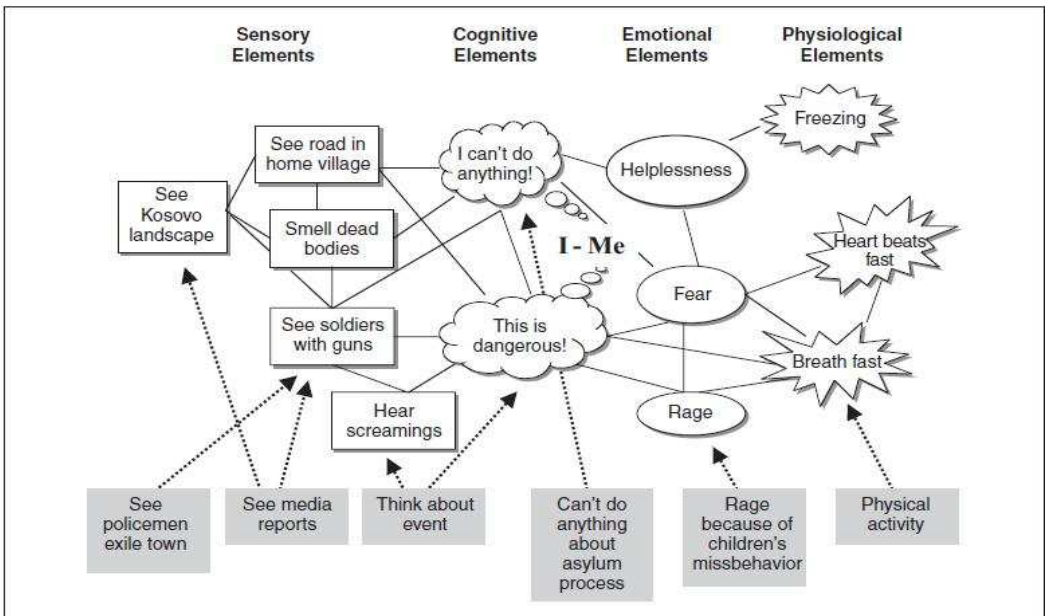


Figure 2. Schematic presentation of a hypothetical sensory-perceptual representation – a so-called "fear network," including sensory, cognitive, emotional, and physiological elements. The network represents a Kosovar refugee's memory of the attack on his home village. The boxes below indicate environmental stimuli with the potential to activate the representation.

Schauer, M., Neuner, F., & Elbert, T. (2005). *Narrative Exposure Therapy. A Short Term Intervention for Traumatic Stress Disorders after War, Terror, or Torture.* Gõttingen: Hogrefe & Huber Publishers, p. 16.

Figure 5. Cognitive restructuring during NET.

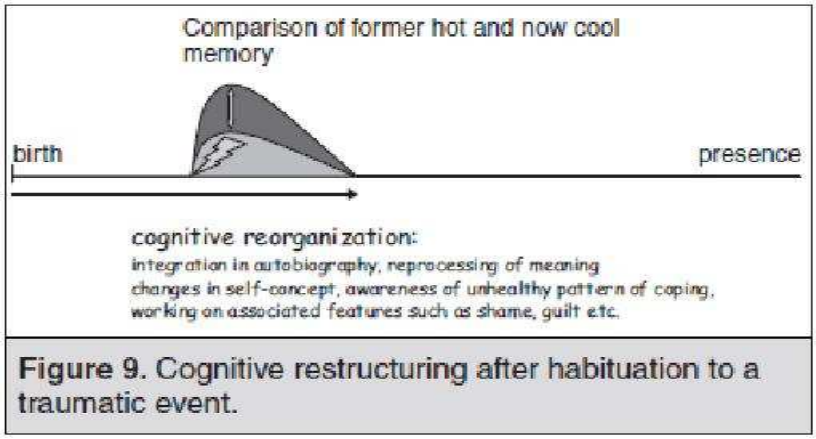


Figure 9. Cognitive restructuring after habituation to a traumatic event.

Schauer, M., Neuner, F., & Elbert, T. (2005). *Narrative Exposure Therapy. A Short Term Intervention for Traumatic Stress Disorders after War, Terror, or Torture.* Gõttingen: Hogrefe & Huber Publishers, p. 40.

Figure 6. A complete narration after NET.

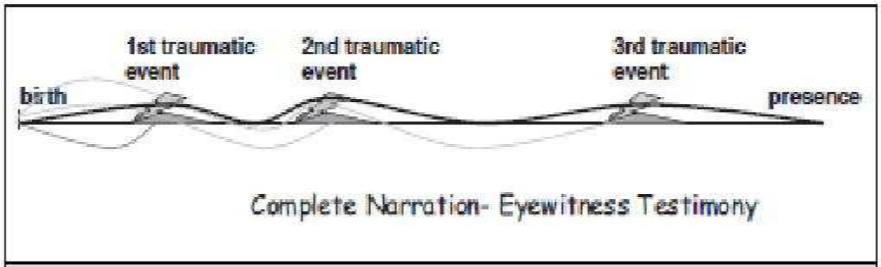
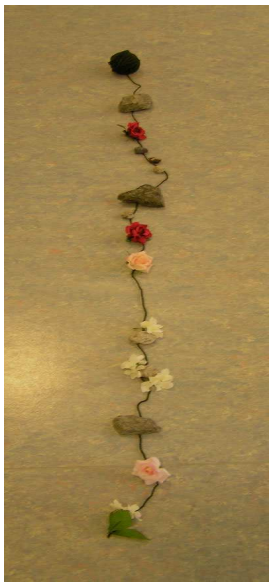


Figure 11. An entire narrative following the patient's life line is completed.

Schauer, M., Neuner, F., & Elbert, T. (2005). *Narrative Exposure Therapy. A Short Term Intervention for Traumatic Stress Disorders after War, Terror, or Torture.* Gõttingen: Hogrefe & Huber Publishers, p. 42.

Figure 7. "The Life-Line Exercise".



Appendix C

The approval from the Committee for Medical Research Ethics (Regional Etisk Komité)

UNIVERSITETET I BERGEN
Regional komité for medisinsk forskningsetikk, Vest-Norge (REK Vest)

Førsteamanuensis Anne Marita Milde
Institutt for biologisk og medisinsk psykologi, UiB
Jonas Lies vei 91
5009 BERGEN

Deres ref	Vår ref	Dato
	033.07 – 07/3029/ars	05.03.2007.

Ad. prosjekt: Behandlingseffekt av Narrativ eksponeringsterapi og kortisolnivå hos pasienter med posttraumatisk stress lidelser. (033.07).

Det vises til din søknad om etisk vurdering datert 06.02.07, inklusiv søknad om opprettelse av forskningsbiobank datert 16.02.07. REK Vest vurderte studien i møte den 22.02.07.

Komiteen mener studien har en relevant problemstilling og design som er egnet til å besvare forskningsspørsmålet. En har derfor ingen innvendinger til gjennomføring. I skrivet til deltakerne bør en imidlertid si litt mer om hva deltakelse innebærer.

Vi ønsker dere lykke til med gjennomføringen og minner om at komiteen setter pris på en sluttrapport, eventuelt en kopi av trykt publikasjon når dette foreligger.

Med vennlig hilsen

Jon Lekven
leder

Arne Salbu
sekretær

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