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Long-term Effects of Trauma Exposure on Inhibition and Rumination among Utøya Survivors

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Preface

The terror attack at Utøya in 2011 highlighted the importance of understanding the effects of trauma exposure on adolescent development. Unfortunately, terror continues to be a global problem with major ramifications for affected individuals as well as the society as a whole. Throughout the process of writing our thesis, global media coverage of tragic events around the world, including terror attacks and school shootings, has frequently reminded us of the persistent relevance of investigating the topics of our thesis.

We would like to thank our supervisor, Åsa Hammar, for her encouragement, enthusiasm, and confidence in our abilities. Specifically, her genuine interest and ability to link research to its clinical relevance have been of major inspiration. Furthermore, special thanks are directed to Thale H. Eggen, Ola U. Bergeland, and Tora P. Skagestad for their helpful comments and informative feedback. We would also like to express our gratefulness to our significant others for their tolerance and understanding in our process of finishing the thesis. Finally, we would like to thank each other for motivation, patience and excellent teamwork.

Sammendrag

Formålet med denne studien var å undersøke langtidseffekter av traumeeksponering på inhibisjon og ruminering blant ungdommer som overlevde terroren på Utøya 22. juli, 2011. Traumeeksponering er assosiert med svekket eksekutiv fungering. Inhibisjon betraktes som en kjernekomponent i eksekutiv fungering, og disse funksjonene er nødvendige for adekvat fungering i hverdagen. Dårlige inhibitoriske evner forbindes med økt ruminering. Å utforske effekter av traumeeksponering blant ungdom er relevant ettersom tilegnelsen av inhibitoriske ferdigheter er et sentralt aspekt i denne utviklingsfasen. I denne tverrsnittsstudien ble 20 traumeeksponerte ungdommer sammenlignet med 20 kontroller på nevropsykologiske tester av inhibisjon og selvrapporterte mål av inhibisjon og ruminering. Oppfølgende undersøkelser av undergrupper ble foretatt for å studere forskjeller i inhibisjon og ruminering relatert til PTSD symptomatologi. Analysene avdekket assosiasjoner mellom svekkelser i inhibisjon og grad av PTSD symptomatologi. Traumeeksponerte deltakere rapporterte signifikant større problemer med ruminering enn ikke-eksponerte kontrolldeltakere. Alle rapporterte resultater var signifikante på $p<.05$ nivå og effektstørrelsen var sterk for alle signifikante resultater ($n>.14$). Det konkluderes med at svekket inhibisjon i større grad er relatert til PTSD symptomatologi enn traumeeksponering i seg selv, og at traumerelatert ruminering ser ut til å være mindre avhengig av PTSD symptomatologi. Funnene antyder at inhibitorisk dysfunksjon kan være en risikofaktor og konsekvens av PTSD symptomatologi hos traumeeksponerte ungdommer. Longitudinelle studier trengs for å undersøke videre effekter av traumer i en større utviklingssammenheng.

Abstract

The aim of the present study was to examine long terms effects of trauma exposure on inhibition and rumination in adolescent survivors of the terror at Utøya 22nd of July, 2011. Exposure to trauma is associated with impairments in executive function. Inhibition is a core component of executive functioning, and these functions are essential for adequate adjustment in everyday life. Poor inhibitory abilities relate to increased rumination. It is relevant to explore the effects of trauma exposure in adolescents, as acquisition of inhibitory skills represents a central aspect of this developmental phase. In this cross-sectional case-control study, 20 trauma exposed adolescents and 20 controls were compared on neuropsychological tests of inhibition and self-report measures of inhibition and rumination. Follow-up analyses of subgroups were conducted to assess differences in inhibition and rumination related to PTSD symptomatology. Analyses revealed associations between impaired inhibition and more severe PTSD symptomatology. Trauma exposed subjects reported significantly more problems with rumination than non-exposed controls. All reported results were significant with alpha level $p<.05$, and effect sizes were large for all significant results (n>.14). We conclude that impaired inhibition is more related to PTSD symptomatology than mere trauma exposure, and that trauma-related rumination appears to be less dependent on PTSD symptomatology. Findings suggest that inhibitory dysfunctions may be a risk factor and consequence of PTSD symptomatology in trauma exposed adolescents. Longitudinal designs are warranted to further investigate effects of trauma in a broader developmental context.

Keywords: Trauma exposure, posttraumatic stress disorder, adolescence, neuropsychology, executive function, prefrontal cortex, inhibition, rumination.

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Long-term Effects of Trauma Exposure on Inhibition and Rumination Among Utøya Survivors

The research literature on adults solidly documents the impact of trauma on several areas of functioning, including emotion, cognition, and biology. The field of developmental psychology describes transitions in these areas through development. Integrating the field of developmental psychology with trauma research results in a developmental perspective on the effects of trauma. Trauma research has predominately focused on adults, leaving these associations nearly unexplored in children and adolescents. With adolescents being more similar to children in some areas and more like adults in others, it is reasonable to inquire whether they may respond to trauma in corresponding or distinct manners. Central in adolescent development is acquisition of executive control functions essential for successful transition into adult life. Inhibition is an executive function (EF), and impairments in this function have been associated with trauma exposure. Inadequate inhibition has been associated with rumination, a maladaptive thinking pattern which has been found in patients with posttraumatic stress disorder and other psychological disorders. The current study aims to explore the effects of trauma exposure on an adolescent developing brain by examining inhbition and rumination in survivors of the terror attack at Utøya.

On July 22nd, 2011, two terror attacks were executed in Norway. A bomb explosion in the government district in central Oslo was followed by a brutal massacre at Utøya Island, killing 77 innocent people in total. These attacks represent the worst violent acts committed in Norway during peacetime (Norwegian Center for Violence and Traumatic Stress Studies, n.d.). Unfortunately, according to reports from the National Counterterrorism Center (2012), the terror on July 22nd adds to about 10.000 other terror attacks worldwide in 2011.

Acts of terrorism vary on several dimensions, and a well agreed upon conceptualization of this phenomenon seems difficult to capture. In an attempt to construct a

universally applicable definition of terrorism, Arnold and colleagues (2003) suggest that terrorism is "The intentional use of violence — real or threatened — against one or more non-combatants and/or those services essential for or protective of their health, resulting in adverse health effects in those immediately affected and their community, ranging from a loss of well-being or security to injury, illness, or death" (p. 49). There are numerous definitions of terror, and this lack of agreement probably reflects the heterogeneity among terror attacks (Victoroff, 2005). However, the attacks in Norway on July 22nd clearly resemble well-known characteristics of terror as described above.

The terror attacks in Norway this day left an entire nation in fear and grief. In addition to those killed or physically injured, many were indirectly affected as next-of-kin or rescue workers, or through material damage. At the time of the attack, The Norwegian Labor Party's youth organization hosted their annual summer camp at Utøya. Fivehundred-and-sixtyfour people were on the island when the perpetrator arrived, most of them politically active adolescents (Dyb, Jensen, Glad, Nygaard, & Thoresen, 2014). The perpetrator walked around the island, shooting and killing everyone in sight, until the police arrested him roughly 90 minutes later. Sixtynine innocent people were murdered, and many more injured. The 495 registered survivors were exposed to major psychological traumas, and there are reasons to believe that they still are affected by this tragedy.

Traumatic Events and Posttraumatic Stress Disorder

The concept of trauma has often been used in a twofold manner as referring to stressful events as well as psychological reactions to such events. A general, objective definition of trauma has proved difficult to capture: Stressful events vary in a number of ways, and the perception of an event as stressful depends on subjective appraisal (Weathers & Keane, 2007). The understanding of whether an event should be defined as traumatic or not is to a large extent based on knowledge about individuals´ reactions to these events, and

this knowledge is mainly based on examinations of posttraumatic stress disorder (PTSD; Bonanno, 2004; Bonanno, Galea, Bucciarelli, & Vlahov, 2006). PTSD is a diagnosis that comprises common psychological reactions to stressful events, which means that it by definition presupposes the experience of a traumatic event (World Health Organization [WHO], 1992). Although studies on lifetime prevalence have found a higher possibility of experiencing a traumatic event during a lifetime than not experiencing one, not all of those who experience a trauma develop PTSD (Breslau, Davis, Andreski, & Peterson, 1991; Frans, Rimmö, Åberg, & Fredrikson, 2005; Galea, Nandi, & Vlahov, 2005; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995; Norris, 1992). Research on trauma and PTSD has contributed to identify common features of traumatic events, to define these events, and to describe them as more or less predictive of PTSD.

Traumatic events. Definitions of traumatic events are often based on diagnostic criteria for PTSD. The International Classification of Mental and Behavioral Disorders (ICD-10; WHO, 1992) proposes the following definition of a traumatic event:

> A stressful event or situation (of either brief or long duration) of an exceptionally threatening or catastrophic nature, which is likely to cause pervasive distress in almost anyone (i.e., natural or manmade disasters, combat, serious accidents, witnessing the violent death of others, or being the victim of torture, terrorism, rape, or other crime). (p. 120).

Similar to ICD-10, traumatic events are described as events that involve exposure to actual or threatened death, serious injury or sexual violence in the fifth and most recent edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association, 2013). Further nuances are accounted for in DSM-5, where witnessing or learning about events occurring to others, as well as repeated or extreme exposure to aversive details of the events, are also included as traumatic experiences. Thus, diagnostic

conceptualizations of traumatic events correspond to events or situations frequently reported by PTSD patients.

Risk and protective aspects of traumatic events. Variations in the kind of events or situations that may be defined as traumatic experiences, and dependence on subjective appraisal, make it challenging to objectively define stressors. This complexity is reflected in the research on aspects of traumatic events considered to exacerbate the severity of exposure and increase the likelihood for posttraumatic stress reactions. Various aspects of trauma exposure are identified as risk factors for PTSD.

Variations in type and characteristics of traumatic events are found to be more or less predictive of posttraumatic stress reactions. For instance, research on types of trauma suggest that exposure to sexual and physical assault pose a greater risk for PTSD than exposure to traffic road accidents (Frans et al., 2005), and victims of mass violence (e.g., terrorism, mass shooting) are more likely to be negatively affected than victims of natural or technological disasters (Neria, Nandi, & Galea, 2008; Norris et al., 2002). In addition to the variation in types of traumatic experiences, an event may vary on aspects such as frequency, duration, predictability, controllability, and magnitude of exposure (Weathers & Keane, 2007). The latter aspect, magnitude or degree of exposure, is consistently documented as a predictor of PTSD in adult populations (Brewin, Andrews, & Valentine, 2000; Frans et al., 2005; Neria et al., 2008), and predictive of posttraumatic stress symptoms in children and adolescents who survive terror (Dyb et al., 2014; Scrimin et al., 2011; Suomalainen, Haravuori, Berg, Kiviruusu, & Marttunen, 2011). Neria and colleagues (2008) further describe other important aspects included in the magnitude of exposure to be a direct risk of life, degree of physical injury, number of deaths, and severity of destroyed property. These aspects seem to be especially predictive of PTSD.

Traumatizing aspects of the terror attack at Utøya. Several aspects of the massacre at Utøya may have exacerbated the perceived threat of the attack. Mappings of degree of exposure among survivors of Utøya revealed that more than 90% heard screams during the attack and ran or hid from the perpetrator, the majority saw dead bodies, around 50% saw someone getting injured or killed, and more than two thirds reported that they had feared for their life (Dyb, Jensen, Nygaard, et al., 2014; Hafstad, Dyb, Jensen, Steinberg, & Pynoos, 2014). Other aspects may have further amplified the severity of this attack.

As the mass shooting took place on an island, the victims had limited opportunities to escape or hide. The perpetrator´s appearance as a trustworthy policeman also confused the victims about whom to trust, and around 40% of the survivors reported that they had felt threatened by the police (Dyb, Jensen, Nygaard, et al., 2014). Many of the adolescents knew each other, and witnessing friends and acquaintances being wounded or killed may have intensified the traumatic experience. An examination of survivors of Utøya found that loss of a friend could predict posttraumatic reactions (Dyb, Jensen, Nygaard, et al., 2014).

Furthermore, media exposure following terror attacks has been found to trigger psychological distress in unaffected individuals through mechanisms of indirect exposure (Holman, Garfin, & Silver, 2014). Among directly exposed individuals, media exposure may have served as trauma reminders, and frequency of trauma reminders are found to be predictive of PTSD (Scrimin et al., 2011). Other aspects of the Utøya tragedy may have been as protective factors. Studies of children and adolescents exposed to trauma show that social support protects against and reduces posttraumatic stress reactions (Dyb, Jensen, Nygaard, et al., 2014; Pine & Cohen, 2002; Suomalainen et al., 2011), and high levels of perceived social support is found among Utøya survivors (Dyb, Jensen, Nygaard, et al., 2014). By emphasizing that Norway was attacked as a nation and presenting the victims as innocent targets, the media coverage following 22nd of July may have contributed to perceived social

support among survivors. District-based follow-up interventions that were implemented in the aftermath of the disaster at Utøya (Dyb & Glad, 2013) may further have reinforced the perception of support and cohesion among victims. Nevertheless, survivors of the attack at Utøya were exposed to major psychological trauma, as indicated by research on prevalence of PTSD among survivors.

PTSD. A study on the prevalence of PTSD among survivors of Utøya reveals that levels of posttraumatic stress were more than six times higher in survivors than in the general population (Dyb, Jensen, Nygaard, et al., 2014). More specifically, 47% of the 325 survivors included in the study reported clinical levels of PTSD, whereof 11% met criteria for full PTSD and 36% for partial PTSD. An unpublished study of 27 survivors found that 29.6% fulfilled the diagnostic criteria for PTSD (Bøen & Matre, 2014).

PTSD has been documented as the most common psychiatric disorder after trauma exposure, with a prevalence rate of 30-40% in direct victims of trauma (Galea et al., 2005). PTSD is characterized by symptoms of intrusion (e.g., intrusive memories or recurrent dreams), avoidance (e.g., efforts to avoid thoughts, feelings, or external reminders of the trauma), and hyperarousal (e.g., hypervigilance, increased startle response, sleep problems; American Psychiatric Association, 2013). Negative alterations in cognition and mood are recognized as a new symptom cluster in the fifth edition of DSM (American Psychiatric Association, 2013). In order to be diagnosed with PTSD, a certain number of symptoms from each of the clusters must have persisted according to specified time periods and caused significant distress or interference with everyday functioning (American Psychiatric Association, 2013).

Exposure to stressful experiences is a necessary, though not a sufficient, causal factor in PTSD (Frans et al., 2005), and most studies about reactions to trauma exposure are therefore focused on this diagnosis. However, PTSD is frequently comorbid with other

psychiatric disorders (Breslau et al., 1991). There is an extensive overlap of PTSD and depressive symptoms, and the two conditions frequently co-occur in trauma exposed patients (Galea et al., 2002; Shalev, 1998). Major depressive disorder (MDD) is found to be the most prevalent trauma-related disorder secondary to PTSD (Norris et al., 2002; Shalev, 1998). Studies reveal that PTSD patients have a 65% prevalence of comorbid MDD (Bleich, Koslowsky, Dolev, & Lerer, 1997). Even though both MDD and PTSD are common disorders after traumatic experiences, PTSD is the only diagnosis that presupposes trauma exposure and is therefore the main basis of research on reactions to trauma.

PTSD symptomatology has been linked to cognitive impairments such as disturbed memory of the traumatic event and attention deficits as a result of disturbing symptoms (Johnsen, Kanagaratnam, & Asbjornsen, 2013). Furthermore, research has indicated that trauma exposure can be associated with primary cognitive dysfunctions that go beyond those related to processing of trauma-related material (Johnsen et al., 2013). Studies of PTSD patients reveal problems with concentration and memory as measured by self-reports (Johnsen et al., 2013), and neuropsychological tests (Horner & Hamner, 2002; Vasterling et al., 2002). Trauma exposure is also associated with primary impairments in EF (for reviews: Aupperle, Melrose, Stein, & Paulus, 2012; Polak, Witteveen, Reitsma, & Olff, 2012). EF is important for the regulation of other cognitive functions and impairments can therfore extensive impact on adjustment in many areas of life (A. Miyake & Friedman, 2012; A. Miyake et al., 2000; Vaughan & Giovanello, 2010). EF develops relatively late compared to other cognitive functions (Atkins, Bunting, Bolger, & Dougherty, 2012; Barkley, 2012; Bunge & Wright, 2007; Davidson, Amso, Anderson, & Diamond, 2006), and trauma-related impairments in EF is therefore relevant to study in a developmental context.

Understanding Trauma-Related Impairments in EF in a Developmental Context

Many domains of life may be affected by exposure to traumatic events. Trauma exposed individuals have demonstrated disturbances in emotion, cognition, and biology (Landsman, 2002). All of these levels of functioning influence each other and rely on EF for adequate adjustment in everyday life. EF refers to multiple higher order cognitive skills that are necessary to perform goal-directed activity (A. Miyake et al., 2000), such as planning, problem solving, inhibition, and set shifting (Leskin & White, 2007). The relatively late acquisition of EF has to do with their reliance on the prefrontal part of the brain (Anderson, 2002). The prefrontal cortex (PFC) is the last part of the brain to fully mature around the mid-20s (Reyna, Chapman, Dougherty, & Confrey, 2012). EF is commonly referred to as the function of PFC, and these terms are often used interchangeably when describing higher order cognitive function (e.g., Alvarez & Emory, 2006; A. Miyake et al., 2000; Stuss & Alexander, 2000). A slightly more appropriate simplification of this relationship may be so say that EF "happens" in the region of PFC.

The coinciding maturation of PFC and acquisition of EF represents an important hallmark of the transition from childhood to adulthood, and a central component of adolescent development according to neuropsychological standards (Reyna et al., 2012; Steinberg, 2011). Associations beween trauma exposure and impaired EF is thoroughly studied among adult PTSD populations and reviews on this this field generally conclude that adults with a PTSD diagnosis perform poorer on neuropsychological tests tapping EFs (e.g., Aupperle et al., 2012; Polak et al., 2012). Compared to adults, these associations are less explored in children and adolescents (Dalgleish, Meiser-Stedman, & Smith, 2005; Turley & Obrzut, 2012). It is suggested particularly relevant to study associations between trauma exposure and EF in adolescents because of distinct characteristics of this developmental phase (Schoeman, Carey, & Seedat, 2009). Despite this, few studies exist on isolated groups

of adolescents who have been exposed to trauma. In all its infortune, the survivors of the Utøya tragedy represent a unique and naturally occurring opportunity to study the effect of trauma on adolescent development.

Characteristics of adolescent development. Adolescence is typically defined as the development and growth that takes place between the ages of 10 and 19 (WHO, 2015). However, there is a lack of consensus about the defining boundaries of adolescence, and the issue can be approached from several perspectives (Steinberg, 2011). Adolescence involves a gradual transformation towards increased maturity, independence and responsibility (Spear, 2007) which includes several transitions on biological, emotional, cognitive, and social levels (Steinberg, 2011). These transitions have different starting points and various durations for every individual, and leave the person with increased maturity in some areas before others (Steinberg, 2011). From a neuropsychological point of view, the brain is not considered fully developed until the frontal regions complete their maturation process halfway through the third decade of life (Reyna et al., 2012). As such, the PFC is in continuous development through, and beyond, adolescence as it is defined by age. In the context of this paper, adolescence will be discussed according to a neuropsychological perspective with emphasis on prefrontal brain development as an important hallmark of adolescence.

The relatively late maturation of PFC and corresponding EFs becomes particularly evident in adolescence because of other characteristics of this developmental phase. Adolescence is characterized by increases in reckless behavior traits such as novelty seeking, sensation seeking, risk taking, and impulsivity (Arnett, 1992; M. Wilson & Daly, 1985). Many risky decisions typically made by adolescents can have serious consequences, as reflected in accident statistics, risky driving, unintentional pregnancies, and experimental substance use (Arnett, 1992; Blackmore & Robbins, 2012; Viner et al., 2012). As Steinberg (2005) points out, puberty-related alterations in arousal and motivation appear to proceed the

maturation of PFC and corresponding regulatory functions. As such, the combination of the delayed prefrontal brain development and prototypical adolescent behaviors has been described as resulting in a temporal gap between increases in behaviors that need regulation and the necessary regulatory skills (Steinberg, 2005). This apparent disjunction has been described metaphorically as "starting the engine with an unskilled driver" (Nelson et al., 2002, p. 515).

Development of PFC and EF in adolescence. Understanding the pathways of development related to maturation of PFC and acquisition of EF constitute an important framework for exploring the possible impact of trauma in adolescence. As a result of brain maturation, especially in frontal regions, EF seems to develop continually from infancy through childhood and into early adulthood (Atkins et al., 2012; Barkley, 2012; Bunge & Wright, 2007; Davidson et al., 2006). Maturation of the adolescent brain unfolds through three basic steps: Synaptic overproduction, pruning, and myelination (Johnson, Blum, & Giedd, 2009). Synaptic overproduction leads to a peak in gray matter volumes in frontal regions around the age of 12 (Giedd et al., 1999). Following this, a pruning process takes place with the shared purpose of eliminating neural connections that are rarely used and allowing the remaining ones to become more efficient and specialized (Giedd, 2008). Subsequent myelination of these surviving connections ensures a more integrated brain with faster and more efficient communication within and between regions (Anderson, 2002). These changes facilitate improvements in EF and accompanying regulatory skills (Compas, 2004), including inhibition (Luna et al., 2001). The fact that the pruning and myelination processes that facilitate these advancements do not occur in the PFC until the third decade of life explains why PFC and EF are central aspects of biological development in adolescence.

Understanding developmental gaps in adolescence. Adolescent-related changes in biology and behavior appear to follow uneven pathways. This lack of synchrony implies that

the capacity for behavioral expression will be ahead of the capacity for behavioral regulation. This may immediately sound unfavourable, but has been suggested to be adaptive as similar behavior changes are seen among other mammals in corresponding phases of life (Spear, 2007). From an evolutionary perspective, adolescence-related increases in novelty seeking and risk taking are considered highly preserved because they facilitate successful completion of central developmental tasks in adolescence (M. Wilson & Daly, 1985), such as emigration away from home and successful reproduction without inbreeding (Bixler, 1992). In this sense, delayed maturation of PFC and related inhibitory functions may actually be both necessary and beneficial for the survival of our species.

The concept of developmental gaps may be further clarified by consulting the terms "hot" and "cold" cognition. Whereas hot cognition refers to thought processes influenced by emotion and high arousal, cold cognition corresponds to more emotionally neutral and rational thought processes (e.g., Goel & Dolan, 2003; Steinberg, 2005). In line with this terminology, adolescent risk taking behavior and impulsive responses may be understood in the framework of hot cognition, while typical EFs would represent the operation of cold cognition (Steinberg, 2005). As such, it is possible to describe adolescence as a period in life characterized by underdeveloped cold cognition combined with overactive hot cognition. The terms of hot and cold cognition may also relate to a more detailed account for development within PFC itself. The functions of ventromedial and dorsolateral systems in PFC have been implicated in hot and cold cognition, respectively (Steinberg, 2011). As Phillips and colleagues (2003) point out, the ventral system may recognize emotional salience and produce emotional responses, while the dorsal system inhibits or modulates these emotional states and expressions in order to be in accordance with contextual norms. It has been found that the brain appears to develop from ventral to dorsal (Mackiewicz, 2011), which implies

that the ventral hot system might be more developed than the dorsal cold system in adolescents.

Viewing the delayed aquisition of EF seen in the context of other characteristics of adolescence highlights the distinctiveness of this period. Although the developmental gaps of adolescence may be adaptive in an evolutionary framework, they may also leave the individual exposed and vulnerable when faced with developmental demands. Protracted development of the PFC also renders the adolescent brain particularly vulnerable for the influence of environmental factors such as traumatic stress (Augusti & Melinder, 2013; Gralton, Muchatuta, Morey-Canellas, & Lopez, 2008; Insana, Banihashemi, Herringa, Kolko, & Germain, 2015; Thompson-Schill, Ramscar, & Chrysikou, 2009; K. R. Wilson, Hansen, & Li, 2011). However, in spite of increased vulnerability due to brain plasticity, it is also important to highlight that potentials and strengths may be associated with a plastic brain (Johnson et al., 2009; Steinberg, 2005). The relatively protracted development of EF appears to result in a characteristic gap in the transition phase from childhood to adulthood. Understanding the effect of trauma on the brain becomes relevant as areas responsible for such cold cognition, namely PFC, may be directly affected when exposed to trauma.

The Effects of Trauma on the PFC

Exposure to traumatic experiences activates a cascade of neurochemical processes in the brain (Carrion & Wong, 2012; K. R. Wilson et al., 2011). This response is adaptive and necessary to fight or flight from an immediate stressor. However, if it becomes too frequent or prolonged, it can disturb brain development and alter neural circuits involved in emotion and cognition (Carrion & Wong, 2012; Weber & Reynolds, 2004; K. R. Wilson, 2010). This is known as a traumatic stress response and involves the interaction of the limbic system, the prefrontal cortex (PFC), and the hypothalamic-pituitary-adrenal-axis (HPA-axis). Amygdala is a part of the limbic system – the emotional control centre of the brain – and is responsible

for detecting and initiating a response to threats (Arnsten, 1998; Southwick, Rasmusson, Barron, & Arnsten, 2005). Catecholamine neurotransmitters from amygdala further activate the HPA-axis, which initiates the release of stress hormones, such as cortisol (Arnsten, 1998; Southwick et al., 2005), and conveys the message of a perceived stressor to other brain region, such as the PFC. The PFC, which is responsible for higher cognitive processes, terminates the stress response by inhibiting the activity of the amygdala and the HPA-axis (Cohen, Perel, DeBellis, Friedman, & Putnam, 2002). The main result of these neurochemical processes is an enhanced functioning of the amygdala and impaired functioning of the PFC, which implies a risk of poorly regulated emotional activity (Arnsten, 1998; Southwick et al., 2005).

Exposure to trauma may cause structural and functional changes in the PFC. Reviews on adults with PTSD show that the PFC is hyporesponsive and structurally smaller among these patients (e.g., Francati, Vermetten, & Bremner, 2007; Shin, Rauch, & Pitman, 2006). This is supported by fMRI studies that have documented increased limbic activation in the amygdala and decreased activation in prefrontal areas in adults who suffer from PTSD (Francati et al., 2007). The high levels of catecholamine stimulation is also known to impair the functioning of the PFC (Arnsten, 1998).

Neuroimaging research on the effects of trauma on PFC in children have revealed similar findings. Carrion and colleagues (2001) found that children with posttraumatic stress symptoms had smaller total brain volumes compared to healthy controls. Related findings reveal that traumatized children had larger gray matter volumes in middle and ventral regions of the PFC, but smaller volumes in the dorsal area (Richert, Carrion, Karchemskiy, & Reiss, 2006) – the area previously implicated in cold cognition. Similar findings have been reported elsewhere (e.g., De Bellis et al., 1999; Hanson et al., 2012; Richert et al., 2006). Traumatic stress in children and adolescents has further been associated with high levels of cortisol

secretion (Carrion & Wong, 2012; Weber & Reynolds, 2004; K. R. Wilson, 2010), and evidence shows that chronic cortisol exposure contributes to neuronal atrophy in the brain (Luine, Villegas, Martinez, & McEwen, 1994).

In summary, findings indicate that trauma exposure and posttraumatic stress symptoms have been associated with impairments in areas of the PFC that are thought to be responsible for EF (Carrion & Wong, 2012). Alterations in PFC have been suggested to mediate the link between stress and impaired EF (Hanson et al., 2012).

The Effects of Trauma on EF

As described, impairments in EF are solidly documented in adult PTSD populations. EF is a broad concept, and more detailed accounts reveal that specific aspects of EFs seem to be selectively impaired in trauma exposed individuals(Aupperle et al., 2012). Frequently addressed topics in the literature concern whether EF should be understood as one uniform mechanism, or an umbrella term that includes various separable sub-functions (Barkley, 2012). Many attempts have been made in order to investigate the organization of executive sub-functions (e.g., Burgess, Alderman, Evans, Emslie, & Wilson, 1998). According to Miyake and colleagues (2000), executive sub-functions commonly addressed in the literature are: "(a) Shifting between tasks or mental sets, (b) updating and monitoring of working memory representations, and (c) inhibition of dominant or prepotent responses" (pp. 54). The relevance of these sub-functions (i.e., shifting, updating, and inhibition) is also reported elsewhere (Baddeley, 1996; DePrince, Weinzierl, & Combs, 2009; Hall & Marteau, 2014; Smith & Jonides, 1999). In their review, Polak and colleagues (2012) found significant associations between PTSD and specific tests tapping EF. Such findings suggest that sub-functions, rather than unitary EF, may be affected in the aftermath of trauma exposure.

Inhibition as a central sub-function of EF. A review by Aupperle and colleagues (2012) conclude that deficits in inhibition and attention sub-functions of EF may be

particularly central in PTSD patients. Inhibition is acknowledged as a core component of EF (Carrion, Garrett, Menon, Weems, & Reiss, 2008), and described as the ability to purposefully stop prepotent, dominant, or automatic responses (A. Miyake et al., 2000). Miyake and Friedman (2012) postulate that inhibition is the purest function of EF as it is highly correlated with unitary EF, as opposed to other sub-functions that to a greater extent are considered a combination of unitary EF and other executive sub-functions. Furthermore, researchers have suggested that inhibition is involved in a range of other EFs (Burgess et al., 1998). Thus, inhibition is among some researchers considered a primary component and an important sub-function of EF, and impairments in EF seem to be especially evident in neuropsychological assessments of trauma exposed individuals.

Inhibition and rumination. Research on EF has revealed connections between inhibition and rumination (Davis & Nolen-Hoeksema, 2000; Schmid & Hammar, 2013). Rumination is a maladaptive thinking pattern characterized by a repeated focus on distressing symptoms and causes or consequences thereof (Ehlers & Clark, 2000; S. Nolen-Hoeksema, 1991). Inhibitory dysfunctions have been found to correlate with increased rumination (Davis & Nolen-Hoeksema, 2000; Joormann, 2006; Philippot & Brutoux, 2008; Whitmer & Gotlib, 2013). It has been suggested that impaired inhibition in some cases serve to maintain the ruminative pattern despite of its maladaptive effects (Elwood, Hahn, Olatunji, & Williams, 2009). Taken together, rumination may be said to reflect poor regulatory skills, which again may in some cases be attributable to inhibitory dysfunctions. The association between inhibitory dysfunction and rumination is well established in research on depression (Davis & Nolen-Hoeksema, 2000; Philippot & Brutoux, 2008; Schmid & Hammar, 2013; Whitmer & Gotlib, 2012), and both constructs have separately been implicated in PTSD.

Core symptoms of PTSD has been suggested to reflect a breakdown in cognitive control functions so that primary impairments in EF lead to poor performance on cognitive

tasks as well as poorer ability to inhibit symptoms (Johnsen et al., 2013). More specifically, it has been suggested that each of the symptom clusters of PTSD may be attributable to underlying impairments in inhibition (Leskin & White, 2007). Various studies have found associations between PTSD and rumination (Ehring, Frank, & Ehlers, 2008; Elwood et al., 2009; Roley et al., 2015). It has been suggested that rumination represents a maintaining factor in PTSD (Elwood et al., 2009) and that it is predictive of symptom severity in PTSD (Ehring et al., 2008). Furthermore, anticipatory and repetitive rumination has been found to mediate between PTSD and depression (Roley et al., 2015). Trauma related rumination involves pondering about causes and consequences of the trauma rather than processing of traumatic experiences (Ehlers & Clark, 2000). By preventing processing of trauma related memories, rumination tendencies may result in increased re-experiencing symptoms (Ehlers, Mayou, & Bryant, 1998). Taken together, findings indicate that poor inhibitory abilities may cause PTSD symptoms directly as well as indirectly through its maintaining effect on rumination.

Neuropsychological research on EF and inhibition in adults. Research on the neuropsychological effects of trauma exposure in adults is predominantly based on PTSD populations, resulting in less knowledge about trauma exposed samples without symptomatology (Bonanno et al., 2006; Stein, Kennedy, & Twamley, 2002). Although the association between PTSD and impairments in EF and inhibition is relatively well established (Aupperle et al., 2012; Polak et al., 2012), the direction of the relationship remains somewhat unclear. Many attempts have been made at determining whether executive dysfunctions represent cause or effect of PTSD, or a combination of both (Hart et al., 2008; Olff, Polak, Witteveen, & Denys, 2014; Steudte-Schmiedgen et al., 2014). Some researchers find that those who develop PTSD following trauma show greater cognitive deficits than those who do not, and interpret this as PTSD causing the difficulties rather than trauma per se (Leskin &

White, 2007; Olff et al., 2014). For instance, symptoms of hyperarousal have been suggested to be so demanding that less cognitive capacity remains for other tasks (Johnsen et al., 2013). In this line of thought, impairments in EF may be understood as a consequence of coping with PTSD symptoms. However, methodological and ethical issues challenge the inference that PTSD causes executive dysfunction. Premorbid cognitive functioning is usually unknown and can therefore not be excluded as an explanation for why some develop PTSD and some do not (Aupperle et al., 2012; Polak et al., 2012). Reviews suggest that executive and inhibitory dysfunctions following trauma should be considered a risk factor for development of PTSD as well as a plausible consequence of the disorder (Aupperle et al., 2012; Johnsen et al., 2013).

Other studies conclude that executive dysfunction may result from the trauma itself, as they find poorer functioning among trauma exposed individuals compared to unexposed controls regardless of diagnostic status (Stein et al., 2002). Polak and colleagues (2012) further found that PTSD subjects differed more from mere trauma exposed participants than non-exposed controls in measures of EF. They propose that these results can be understood through a posttraumatic growth approach. Posttraumatic growth refers to positive personal changes following trauma (Bostock, Sheikh, & Barton, 2009). In this line of thought, those who do not develop PTSD after trauma exposure may represent a group that acquire effective coping strategies that both prevent them from developing PTSD and enable them to adjust better to life after trauma (Bonanno et al., 2006). As mentioned, the adult literature mainly concerns PTSD samples and less is therefore known about aspect that prevent or protect against development of PTSD.

Neuropsychological research on EF and inhibition in children and adolescents. Associations between trauma exposure, PTSD, and neuropsychological impairments are nearly unexplored in children and adolescents. As far as we know, only two reviews are

published on neuropsychological functioning among children and adolescents exposed to trauma. Both of these publications indicate associations between impairments in EF and exposure to trauma among children and adolescents (Dalgleish et al., 2005; Turley & Obrzut, 2012). An unpublished review that also summarizes studies on these associations yields similar results (Holmeng & Stavestrand, 2014), with six of the eight included studies concluding that exposure to trauma and PTSD is associated with impairment in EF (Barrera, Calderón, & Bell, 2013; Beers & De Bellis, 2002; De Bellis, Hooper, Spratt, & Woolley, 2009; De Bellis, Woolley, & Hooper, 2013; Leskin & White, 2007; Samuelson, Krueger, Burnett, & Wilson, 2010; Schoeman et al., 2009).

This field of research is mainly concerned with children and adolescents exposed to trauma involving maltreatment, neglect and intimate partner violence. However, research on neuropsychological function on children and adolescents exposed to terror attacks seems to yield similar findings. Scrimin and colleagues (2006) found that children surviving the terror attack in Beslan had difficulties in sustaining attention and in short-term memory, and Melinder and colleagues (2015) found an association between set-shifting and PTSD symptoms in a sample of Utøya survivors. Consistent with the literature on adults, exposure to trauma appears to affect EF in children and adolescents exposed to various types of trauma. In further consistence, sub-functions of EF seem to be specifically affected, and struggles with inhibition are indicated as especially evident in trauma exposed children and adolescents (Barrera et al., 2013; Holmeng & Stavestrand, 2014). Despite shortcomings, the literature accumulated over last decade suggests that traumatized children and adolescents are likely to struggle with EF and inhibition.

The literature on associations between trauma exposure, PTSD, and neuropsychological functioning has requested research on subgroup comparisons that differentiate between trauma exposed individuals, with and without PTSD, and non-exposed

control subjects (e.g., Dalgleish et al., 2005; Holmeng & Stavestrand, 2014). As mentioned, the literature on adult populations tend to reveal greater differences between traumatized individuals, with and without PTSD, compared with comparisons of PTSD and non-exposed controls (for review see Polak et al., 2012). The neuropsychological literature on traumatized children and adolescents is too scarce to summarize consistent findings of differentiations between the relative impact of PTSD and trauma exposure per se. However, some tendencies that support a similar notion among children and adolescents are revealed in the existing literature.

Studies of children and adolescents do find impaired EF to be more associated with PTSD than trauma exposure per se (Leskin & White, 2007; Schoeman et al., 2009). Furthermore, impairments in specific sub-functions of EF, are found to be associated with posttraumatic stress symptoms (Melinder et al., 2015). Other results, though not significant, also indicate that neuropsychological deficits may be attributed to PTSD rather than mere trauma exposure (Samuelson et al., 2010), and that children with PTSD tend to exhibit more difficulties than traumatized children without PTSD (De Bellis et al., 2009). Other studies do not reveal pronounced differences between PTSD and mere trauma exposure (De Bellis et al., 2013; Twamley, Hami, & Stein, 2004). However, DeBellis and colleagues (2013) did find that PTSD participants struggled more with one out of several neuropsychological measures compared to traumatized participants without PTSD. Despite non-significant results, DeBellis, Hooper, Spratt and Woolley De Bellis, et al. (2009) report that PTSD subjects tended to be worse off than exposed participants without PTSD in several neuropsychological measures. Contradicting results are also reported, for instance, Barrera and colleagues (2013) found equivalent impairments in PTSD and trauma exposed groups. The inconsistency in reported results is natural given the scarce research literature, and more research is needed in order to establish general findings. However, based on existing literature, it is considered

appropriate to affirm that preliminary findings in children and adolescents points towards similar tendencies as found in the adult literature: Children and adolescents who develope PTSD appear to struggle more with EF than trauma exposed children and adolescents without PTSD.

Few consistent findings exist in trauma exposed adolecents as this group is frequently merged with studies on younger populations. Although the relative lack of studies may reflect the absense of clearly defined boundaries of adolescence, several unique characteristics of adolescence highlight the relevance of studying this group separately.

Aim of the present study

Associations between trauma exposure, PTSD and impairments in EF are well-established in the neuropsychological literature on adult populations. Central suggestions in this field of research include that impairments in EF are more strongly associated with PTSD than with exposure to trauma per se, that specific aspects of EF may be affected following trauma exposure, and that inhibition may be one especially influenced aspect of EF related to trauma exposure and PTSD. These associations are nearly unexplored among children and adolescents, but the existing literature generally indicate consistent findings with the adult literature. Inhibition is crucial for the regulation of thoughts, emotions and behavior, and impairments have been found to maintain ruminating tendencies. Poor inhibitory skills and rumination have both been suggested to contribute to symptoms of PTSD. However, research on the interplay of rumination and inhibition in the understanding of trauma-related suffering is still in its infancy.

As adolescence is associated with immature inhibitory functions, it is particularly relevant to examine the effect of trauma that occurs in this phase of life. With their recklessness and immature regulatory functions, combined with increased societal demands of independence and responsibility, adolescents can be said to have one foot in each camp of

childhood and adulthood. Despite this unique developmental position, they are often merged with children or adults in studies of trauma exposure and PTSD. Studying survivors of Utøya represent an unique opportunity to shed light on developmental impact of trauma exposure in adolescence.

The main aim of the present study is to explore the neuropsychological effects of trauma exposure in a developmental perspective. In a cross-sectional case-control study, multivariate- and univariate analyses of variance (MANOVAs and ANOVAs, respectively) will be carried out to investigate how trauma exposure in adolescence may affect inhibition and rumination, and to examine associations between these variables and PTSD symptomatology. In order to investigate the latter, trauma exposed participants are divided in subgroups based on reported PTSD symptoms. Correlational analyses will be carried out to explore associations between inhibition and rumination.

Existing literature on children and adults enables certain predictions, as it is considered appropriate to inquire similar tendencies among adolescents. However, studying the neuropsychological effects of trauma in adolescence is explorative as it adds to a nearly unexplored research field on effects of trauma exposure on adolescent development. The impact of trauma exposure on inhibition and rumination in adolescents will be addressed through investigating the following research questions:

1. Do trauma exposed adolescents differ from controls on neuropsychological and self-reported measures of inhibition? Will follow-up comparisons of sub-groups reveal differences in inhibition related to PTSD symptomatology?
2. Do trauma exposed adolescents differ from controls on self-reported measures of rumination? Will follow-up comparisons of subgroups reveal differences in rumination related to PTSD symptomatology?
3. Is there an association between inhibition and rumination?

We predict that difficulties with inhibition and ruminative tendencies will be more pronounced in trauma exposed adolescents than in controls. Furthermore, we expect that sub-groups with higher PTSD symptom load will exhibit more problems with inhibition and rumination than trauma exposed sub-groups with less or no PTSD symptoms and the control group. Finally, we expect to find that greater difficulties with inhibition are associated with higher levels of rumination.

Methods

Participants

The sample consisted of 40 subjects whereof 20 were Utøya survivors and 20 were control participants. Participants in the two groups were matched by age, gender and level of education. There were 14 males and 26 females in the sample. They were between the ages of 17-24, with a total mean age of 19.98 (*SD* = 1.51; Males: *M* = 20, *SD* = 1.88; Females: *M* = 19.96 , *SD* = 1.31). All participants were either employed or under education.

Recruitment and Sample

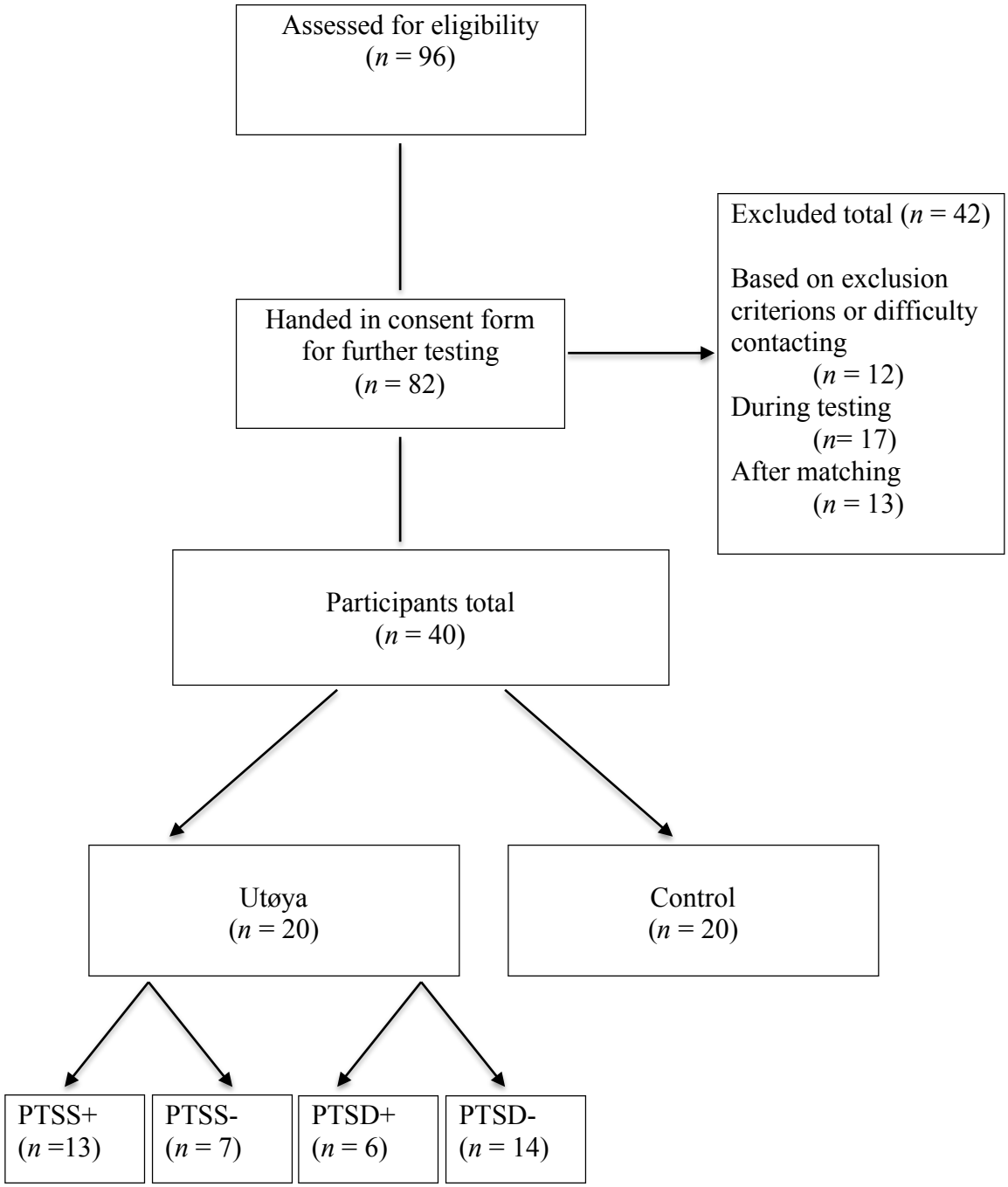
The trauma exposed group. Written invitations were sent to 45 Utøya survivors between the ages of 16-25 in the Hordaland and Rogaland counties. Invitations were sent through the Resource center for Violence, Traumatic stress and Suicide prevention region West (RVTS West) and contained information about the study and the tests. Participants who were interested answered by returning a signed consent form. Participants were excluded if they had a history of endocrinological, psychiatric or neurologic illness prior to the Utøya attack, previous experience of head trauma leading to unconsciousness longer than 10 minutes, metal implants not compatible with fMRI scanning, substance abuse, or current pregnancy. Travel expenses related to participation were covered and all subjects received a honorarium of 500 NOK.

After receiving an invitation, 29 of the 45 survivors handed in the consent form, resulting in a response rate of 64%. In addition to the 29 potential participants, two Utøya survivors learned about the study in the media and contacted the research team directly with requests to participate. Phone interviews of the 31 potential participants were carried out to ensure that the inclusion criterias were met, and to check for fMRI suitability. The interviews led to exclusion of four survivors who were difficult to contact, or who did not meet the inclusion criteria. All subjects who were interested were asked if they had been physically injured in the shooting. One survivor confirmed and was included despite inability to complete the fMRI testing. None of the participants withdrew during testing. After testing, seven participants were further excluded in the present study due to various reasons.

The control group. The control participants were politically active youths from other political parties. None of these subjects were involved in the trauma or related to any of the survivors. Several approaches were used to recruit control subjects. Leaders of political youth organizations in Hordaland county were contacted for permission to visit the political party meetings and hand out invitations to participate in the study. After recruiting some participants, these participants collected contact information from other interested youths. In addition, adolescents known to be politically active were contacted directly and invited to participate. Prior to participation, all interested subjects were given information handouts describing the main purpose of the study along with consent forms for participation.

In addition to the exclusion criteria mentioned above, subjects with ongoing or previous history of psychiatric illness (assessed through Mini International Neuropsychiatric Interview [MINI]; Sheehan et al., 2009) were screened out as this would affect the results. 51 control subjects were recruited, whereof eight were excluded based on not meeting different inclusion criteria. During the testing, further ten participants were excluded, due to inconveniences such as artifacts on fMRI scans, technical failure in a computer program used

in the fMRI session, and psychiatric symptoms reported in the neuropsychiatric interview. After collecting all data, controls were matched to the trauma exposed group, which further caused exclusion of 13 control participants. All control subjects received a honorarium of 500 NOK to compensate for their participation (See figure 1 for participants).



Figur 1. Flowchart illustrating inclusion of participants, and separation into different groups for different analysis.

Procedures

The data for this study were collected 32 - 46 months after the attack at Utøya. The participants were a part of an extensive research project on long-term outcomes of the Norwegian terror attack. In this research project, they were assessed through a neuropsychiatric interview and neuropsychological tests. They further underwent an fMRI session and assessments of circadian rythm and diurnal cortisol. On the testing day, they started out with the fMRI session, followed by the neuropsychiatric interview and finally the neuropsychiatric assessment. The current study concerns a subset of the neuropsychological assessments.

Diagnostic Evaluation and Procedure

Mini International Neuropsychiatric Interview (MINI). Qualified health professionals administered the Norwegian version of MINI (MINI, 6.0.0;Sheehan et al., 2009). MINI is a structured interview that detects psychiatric diagnoses according to diagnostic manuals (ICD-10 and DSM-IV). It is known to be applicable, with good results on measures of validity and reliability (Lecruiber et al., 1997). MINI explores both ongoing and previous disorder based on criterion symptoms, as it differentiates between current symptoms and symptoms from an earlier time. When questions are answered confirmatory, follow-up questions determine the presence of sufficient symptoms to fulfill diagnostic criteria. The 27 MINI diagnoses assessed in the present study were clustered into six categories: Eating disorder, abuse/addiction, mania, depression, anxiety and suicidality. In addition, occurrence of subclinical posttraumatic stress disorder (Posttraumatic Stress Symptoms; PTSS) was examined to detect those who confirmed symptoms of PTSD but who did not fulfill the diagnostic criteria. In the present study, the only diagnosis used in the analyses will be PTSD, PTSS.

Neuropsychological Assessment

The participants underwent a comprehensive neuropsychological assessment. The test battery included measures of IQ and other standardized and experimental tests. All testing was performed during regular work hours, administered in the same sequence to all participants, and took approximately 4 hours to complete.

Measurement of general intelligence.

Wechsler Abbreviated Scale of Intelligence (WASI). WASI is a short test that estimates verbal IQ, performance IQ and full scale IQ (Ryan et al., 2003). It was developed at the same time as the Wechsler Adult Intelligence Scale – III (WAIS-III), and its measures are thus linked to the WAIS-III (Axelrod, 2002). The WASI generates a general intellectual function based on two subtests of verbal abilities and two subtests of performance. It is known to have sufficient internal consistency and test-retest reliability for the three IQs (Axelrod, 2002).

Measurements of inhibition and EF. Two measurements of inhibition were carried out: A standardized test from the Delis-Kaplan Executive Function System (D-KEFS; Delis, Kaplan, & Kramer, 2001), and The Behavior Rating Inventory of Executive Function – Adult version (BRIEF-A). D-KEFS was administered by a trained technician and BRIEF-A was self-reported by the participants.

D-KEFS Color-Word Interference Test (CWIT). D-KEFS CWIT is based on the Stroop (1935) procedure. The test consists of four conditions: color naming (C), word reading (W), inhibition (CW), and inhibition/switching (IS). In the first two conditions (C and W), the participant is presented with cards containing colored patches (C) and names of colors written in black ink (W), and instructed to name the colors and read the words as fast as possible. In the CW condition, the participant is presented with color names written in congruent and incongruent colors. The subject is instructed to name the ink color rather than

read the word. In the IS condition, some words are presented in a frame. The subject is instructed to continue as in the previous condition, except when the word is presented in a frame. When this is the case, the subject is told to read the word instead of naming the color. The participant is then switching back and forth between naming the dissonant ink colors and reading the words. This way the IS condition measures both inhibition and the ability to shift mental sets (mental flexibility; Delis et al., 2001). D-KEFS and CWIT have been found to hold good clinical utility, as well as discriminant validity (Oasay, 2013).

BRIEF-A. The BRIEF-A is a standardized measure of adult EF and self-regulation, which consists of a self-reported rating form. It is based on the original BRIEF and contains 75 items in nine clinical scales and three validity scales. The clinical scales are theoretically and empirically derived, and do not overlap (Gioia, Isquith, Guy, & Kenworthy, 2000). These scales form two indexes - Behavioral Regulation and Metacognition - which further are combined in the overall summary score termed Global Executive Composite. Inhibit, Shift, Emotional Control and Self-Monitor represent the four subscales subsumed in the Behavioral Regulation scale. In the Metacognition scale, there are five subscales: Initiate, Working Memory, Plan/Organize, Organization of Materials, and Task Monitor (Gioia et al., 2000). Only results from the overall summary score (BRIEF total) and the inhibition subscale (BRIEF inhibition) will be reported as these are relevant for the research questions in the present study. BRIEF-A has been found to be reliable and valid measure of EF in adults (Roth, Isquith, & Gioia, 2005).

Measurement of rumination. Rumination was measured by using the Measurement Ruminative Response Scale (RRS) that measures state rumination.

RRS. The RRS is a subscale of the Response Styles Questionnaires (RSQ). The scale consists of 22 self-report items that assess responses to self-focused and symptom-focused dysphoric mood, and potential causes and possible consequences of this. The RRS maps

ruminating tendencies by instructing the subject to rate statements like “Think about how sad you feel”, “Think about all your shortcomings, failings, faults, mistakes” (depression related items),”Go away from yourself and think about why you feel this way” (reflective items), and “Think “Why do I have problems other people don’t have” ”(brooding items) (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). Each item is rated on a likert scale from 1-4, where 1 is almost never and 4 is almost always (Roelofs, Muris, Huibers, Peeters, & Arnts, 2006). High scores indicate high levels of rumination, and lower scores indicate less rumination.

Statistical Analyses

All statistical analyses were conducted using the IBM SPSS, version 22.0 and 23.0. Statistical significance was defined as $p < .05$ for all results.

Preliminary analyses. The trauma exposed group and the control group were matched on the variables gender, age, and education. The matching procedure was carried out using FUZZY extension commands of 0, 2 and 3 for gender, education level, and age, respectively. Independent t-tests were subsequently conducted to ensure that the matching on age and education level had been successful in eliminating differences between the groups. To further assure that the groups were sufficiently similar before conducting analysis, we compared the exposed group and the non-exposed group on WASI total intelligence quotient (IQ). Preliminary t-tests were also conducted to control for differences between the groups on the C and W conditions of the CWIT, as potential differences on these variables could distort the interpretation of results on the CW and IS conditions.

Missing values were replaced with mean in all cases (4.27% of the total amount of test values). In the RRS questionnaire, several participants did not receive the last page of the questionnaire. Eleven participants seven from of the trauma exposed group and four from the control group were missing nine questions. These values were replaced with mean. Due to the large proportion of missing data on this scale, additional procedures were carried out to

ensure that these subjects did not differ from other participants in any significant matter. Using independent t-tests, the 11 participants were compared to a random selection of 11 participants from corresponding groups on related variables. No differences were found when comparing the them on related variables that could have been reflected on the RSS. It was therefore assumed that participants who missed a page of the questionnaire were sufficiently similar to other members of their respective groups, and that replacing with mean could be justified.

Main analyses. Preliminary assumption testing for MANOVA and ANOVA was conducted by assessing for univariate and multivariate outliers, linearity, homogeneity of variance, covariance matrices, and multicollinearity. No serious violations were noted when testing the preliminary assumptions to execute univariate and multivariate analysis of variance.

MANOVA. One-way between groups MANOVAs were performed to investigate the effect of trauma exposure on inhibition and rumination. The two groups, trauma exposure and control, were used as levels of the independent variable "group", and the results on the tests CWIT, BRIEF, and RRS were used as dependent variables. Due to strong correlations between the BRIEF total and the BRIEF inhibition measures, these variables were assessed consecutively in the MANOVAs. The follow-up MANOVAS were then conducted, where the original trauma exposed group was divided into different subgroups based on PTSD symptom criteria. The trauma exposed group was first subdivided based on presence (+) or absence (-) of posttraumatic stress disorder (PTSD+/-). Those with diagnosed PTSD as determined by MINI were grouped together in the PTSD+ group (n = 6), while the remaining ones were placed in a PTSD- group (n = 14). This made it possible to isolate the effect of PTSD. Next, the trauma exposed group was subdivided based on PTSS status. In this case, the PTSS+ group (n = 13) included participants who qualified for a PTSD diagnosis or

reported a subclinical level of PTSD symptoms according to MINI, while the PTSS- group (n = 7) comprised those without reported symptoms. Although grouping together those with PTSD and PTSS makes the effect of PTSD more ambiguous, this subdivision created a symptom-free trauma exposed group. In the first follow-up, PTSD status was used as the independent variable, and the response on CWIT conditions CW and IS, BRIEF inhibition, and RRS as dependent variables. The same analysis was repeated with BRIEF total instead of BRIEF inhibition. Following this, the same procedure was done using PTSS status as the independent variable. As the sample consisted of only 40 participants, the alpha level of $p>.05$ was not Bonferroni adjusted in the MANOVA to control for a possible Type 2 error.

ANOVA. After performing the MANOVAs, three separate ANOVAs were carried out for the significant MANOVA results. The same ANOVA on the two subgroup combinations ([PTSD+, PTSD- and control],[PTSS+, PTSS- and control]) was performed, with group as the independent variable, and the response on CWIT, BRIEF, and RRS as the dependent variables. Post-hoc tests using Tukey honest significance difference (Tukey HSD) were applied on all the ANOVAs.

Correlation analysis. A correlation analysis was carried out on the variables CW and IS of the CWIT, BRIEF inhibition, BRIEF total, and RRS.

Results

Descriptive Analyses

Preliminary t-tests. Results of the preliminary t-tests revealed that there were no significant differences between the trauma exposed group and the control group on the variables age, level of education, and WASI IQ. The results of the t-tests on the C and W conditions of the CWIT indicated that there were no significant differences between the groups. Results of all the preliminary analyses are illustrated in Table 1.

Table 1.

Means and Standard deviations on the variables age, years of education, WASI IQ and CWIT conditions C and W for the trauma exposed group and the control group.

	Trauma exposed		Control			
Descriptives	*M*	*SD*	*M*	*SD*	*t*	*p*
Age	20.00	1.49	29.95	1.57	.103	.354
Years of education	14.45	1.05	13.95	1.43	-1.259	.217
WASI IQ	113.05	10.97	112.30	8.50	.242	.284
CWIT C	26.50	4.097	28.20	5.606	-1.095	.140
CWIT W	21.55	3.471	20.70	3.672	.752	.863

Note. Scores on the CWIT test are measured in seconds.

Main Analyses

A one-way between groups MANOVA was carried out, using group membership (i.e., trauma exposed, control) as the independent variable and CWIT CW, CWIT IS, BRIEF inhibition and RRS as dependent variables. There was a statistically significant difference between the groups on the dependent variables, F (4, 35) = 3.10; Wilks´ Lambda = .74; partial eta squared = .26. When the results for the dependent variables were considered separately, there was a significant difference on the BRIEF inhibition and RRS variables. An investigation of the mean scores indicated that the trauma exposed group reported more rumination and greater difficulties with inhibition than the control group. Replacing BRIEF inhibition with BRIEF total did not result in any additional significant findings (significant results are indicated in table 2).

Table 2.

Means and Standard deviations of CWIT conditions CW and IS, BRIEF inhibition, BRIEF total, and RRS scores when using the PTSD+, PTSD-, and control grouping.

	Trauma exposed		Control				
Test	*M*	*SD*	*M*	*SD*	*F*(2,37)	*p*	*Partial* η^2
CWIT CW	47.75	11.58	47.40	10.61	.010	.921	.00
CWIT IS	58.20	11.31	54.70	8.48	1.23	.275	.03
BRIEF inhibition	13.70	3.11	11.70	2.36	5.23	.028*	.12
BRIEF total	47.45	9.58	42.20	7.93	3.56	.067	.86
RRS	45.00	12.55	36.20	5.35	8.34	.006*	.18

Note. Scores on CWIT are measured in seconds. High scores indicate longer time needed to perform the test. High scores on BRIEF indicate more reported difficulties with inhibition or general EFs. High scores on RRS indicate higher levels of reported rumination.
* The result is significant at the 0.05 level

Follow-up MANOVA with groups determined by PTSD status. MANOVA using PTSD status as the independent variable, and CWIT CW, CWIT IS, BRIEF inhibition and RRS as dependent variables was carried out. There was a statistically significant difference between the groups on the dependent variables, *F* (4, 34) = 3.87; Wilks´Lambda =.47; partial eta squared = .31. When the results for the dependent variables were considered separately, there was a significant difference on the CWIT IS, BRIEF inhibition, and RRS variables. An investigation of the mean scores indicated poorer scores for the PTSD group on the measures CWIT IS, BRIEF inhibition and RRS than PTSD- and the control group, respectively. When using BRIEF total instead of BRIEF inhibition, results showed that the PTSD+ group reported more problems than the PTSD- group and the control group, respectively (see table 3 for results on MANOVA using groups determined by PTSD status).

Table 3.

Means and Standard deviations of CWIT conditions CW and IS, BRIEF inhibition, BRIEF total, and RRS scores when using the PTSD+, PTSD-, and control grouping.

	PTSD+		PTSD-		Control				
Test	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*(2,37)	*p*	*Partial* η^2
CWIT CW	52.50	10.95	45.71	11.62	47.40	10.61	.80	.46	.04
CWIT IS	66.17	10.93	54.79	9.96	54.70	8.48	3.79	.032*	.17
BRIEF inhibition	16.33	1.37	12.57	2.98	11.70	2.36	7.97	.001*	.30
BRIEF total	53.67	6.65	44.79	9.58	42.20	7.93	4.31	.021*	.19
RRS	49.45	14.21	43.10	11.81	36.20	5.35	5.20	.010*	.22

Note. Scores on CWIT are measured in seconds. High scores indicate longer time needed to perform the test. High scores on BRIEF indicate more reported difficulties with inhibition or general EFs. High scores on RRS indicate higher levels of reported rumination.
* The result is significant at the 0.05 level

Follow-up MANOVA with groups determined by PTSS status. In this MANOVA, PTSS status was used as the independent variable, and the same four dependent variables were used: CWIT CW, CWIT IS, BRIEF inhibition and RRS. There was a statistically significant difference between the groups on the dependent variables, F (4, 34) = 4.57; Wilks´Lambda =.42; partial eta squared = .35. When the results for the dependent variables were considered separately, there was a significant difference on all the variables: CWIT CW, CWIT IS, BRIEF inhibition, and RRS. An investigation of the mean scores indicated that PTSS+ reported higher difficulties with CWIT CW, CWIT IS, BRIEF inhibition, and RRS, than PTSD- and the control group. When using BRIEF total instead of BRIEF inhibition, no significant differences were found (see table 4 for results on MANOVA using groups determined by PTSS status).

Table 4.

Means and Standard deviations of CWIT conditions CW and IS, BRIEF inhibition, BRIEF total, and RRS scores when using the PTSS+, PTSS-, and control grouping.

	PTSS+		PTSS-		Control				
Test	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*(2,37)	*p*	*Partial* η^2
CWIT CW	53.23	8.58	37.57	9.57	47.40	10.16	5.79	.007*	.24
CWIT IS	62.46	10.00	50.29	9.64	54.70	8.48	4.72	.015*	.20
BRIEF inhibition	14.85	2.94	11.57	2.30	11.70	2.36	6.80	.003*	.27
BRIEF total	49.38	9.00	43.86	10.29	42.20	7.93	2.74	.078	.13
RRS	46.63	12.92	41.99	12.19	36.19	5.35	4.70	.015*	.20

Note. Scores on CWIT are measured in seconds. High scores indicate longer time needed to perform the test. High scores on BRIEF indicate more reported difficulties with inhibition or general EFs. High scores on RRS indicate higher levels of reported rumination.
* The result is significant at the 0.05 level

Follow-up ANOVA using PTSD status as independent variable.

Neuropsychological measures of inhibition. A univariate between groups comparison ANOVA with the subgroups PTSD+ and PTSD- in addition to the trauma unexposed control group was carried out. First, the groups were measured on the CWIT test, condition IS, with results indicating significant differences. Post hoc comparison using the Tukey HSD test revealed significant differences between the PTSD+ group and the PTSD- group as well as between the PTSD+ group and the control group. In both cases, the PTSD+ group showed the poorest performance.

Self-report measures of inhibition and EF. As with objective measures of inhibition and EF, univariate ANOVAs were carried out using group membership (PTSD+, PTSD-, and control) as the independent variable, and BRIEF total and BRIEF inhibition as the dependent variables. Results illustrated significant differences on both measures. Post hoc Tukey HSD on BRIEF inhibition indicated that the PTSD+ group was significantly different from the PTSD- group and the control group. The PTSD+ group reported greater difficulties than both of the two other groups. On BRIEF total, post hoc Tukey HSD revealed significant differences between the PTSD+ group and the control group, with greater difficulties reported by the PTSD+ group.

Self-report measures of rumination. When using RRS as the dependent variable, significant differences were found between the groups. The Tukey HSD post-hoc test illustrated that the significant differences were between the means of the PTSD+ group and the control group, but no significant differences in the means were found between the PTSD+ and the PTSD- group, nor the PTSD- and the control group. There was a tendency for the PTSD+ group to report most difficulties with rumination, followed by the PTSD- group and finally the non-exposed group, respectively (see table 5 for results on ANOVA using groups determined by PTSD status).

Table 5.

Means and Standard deviations of CWIT conditions IS, BRIEF inhibition, BRIEF total, and RRS scores when using the PTSD+, PTSD-, and control grouping.

	PTSD+		PTSD-		Control				
Test	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*(2,37)	*p*	*Pairwise group differences*[a]
CWIT IS	66.17	10.93	54.79	9.94	54.70	8.48	3.79	.032	1 > 2, 3
BRIEF inhibition	16.33	1.37	12.57	2.98	11.70	2.36	7.97	.001	1 > 2, 3
BRIEF total	53.67	6.65	44.79	9.58	42.20	7.93	4.31	.021	1 > 3
RRS	49.45	14.21	43.10	11.81	36.19	5.35	5.20	0.2	1 > 3

Note. Scores on CWIT are measured in seconds. High scores indicate longer time needed to perform the test. High scores on BRIEF indicate more reported difficulties with inhibition or EFs. High scores on RRS indicate higher levels of reported rumination.
[a]Comparisons for all pairs using the post hoc test Tukey, $p<.05$

Follow-up ANOVA using PTSS status as the independent variable.

Neuropsychological measures of inhibition. When applying the same procedure to the PTSS+ and PTSS- subgroups on the CWIT conditions CW and IS, significant differences were found in both conditions. Post hoc comparisons using Tukey HSD revealed that the mean score of the PTSS+ group was significantly different from the mean score of the PTSS- group on both the CW and the IS variables. On both variables, the PTSS- group performed better on the EF tasks than the PTSS+ group. The control group did not differ significantly from any of the trauma exposed groups on the variables in question.

Self-report measures of inhibition. When using BRIEF inhibition as dependent variable, ANOVA revealed significant differences between the groups. The Tukey HSD post-hoc test illustrated that the differences were between the PTSS+ group and the control group, as well as between the PTSS+ and the PTSS- groups. The PTSS+ group reported significantly higher difficulties with inhibition than both of the remaining groups.

Self-report measures of rumination. Significant differences were found when using RRS as the dependent variable. The Tukey HSD post hoc indicated significant differences between the PTSS+ group and the control group. No significant differences were found in the remaining group combinations. There was a tendency for the PTSS+ group to report most difficulties with rumination, followed by the PTSS- group and finally the control group, respectively (see table 6 for results on ANOVA using groups determined by PTSS status).

Table 6.

Means and Standard deviations of CWIT conditions CW and IS, BRIEF inhibition, and RRS scores when using the PTSS+, PTSS-, and control grouping.

	PTSS+		PTSS-		Control				
Test	*M*	*SD*	*M*	*SD*	*M*	*SD*	*F*(2,37)	*p*	*Pairwise group differences*[a]
CWIT CW	53.23	8.58	37.57	9.57	47.40	10.61	5.79	.007	1 > 2
CWIT IS	62.46	10.00	50.29	9.64	54.70	8.48	4.72	.015	1 > 2
BRIEF inhibition	14.85	2.94	11.57	2.30	11.70	2.36	6.80	.003	1 > 2, 3
RRS	46.63	12.92	42.00	12.19	36.19	5.35	4.70	.015	1 > 3

Note. Scores on CWIT are measured in seconds. High scores indicate longer time needed to perform the test. High scores on BRIEF indicate more reported difficulties with inhibition. High scores on RRS indicate higher levels of reported rumination.
[a]Comparisons for all pairs using the post hoc test Tukey, $p<.05$

Correlation analyses. The correlation analysis indicated a positive correlation between BRIEF total and RRS. A correlation matrix summarizes the results (Table 7).

Table 7.

Correlations all subjects

Variable	CWIT CW	CWIT IS	BRIEF inhibition	BRIEF total	RRS
CWIT CW	-	.646**	-.002	.087	.102
CWIT IS	.646**	-	.075	.029	.159
BRIEF inhibition	-.002	.075	-	.828**	.271
BRIEF total	.087	.029	.828**	-	.468**
RRS	.102	.159	.271	.468**	-

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

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

Discussion

There are two important findings in the present study. The first main finding is that impaired inhibition was more related to PTSD symptomatology than to trauma exposure per se. Self-reports revealed that trauma exposed subjects experience greater problems with inhibition than controls, but such group differences were not seen on neuropsychological tests. Subgroup analyses revealed that the greatest inhibitory dysfunction was seen in the groups with the highest symptom load (PTSD+, PTSS+). Furthermore, the least problems were seen for the trauma exposed group without symptoms (PTSS-), which also demonstrated better inhibitory skills than the control group. These follow-up findings were consistent across neuropsychological and self-report measures. The second main finding was that rumination was related to trauma exposure. Subgroup analyses indicated that rumination tendencies were increased by trauma exposure and that they were further exacerbated by PTSD symptomatology. As such, rumination tendencies seem to be less dependent on PTSD symptomatology than inhibition. There was a positive correlation between rumination and self-reported problems with EF, but beyond this no consistent associations were found between rumination and inhibition. Several inferences may be drawn from these findings.

The results of the main analyses revealed that trauma exposed adolescents experience significantly more problems with inhibition than controls according to self-report measures. These differences were not reflected in neuropsychological measures of inhibition, which

means that the predictions were only partly supported. Consistent results on inhibition were mainly detectable when the trauma exposed group was subdivided based on PTSD and PTSS status in follow-up analyses. Self-report results from the follow-up analyses reflected the main tendencies obtained from the neuropsychological measures.

In line with predictions, higher levels of posttraumatic symptomatology (PTSD+, PTSS+) were associated with the poorest performance on neuropsychological measures of inhibition and the most self-reported problems with inhibition. This finding was consistent across subgroup analyses. In addition, results from each of the two sub-group divisions added further nuance to the total picture. Firstly, the trauma exposed adolescents with diagnosed PTSD (PTSD+) performed significantly worse on cognitive measures of inhibition/shifting than other trauma exposed adolescents (PTSD-) and controls, whose group results did not differ significantly from each other. This shows that trauma exposed adolescents without PTSD were more similar to controls than to trauma exposed adolescents with PTSD, which supports the finding that inhibitory dysfunctions are primarily associated with PTSD. Secondly, the trauma exposed adolescents with diagnosed or subclinical PTSD (PTSS+) performed significantly worse than the trauma exposed adolescents without symptoms (PTSS-) on both neuropsychological measures of inhibition. The control group was intermediate the two trauma exposed groups (PTSS+, PTSS-), without being significantly different from any of them. In addition to supporting the association between inhibitory dysfunctions and PTSD symptomatology, these results support the minimal association between inhibitory dysfunctions and trauma exposure per se. Although the symptom-free trauma exposed group (PTSS-) did not perform significantly better than the control group, differences were clearly observable.

The main analyses of rumination revealed that the trauma exposed group reported significantly higher levels of rumination than the control group. This was consistent with

expectations. Follow-up analyses of subgroups indicated that the groups with the greatest symptom load (PTSD+, PTSS+) reported significantly higher levels of rumination than the control group, which was also in line with expectations. The other trauma exposed groups (PTSD-, PTSS-) were intermediate without being significantly different from any of the extremes. The group trends indicate that trauma exposure is associated with increased rumination, and that PTSD symptomatology is associated with additional increases in rumination.

Results of the correlation analyses did not indicate any associations between inhibition and rumination. Besides the self-reported measure of general EF, no other measures reflecting inhibition correlated significantly with measures of rumination. The positive correlation between self-reported EF and rumination indicate that subjects who experience greater difficulties with EF tend to ruminate more. However, conclusions cannot be made about causality because the direction of the relationship and the potential impact of confounding variables on the observed relationship are not controlled for.

Taken together, the results from the main analyses and follow-up analyses of inhibition and rumination support the existence of trauma-related impairments in inhibition and increases in rumination, but suggest that the impairments in inhibition are more associated with PTSD than with trauma exposure per se. However, inhibition and rumination did not appear to be related in this sample. The findings are partly consistent and partly inconsistent with indications from previous research, and may be understood in several ways.

The inconsistent results on neuropsychological and self-report measures of inhibition in trauma exposed adolescents and control may be interpreted in several ways. One possible explanation is that trauma exposed individuals feel impaired although their experienced problems do not have a neuropsychological basis (Melinder et al., 2015). This is consistent with findings and conclusions by Melinder and colleagues (2015) in their study of the same

trauma exposed population. They suggest that this is an important message to convey to affected subjects because it indicates good prognosis for recovery. Alternatively, neuropsychological tests may be poorly suited to detect existing inhibitory dysfunctions in trauma exposed adolescents. Inhibitory difficulties that are experienced and expressed in daily life may not necessarily be reflected in the standardized and controlled environment that characterizes a test situation. This is consistent with principles of ecological validity, which suggest that neuropsychological test results do not necessarily generalize to real world settings (Barkley, 2012; Burgess et al., 1998). Either way, the results support the supplementary use of self-report measures in future neuropsychological trauma research to investigate this matter more closely.

The finding of trauma-related impairments in inhibition in adolescents is consistent with previous research on EF in adults (Aupperle et al., 2012; Polak et al., 2012) and children and adolescents (Dalgleish et al., 2005; Turley & Obrzut, 2012). Regardless of whether inhibition is considered a sub-function of a diverse EF or a construct reflecting unitary EF, the findings support the importance of inhibition in understanding the effects of trauma. This contrasts the results of Melinder et al. (2015), whose study is also based on survivors of the terror attack at Utøya. Their study showed that PTSD symptoms were not associated with inhibition, but with shifting. However, Melinder and colleagues tested their subjects on the Stop Signal Task, which is a measure of behavioral inhibition. They suggested that the Stroop test might be better suited to detect differences in cognitive inhibition, which may explain why associations between PTSD symptomatology and inhibition were found in the present study.

The current study is not the first to suggest that trauma-related impairments in inhibition are more strongly related to PTSD symptomatology than to trauma exposure per se. Similar inferences have been discussed by other researchers who have studied EF in

trauma exposed children and adolescents (De Bellis et al., 2009; Leskin & White, 2007; Schoeman et al., 2009) and adults (Olff et al., 2014; Polak et al., 2012). Given this association, it still remains unclear whether and to what extent inhibitory dysfunction is primary or secondary to development of PTSD symptomatology. One possibility is that inhibitory dysfunctions preexist in some individuals and make them more prone to develop PTSD in the aftermath of a traumatic event. However, pre-trauma data on cognitive functioning are rarely available, which complicates further investigations of this matter. This is usually an inevitable methodological limitation in trauma research, as it would be unethical to inflict trauma upon individuals or gather information from groups prone to future trauma in order to improve research findings. Another potential explanation is that inhibitory dysfunctions arise as a consequence of PTSD symptoms because symptoms demand cognitive resources at the expense of important EFs such as inhibition. Both of the suggested interpretations have been mentioned in previous research on EF in trauma exposed children and adolescents (Schoeman et al., 2009; Twamley et al., 2004), and support for one does not necessarily rule out the other.

The lack of findings of consistent impairments in inhibition related to trauma exposure itself can be understood in several ways. One possible explanation is that the trauma exposed group who did not show signs of PTSD represent a pre-trauma cognitively resilient group whose cognitive resources protected against the appearance or unfolding of PTSD symptoms after experiencing a traumatic event (Schoeman et al., 2009). This is in line with suggestions made by other authors (Aupperle et al., 2012). However, the possibility of improved cognitive functioning related to posttraumatic growth (Bostock et al., 2009) may also be relevant to consider here. Indirectly, the absence of an observed negative effect of trauma exposure alone on inhibition strengthens the suggestion that inhibitory dysfunction is more strongly related to PTSD. However, the existence of an association between mere

trauma exposure and inhibitory dysfunction should not be discarded based on the current study, as previous studies of EF in trauma exposed children and adolescents have yielded opposite results (Barrera et al., 2013; DePrince et al., 2009).

No significant correlation was found between rumination and pure inhibition measures. However, finding correlations of rumination and experienced EF may somehow be in accordance with other studies revealing associations between inhibition and rumination

It is unknown why there was a lack of finding of an association between inhibition (Davis & Nolen-Hoeksema, 2000; Joormann, 2006; Philippot & Brutoux, 2008; Whitmer & Gotlib, 2013) as inhibition is considered a core function of EF (Carrion et al., 2008). Results may be considered as neither supporting nor falsifying assumptions of a correlative relationship between inhibition and rumination. Finding that trauma exposed participants tend to ruminate more, and that ruminative patterns may be associated with severity PTSD symptom load are consistent with previous research revealing associations between PTSD and rumination (Ehring et al., 2008; Elwood et al., 2009; Roley et al., 2015). Uncovering associations between rumination and mere trauma exposure may be of particular interest in a developmental context. Increased rumination elicited by mere trauma exposure may pose greater challenges for trauma inflicted adolescents, as inhibitory abilities are considered immature in adolescence and thereby set youths in a vulnerable position to cope with increases in rumination. However, findings and interpretations of associations between rumination, inhibition and PTSD are considered preliminary indications in a nearly non-existing field of research.

Factors that are outside the scope of the present study may also contribute to the observed associations in the present study, and a number of issues arise when attempting to compare the findings to those of previous studies. Although most studies appear to find some association between trauma and EF in children and adolescents, a closer look at the results of

the separate studies reveals a range of apparent inconsistencies among them (e.g., Leskin & White, 2007; Schoeman et al., 2009; Twamley et al., 2004). Researchers who study EF in trauma exposed individuals have approached the topic by extracting various sub-functions and using different measurements, both of which are likely to affect the results. Moreover, great variation can exist in the trauma histories of participants within and across studies, including type of trauma and time after trauma exposure. Samples considered to represent children or adolescents may also be composed of participants of various ages. Moreover, researchers deal with the issue of comorbidity in different ways. As other psychiatric disorders than PTSD are also known to be associated with executive and inhibitory dysfunctions (e.g., depression, see Davis & Nolen-Hoeksema, 2000; Whitmer & Banich, 2007; Whitmer & Gotlib, 2012), presence of comorbid disorders not controlled for can confound the results and potentially lead to misattribution of findings to trauma exposure or PTSD (Leskin & White, 2007; Olff et al., 2014). The need to address comorbidity is emphasized by knowledge of PTSD as one of the psychiatric disorders most frequently associated with comorbidity (Breslau et al., 1991). However, this is a complex matter with many issues involved related to methodology and generalizability. Excluding subjects with PTSD and comorbid disorders would also challenge the generalizability of findings, as pure PTSD samples may be less representative of the real world population of PTSD patients (Leskin & White, 2007; Samuelson et al., 2010). All of these factors may influence the results and further lead to inconsistencies across studies. This must be taken into consideration in interpretation of the current findings as well.

Discussing the findings on inhibition also requires consideration of the time of measurement in light of knowledge about maturation of PCF and acquisition of EF. Assuming that the present sample is not fully developed in these areas, impairments could potentially be more or less likely to show on neuropsychological tests of this group as

compared to other age groups. The transitional phase and its related plasticity may protect against the negative effects of trauma on EF that have been observed in children (Dalgleish et al., 2005; Turley & Obrzut, 2012) and adults (Aupperle et al., 2012; Polak et al., 2012). Alternatively, it may conceal negative effects of trauma that will become evident over time as EFs mature. This highlights the necessity of studying adolescents over time and in relation to other age groups in order to understand whether the long-term effects of trauma are comparable or different in any way.

Strengths and Limitations

The present study has several limitations and strengths that should be considered in interpretation of the results. The small sample raised the issue of a possible low power, and had to be taken into consideration. This is a common limitation of psychological research, and it is often controlled for by adjusting the alpha level up. This was not done in the present study, in order to control for Type 1 error. The attempt to control for Type 1 error also increased the possibility for a Type 2 error. A strength in not adjusting the alpha level up is that significant results are difficult to obtain from small samples, and where there were findings were significant in this study the effect size was also large. In addition, the alpha level of $p > .05$ was not Bonferroni adjusted down in the MANOVA to further control for a possible Type 2 error.

Several characteristics of the sample limit the generalizability of the findings. Primary concerns regard the relatively small sample size. The main analyses were conducted with only 20 participants in each group, and follow-up analyses were based on even smaller subgroups. Furthermore, the trauma exposed group consisted of 20 out of the 495 registered survivors of the Utøya terror (Hafstad et al., 2014), which corresponds to roughly 4% of the total group. This relatively small proportion may not be representative of the remaining survivors.

Other aspects further complicate the representativeness of the trauma exposed sample for Utøya survivors in general. More specifically, it is plausible that the current sample is a relatively well-functioning and resourceful group of survivors. There are several reasons for this assumption. First of all, the survivors with the most severe injuries may be less represented in the sample because hospitalization and treatment programs make participation inconvenient or impossible. Second, several subjects were ruled out during the recruitment process because of fulfilling exclusion criteria that can possibly have involved poorer psychological health. Third, those who received the invitation and chose to participate may differ from those who refrained. They may suffer fewer aftereffects or cope better with difficulties and as a result have more capacity for engagement in testing that will demand them to confront trauma-related topics. Furthermore, they may be more informed about the purposes and benefits of research and find it more rewarding to contribute to the "greater good" of science. This effect may have been enhanced by the inclusion of two survivors who learned about the research project through other sources than direct invitation, and the preparatory screening out of participants who were hard to stay in touch with. However, it must also be considered that survivors with greater difficulties may have been more motivated to participate because of the opportunity to convey their experience. Finally, the trauma exposed sample was recruited from two counties in the South-West of Norway and is therefore not geographically representative. District-based follow-up interventions were implemented in the aftermath of the disaster at Utøya (Dyb & Glad, 2013). The extent to which these efforts have been successful may have varied across municipalities, contributing to geographic variations in the survivors´ adjustment.

The fact that nationwide efforts were initiated for the survivors of the Utøya terror may have served a protective purpose for this particular trauma exposed group (Dyb, Jensen, Nygaard et al., 2014). The social support encouraged by media coverage combined with the

collective nature of the trauma may have protected the survivors from certain negative effects that are commonly associated with other traumas, such as taboo and self-blame. The Utøya survivors may differ from other trauma exposed individuals in other aspects as well. All of the victims were politically active, which may be associated with personality characteristics such as conscientiousness, societal responsibility and motivation. Accumulation of such characteristics may challenge the generalizability of findings of this group to other trauma exposed populations.

The control group also consisted of politically active adolescents. This was a necessity for comparative purposes, although it may compromise the representativeness of adolescents in general. This may have been reinforced by the convenient sampling procedure with its inherent self-selection bias. Bias in the control group may also come from exclusion of subjects with ongoing or previous psychiatric illness. Adolescents are assumed to be at increased risk of developing psychological disorders (Schoeman et al., 2009), and screening these out of the control group may have contributed to a relatively well-adjusted sample that may not represent the general population adolescents. However, this was a necessary precaution, as psychopathology among control participants could have affected test results and potentially conceal trauma-related effects.

Certain variables that were not controlled for in the present study may have affected the results. First of all, the trauma exposed group and control group were matched on gender distribution, but no further control regarding gender was undertaken on subgroups in the analyses. This could potentially have influenced the findings as PTSD symptoms are found to be more prevalent in trauma exposed females than in males across age groups (Garvranidou & Rosner, 2003; Ullman & Filipas, 2005). Moreover, Polak and colleagues (2012) postulate that trauma-related differences in EF are greater between males with and without PTSD than between corresponding groups of females. Second, history of trauma exposure was not

controlled for among participants. Any history of trauma exposure among control participants could reduce group differences and lead to risk of undermining the effects of trauma in survivors of the Utøya terror. Lack of screening for previous trauma exposure among the Utøya survivors prevents us from controlling for cumulative effects of trauma, which has been associated with increased rates of PTSD (Suliman et al., 2009). Third, depression was not controlled for in the present study. Depression is known to be common in trauma exposed individuals and frequently comorbid with PTSD (Galea et al., 2002; Shalev, 1998). Moreover, research has described inhibitory dysfunction and rumination as common characteristics of depression (Susan Nolen-Hoeksema, 2000; Schmid & Hammar, 2013). Controlling for depression as a potential confounding variable would therefore have been relevant to strengthen or dismiss the observed associations between trauma exposure, PTSD, inhibition and rumination.

Potential limitations of our study also concern the understanding of adolescence as a unique developmental phase that can and should be separately studied. No clear consensus exists about the defining boundaries of adolescence, and the question can be approached from several perspectives on development (Steinberg, 2011). It therefore remains ambiguous to what extent the results should be considered to reflect adolescence as a unitary developmental phase, a sub-phase of adolescence, or rather one aspect of adolescent development (i.e., maturation of PFC and EF). Ultimately, the relative lack of research on trauma exposed adolescents provides limited context for comparing and interpreting the findings.

Despite limitations, the current study is unique in many ways. In all its tragedy, the survivors of the Utøya massacre represent a rare opportunity to study the effects of trauma exposure in adolescence. There are few existing studies on adolescents who have been exposed to the same type of traumatic event and this has been requested by other researchers (e.g., Melinder et al., 2015; Scrimin et al., 2006). As opposed to samples of subjects who

have been exposed to individual complex traumas such as sexual abuse, the current sample is unique in that confounding relational factors may be less of a concern and shared aspects of the traumatic experience may be more prominent.

Clinical Implications

The main finding of an association between PTSD and impairments in inhibition has important clinical implications. One interpretation of this finding is that inhibitory function is a risk factor for development of PTSD after trauma exposure (e.g., Aupperle et al., 2012; Twamley et al., 2004). If this is the case, all trauma exposed adolescents should be assessed for problems with inhibition to identify vulnerable individuals who should be offered preventive interventions in order to counteract development of PTSD. Another interpretation of the finding is that impairments in inhibition are elicited or exacerbated by PTSD symptoms (e.g., Johnsen et al., 2013). This would mean that treatment for PTSD should include interventions that target inhibition directly. If inhibitory dysfunction and PTSD symptoms reinforce each other, it can be expected that improvements in inhibition would lead to subsequent reduction in PTSD symptoms.

The second main finding of an association between trauma exposure and rumination may have particular clinical relevance in adolescents. The consequences of rumination may be especially severe for adolescents with immature inhibitory skills, as rumination will be allowed to flourish uninhibitedly and stronger inhibitory capacities will constantly be required to discontinue the ruminating tendency. This potentially vicious cycle emphasizes the need to assess rumination in trauma exposed adolescents and offer appropriate interventions. Cognitive behavior therapy (CBT) has proved effective in reducing rumination (Roley et al., 2015). Reduced rumination through CBT may further reduce the blockade to efficient cognitive restructuring and facilitate maturation of adaptive cognitive functions in adolescents.

The current study highlights the clinical importance of targeting adolescents specifically in research on trauma. Developmental aspects of adolescence may not only make this group more prone to experience trauma (Viner et al., 2012), but may also make it particularly hard to inhibit the unfolding of PTSD symptoms. Moreover, trauma exposure and PTSD in adolescence may have broad ramifications for maturation of cold cognitive functions that are thought to be incomplete at this stage of life. It could be that impairments such as those observed in this cross-sectional study will readjust with time and treatment, and that development will again resume a normal course. However, the nature of adolescence implies a need to examine whether trauma exposure or PTSD may interfere with development by stagnating or slowing down the maturation of affected regulatory functions. Evening out the relationship between hot and cold cognitions is crucial for successful adjustment in many areas of adult life. Integration of research from this field with clinical applications is essential not only to relieve individual suffering, but also for the sake of societal expenditure.

Warrants for Future Research

Future research should use longitudinal designs to investigate effects of trauma exposure in adolescence. Studying trauma-exposed adolescents and comparable controls over time would enable within-group and between-groups comparisons of inhibition and other EFs. This could tell us more about if and how trauma in adolescence affects the developmental course of these regulatory functions. Another approach is to compare trauma-exposed adolescents to trauma-exposed children and adults over time would. This could increase the understanding of the risk versus protective aspects associated with trauma exposure in different developmental phases. Longitudinal studies are also relevant to examine whether trauma-related effects on inhibition and EF affect long-term adjustment in various domains of life.

Results of the current study highlight the need for further investigation of the relationship between subjective and objective indications of dysfunction in trauma exposed adolescents. Furthermore, the relevance of EF and PFC in adolescent development makes it particularly interesting to supplement neuropsychological research findings with results from neuroimaging studies. Although the current study solely concerns the neuropsychological findings, fMRI data on the current sample exist and may be relevant for later investigations.

Interesting results could also be achieved in future research by examining associations between specific subsets of the variables used in the current study. For instance, PTSD could be assessed in terms of degree of total symptoms or specific symptom clusters and examined in relation to inhibitory function (Melinder et al., 2015). Examining degree of trauma exposure rather than presence or absence of trauma exposure could yield more nuanced information about the association between trauma exposure and EF. Using larger samples of adolescents that can be subdivided based on age could shed light on subtle developmental differences in adolescence.

Based on the clinical implications discussed in the current study it is suggested that effect studies are carried out with the purpose of increasing our understanding of inhibitory dysfunctions as causes or consequences of PTSD symptomatology. If effect studies were conducted on the procedures to screen for inhibitory dysfunctions in trauma exposed adolescents and corresponding preventive efforts, it could be easier to predict and prevent development of PTSD. Furthermore, trauma exposed adolescents with PTSD symptomatology should be offered cognitive interventions to improve their inhibitory skills and these should be assessed for their effectiveness in reducing PTSD symptoms.

If trauma utløser rumination in adolescents, and they have dårligere inhibition, this should be further researched.

Conclusion

The present study examined long-term effects of trauma exposure on inhibition and rumination in a sample of adolescent survivors of the terror attack at Utøya. Results of the current study indicated that impaired inhibition appeared to be more related to PTSD symptomatology than to trauma exposure per se. Although mere trauma exposure was associated with an experience of greater problems with inhibition, this was not reflected in neuropsychological tests. Furthermore, rumination was more pronounced among trauma exposed adolescents than controls, and appeared to be less dependent on PTSD symptomatology than impairments in inhibition. Rumination was associated with experienced difficulties with EF, but not inhibition specifically. In line with previous research on various age groups, the findings suggest that inhibitory dysfunctions may be a risk factor for development of PTSD in adolescent survivors of trauma, a consequence of PTSD symptomatology, or both. This implies that it may be relevant for clinicians to assess for inhibitory dysfunctions in trauma exposed adolescents and implement interventions targeting inhibition in order to prevent and reduce PTSD symptomatology. Findings of rumination tendencies among trauma exposed adolescents should be investigated further because of their developmental position. Rumination triggered by trauma exposure may add on adolescent-related shortcomings in inhibitory functioning, and thereby pose a greater challenge for those affected. These results suggest further investigations of the interaction between inhibition and rumination in the understanding of trauma-related suffering. Inferences about the effects of trauma exposure on developmental paths cannot be made due to the cross-sectional design of the present study, and longitudinal examinations are warranted in order to investigate the possible impact in a broader developmental context. However, the current study reveals significant long-term effects of trauma exposure on EF in adolescents, thus adding important contributions to this nearly unexplored field of research.

References

Alvarez, J. A., & Emory, E. (2006). Executive function and the frontal lobes: A meta-analytic review. *Neuropsychology Review, 16*, 17-42. doi:10.1007/s11065-006-9002-x

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders: DSM-V* (5th ed.). Washington, D.C.: American Psychiatric Association.

Anderson, P. (2002). Assessment and development of executive function (EF) during childhood. *Child Neuropsychology, 8*, 71-82. doi:10.1076/chin.8.2.71.8724

Arnett, J. (1992). Reckless behavior in Adolescence: A developmental perspective. *Developmental Review, 12*, 339-373. doi:10.1016/0273-2297(92)90013-r

Arnold, J. L., Örtenwall, P., Birnbaum, M. L., Sundnes, K. O., Aggrawal, A., Arantharaman, V., . . . Tsai, M.-C. (2003). A proposed universal medical and public health definition of terrorism. *Prehospital and Disaster Medicine, 18*, 47-52. doi:10.1017/S1049023X00000753

Arnsten, A. F. T. (1998). Catecholamine modulation of prefrontal cortical cognitive function. *Trends in Cognitive Sciences, 2*, 436-447. doi:10.1016/S1364-6613(98)01240-6

Atkins, S. M., Bunting, M. F., Bolger, D. J., & Dougherty, M. R. (2012). Training the adolescent brain: Neural plasticity and the acquisition of cognitive abilities. In V. F. Reyna, S. B. Chapman, M. R. Dougherty & J. Confrey (Eds.), *The adolescent brain: Learning, reasoning, and decision making* (pp. 211-241). Washington, DC: American Psychological Association.

Augusti, E.-M., & Melinder, A. (2013). Maltreatment is associated with specific impairments in executive functions: A pilot study. *Journal of Traumatic Stress, 26*, 780-783. doi:10.1002/jts.21860

Aupperle, R. L., Melrose, A. J., Stein, M. B., & Paulus, M. P. (2012). Executive function and PTSD: Disengaging from trauma. *Neuropharmacology, 62*(2)(, 686-694. doi:10.1016/j.neuropharm.2011.02.008

Axelrod, B. N. (2002). Validity of the Wechsler Abbreviated Scale of Intelligence and other very short forms of estimating intellectual functioning. *Assessment, 9*, 17-23. doi:10.1177/1073191102009001003

Baddeley, A. (1996). Exploring the central executive. *The Quarterly Journal of Experimental Psychology Section A, 49*, 5-28. doi:10.1080/713755608

Barkley, R. A. (2012). *Executive functions: What they are, how they work, and why they evolved.* New York, NY: The Guilford Press.

Barrera, M., Calderón, L., & Bell, V. (2013). The cognitive impact of sexual abuse and PTSD in children: A neuropsychological study. *Journal of Child Sexual Abuse, 22*, 625-638. doi:10.1080/10538712.2013.811141

Beers, S. R., & De Bellis, M. D. (2002). Neuropsychological function in children with maltreatment-related posttraumatic stress disorder. *The American Journal of Psychiatry, 159*, 483-486. doi:10.1176/appi.ajp.159.3.483

Bixler, R. H. (1992). Why littermates don't: The avoidance of inbreeding depression. *Annual Review of Sex Research, 3*(1), 291-328. doi:10.1080/10532528.1992.10559882

Blackmore, S.-J., & Robbins, T. W. (2012). Decision-making in the adolescent brain. *Nature Neuroscience, 15*, 1184-1191. doi:10.1038/nn.3177

Bleich, A., Koslowsky, M., Dolev, A., & Lerer, B. (1997). Post-traumatic stress disorder and depression. An analysis of comorbidity. *The British Journal of Psychiatry, 170*, 497-482. doi:10.1192/bjp.170.5.479

Bonanno, G. A. (2004). Loss, trauma, and human resilience: Have we underestimated the human capacity to thrive after extremely aversive events? *American Psychologist, 59*, 20-28. doi:10.1037/0003-066X.59.1.20

Bonanno, G. A., Galea, S., Bucciarelli, A., & Vlahov, D. (2006). Psychological resilience after disaster: New York City in the aftermath of the september 11th terrorist attack. *Psychological Science, 17*, 181-186. doi:10.1111/j.1467-9280.2006.01682.x

Bostock, L., Sheikh, A. I., & Barton, S. (2009). Posttraumatic growth and optimism in health-related trauma: A systematic review. *Journal of Clinical Psychology in Medical Settings, 16*, 281-296. doi:10.1007/s10880-009-9175-6

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. doi:10.1001/archpsyc.1991.01810270028003

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*, 748-766. doi:10.1037//0022-006x.68.5.748

Bunge, S. A., & Wright, S. B. (2007). Neurodevelopmental changes in working memory and cognitive control. *Cognitive Neuroscience, 17*, 243-250. doi:10.1016/j.conb.2007.02.005

Burgess, P. A., Alderman, N., Evans, J., Emslie, H., & Wilson, B. A. (1998). The ecological validity of tests of executive function. *Journal of the International Neuropsychological Society, 4*, 547-558.

Bøen, B. W., & Matre, L. (2014). *Long-Term Psychiatric Symptoms Among Survivors of the 2011 Terrorist Attack at Utøya: A Descriptive Study.* (Unpublished master's thesis), University of Bergen, Norway.

Carrion, V. G., Garrett, A., Menon, V., Weems, C. F., & Reiss, A. L. (2008). Posttraumatic stress symptoms and brain function during a response-inhibition task: An fMRI study in youth. *Depression and Anxiety, 25*, 514-526.

Carrion, V. G., Weems, C. F., Eliez, S., Patwardhan, A., Brown, W., Ray, R. D., & Reiss, A. L. (2001). Attenuation of frontal asymmetry in pediatric posttraumatic stress disorder. *Biological Psychiatry, 50*, 943-951. doi:http://dx.doi.org/10.1016/S0006-3223(01)01218-5

Carrion, V. G., & Wong, S. S. (2012). Can traumatic stress alter the brain? Understanding the implications of early trauma on brain development and learning. *Journal of Adolescent Health, 51*(Suppl. 2), S23-S28. doi:10.1016/j.jadohealth.2012.04.010

Cohen, J. A., Perel, J. M., DeBellis, M. D., Friedman, M. J., & Putnam, F. W. (2002). Treating traumatized children: Clinical implications of the psychobiology of posttraumatic stress disorder. *Trauma, Violence, & Abuse, 3*, 91-108. doi:10.1177/15248380020032001

Compas, B. E. (2004). Processes of risk and resilience during adolescence: Linking context and individuals. In R. M. Lerner & L. Steinberg (Eds.), *Handbook of adolescent psychology, Second Edition* (pp. 263-298). Hoboken, NJ: John Wiley & Sons Inc.

Dalgleish, T., Meiser-Stedman, R., & Smith, P. (2005). Cognitive aspects of posttraumatic stress reactions and their treatment in children and adolescents: An empirical review and some recommendations. *Behavioural and Cognitive Psychotherapy, 33*, 459-486. doi:10.1017/S1352465805002389

Davidson, M. C., Amso, D., Anderson, L. C., & Diamond, A. (2006). Development of cognitive control and executive functions from 4 to 13 years: Evidence from manipulations of memory, inhibition, and task switching. *Neuropsychologia, 44*, 2037-2078. doi:10.1016/j.neuropsychologia.2006.02.006

Davis, R. N., & Nolen-Hoeksema, S. (2000). Cognitive inflexibility among ruminators and nonruminators. *Cognitive Therapy and Research, 24*, 699-711. doi:10.1023/A:1005591412406

De Bellis, M. D., Hooper, S. R., Spratt, E. G., & Woolley, D. P. (2009). Neuropsychological findings in childhood neglect and their relationships to pediatric PTSD. *Journal of the International Neuropsychological Society, 15*, 868-878. doi:10.1017/S1355617709990464

De Bellis, M. D., Keshavan, M. S., Clark, D. B., Casey, B., Giedd, J. N., Boring, A. M., . . . Ryan, N. D. (1999). Developmental traumatology part II: Brain development. *Biological Psychiatry, 45*, 1271-1284. doi:10.1016/s0006-3223(99)00045-1

De Bellis, M. D., Woolley, D. P., & Hooper, S. R. (2013). Neuropsychological findings in pediatric maltreatment: Relationship of PTSD, dissociative symptoms, and abuse/neglect indices to neurocognitive outcomes. *Child Maltreatment, 18*, 171-183. doi:10.1177/1077559513497420

Delis, D. C., Kaplan, E., & Kramer, J. H. (2001). *D-KEFS examiners manual*. San Antonio, Texas: Psychological Corporation.

DePrince, A. P., Weinzierl, K. M., & Combs, M. D. (2009). Executive function performance and trauma exposure in a community sample of children. *Child Abuse & Neglect, 33*, 353-361. doi:10.1016/j.chiabu.2008.08.002

Dyb, G., & Glad, K. A. (2013). *"Opplevelser og reaksjoner hos de som var på Utøya 22.juli 2011". En oppsummering av andre intervjurunde*. Retrieved from http://www.nkvts.no/fu/Sider/ProsjektInfo.aspx?prosjektid=1333

Dyb, G., Jensen, T. K., Glad, K. A., Nygaard, E., & Thoresen, S. (2014). Early outreach to survivors of the shootings in Norway on the 22nd of July 2011. *European Journal of Psychotraumatology, 5*. doi:10.3402/ejpt.v5.23523

Dyb, G., Jensen, T. K., Nygaard, E., Ekeberg, O., Diseth, T. H., Wentzel-Larsen, T., & Thoresen, S. (2014). Post-traumatic stress reactions in survivors of the 2011 massacre on Utøya Island, Norway. *The British Journal of Psychiatry, 204*, 361-367. doi:10.1192/bjp.bp.113.133157

Ehlers, A., & Clark, D. M. (2000). A cognitive model of posttraumatic stress disorder. *Behaviour Research and Therapy, 38*, 319-345. doi:10.1016/S0005-7967(99)00123-0

Ehlers, A., Mayou, R. A., & Bryant, B. (1998). Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. *Journal of Abnormal Psychology, 107*, 508-519. doi:10.1037/0021-843X.107.3.508

Ehring, T., Frank, S., & Ehlers, A. (2008). The role of rumination and reduced concreteness in the maintenance of posttraumatic stress disorder and depression following trauma. *Cognitive Therapy and Research, 32*, 488-506. doi:10.1007/s10608-006-9089-7

Elwood, L. S., Hahn, K. S., Olatunji, B. O., & Williams, N. L. (2009). Cognitive vulnerabilities to the development of PTSD: A review of four vulnerabilities and the proposal of an integrative vulnerability model. *Clinical Psychology Review, 29*, 87-100. doi:10.1016/j.cpr.2008.10.002

Francati, V., Vermetten, E., & Bremner, J. D. (2007). Functional neuroimaging studies in posttraumatic stress disorder: Review of current methods and findings. *Depression and Anxiety, 24*, 202-218. doi:10.1002/da.20208

Frans, Ö., Rimmö, P.-A., Åberg, L., & Fredrikson, M. (2005). Trauma exposure and post-traumatic stress disorder in the general population. *Acta Psychiatrica Scandinavica, 111*, 291–290. doi:10.1111/j.1600-0447.2004.00463.x

Galea, S., Ahern, J., Resnick, H., Kilpatrick, D., Bucuvalas, M., Gold, J., & Vlahov, D. (2002). Psychological sequalae of the september 11 terrorist attacks in New York

City. *The New England Journal of Medicine, 346*, 982-987. doi:10.1056/NEJMsa013404

Galea, S., Nandi, A., & Vlahov, D. (2005). The epidemiology of post-traumatic stress disorder after disasters. *Epidemiologic Reviews, 27*, 78-91. doi:10.1093/epirev/mxi003

Garvranidou, M., & Rosner, R. (2003). The weaker sex? Gender and post-traumatic stress disorder. *Depression and Anxiety, 17*, 130-139. doi:10.1002/da.10103

Giedd, J. N. (2008). The teen brain: Insights from neuroimaging. *Journal of Adolescent Health, 42*, 335-343. doi:10.1016/j.jadohealth.2008.01.007

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, F., Liu, H., Zijdenbos, A., . . . Rapoport, J. L. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience*(10), 861-863. doi:10.1038/13158

Gioia, G. A., Isquith, P. K., Guy, S. C., & Kenworthy, L. (2000). *Behavior rating inventory of executive function (BRIEF)*. Lutz, FL: Psychological Assessment Resources.

Goel, V., & Dolan, R. J. (2003). Reciprocal neural response within lateral and ventral medial prefrontal cortex during hot and cold reasoning. *NeuroImage, 20*, 2314-2321. doi:10.1016/j.neuroimage.2003.07.027

Gralton, E., Muchatuta, A., Morey-Canellas, J., & Lopez, C. D. (2008). Developmental traumatology: Its relevance to forensic adolescent settings. *The British Journal of Forensic Practice, 10*, 33-39. doi:10.1108/14636646200800012

Hafstad, G. S., Dyb, G., Jensen, T. K., Steinberg, A. M., & Pynoos, R. S. (2014). PTSD prevalence and symptom structure of DSM-5 criteria in adolescents and young adults surviving the 2011 shooting in Norway. *Journal of Affective Disorders, 169*, 40-46. doi:10.1016/j.jad.2014.06.055

Hall, P. A., & Marteau, T. M. (2014). Executive function in the context of chronic disease prevention: Theory, research and practice. *Preventive Medicine, 68*, 44-50. doi:10.1016/j.ypmed.2014.07.008

Hanson, J. L., Chung, M. K., Avants, B. B., Rudolph, K. D., Shirtcliff, E. A., Gee, J. C., . . . Pollak, S. D. (2012). Structural variations in prefrontal cortex mediate the relationship between early childhood stress and spatial working memory. *The Journal of Neuroscience, 32*, 7917-7925. doi:10.1523/JNEUROSCI.0307-12.2012

Hart, J., Jr., Kimbrell, T., Fauver, P., Cherry, B. J., Pitcock, J., Booe, L. Q., . . . Freeman, T. W. (2008). Cognitive dysfunctions associated with PTSD: Evidence from World War II prisoners of war. *The Journal of Neuropsychiatry and Clinical Neurosciences, 20*, 309-316. doi:10.1176/appi.neuropsych.20.3.309

Holman, E. A., Garfin, D. R., & Silver, R. C. (2014). Media's role in broadcasting acute stress following the Boston Marathon bombings. *Proceedings of the National Academy of Sciences, 111*, 93-98. doi:10.1073/pnas.1316265110

Holmeng, M. E., & Stavestrand, S. H. (2014). *Posttraumatic stress disorder and executive function in the developing brain - a systematic review.* (Unpublished master's thesis), University of Bergen, Norway.

Horner, M. D., & Hamner, M. B. (2002). Neurocognitive functioning in posttraumatic stress disorder. *Neuropsychology Review, 12*, 15-30. doi:10.1023/A:1015439106231

Insana, S. P., Banihashemi, L., Herringa, R. J., Kolko, D. J., & Germain, A. (2015). Childhood maltreatment is associated with altered frontolimbic neurobiological activity during wakefulness in adulthood. *Development and Psychopathology*, 1-14. doi:10.1017/s0954579415000589

Johnsen, G. E., Kanagaratnam, P., & Asbjornsen, A. E. (2013). Posttraumatisk stressforstyrrelse er forbundet med kognitive dysfunksjoner. [Post traumatic stress

disorder is associated with cognitive impairments]. *Tidsskrift for Norsk Psykologforening, 50*, 201-207.

Johnson, S. B., Blum, R. W., & Giedd, J. N. (2009). Adolescent maturity and the brain: The promise and pitfalls of neuroscience research in adolescent health policy. *Journal of Adolescent Health, 45*, 216-221. doi:10.1016/j.jadohealth.2009.05.016

Joormann, J. (2006). Differential effects of rumination and dysphoria on the inhibition of irrelevant emotional material: Evidence from a negative priming task. *Cognitive therapy and research, 30*, 149-160. doi:10.1007/s10608-006-9035-8

Kessler, R., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the national comorbidity survey. *Archives of General Psychiatry, 52*(12), 1048-1060. doi:10.1001/archpsyc.1995.03950240066012

Landsman, I. S. (2002). Crisis of meaning in trauma and loss. In J. Kauffman (Ed.), *Loss of the assumptive world: A theory of traumatic loss* (pp. 13-30). Retrieved from http://www.google.no/books?id=bfav34bNIPoC&dq=Loss+of+the+assumptive+world:+A+theory+of+traumatic+loss+(&lr=.

Lecruiber, Y., Sheenan, D. V., Weiller, E., Amorim, P., Bonora, I., Harnett Sheenan, K., . . . Dunbar, G. C. (1997). The Mini International Neuropsychiatric Interview (MINI). A short diagnostic structured interview: Reality and validity according to the CIDI. *European Psychiatry, 12*, 224-231. doi:10.1016/S0924-9338(97)83296-8

Leskin, L. P., & White, P. M. (2007). Attentional networks reveal executive function deficits in posttraumatic stress disorder. *Neuropsychology, 21*, 275-284. doi:10.1037/0894-4105.21.3.275

Luine, V., Villegas, M., Martinez, C., & McEwen, B. S. (1994). Repeated stress causes reversible impairments of spatial memory performance. *Brain Research, 639*, 167-170. doi:10.1016/0006-8993(94)91778-7

Luna, B., Thulborn, K. R., Munoz, D. P., Merriam, E. P., Garver, K. E., Minshew, N. J., . . . Sweeney, J. F. (2001). Maturation of widely distributed brain function subserves cognitive development. *NeuroImage, 13*, 786–793. doi:10.1006/nimg.2000.0743

Mackiewicz, S. K. (2011). *Cognitive and brain processes in young adults with a history of childhood interpersonal trauma.* (Doctoral Dissertation, University of Colorado). Retrieved from http://search.proquest.com/docview/887719963

Melinder, A., Augusti, E.-M., Matre, M., & Endestad, T. (2015). Associations between executive functions and long-term stress reactions after extreme trauma: A two-year follow-up of the utøya survivors. *Psychological Trauma: Theory, Research, Practice, and Policy, 7*, 583-590. doi:10.1037/tra0000048

Miyake, A., & Friedman, N. P. (2012). The nature and organization of Individual differences in executive functions: Four general conclusions. *Current directions in psychological science, 21*, 8-12. doi:10.1177/0963721411429458

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex "frontal lobe" tasks: A latent variable analysis. *Cognitive Psychology, 41*, 49-100. doi:10.1006/cogp.1999.0734

National Counterterrorism Center. (2012). *2011 Report on terrorism*. Retrieved from http://fas.org/irp/threat/nctc2011.pdf

Nelson, C. A., Bloom, F. E., Cameron, J. L., Amaral, D., Dahl, R. E., & Pine, D. (2002). An integrative, multidisciplinary approach to the study of brain-behavior relations in the context of typical and atypical development. *Development and Psychopathology, 14*, 499-520. doi:10.1017/s0954579402003061

Neria, Y., Nandi, A., & Galea, S. (2008). Post-traumatic stress disorder following disasters: A systematic review. *Psychological Medicine, 38*(4), 467-480. doi:10.1017/S0033291707001353

Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology, 100*, 569-582. doi:10.1037/0021-843X.100.4.569

Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology, 109*, 504-511. doi:10.1037/0021-843X.109.3.504

Norris, F. H. (1992). Epidemiology of trauma: Frequency and impact of different potentially traumatic events on different demographic groups. *Journal of Consulting and Clinical Psychology, 60*, 409-418. doi:10.1037/0022-006X.60.3.409

Norris, F. H., Friedman, M. J., Watson, P. J., Byrne, C. M., Diaz, E., & Kaniasty, K. (2002). 60,000 disaster victims speak: Part I. An empirical review of the empirical literature, 1981–2001. *Psychiatry: Interpersonal and Biological Processes, 65*, 207-239. doi:10.1521/psyc.65.3.207.20173

Norwegian Center for Violence and Traumatic Stress Studies. (n.d.). Terrorangrepet. Opplevelser og reaksjoner hos de som overlevde på Utøya [The terrorist attack. Experiences and reactions from the survivors at Utøya]. Retrieved 05.11.2015, from http://www.nkvts.no/fu/Sider/ProsjektInfo.aspx?prosjektid=1333

Oasay, L. M. K. (2013). *ADHD subtypes and the convergent validity of the BRIEF three-factor approach and D-KEFS color-word interference test.* (Master's thesis). Retrieved from http://hdl.handle.net/2152.5/1409

Olff, M., Polak, A. R., Witteveen, A. B., & Denys, D. (2014). Executive function in posttraumatic stress disorder (PTSD) and the influence of comorbid depression. *Neurobiology of Learning and Memory, 112*, 114-121. doi:10.1016/j.nlm.2014.01.003

Philippot, P., & Brutoux, F. (2008). Induced rumination dampens executive processes in dysphoric young adults. *Journal of Behavior Therapy and Experimental Psychiatry, 39*, 219-227. doi:10.1016/j.jbtep.2007.07.001

Phillips, M. L., Drevets, W. C., Rauch, S. L., & Lane, R. (2003). Neurobiology of emotion perception I: The neural basis of normal emotion perception. *Biological Psychiatry, 54*, 504-514. doi:10.1016/S0006-3223(03)00168-9

Pine, D. S., & Cohen, J. A. (2002). Trauma in children and adolescents: Risk and treatment of psychiatric sequelae. *Biological Psychiatry, 51*, 519 –531. doi:10.1016/S0006-3223(01)01352-X

Polak, A. R., Witteveen, A. B., Reitsma, J. B., & Olff, M. (2012). The role of executive function in posttraumatic stress disorder: A systematic review. *Journal of Affective Disorders, 141*, 11-21. doi:10.1016/j.jad.2012.01.001

Reyna, V. F., Chapman, S. B., Dougherty, M. R., & Confrey, J. (2012). Introduction to the adolescent brain: Learning, reasoning, and decision making. In V. F. Reyna, S. B. Chapman, M. R. Dougherty & J. Confrey (Eds.), *The adolescent brain: Learning, reasoning, and decision making* (pp. 3-11). Washington, DC: American Psychological Association.

Richert, K. A., Carrion, V. G., Karchemskiy, A., & Reiss, A. L. (2006). Regional differences of the prefrontal cortex in pediatric PTSD: An MRI study. *Depression and Anxiety, 23*, 17-25. doi:10.1002/da.20131

Roelofs, J., Muris, P., Huibers, M., Peeters, F., & Arnts, A. (2006). On the measurement of rumination: A psychometric evaluation of the ruminative response scale and the

rumination on sadness scale in undergraduates. *Journal of Behavior Therapy and Experimental Psychiatry, 37*, 299-313. doi:10.1016/j.jbtep.2006.03.002

Roley, M. E., Claycomb, M. A., Contractor, A. A., Dranger, P., Armour, C., & Elhai, J. D. (2015). The relationship between rumination, PTSD, and depression symptoms. *Journal of Affective Disorders, 180*, 116-121. doi:10.1016/j.jad.2015.04.006

Roth, R. M., Isquith, P. K., & Gioia, G. A. (2005). *Behavior rating inventory of executive function-adult version (BRIEF-A)*. Lutz, FL: Psychological Assessment Resources.

Ryan, J. J., Carruthers, C. A., Miller, L. J., Souheaver, G. T., Gontkovsky, S. T., & Zehr, M. D. (2003). Exploratory factor analysis of the wechsler abbreviated scale of intelligence (WASI) in adult standardization and clinical samples. *Applied Neuropsychology, 10*, 252-256. doi:10.1207/s15324826an1004_8

Samuelson, K. W., Krueger, C. E., Burnett, C., & Wilson, C. K. (2010). Neuropsychological functioning in children with posttraumatic stress disorder. *Child Neuropsychology, 16*, 119-133. doi:10.1080/09297040903190782

Schmid, M., & Hammar, Å. (2013). Cognitive function in first episode major depressive disorder: Poor inhibition and semantic fluency performance. *Cognitive Neuropsychiatry, 18*, 515-530. doi:10.1080/13546805.2012.754748

Schoeman, R., Carey, P., & Seedat, S. (2009). Trauma and posttraumatic stress disorder in South African adolescents: A case-control study of cognitive deficits. *Journal of Nervous and Mental Disease, 197*, 244-250. doi:10.1097/NMD.0b013e31819d9533

Scrimin, S., Axia, G., Capello, F., Moscardino, G., Steinberg, A. M., & Pynoos, R. S. (2006). Posttraumatic reactions among injured children and their caregivers 3 months after the terrorist attack in Beslan. *Psychiatry Research, 141*, 333-336.

Scrimin, S., U., M., Capello, F., Altoè, G., Steinberg, A. M., & Pynoos, R. S. (2011). Trauma reminders and PTSD symptoms in children three years after a terrorist attack in

Beslan. *Social Science & Medicine, 72*, 694-700. doi:10.1016/j.socscimed.2010.11.030

Shalev, A. Y., Freedman, S., Peri, T., Brandes, D., Sahar, T., Orr, S., & Pitman, R. K. (1998). Prospective study of posttraumatic stress disorder and depression following trauma. *The American Journal of Psychiatry, 155*, 630-637. doi:10.1176/ajp.155.5.630

Sheehan, D., Janavs, J., Harnett-Sheehan, K., Sheehan, M., Gray, C., Lecruiber, Y., . . . Even, C. (2009). M.I.N.I.: Mini International Neuropsychiatric Interview, Norwegian version 6.0.0 (DSM-IV). Retrieved from http://webcache.googleusercontent.com/search?q=cache:Yc4F4GI1fx0J:https://www.helsebiblioteket.no/_attachment/196313+&cd=2&hl=en&ct=clnk&gl=no

Shin, L. M., Rauch, S. L., & Pitman, R. K. (2006). Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. *Annals of the New York Academy of Sciences, 1071*, 67-79. doi:10.1196/annals.1364.007

Smith, E. E., & Jonides, J. (1999). Storage and executive processes in the frontal lobes. *Science, 283*, 1647-1661. doi:10.1126/science.283.5408.1657

Southwick, S., Rasmusson, A., Barron, J., & Arnsten, A. (2005). Neurobiological and neurocognitive alterations in PTSD: A focus on norepinephrine, serotonin, and the HPA axis. In J. J. Vasterling & C. Brewin (Eds.), *Neuropsychology of PTSD: Biological, cognitive, and clinical perspectives* (pp. 27-58). New York, NY: The Guilford Press.

Spear, L. (2007). The developing brain and adolescent-typical behavior patterns. In D. Romer & E. F. Walker (Eds.), *Adolescent psychopathology and the developing brain: Integrating brain and prevention science* (pp. 9-30). New York, NY: Oxford University Press. doi:10.1093/acprof:oso/9780195306255.003.0001

Stein, M. B., Kennedy, C. M., & Twamley, E. W. (2002). Neuropsychological function in female victims of intimate partner violence with and without posttraumatic stress disorder. *Biological Psychiatry, 52*, 1079-1088. doi:10.1016/S0006-3223(02)01414-2

Steinberg, L. (2005). Cognitive and affective development in adolescence. *Trends in Cognitive Sciences, 9*, 69-74. doi:10.1016/j.tics.2004.12.005

Steinberg, L. (2011). *Adolescence* (9 ed.). New York, NY: McGraw-Hill.

Steudte-Schmiedgen, S., Stalder, T., Kirschbaum, C., Weber, F., Hoyer, J., & Plessow, F. (2014). Trauma exposure is associated with increased context-dependent adjustments of cognitive control in patients with posttraumatic stress disorder and healthy controls. *Cognitive, Affective & Behavioral Neuroscience, 14*, 1310-1319. doi:10.3758/s13415-014-0299-2

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*, 643-662. doi:10.1037/h0054651

Stuss, D. T., & Alexander, M. P. (2000). Executive functions and the frontal lobes: A conceptual view. *Psychological Research, 63*, 289-298. doi:10.1007/s004269900007

Suliman, S., Mkabile, S. G., Fincham, D. S., Ahmed, R., Stein, D. J., & Seedat, S. (2009). Cumulative effect of multiple trauma on symptoms of posttraumatic stress disorder, anxiety, and depression in adolescents. *Comprehensive Psychiatry, 50*, 121-127. doi:10.1016/j.comppsych.2008.06.006

Suomalainen, L., Haravuori, H., Berg, N., Kiviruusu, O., & Marttunen, M. (2011). A controlled follow-up study of adolescents exposed to a school shooting – Psychological consequences after four months. *European Psychiatry, 26*, 490-497. doi:10.1016/j.eurpsy.2010.07.007

Thompson-Schill, S. L., Ramscar, M., & Chrysikou, E. G. (2009). Cognition without control: When a little frontal lobe goes a long way. *Current Directions in Psychological Science, 18*, 259-263. doi:10.1111/j.1467-8721.2009.01648.x

Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive therapy and research, 27*, 247-259. doi:10.1023/A:1023910315561

Turley, M. R., & Obrzut, J. E. (2012). Neuropsychological effects of posttraumatic stress disorder in children and adolescents. *Canadian Journal of School Psychology, 27*, 166-182. doi:10.1177/0829573512440420

Twamley, E. W., Hami, S., & Stein, M. B. (2004). Neuropsychological function in college students with and without posttraumatic stress disorder. *Psychiatry Research, 126*, 265-274. doi:10.1016/j.psychres.2004.01.008

Ullman, S. E., & Filipas, H. H. (2005). Gender differences in social reactions to abuse disclosures, post abuse coping, and PTSD of child sexual abuse survivors. *Child Abuse & Neglect, 29*, 767-782. doi:10.1016/j.chiabu.2005.01.005

Vasterling, J. J., Duke, L. M., Brailey, K., Constans, J. I., Allain, A. N., & Sutker, P. B. (2002). Attention, learning, and memory performances and intellectual resources in Vietnam veterans: PTSD and no disorder comparison. *Neuropsychology, 16*, 5-14. doi:10.1037/0894-4105.16.1.5

Vaughan, L., & Giovanello, K. (2010). Executive function in daily life: Age-related influences of executive processes on instrumental activities of daily living. *Psychology and Aging, 25*, 343-355. doi:10.1037/a0017729

Victoroff, J. (2005). The mind of the terrorist: A review and critique of psychological approaches. *The Journal of Conflict Resolution, 49*, 3-42. doi:10.1177/0022002704272040

Viner, R. M., Ozer, E. M., Denny, S., Marmot, M., Resnick, M., Fatusi, A., & Currie, C. (2012). Adolescence and the social determinants of health. *The Lancet, 379*, 1641-1652. doi:10.1016/s0140-6736(12)60149-4

Weathers, F. W., & Keane, T. M. (2007). The criterion a problem revisited: Controversies and challenges in defining and measuring psychological trauma. *Journal of Traumatic Stress, 20*, 107-121. doi:10.1002/jts.20210

Weber, D. A., & Reynolds, C. R. (2004). Clinical perspectives on neurobiological effects of psychological trauma. *Neuropsychology Review, 14*, 115-129. doi:10.1023/b:nerv.0000028082.13778.14

Whitmer, A. J., & Banich, M. T. (2007). Inhibition versus switching deficits in different forms of rumination. *Psychological Science, 18*, 546-553. doi:10.1111/j.1467-9280.2007.01936.x

Whitmer, A. J., & Gotlib, I. H. (2012). Switching and backward inhibition in major depressive disorder: The role of rumination. *Journal of Abnormal Psychology, 121*, 570-578. doi:10.1037/a0027474

Whitmer, A. J., & Gotlib, I. H. (2013). An attentional scope model of rumination. *Psychological Bulletin, 139*, 1036-1061. doi:10.1037/a0030923

Wilson, K. R. (2010). *The neuropsychological effects of the traumatic stress response in sexually abused adolescents throughout treatment.* (Doctoral Dissertation). Retrieved from http://digitalcommons.unl.edu

Wilson, K. R., Hansen, D. J., & Li, M. (2011). The traumatic stress response in child maltreatment and resultant neuropsychological effects. *Aggression and Violent Behavior, 16*, 87-97. doi:10.1016/j.avb.2010.12.007

Wilson, M., & Daly, M. (1985). Competitiveness, risk taking, and violence: The young male syndrome. *Ethology and Sociobiology, 6*, 59-73. doi:10.1016/0162-3095(85)90041-x

World Health Organization. (1992). *International statistical classification of diseases and related health problems 10th Revision (ICD-10)*. Geneva: World Health Organization.

World Health Organization. (2015). Adolescent development. Retrieved from http://www.who.int/maternal_child_adolescent/topics/adolescence/dev/en/

Appendix

Approval Letter From the Regional Committees for Medical and Health Research Ethics

Komiteen legger merke til at informasjonsskrivet mangler informasjon om at biologisk materiale oppbevares i en biobank. Det bes om at dette tas inn.

På denne bakgrunn settes følgende vilkår for godkjenning:

1) I søvntesten skal formuleringen "sovne inn" skrives om

2) Det må informeres om datadeling med forskningsgruppen ved Psykologisk institutt, UiO, i skrivet til deltagerne

3) Informasjonsskrivet må inneholde informasjon om at biologisk materiale oppbevares i en biobank

Vedtak
Komiteen godkjenner prosjektendringssøknaden, jf. helseforskningslovens § 11, under forutsetning av at ovennevnte vilkår oppfylles.

I tillegg til vilkår som fremgår av dette vedtaket, er tillatelsen gitt under forutsetning av at prosjektendringen gjennomføres slik det er beskrevet i prosjektendringsmeldingen og endringsprotokoll, og de bestemmelser som følger av helseforskningsloven med forskrifter.

Forskningsprosjektets data skal oppbevares forsvarlig, se personopplysningsforskriften kapittel 2, og Helsedirektoratets veileder for Personvern og informasjonssikkerhet i forskningsprosjekter innenfor helseog omsorgssektoren.

Klageadgang
Du kan klage på komiteens vedtak, jf. forvaltningslovens § 28 flg. Klagen sendes til REK sør-øst D. Klagefristen er tre uker fra du mottar dette brevet. Dersom vedtaket opprettholdes av REK sør-øst D, sendes klagen videre til Den nasjonale forskningsetiske komité for medisin og helsefag for endelig vurdering.

Vi ber om at alle henvendelser sendes inn på korrekt skjema via vår saksportal: http://helseforskning.etikkom.no. Dersom det ikke finnes passende skjema kan henvendelsen rettes på e-post til: post@helseforskning.etikkom.no.

Vennligst oppgi vårt referansenummer i korrespondansen.

Med vennlig hilsen

Stein A. Evensen
Professor dr. med.
Leder

Gjøril Bergva
Rådgiver

Kopi til: *helge.nordby@psybp.uib.no; post@uib.no*

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