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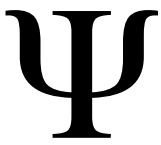

Long-Term Effects of Extreme Trauma on Sleep Quality and the Circadian Rhythm of Sleep and Wakefulness: An Actigraphy Study of Utøya Survivors

HOVEDOPPGAVE

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Working close with these data over time have given us tremendous compassion for everybody involved and affected by this horrible day in July 2011. The story of this day puts the work of this thesis in a much larger perspective. We will express our utmost respect for the ongoing and previous work that is dealing with the consequences of the worst terror attack in Norwegian history.

We would like to express our gratitude to all participants for their contribution in this project. We will especially thank survivors of the Utøya massacre for contributing to the project after what they experienced, making it possible to understand more about the complexity of post-traumatic reactions and how trauma affects sleep.

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Abstract

The terror attack at Utøya Island in 2011 was a national tragedy. Most of the survivors were adolescents. The aim of this study was to explore how sleep and circadian rhythm of sleep and wakefulness was affected after the Utøya massacre. In addition we wanted to examine the general sleep pattern among adolescents.

Methods: 42 Utøya survivors and 46 control subjects matched on gender, age and socio-demographical variables were studied 18-30 months after the attack. Sleep was assessed by actigraphy.

Results: Utøya survivors display a delayed circadian rhythm of sleep and wakefulness (bedtime, sleep onset, wake up time and get up time; $F_{1,161}$'s >=5,7, p<0.018) compared to control subjects. No significant differences were revealed on sleep quality (sleep onset latency, wake after sleep onset, total sleep time and sleep efficiency). All participants display a delayed circadian rhythm during weekends compared to weekdays ($F_{1,161}$'s >22,5, p<0.001). Preliminary analyses and several miscalculated actograms favor the use of manually coded rest intervals. We discuss the findings in relation to results of subjective sleep measurements of the same sample, previous research, therapeutic relevance and further research.

Conclusion: Assessment of actigraphy gives important information about long-term changes in sleep and the circadian rhythm of sleep and wakefulness after traumatic exposure. It illustrates the importance of addressing circadian rhythm and sleep after trauma among all survivors regardless of any major sleep-or psychiatric disorders at the moment. It supports earlier research about adolescents sleep patterns, and encourage researchers to use manually coded rest intervals when measuring sleep by actigraphy.

Sammendrag

Terrorahandlingene på Utøya i 2011 var en nasjonal tragedie. De fleste på øya var ungdommer, og 69 personer ble drept. Målet med studien var å undersøke hvordan søvnen og døgnrytmen blant de overlevende har blitt påvirket etter Utøya massakren. I tillegg ønsket vi å utforske døgnrytmen generelt blant ungdom.

Metode: 42 Utøya overlevende og 46 kontroll personer ble matchet på alder, kjønn og sosiodemografiske variabler. De ble studert 18-30 måneder etter terroren. Aktigrafi ble brukt for å måle søvnen og døgnrytmen.

Resultat: Utøya overlevende hadde en forsinket søvnfase sammenlignet med kontroll deltakerne. Ingen betydelige forskjeller ble funnet for søvnkvaliteten (søvnlatens, antall oppvåkninger i løpet av natten, total søvntid og søvneffektivitet; $F_{1,161}$'s >=5,7, p<0.018). Alle deltakerne hadde en forsinket søvnfase i helgene ($F_{1,161}$'s >22,5, p<0.001). Preliminære analyser og feilregistrerte aktogrammer støttet bruken av manuelt kodede hvileintervaller. Funnene ble diskutert i forhold til subjektive data fra samme utvalg, tidligere forskning, klinisk relevans og forslag til videre forskning.

Konklusjon: Bruk av aktigraf gir viktig informasjon om langtidsendringer i døgnrytme og søvn etter traumatisk eksponering. Den illustrerer viktigheten av å undersøke søvn og døgnrytme blant ungdom utsatt for traume, uavhengig av om de forteller om søvn eller psykiatriske problemer. Den støtter tidligere funn om en forsinket døgnfase blant ungdom i helgene, og anbefaler manuelt kodede hvileintervall ved bruk av aktigrafi.

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Introduction

Utøya Attack

On the 22nd of July 2011, Norway experienced the worst terror attack since World War II. After detonating a bomb in the Government Quarter in Oslo, the assailant attacked the Workers' Youth League (AUF)-run summer camp at Utøya Island. By posing as a police officer, he entered the island claiming to secure the people, 564 in total, most of them adolescents. During almost 80 minutes he shot and killed 69 people. More than 100 were injured, and survivors were all direct witnesses of the brutality. They survived by hiding from the assailant for over an hour. Some pretended to be dead among killed friends, while others swam for their lives. Several survivors were witnessing brutal executions, watching the terrorist as he aimed to shoot and kill all adolescents at Utøya. It is likely that the adolescents are still affected by what happened, 2 years after the attack. In this thesis we will examine the long-term effects on sleep and the circadian rhythm of sleep and wakefulness among the survivors. First we will give a background of behavioral, psychological and physiological consequences that can occur after trauma.

Reactions to Traumatic Events

It is registered over 125 000 terror attacks around the world between 1970 and 2013 (National Consortium for the Study of Terrorism and Responses to Terrorism (START), 2013). The terror attacks in recent years are almost 17 % more likely to involve death or injury compared to earlier attacks (Enders & Sandler, 2000). Terrorism is an extreme form of violent trauma that happens suddenly and unexpected, attacking many innocent individuals who are unaware that the attack may occur (Shaw, 2003). Kofi Annan, the former Secretary General of the United Nations describes terrorism as an action that

cause death or serious injury to harm the population, or to force a government or an international organization to act or refrain from acting. Terrorism is a critical and severe problem among many nations, and may lead to a growing fear among individuals across nations (United Nations Association of Norway, 2013).

To be subjected to terror is considered a traumatic experience. The Worlds Health Organizations official system of diagnosis (ICD-10) describe a traumatic event as a "[...] situation (of either brief or long duration) of an exceptionally threatening or catastrophic nature, which is likely to cause pervasive distress in almost anyone (such as natural or human made disasters, acts of war, serious accidents, facing violent death of others, being subjected to torture, terrorism, rape or other criminal acts)" (World Health Organization, 1992, p. 146). The lifetime likelihood of experiencing a trauma ranges from 39-84% (Breslau, Davis, Andreski, & Peterson, 1991; Davidson & Smith, 1990; Norris, 1992; Vrana & Lauterbach, 1994), depending on the national stability in the studied population.

Normal reactions to a traumatic event are often divided into short-term and long-term reactions. Immediately after being subjected to an attack or a disaster, the affected victim normally experiences a variety of emotional reactions (Rubin, Brewin, Greenberg, Simpson, & Wessely, 2005; Schuster et al., 2001; Thoresen, Aakvaag, Wentzel-Larsen, Dyb, & Hjemdal, 2012). Short-term reactions such as anxiety, anger, fear, increased muscle tension and greater alertness affecting sleep and concentration, are in most cases considered normal reactions and usually fade after some time (Rubin et al., 2007; Silver, Holman, McIntosh, Poulin, & Gil-Rivas, 2002). Immediately after the Utøya attack, survivors reported high levels (2.8 on a scale from 0 to 4) of stress reactions such as fear, confusion, peritraumatic dissociation, rapid heartbeat,

helplessness and horror (Dyb et al., 2014). In addition, many Norwegian citizens experienced emotions such as sadness and feeling of unreality in the days after the attack. Similar results have been reported after other terror attacks affecting a whole nation. Three to five days after the terror attack on 11th of September 2001 (9/11) in New York, children and adults all over the nation reported sadness, impaired sleep, invasive thoughts, dreams and memories of what happened, difficulties concentrating, and feeling irritable or angry (Schuster, et al., 2001).

Sleep is normally disrupted after a traumatic exposure (Bradburn, 1991; Dollinger, 1986; Lavie, 2001; Sadeh, 1996). After the Hanshin earthquake in Japan in 1995, 58% of the evacuees reported sleep difficulties 3 weeks after the event (Kato, Asukai, Miyake, Minakawa, & Nishiyama, 1996). The difficulties were still present for 46% of the respondents, 8 weeks after the earthquake. Similarly, substantial sleep difficulties are reported among affected adults and children 3 days to 6 months after events such as the 9/11 attack and the Oklahoma City bombing in 1995 (North et al., 1999; Schuster, et al., 2001). The earthquake in San Francisco in 1989 lead to sleep difficulties and bad dreams in 27% of the children, 6-8 months after the disaster (Bradburn, 1991). Thus, disrupted sleep is a common consequence of traumatic experience, and may persist for some time.

In the long term, there will normally be a reduction in activation and stress reactions (Rubin, et al., 2007). On the 7th of July 2005, a bomb attack destroyed London's transport network, and 52 people died and more than 700 were injured. Two years after the attack, citizens of London reported a reduction in feelings of substantial stress and fear compared to immediately after (Rubin, et al., 2005). However, 61% of the citizens stated that this event had altered their view of the world. Behaviors such as

travelling and shopping in central London were reduced as a result of the bombing. Londoners still felt an increased threat level to their closest and most beloved ones, and a higher perceived likelihood of future attacks (Rubin, et al., 2007). Similar findings are described 6 months after the 9/11 attack (Silver, et al., 2002). Although there is a decline in reactions over time, terror attacks typically influence the perception of safety, as well as subjective well-being and mental health in the general population (Miguel-Tobal et al., 2006; North, et al., 1999; Rubin, et al., 2007; Schuster, et al., 2001). These findings show how a whole nation gets affected after a terror attack, both immediately after and in the long-term. However, the reactions may persist and develop into pathological disorders for some survivors.

Pathological Consequences After Trauma

Psychiatric disorders. When the activation and stress reactions exceed the expected intensity and duration, it may qualify as a psychiatric diagnosis (North, et al., 1999; Sutker & Allain Jr, 1996; Vrana & Lauterbach, 1994). Being exposed to a traumatic event increases the risk of several psychiatric disorders, such as post-traumatic stress disorder (PTSD), depression, anxiety and panic disorder (North, et al., 1999; Sutker & Allain Jr, 1996; Vrana & Lauterbach, 1994).

ICD-10 defines PTSD as "[...] a delayed or protracted response to 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" (World Health Organizatin, 1992, p. 146). PTSD is the most common pathological consequence after trauma, and the disorder is closely related to the event itself (Bleich, Koslowsky, Dolev, & Lerer, 1997). Among those exposed to a traumatic event, the incidence of PTSD ranges from 7% to more than 27% (Breslau, et al., 1991; Davidson

& Smith, 1990; Norris, 1992; Vrana & Lauterbach, 1994). This gives a lifetime prevalence of PTSD at about 5-10% (Breslau, et al., 1991; Schnurr, Friedman, & Bernardy, 2002).

Patients with chronic depression often have a childhood characterized by psychological trauma, adversity, protracted environmental stress and neglect (G. W. Brown & Moran, 1994; Riso, Miyatake, & Thase, 2002). Similarly, a childhood characterized by physical and sexual abuse is more common among people with panic disorders than in the normal population (Stein et al., 1996). These tendencies indicate a close relationship between traumatic experiences during the childhood and both depression and panic disorders. Studies of the terror attack on Utøya and other terror attacks have reported similar relationships between traumatic exposure and psychiatric disorders. Eleven percent of the surviving adolescents at Utøya met the criteria for full PTSD and 36% for partial PTSD, 4-5 months after the attack (Dyb, Jensen, Nygaard, et al., 2014). Similar patterns were replicated 18-30 months after the attack, where 30% of the Utøya survivors had an ongoing PTSD diagnosis, compared to 0 % of the control subjects (Milde et al., 2014). There was also a higher prevalence of panic disorder (29 % versus 0 %) and earlier depressive disorder (33 % versus 12 %) among Utøya survivors than control subjects (Milde, et al., 2014). Six months after the Oklahoma City bombing, 34.3% of the victims were diagnosed with PTSD, 22.5% were diagnosed with major depression, and 6.6% suffered from panic disorder (North, et al., 1999). In comparison, the percentage of PTSD, major depression and panic disorder in Oklahoma before the bombing were 15%, 12.6% and 2.8% respectively (North, et al., 1999), indicating a doubling of these diagnoses as a consequence of the bombing.

The development of PTSD is also found to increase the risk for other co-occurring disorders (Breslau, et al., 1991; Davidson, Hughes, Blazer, & George, 1991). Ohayon and Shapiro (2000) report that 75.7% of patients with PTSD also qualifie for at least one additional psychiatric diagnosis. People with PTSD are more than 10 times as likely to get diagnosed with a major depression, and more than 20 times as likely to be diagnosed with panic disorder, compared to non-PTSD subjects (Davidson, et al., 1991). If the trauma consists of a traumatic loss in addition to directly life threat, the risk of developing a comorbid PTSD and depression increases (Momartin, Silove, Manicavasagar, & Steel, 2004). One study reported that 70% of adolescents with comorbid depression and PTSD developed the first episode of depression during the same year as they developed PTSD. Moreover, the same developmental pattern applied for 66.7% with comorbid PTSD and drug dependence, and 45.5% with comorbid PTSD and alcohol dependence (Giaconia et al., 1995). These associations indicate that for adolescents, a potential traumatic event may lead to the development of several psychiatric diagnoses within a short period of time (Giaconia, et al., 1995). Altogether, these studies give an indication of the prevalence and comorbidity of psychiatric disorders after terror attacks.

Knowledge about both expected reactions after traumatic experiences and what is pathological is important for successful differentiation between what is considered normal reactions, and what is in need for treatment. There are multiple risk factors that may contribute to the development of psychiatric disorders after trauma.

Risk factors to develop psychiatric disorders. Individual differences and social context may be of great importance for the perception and interpretation of the event, as well as the survivor's perceived level of control (Gjestad, 2005; North, et al., 1999;

Rubin, et al., 2005; Udwin, Boyle, Yule, Bolton, & O'Ryan, 2000). Biological predispositions, personality traits, and history of psychiatric illness affect the risk of developing pathological disorders after traumatic events (North, et al., 1999; Paris, 2000; Silver, et al., 2002). Studies conducted after 9/11 and the bombing of London transport network found that being female increased the risk of PTSD (Rubin, et al., 2005; Schuster, et al., 2001). Likewise, being young increased the risk of PTSD among military peacekeepers exposed to war (Bramsen, Dirkzwager, & Van der Ploeg, 2000). Personality traits, such as neuroticism, are also associated with development of PTSD (Lawrence & Fauerbach, 2003). Further, a previous history of psychiatric disorders was found among 57% of the people with PTSD related to the Oklahoma City bombing (North, et al., 1999). Added together, there are multiple individual factors that may contribute to increase the risk of severe psychiatric disorders after experiencing horrible traumas.

Social factors such as being part of an ethnic minority (Galea et al., 2002; Neria, DiGrande, & Adams, 2011; Rubin, et al., 2005; Thoresen, et al., 2012) or low socioeconomic status also seem to affect the mental health among survivors after traumatic events (Cohen & Roth, 1987). Ethnic minorities often have low socioeconomic status, indicating a close relationship between these factors. Further, ethnic minorities often have a history of previous traumas, as they often are refugees from war affected areas (Thoresen, et al., 2012). Several studies have found that the number of previously experienced traumas also increase the risk of developing psychiatric disorders such as anxiety, depression and PTSD (Cohen & Roth, 1987; North, et al., 1999; Silove, Steel, McGorry, & Mohan, 1998; Sutker & Allain Jr, 1996).

The character and severity of the traumatic event are associated with future psychiatric disorders (Norris et al., 2002). Prevalence of psychopathology is found to be greater after an event of mass violence compared to natural or technological disasters (Goenjian et al., 2001; Norris, et al., 2002). Further, the prevalence of psychiatric disorders increases when the level of exposure increases (Goenjian, et al., 2001; Hoven et al., 2005; Neria, Nandi, & Galea, 2008; Silver, et al., 2002). All survivors at Utøya heard gun shots, 93.8 % heard people screaming, 82.6% heard someone be injured or killed, 86.7% saw dead bodies, 46 % touched dead bodies or injured people, and 45.1% saw the terrorist point the gun at him/her or realised that he had shot at him/her. The level of exposure to injuries and the assailant's brutality predicted the level of post-traumatic stress reactions 4-5 months after the attack (Dyb, Jensen, Nygaard, et al., 2014). Similarly, after Hurricane Mitch in Nicaragua in 1998, the prevalence of PTSD and depression among adolescents were highly correlated with the extent to which their region was affected (Goenjian, et al., 2001).

Psychological and geographical proximity is also considered risk factors (Thoresen, et al., 2012). Psychological proximity, as being worried about the safety of someone close or having personal relationships with victims of the attack, was highly correlated with PTSD symptoms among Norwegian citizens 4-5 months after the terror attack (Thoresen, et al., 2012). Further, being geographically close to the event is closely related to the level of exposure, and to development of PTSD (Hansen, Nissen, & Heir, 2013). The citizens of Oslo reported significantly greater feelings of jumpiness and fear shortly after the attack compared to the rest of the nation (Thoresen, et al., 2012). These immediate reactions were further associated with symptoms of PTSD, 4-5 months after the attack (Thoresen, et al., 2012). The prevalence of PTSD among

ministerial employees whose workplace was attacked was assessed 10 months after the terror attack in Oslo. Twenty-four percent of those present at work that day had PTSD, compared to 4% of those not present that day (Hansen, et al., 2013). Similarly, 20 % of those living close to the World Trade Center had PTSD about 2 months after the 9/11 terrorist attack, compared to 7.5% of the adults in the rest of Manhattan (Galea, et al., 2002).

After the traumatic event, coping strategies (Cohen & Roth, 1987; Silver, et al., 2002), level of social support (Dyb, Jensen, Nygaard, et al., 2014; Galea, et al., 2002) and different consequences of the event such as loss (Galea, et al., 2002) are associated with the risk of developing psychiatric disorders. Loss of a job or a possession, death of a friend or relative and low levels of social support are found to increase the risk of developing PTSD and/or depression (Dyb, Jensen, Nygaard, et al., 2014; Galea, et al., 2002; Hobfoll, Tracy, & Galea, 2006). Almost 3/4 of the Utøya survivors reported having lost a significant person; almost all of them (96.3%) describe the person as a friend. Further, 4.6% lost a boyfriend/girlfriend, and 4.6% lost a family member (Dyb, Jensen, Nygaard, et al., 2014). These losses put the survivors at greater risk of developing pathological reactions. Notably, the same study found that almost all of the survivors received high levels of social support after the attack, and the social support was found to reduce PTSD and depression among the survivors (Dyb, Jensen, Nygaard, et al., 2014; Moscardino, Scrimin, Capello, & Altoè, 2010).

People experiencing sleep disturbances immediately before (Bryant, Creamer, O'Donnell, Silove, & McFarlane, 2010) or after a traumatic event have a greater risk to develop future physical and psychiatric symptoms (Lavie, 2001), such as depression, any anxiety disorder, alcohol- and substance abuse and PTSD (Breslau, Roth,

Rosenthal, & Andreski, 1996; Hobfoll, et al., 2006; Lavie, 2001). In addition, these psychiatric disorders can in turn lead to the development of sleep disturbances (Ohayon & Shapiro, 2000; Uhde et al., 1984), suggesting a mutual influence. Patients with panic disorder and/or PTSD report substantial sleep disturbances, and as many as 90% of patients with depressive disorders describe having sleep difficulties at some point (American Academy of Sleep Medicine, 2005). Sleep is an important risk factor and may serve as a predictor of developing future psychiatric disorders. Before further investigation of how sleep is affected after trauma, it is helpful to explore basic mechanisms of sleep.

Sleep

A third of our lives is spent sleeping. It is usually easy to determine if a person is asleep by the inactivity and inattention to the environment. Even though externally it looks like the brain is inactive during sleep, some brain cells are actually more active when sleeping than when awake (Siegel, 2003). The question of why we sleep has been of interest for many decades, and researchers have progressed to reveal the etiology of sleep. We know that sleep is essential to restore information to memory, restore synaptic function and repair neuronal circuits (Horne, 1988; Jenkins & Dallenbach, 1924; Krueger & Obál, 1993; Siegel, 2003). Recently it has been found that neurotoxic waste is effectively removed during sleep (Xie et al., 2013). Further, several different physiological systems are maintained by sleep: the immune system (Marshall & Born, 2002), the cardiovascular system (Wolk, Gami, Garcia-Touchard, & Somers, 2005) and the metabolic system (Grønli, Soulé, & Bramham, 2013; Laposky, Bass, Kohsaka, & Turek, 2008). Studies of longer periods with sleep deprivation demonstrate how sleep loss may affect our subjective well-being, concentration (Lim & Dinges, 2010), the

ability to withstand pain and stress, our cognitive performance and alertness, school performance (Siegel, 2003), sleepiness during daytime (Gradisar, Gardner, & Dohnt, 2011), mood, risk for accidents, vulnerability to abuse alcohol and drugs, and vulnerability to develop sleep- (Carskadon, 1990) and psychiatric disorders (Bianchi & Nyer, 2014; Boyne, Sherry, Gallagher, Olsen, & Brooks, 2013). These findings illustrate the importance of sleep for optimal functioning, and how poor sleep may affect individuals in various ways.

The sleep duration depends on individual differences, however a normal night is considered to consist of 6-8 hours of sleep (Grønli & Saxvig, 2014). Sleep itself consists of two different physiological states - an active state of rapid eye movement (REM) and more quiet state of non-REM (NREM) sleep. NREM-REM sleep cycle alternates cyclically through the night lasting approximately 90 minutes, hence a normal night usually consists of four to six sleep cycles (Carskadon & Dement, 2000). Based on the frequency of the brain waves measured by electroencephalography (EEG), NREM is subdivided into the stages N1, N2 and N3. Stage N1 and N2 are considered light sleep and easy to wake up from. N3 is defined by low frequency waves, show a higher waking threshold and is considered a deep sleep stage (Carskadon & Dement, 2000). The stage N3 has a restorative effect on the brain where proteins are synthesized and growth hormones are secreted (Grønli, et al., 2013; Siegel, 2003). N3 predominates the first sleep cycles during the night, while time in REM sleep increases in duration towards the morning (Keenan & Hirshkowitz, 2011). It is natural to experience awakenings between sleep cycles, but due to the short duration most of us do not recall this in the morning (Grønli & Saxvig, 2014).

Dreaming may occur during all sleep stages, and may consist of both positive, neutral and negative emotions (Cartwright, Luten, Young, Mercer, & Bears, 1998; Foulkes, 1962). The dreams that we recall happens primarily during REM sleep (Dement & Kleitman, 1957; Norsk Helseinformatikk, 2009; Siegel, 2003). Dreams arise from centers in the brain associated with the recognition of places and faces, and processing of feelings and visual memories (Bischof & Bassetti, 2004).

Sleep is regulated by three factors: homeostatic, circadian and behavior (Carskadon, 2010; Pallesen & Bjorvatn, 2009). These factors contribute to the experience of sleepiness during the night and wakefulness during the day. The homeostatic factor reflects the need for sleep, which increases during time spent awake. The built up need for sleep is further reflected in the amount of N3 sleep in the following night (Zisapel, 2007). The circadian rhythmicity of activation normally follows approximately a 24-hours cycle, and is consistent with cyclic changes in body temperature and the secretion of hormones such as melatonin (Arendt, 2000; Moore, 1992). The endogenous circadian rhythm is often longer than 24 hours in adults, but external stimuli, such as light exposure, activity and social factors help keep the biological circadian rhythm to a 24-hour, every day (Czeisler & Gooley, 2007; Grønli & Ursin, 2009). Our behavior influences our biological sleep regulation and may overrule the homeostatic and circadian factors. Due to this ability to overrule, we are capable to attend a party or work night shifts even though our body is programmed to sleep (Bjorvatn & Pallesen, 2009). There are great individual differences in how much sleep a person needs, but most people need between 6 and 9 hours of sleep a night (Grønli & Saxvig, 2014). Also, there are individual preferences for bedtime and wake up time, which are referred to as “eveningness-types” or “morningness-types”, depending on the

individual's circadian rhythmicity of sleep and wakefulness (Horne & Östberg, 1977). In adolescence there are more eveningness-types than in the older population (Adan et al., 2012; Tonetti, Fabbri, & Natale, 2008), indicating how different developmental stages affect sleep, and how sleep may vary across the lifespan.

Sleep among adolescents. The survivors at Utøya consisted mainly of adolescents (Dyb, Jensen, Nygaard, et al., 2014). Sleep during adolescence is important for physical, cognitive and emotional development (Dahl & Lewin, 2002). A delayed sleep phase is common among adolescents, shown as later bedtimes and wake up times. Later bedtimes are further associated with increased daytime sleepiness during schooldays (Carskadon, Vieira, & Acebo, 1993; Van den Bulck, 2007). Reduced sleep time increases the risk to develop mood and behavior disturbances, abuse of alcohol and drugs, and severe circadian rhythm difficulties (Carskadon, 1990).

Several theories have been proposed to describe delayed sleep phase based on studies of adolescents on different stages of puberty. Some argue that the biological circadian clock changes during adolescence, possibly prolonging the endogenous circadian period. This change may lead to a delayed circadian phase compared to adults (Crowley, Acebo, & Carskadon, 2007). Another theory describes changes in the homeostatic drive during adolescence. Studies of sleep deprived adolescents indicate a slower accumulation of sleep need during prolonged wakefulness among the more mature adolescents (Jenni, Achermann, & Carskadon, 2005). Still, the dissipation of sleep need during the night sleep does not differ among adolescents of different age (Jenni, et al., 2005). Thus, both circadian and homeostatic factor may contribute to the delayed sleep phase seen among adolescents.

Behavior may have a special impact on sleep during adolescence. At this age, the 'children' gain more control over their own bedtime (Carskadon, 1990) and opportunities for social activities in the evening increase (Dahl & Lewin, 2002). Other behaviors associated with a delayed sleep phase is part-time job in addition to school (Carskadon, 1990) and the use of mobile phones and other electronic devices with blue light illumination in bed during night time (Calamaro, Mason, & Ratcliffe, 2009). One, or several of these behaviors combined, are associated with delayed sleep phase observed among adolescents (Calamaro, et al., 2009; Carskadon, 1990).

Sleep pattern often vary between weekdays and weekends, this is usually referred to as "social jet lag"(Tonetti, et al., 2008; Wittmann, Dinich, Merrow, & Roenneberg, 2006). Social jet lag is a combination of preferences for circadian rhythm and social influence, where early mornings because of school/work during weekdays often lead to an increased sleep during weekends to compensate for lost sleep (Roenneberg, Wirz-Justice, & Merrow, 2003; Wittmann, et al., 2006). The delayed sleep phase during weekends are more prominent among older adolescents than younger (Laberge et al., 2001). Eveningness-types have a later bedtime, and a more evident social jet lag than morningness-types (Wittmann, et al., 2006). Even though many adolescents are eveningness-types, some may experience the delayed sleep phase as a major problem impairing their daily lives. Sleep disturbances among adolescents may be associated with psychiatric disorders. Adolescents reporting sleep disturbances are more likely to report symptoms of anxiety, depression, stress, and higher use of alcohol (Kirmil-Gray, Eagleston, Gibson, & Thoresen, 1984; Manni et al., 1997; Saarenpää-Heikkilä, Laippala, & Koivikko, 2001).

Sleep disturbances after trauma. Sleep disturbances are common among several psychiatric disorders, such as PTSD, depression, and anxiety (Benca, 1996) and are one of the strongest risk factors to develop psychiatric disorders (Breslau, et al., 1996; Lavie, 2001). The next section will address frequently reported sleep disturbances after traumatic experiences, and the link to psychiatric disorders associated with trauma. The main focus is on PTSD because it is the most prominent disorder after trauma (Bleich, et al., 1997). In addition, a circadian rhythm disorder and the circadian preference of eveningness are described due to the importance of the sleep changes appearing during adolescence.

Sleep and PTSD. Core symptoms of PTSD are likely to affect sleep (Babson et al., 2011; Caldwell & Redeker, 2005; Pillar, Malhotra, & Lavie, 2000). First, re-experiencing the event may occur during daytime as intrusive thoughts or during nighttime in form of nightmares, affecting the quality of sleep. Second, increased arousal or bodily activation may lead to sleep difficulties due to muscle tension, increased alertness and restlessness (American Psychiatric Association, 2000; World Health Organization, 1992, 2004). The third symptom is avoidance of elements reminding the victims of the traumatic event. These elements serve as stressors, typically increasing the level of anxiety. Victims of traumatic events may become afraid of the dark, of being alone, or closing their eyes and re-experiencing the event in vivid dreams, possibly causing them to avoid going to bed at night (Inman, Silver, & Doghramji, 1990). Frequently reported sleep complaints among PTSD patients are difficulties falling asleep, restless sleep, frequent awakenings from sleep (with further difficulties to again fall asleep), shorter sleep duration, excessive daytime sleepiness, nightmares and early morning awakenings (Dawood, Pillard, Horvath, Revelle, &

Bailey, 2000; Neylan et al., 1998; Pillar, et al., 2000; Waldrop, Back, Sensenig, & Brady, 2008).

Sleep difficulties have traditionally been considered a secondary symptom of PTSD, even though sleep disturbances are reported among 70-91% of those who suffer from PTSD (Maher, Rego, & Asnis, 2006; Ohayon & Shapiro, 2000). Secondary symptoms typically develop during the course of the underlying disorder (Spoormaker & Montgomery, 2008). However, several studies have found sleep difficulties to be present before the development of PTSD, even serving as predictors for future psychopathology (Lavie, 2001). Subjective sleep complaints are found after experiencing motor vehicle accident, prior to the development of PTSD (Koren, Arnon, Lavie, & Klein, 2002). The sleep complaints a month after the accident served as predictors for the development of PTSD a year after the accident (Koren, et al., 2002). Likewise, sleep disturbances among children and adolescents 24 months after Hurricane Katrina predicted posttraumatic stress symptom severity 30 months after the disaster (T. H. Brown, Mellman, Alsano, & Weems, 2011).

Treatment of sleep disturbance after a traumatic experience may often lead to improvement of both sleep and psychiatric diagnoses such as PTSD, depression and anxiety (Krakow et al., 2001; Krakow, Melendrez, Johnston, Clark, et al., 2002). Treatment of PTSD alone has an effect on symptoms of sleep disturbance, but rarely restores a normal sleep pattern (Galovski, Monson, Bruce, & Resick, 2009; Zayfert & DeViva, 2004). Further, untreated sleep disturbances may contribute to maintain PTSD (Krakow et al., 2000). These relations may indicate that disturbed sleep is not just a secondary symptom, but rather a separate condition often seen after traumatic experiences (Spoormaker & Montgomery, 2008), which may be in need of specific

treatment. These findings emphasize the potential importance of the quality of sleep in the aftermath of a traumatic experience.

Empirical studies to date supports that sleep disturbances have a complex but central role in PTSD (Kovachy et al., 2013). Some studies report REM-sleep abnormalities in PTSD patients, but these abnormalities often differ in character (Ross et al., 1994). In spite of inconsistencies there is a consensus that REM- disruptions affect the emotional processing during sleep among these patients (Engdahl, Eberly, Hurwitz, Mahowald, & Blake, 2000; Hurwitz, Mahowald, Kuskowski, & Engdahl, 1998; Ross, et al., 1994). Neurobiological changes like disruptions in the noradrenergic activity are also considered to contribute to sleep difficulties among PTSD patients (Mellman, Kumar, Kulick-Bell, Kumar, & Nolan, 1995). Noradrenergic cells show high firing rate during wakefulness, and decrease during non-REM sleep. Many noradrenergic cells in locus coeruleus cease firing during the transition from non-REM sleep to REM sleep (or during REM sleep). They regain their activity once REM sleep has terminated (Gaillard, 1985). Studies of PTSD patients show that excretion of one norepinephrine metabolite (MHPG) does not decrease during the night as it does among healthy controls (Mellman, et al., 1995). These abnormalities in the central nervous system's noradrenergic system may be related to the hyperarousal state found in PTSD patients, which again are related to development of insomnia (Pillar, et al., 2000).

Insomnia. ICD-10 define insomnia as "a condition of unsatisfactory quantity and/or quality of sleep, which persists for a considerable period of time, including difficulty falling asleep, difficulty staying asleep, or early final wakening" (World health Organization, 2004, p. 180). Insomnia is frequently reported among PTSD patients and people exposed to trauma (Ohayon & Shapiro, 2000). Insomnia patients

report more depression and anxiety disorders than people without insomnia (Taylor, Lichstein, Durrence, Reidel, & Bush, 2005). Among Utøya survivors, 57.1 % met the criteria for insomnia 18-30 months after the terror attack compared to 20.5% of the control subjects. The survivors had shorter sleep duration, more problems falling asleep, more difficulties in their lives because of excessive sleepiness, and were overall less satisfied with their sleep than control subjects. Among the survivors, 83.33 % had insomnia with recurrent depression, 80 % with panic disorders and 75 % with PTSD (Grønli, Ousdal, Melinder, et al., 2014).

Nightmares. Nightmares are described in ICD-10 as "dream experiences loaded with anxiety or fear [...] and usually includes themes involving threats to survival, security, or self-esteem". The dreamer often recalls the content vividly, and the same themes can occur several times (World Health Organization, 2004, p. 187). Nightmares are common after traumatic incidents (Krakow, Schrader, et al., 2002), and the frequency of nightmares is greater among people with psychiatric disorders, such as PTSD, anxiety and depression (Inman, et al., 1990; Levin & Fireman, 2002; Zadra & Donderi, 2000). Among PTSD patients, it is reported that 75 % experienced unpleasant dreams (McFarlane, Bookless, & Air, 2001) and 18.8% had nightmares at least once a month (Ohayon & Shapiro, 2000). PTSD patients who are victims of aggression are more likely to report nightmares compared to PTSD patients experiencing other types of trauma (Ohayon & Shapiro, 2000). Among Utøya survivors, 82.2 % reported nightmares consisting of negative content (42.8 %) and strong emotions (57.1 %). All Utøya survivors that reported nightmares also qualified for a psychiatric disorder, such as recurrent depression, panic disorder and/or PTSD (Grønli, Ousdal, Melinder, et al., 2014).

It is important to emphasize that a nightmare is a normal phenomenon. Many people experience nightmares, although not exposed to trauma or psychiatric disorders (Berge, 2007). It is suggested that the discomfort associated with the nightmares is linked to psychiatric disorders, and not the frequency of nightmares alone (Krakow & Zadra, 2006). Nonetheless, an association may exist between nightmares and sleep difficulties, such as initiating sleep, restless sleep and awakenings during night (Krakow & Zadra, 2006).

Sleep-related movement disorders (SMD). SMD are sleep disorders characterized by simple and stereotyped movements that disturb sleep (Thorpy, 1990). Periodic limb movement disorder and restless legs syndrome (RLS) are examples of sleep movement disorders where repetitive movements of limbs and irresistible urge to move the legs are prominent (Thorpy, 1990). SMD are more common among PTSD patients than among the general population (Maher, et al., 2006; Ohayon & Shapiro, 2000), and are proposed to contribute to the subjective sleep complaints reported in PTSD patients (Lavie, Hefez, Halperin, & Enoch, 1979). Patients with PTSD are more likely to report excessive body movement during sleep compared to insomnia patients without PTSD (Inman, et al., 1990). RLS was detected in 37.1% of the Utøya survivors compared to 7.7 % of the control subjects 18-30 months after the attack. Sixty percent of the survivors with RLS had recurrent depression, 40 % panic disorder and 20 % PTSD (Grønli, Ousdal, Milde, et al., 2014).

Sleep disordered breathing (SDB). SDB is common among PTSD patients (Maher, et al., 2006). SDB is characterized by disordered breathing during sleep, and include disorders such as obstructive sleep apnea (OSA) and central sleep apnea(Thorpy, 1990). Nightmares, anxiety, depression, sleep quality, quality of life, and

PTS symptoms are worse among SDB patients than in the general population (Krakow et al., 2004; Krakow, Melendrez, Johnston, Warner, et al., 2002). Utøya survivors reported more OSA (20% versus 0 %) than control subjects. All Utøya survivors with OSA were diagnosed with PTSD, whilst 50 % were diagnosed with panic disorder and recurrent depression (Grønli, Ousdal, Milde, et al., 2014).

Delayed sleep phase disorder (DSPD) and eveningness-types. DSPD, earlier known as delayed sleep phase syndrome (DSPS), is an involuntary exaggerated sleep phase delay regarded as a problem by the patient (American Academy of Sleep Medicine, 2005). It is considered the most common circadian rhythm sleep disorder (Dagan & Eisenstein, 1999), especially among adolescents (Thorpy, Korman, Spielman, & Glovinsky, 1988). The onset of DSPD usually occur during adolescence, and is characterized by a delayed major sleep interval with symptoms of sleep onset insomnia and difficulties with awakening at times that are desirable in line with the social norm and school schedules (Thorpy, 1990). They typically do not initiate sleep before 02:00 until 06:00, and have difficulties waking up to fulfill their daily chores during morning hours. This typically makes the sleep duration during weekdays shorter than desired (American Academy of Sleep Medicine, 2005). Daytime sleepiness is often observed, in addition to challenges at school/work and trouble functioning during the day (American Academy of Sleep Medicine, 2005; Crowley, et al., 2007). DSPD is associated with multiple negative outcomes such as lower school grades (Saxvig, Pallesen, Wilhelmsen-Langeland, Molde, & Bjorvatn, 2012), learning disabilities (Dagan & Eisenstein, 1999), psychosocial impairment, poor academic performance (Thorpy, et al., 1988), smoking and alcohol use, anxiety, depression (Saxvig, et al., 2012), and personality disorders (Dagan, Stein, Steinbock, Yovel, & Hallis, 1998; Saxvig, et al., 2012). They are usually

frustrated over not finding a way to fall asleep more quickly, and often try to go to bed earlier, ask friends or parents to wake them up etc. (American Academy of Sleep Medicine, 2005). During weekends, adolescents with DPSD often have a rebound-sleep displayed as a significant increase in total sleep time compared to weekdays (Saxvig, et al., 2012; Thorpy, et al., 1988). This emphasizes that the sleep quality and duration is only affected indirectly, due to the abnormalities in the circadian rhythm of sleep and wakefulness (American Academy of Sleep Medicine, 2005).

Prevalence is unclear, ranging from 0.48-16 % (American Academy of Sleep Medicine, 2005; Gradisar et al., 2011; Gradisar, Gardner, et al., 2011; Hazama, Inoue, Kojima, Ueta, & Nakagome, 2008). However, symptoms associated with DSPD appear to be common among Norwegian high school students (Saxvig, et al., 2012). The etiology of DSPD is not fully understood, but it is assumed to include a combination of multiple mechanisms, such as circadian, homeostatic, genetic, psychological traits and behavioral factors (Aoki, Ozeki, & Yamada, 2001; Oren, Turner, & Wehr, 1995; Ozaki, Uchiyama, Shirakawa, & Okawa, 1996). Studies have revealed possible circadian deficits among DSPD patients. They seem to have a longer sleep period between nadir (body temperature at its lowest during night) and wake up time than healthy controls have (Ozaki, et al., 1996). In addition, DSPD patients show an increased suppression of melatonin when exposed to light compared to controls, indicating that increased light sensitivity may be a precipitating or maintaining factor in DSPD (Aoki, et al., 2001). Deficits in the homeostatic system have also been examined as part of the etiology of DSPD. When sleep deprived, DSPD patients have impaired ability to compensate for lost sleep, compared to healthy controls (Uchiyama et al., 1999; Uchiyama et al., 2000).

Even after 24 hours awake, DSPD patients struggle to go to sleep when melatonin levels are low during daytime (Uchiyama, et al., 1999).

Some suggest that an exaggerated sleep phase delay is closely related to psychiatric disorders (Reid et al., 2012). The altered circadian rhythmicity seen among DSPD patients and normal eveningness-types may reduce their possibility to join social activities and impair their ability to have daytime work, which both are associated with better mental health (Reid, et al., 2012). A study of DSPD patients and normal eveningness-types (without DSPD) found that 70 % of all participants had at least one former episode with a psychiatric disorder, such as mood disorder, anxiety disorder or substance abuse in the past. Former PTSD was reported among as many as 15 %, and current PTSD among 8 % of the participants (Reid, et al., 2012). Other studies support the findings that eveningness-types may be at risk for developing psychiatric disorders, even though they do not report the same subjective complaints and impairments during daytime as DSPD patients (Hirata et al., 2007; Mansour et al., 2005; Ong, Huang, Kuo, & Manber, 2007). Even though PTSD seems to be a disorder that is apparent among individuals with preferable eveningness and DSPD patients, specific research on DSPD after trauma is scarce.

The reported findings described in this section depict the complexity and comorbidities between sleep disorders and psychiatric disorders. Sleep difficulties are closely intertwined with both traumatic experiences and several psychiatric disorders. Sleep disturbances may lead to development of psychiatric disorders, and psychiatric disorders may lead to impaired sleep. Further, traumatic experiences may lead to sleep difficulties, and sleep difficulties may lead to impaired recovery from traumatic events

due to lack of the suggested restorative function of sleep (Germain, Buysse, & Nofzinger, 2008). Figure 1 illustrates these connections.

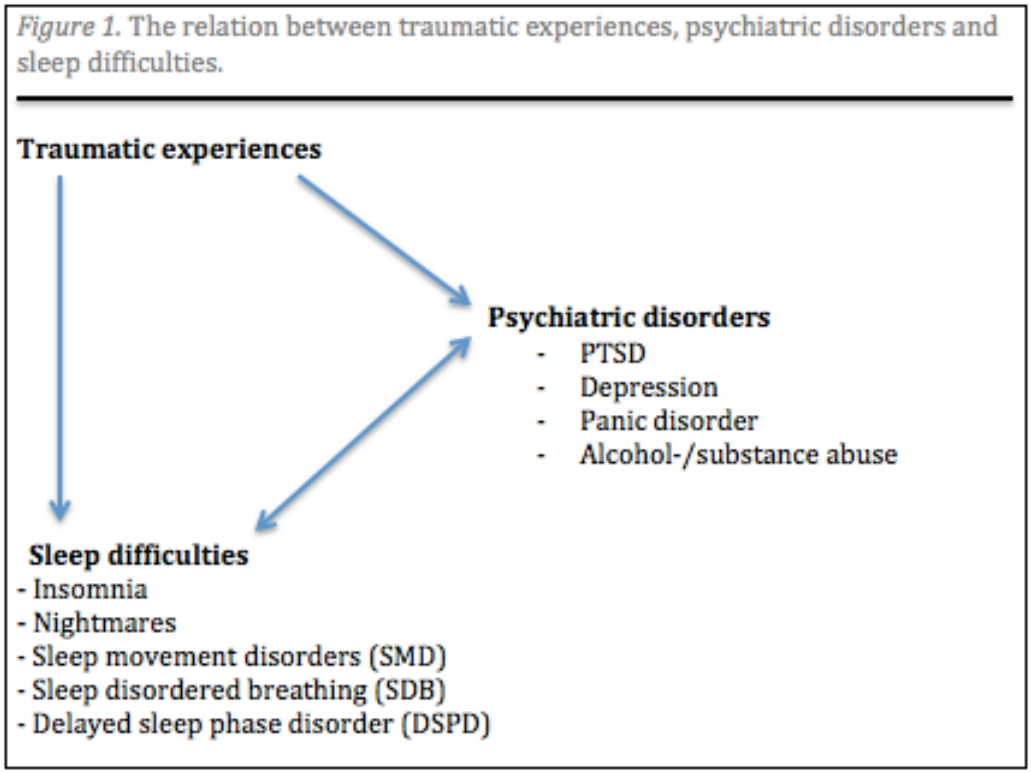

Figure 1. The relation between traumatic experiences, psychiatric disorders and sleep difficulties.

Different Ways to Study and Register Sleep

To investigate sleep quality and the circadian rhythm of sleep and wakefulness, several kinds of registrations can be utilized. Different ways to register sleep is often divided into subjective and objective measurements. What kind of sleep monitoring researchers use depends on relevant sleep parameters, the amount of information required and the size of the population of interest. In addition, there needs to be an assessment of the budget and time consumption; some measurements are quite advanced, time consuming and expensive.

Subjective and objective sleep measurements. Subjective sleep measurements are subjectively reported information about perceived sleep and the circadian rhythm of sleep and wakefulness. Examples of such measurements are sleep diaries, self-reported

questionnaires and interviews (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989; Krystal & Edinger, 2008; Lichstein & Riedel, 1994; Moul, Hall, Pilkonis, & Buysse, 2004; Pallesen et al., 2008; Wolfson et al., 2003). Actigraphy is an example of objective measurement of sleep (Kovachy, et al., 2013), and was developed in the 1970's (Ancoli-Israel et al., 2003). The actigraph is a digital wrist 'watch' registering activity/inactivity (Ancoli-Israel, et al., 2003). The advantage of the actigraph is that it record data for a longer period of time, in a natural sleep environment and it is cost-effective (Ancoli-Israel, et al., 2003; McCall & McCall, 2012). Actigraphy may very well be used to measure the circadian rhythm of sleep and wakefulness, stability of the sleep phase and longitudinal information about the sleep/wake pattern (Ancoli-Israel, et al., 2003; Sadeh, 2011).

The golden standard among objective sleep measurements is polysomnography (PSG), which gets recordings from EEG, electromyography (EMG) and electrooculography (EOG). PSG provides information about the quality and quantity of the different sleep stages. In spite of its advantages, PSG has some considerable limitations; it is an expensive method and it is usually applied only for 1-2 nights of sleep (Chambers, 1994; Meltzer, Walsh, Traylor, & Westin, 2012).

To validate the precision of the actigraph sleep data, PSG is used as a reference standard (Edinger, Means, Stechuchak, & Olsen, 2004; Marino et al., 2013; Meltzer, et al., 2012) comparing sensitivity (detect sleep), specificity (detect wake) and accuracy (detect sleep and wake) by an epoch-by-epoch comparison (Kushida et al., 2001; Meltzer, et al., 2012). Studies support that the actigraph has a high sensitivity (Blood, Sack, Percy, & Pen, 1997), but the specificity is poor (de Souza et al., 2003). The obvious limitation with actigraphy is to detect wakefulness during the sleep interval

(Meltzer, et al., 2012) and to detect accurate onset of sleep (Vallieres & Morin, 2003). Nevertheless, studies conclude with an overall high accuracy for actigraph compared to PSG (Marino, et al., 2013; Meltzer, et al., 2012; Sadeh, Hauri, Kripke, & Lavie, 1995).

The American Academy of Sleep Medicine (AASM) has asked for additional evaluation of the reliability and validity of the actigraph compared to other measurements such as PSG. They want studies to evaluate the standard settings of the actigraph and the accuracy in detection of sleep variables (Morgenthaler et al., 2007).

Measurements of sleep in clinical populations. Actigraphy is commonly used in research of sleep in both general and in clinical populations (McCall & McCall, 2012). Studies of elderly (Marino, et al., 2013), depressed patients with insomnia (McCall & McCall, 2012; Sadeh & Acebo, 2002), patients with sleep apnea (Sadeh & Acebo, 2002), and patients with major depression (Jean–Louis et al., 2000) support the notion that actigraphy may misinterpret quiet wakefulness as sleep. The actigraphy's ability to detect sleep decrease when the quality and amount of sleep decreases, which is common among patients with sleep disorders (Hauri & Wisbey, 1992). Even though actigraph has limitations such as detection of quiet awakeness, studies conclude that the actigraph is accurate enough to estimate different parameters among different clinical populations (Marino, et al., 2013).

Research suggests that PTSD patients may perceive the level of safety in the laboratory during PSG measurements as higher than at home (Hurwitz, et al., 1998). This may give another impression of sleep compared to when registered subjectively or by actigraphy in their home environment (Hurwitz, et al., 1998). Sleep duration and number of awakenings may vary over a period of time, which is found especially among PTSD patients (Khawaja, Hashmi, Westermeyer, Thuras, & Hurwitz, 2013). PSG

measures sleep for a few days only, which may lead to a lack of a clear understanding of sleep disturbances among PTSD patients (Khawaja, Hashmi, Aftab, Westermeyer, & Hurwitz, 1969). It might be helpful to use actigraphy as an objective sleep measurement, to be able to measure sleep over a longer period of time, and in the patient's natural sleep environment (Khawaja, et al., 1969).

Aim of This Study

Most of the previous terror attacks investigated have occurred in large cities such as New York (Galea, et al., 2002), London (Rubin, et al., 2005), Madrid (Miguel- Tobal, et al., 2006) and Oklahoma City (North, et al., 1999), affecting random crowds of people of all ages, and making the level of exposure and proximity heterogenous. In contrast, the terror attack on Utøya Island provided a unique homogenous group of adolescents, all were located in a small geographical area, and the majority of whom had friends or relatives on the island. This scenario allows investigation of sleep after trauma in a population with similar ages, experiences, losses, and level of proximity.

Previous research on how trauma affects sleep has mainly focused on PTSD patients. Hence, knowledge of sleep disturbances in a population exposed to trauma without having developed any psychiatric disorder is scarce, almost not existing (Breslau, et al., 1996; T. H. Brown, et al., 2011; Khawaja, et al., 2013). In addition, most studies on sleep after trauma have been conducted shortly after the event. We aim to examine effects of trauma 18-30 months after the event, in a general population exposed to trauma, to increase our understanding regarding the effect of trauma on sleep.

The goal of the current study is to examine long-term effects of the Utøya attack on sleep quality and the circadian rhythm of sleep and wakefulness among Utøya

survivors, regardless of psychiatric diagnoses. We hypothesize that the circadian rhythm of sleep and wakefulness is affected among Utøya survivors. Due to a normally registered social jet lag among adolescents (Wittmann, et al., 2006), we only expect to find differences between the groups during weekdays. Based on earlier research of sleep after trauma (Waldrop, et al., 2008), we expect to find earlier wake up times among Utøya survivors compared to control subjects. As far as we know, the research is scarce on differences in when individuals exposed to trauma get out of bed in the morning compared to individuals not exposed to trauma. Even though we expect that Utøya survivors wake up earlier, we expect no differences in get up times between the two groups measured by actigraphy. Instead, we expect that Utøya survivors stay in bed trying to sleep even though they wake up early. We also expect that Utøya survivors have poorer sleep quality compared to control subjects.

In addition to investigate the effects of trauma, we want to contribute to the growing amount of research on circadian rhythm of sleep and wakefulness among adolescents in general. Based on previous research on adolescent's sleep patterns and preferences of eveningness (Carskadon, et al., 1993), we hypothesize that all participants have a social jet lag, shown as a delayed sleep pattern during weekends. Summarized, these three main hypotheses are complied:

- Circadian rhythm of sleep and wakefulness: 1) Utøya survivors have significant later bedtimes and sleep onset than control subjects during weekdays. 2) Utøya survivors wake up significantly earlier than control subjects, but there are no significantly differences in when the two groups decide to go out of bed in the morning ("get up time")

- Sleep quality: Utøya survivors have poorer sleep quality than control subjects, shown as longer sleep onset latency (SOL), more wake after sleep onset, (WASO), lower sleep efficiency (SE), and shorter total sleep time (TST).
- Social jet lag: All participants regardless of group have later bedtimes, sleep onset, wake up times, get up times, longer total sleep time (TST) and time in bed (TIB) during weekends than during weekdays.

Methods

Ethics

The Regional Committee for Medical and Health Research Ethics, South East Norway has approved the study. It is conducted in line with the Declaration of Helsinki, including inter alia the written information and the passive consent procedure.

Subjects

The data presented in this study is part of two major research projects where the goal has been to investigate long-term effects on cognition and underlying neural networks among Utøya survivors after the terror attack that happened 22nd of July 2011. One project is headed by Professor Annika Melinder at Department of Psychology, University of Oslo, whilst the other project is headed by associate Professor Anne Marita Milde, Department of Biological and Medical Psychology, University of Bergen. Only objective data on sleep will be reported in this thesis.

Utøya survivors living in Hordaland and Rogaland were recruited from The Resource Centre of Violence, Traumatic Stress and Suicide Prevention Western region (RVTS West) by a posted letter of information and an invitation to participate in the study. Utøya survivors living in Oslo were recruited by letter of invitation distributed through their lawyers or through a letter of invitation and contact information posted on

the homepage of the national support group ("Den nasjonale støttegruppen etter 22. juli hendelsene"). Two recruitment rounds were performed.

Control subjects were matched on age, gender, educational level and socio-demographical variables. Controls living in Hordaland and Rogaland were recruited from political youth parties by written invitations given out on meetings in different political youth parties, while those living in Oslo were recruited from the University of Oslo and from local high schools.

The exclusion criteria among Utøya survivors and control subjects were 1) endocrinological illness, 2) neurological illness or previous head trauma leading to unconsciousness for more than 10 minutes, 3) metal implants not compatible with MRI scanning, and 4) pregnancy. Additional exclusion criteria for control subjects were 1) being involved in the Utøya massacre or knowing anyone who was involved, 2) previous history of psychiatric illness, and 3) substance abuse, determined by a short interview and MINI questionnaire.

There were 495 survivors after the attack at Utøya. Of all the survivors, 8.48% participated in this study. In total, 42 Utøya survivors (Oslo: 23, Bergen: 19) and 46 control subjects (Oslo: 20, Bergen: 26) agreed to wear the actigraph, altogether 88 participants. The age span was between 16 to 25 years. Exact data about age and gender has not yet been decoded for actigraphy data. However, we have information about the participants in general for the main projects in Oslo and Bergen. The mean age was 20.83 (SD=2.56) among Utøya survivors recruited from Oslo, and 20.9 (SD=1.6) recruited from Bergen. The sample from Oslo consisted of 38% women, and the sample from Bergen consisted of 62% women. The mean age was 21.46 (SD=3.07) among

control subject recruited from Oslo, and 20.4 (SD=2.2) from Bergen. The sample from Oslo consisted of 54% women, and the one from Bergen consisted of 59% women.

Design

Eighteen months after the Utøya attack, the two projects started to recruit survivors and controls. To investigate sleep disturbances, actigraphs were distributed to the participants on the laboratory day for neuropsychological- and physiological tests. The data was collected for 7 days during the time window of 18-30 months after the attack.

Measurements of sleep by actigraphy. To be able to monitor sleep ambulatory (as inactivity compared to activity), an actigraph of the type Actiwatch Spectrum (Philips Respironics) was used. This 'watch' is waterproof and includes time and date indicators, event markers and seven data channels to monitor photic illuminance. The participants received a description of how to use the Actiwatch, and were instructed to wear it for at least one week on the non-dominant hand. Also, they were told to prevent clothes from shielding the watch from light exposure. They were instructed to press a button (event marker) on the watch to mark the time they went to bed and turned off the lights (bedtime) and when they finally got out of bed in the morning (get up time). Data were collected during weekdays and weekends. Weekdays were defined as Sunday-, Monday-, Tuesday-, Wednesday- and Thursday nights. Weekends were defined as Friday- and Saturday nights.

Analysis of Actigraphy Data

Analyses of the recorded data were administered by the use of Actiware software (version 6.0.2., Philips Respironics). All activity data was collected in 30-second epochs, scored as wake or sleep by the use of a selected sensitivity threshold. The scoring depends on the activity counts for the particular epoch, in addition to the

adjacent epochs. Wake is scored when number of counts exceeds the selected threshold; Wake = total activity counts > wake threshold value. Sleep is scored if the number of activity counts is less or equal to the wake threshold; Sleep = total activity counts ≤ wake threshold value. Actiware allows different activity thresholds; low, medium and high, corresponding to 20, 40 and 80 counts per epoch, respectively. Based on previous research (Meltzer, et al., 2012), medium wake threshold was applied, which corresponds to 40 activity counts per epoch and is the default threshold set by Actiware.

Rest interval, sleep interval and sleep parameters. The rest interval is defined as "the period between bedtime and get up time". This interval is indicated by low activity, since the subject is probably resting, and defines the in-bed period. Actiware automatic detect 'major' and 'minor' rest intervals based on proprietary algorithms. A 'major' rest interval is defined as periods of more than 3 hours of low activity and indicates when the subject is presumably resting in bed for a longer period of time. 'Minor' rest interval is defined as less than 3 hours and indicates when the subject is presumed to sleep at other times than the main sleep period, like a daytime nap. In this thesis we only analysed the major sleep intervals. The researcher can also establish the rest intervals manually.

Sleep interval is automatically set based on algorithms once the rest interval is set. Sleep interval is defined as "the period between sleep onset and sleep end", the time when the subject is presumed to sleep. To calculate sleep onset and end, Actiware uses the selected inactivity threshold, which is the period of inactive time selected for sleep onset to occur, e.g. 5 minutes or 10 minutes of inactivity. Actiware identifies the first time all epochs but one are scored as inactive for the selected time period, within the rest interval. The first epoch of this sequence will be identified as the sleep onset.

Hence, using 5 minutes of inactivity threshold, 9/10 of the 30 seconds epochs need to be registered as inactive for sleep onset to be set. A similar algorithm is applied to define sleep end, where the last epoch of the sequence is identified as sleep end. Actiware's default setting is a 10 minutes of inactivity threshold. Sleep interval is not possible to establish manually.

Multiple sleep parameters are given as outputs using similar terminology as PSG, but the technologies are distinctly different. The actigraph provide several sleep parameters investigating different aspects such as sleep quality, circadian rhythmicity of sleep and wakefulness, light illuminance and activity. This thesis utilize the following parameters: bedtime (start of the rest interval, when the participant goes to bed), sleep onset (start of sleep interval, when the participant goes to sleep), wake up time (end of sleep interval, when the participant wakes up), get up time (end of the rest interval, when the participant goes out of bed), time in bed (TIB; duration of the rest interval), total sleep time (TST; sum of total sleep time during the sleep interval), sleep onset latency (SOL; the time required for sleep to start after initiating the intent to sleep, registered as the time between bedtime and sleep start), sleep efficiency (SE; the total sleep time divided by time in bed multiplied by 100) and wake after sleep onset (WASO; the total number of minutes scored as awake within the sleep interval). See Appendix for a review.

When we systematically went through the automatically coded rest intervals, we observed that some of the participants were registered as resting or sleeping even though the actogram showed activity (and light exposure). Further, some participants were registered as being awake even though the actogram showed inactivity for a longer period of time (and no light exposure).

Figure 2 is an example of a participant's actogram. On Friday 13.12.2013 Actiware registered bedtime approximately at 01:15, even though there are low activity for a longer period and almost no light exposure from 22:00. The participant marked his/her bedtime at 22:00. A similar pattern is also evident on Monday 16.12.2013. However, Wednesday 18.12.2013 is different. Here it is most likely that Actiware registered bedtime earlier than what is true, since the participant is active, he/she marks the bedtime at a later time point and is exposed to high light illumination.

Figure 2. Automatic Coded Actogram

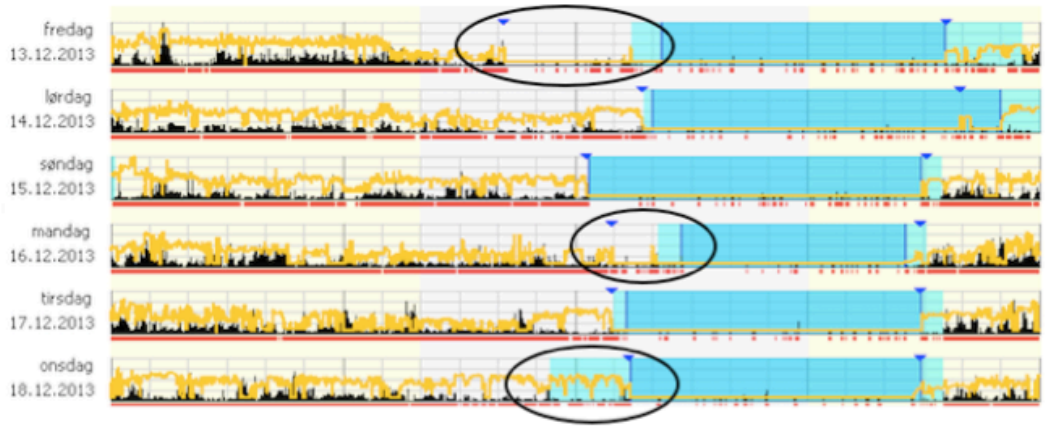

Figure 2. Actogram of participant 109 with automatically coded rest intervals. Ten minutes of inactivity threshold is set for sleep intervals. Rest intervals are presented by "aqua shading". Sleep intervals are presented by "blue shading". Yellow line indicates light illumination, and black bars indicate activity. Sleep/wake scores are shown below activity; red indicates wake and transparency indicates sleep. Event markers for bedtime and get up time are shown as "blue wedges".

Automatic versus manually coded rest interval. To our knowledge, only one study has addressed the differences in use of automatically and manually coded rest intervals using actigraphy as requested by AASM (Boyne, et al., 2013) (Morgenthaler, et al., 2007). In that study, human determination of the rest interval led to better correlations with PSG on sleep onset, wake up time, TST, WASO and SE (mean *r*=

0,640) compared to automatically coded rest intervals (mean *r*= 0,406) (Boyne, et al., 2013). Based on these findings and several questionable observations of the actograms in our sample, we wanted to add a manually coded rest interval in addition to the automatic, to investigate if there were any significant differences in the outcome.

Manually coded rest interval. Together with our supervisor, other sleep researchers and the limited literature on manual coding (Boyne, et al., 2013), we defined specific criteria for how to manually code a rest interval. Our main indicator for onset and end of rest intervals was activity level. Additionally, we used the benefit of light illumination and event marker. Major rest interval was defined according to criteria for onset, maintenance and end (see Figure 3 for an overview). 1) Onset: "significant and sustained decrease in activity". Help criteria were the event marker and/or significant and sustained decrease in light exposure. In cases where a sustained decrease in activity was present but the light intensity was constant, this was interpreted as another person controlling the light. In these cases light exposure was not used as a help criterion. In cases where the event marker differed from the decrease in activity, the event marker was not used as a help criterion. 2) Maintenance: "significant and stable decrease in activity for more than 3 hours". A short period of increased activity and/or light exposure was interpreted as a shorter time out of bed, to go to the bathroom, etc. 3) End of rest interval: "significant and sustained increase in activity". Help criteria were event marker and significant and sustained increase in light exposure. In cases where the activity was low and stable but light intensity increased, this was interpreted as another person controlling the light, and not used as a help criterion. In cases where the event marker differed from increase in activity, the event marker was not used as a help criterion.

Figure 3. Criteria for Manually Coded Rest Interval

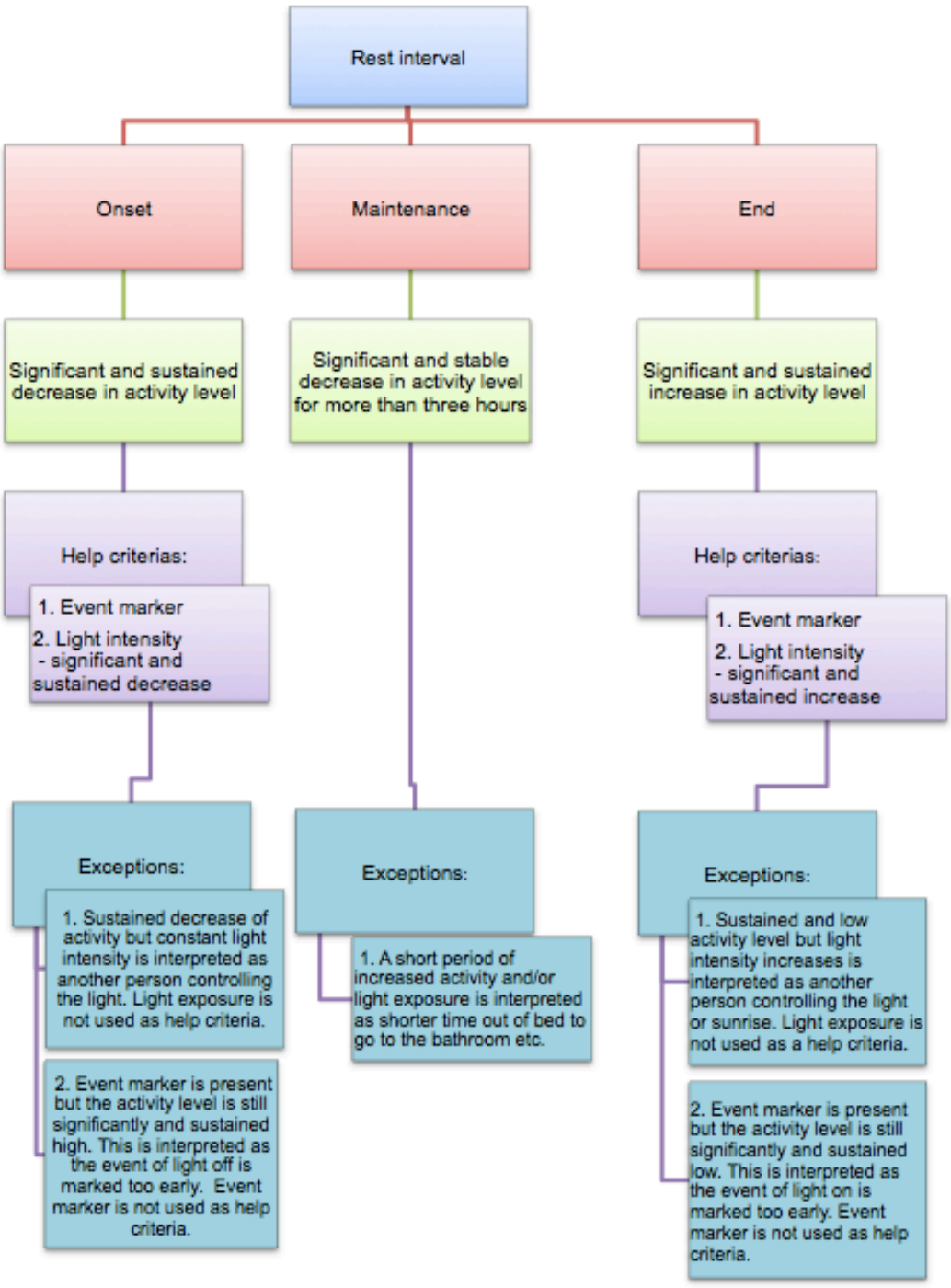

Figure 3. Criteria for manually coded rest interval; onset, maintenance and end. Help criterias and exceptions are described.

The actogram of participant 109 is shown in Figure 4, now with manually coded rest intervals. Here, on Friday 13.12.2013, the bedtime is coded from approximately 22:00, which is more than 3 hours earlier than the automatically coded bedtime. On Monday 16.12.2013, the rest interval is set to approximately 01:00, which is 1 hour earlier than the automatically coded rest interval. The small period with activity and light illumination at 01:30 and 02:00 on these nights are examples of what is interpreted as a shorter period out of bed to go to the bathroom etc. On Wednesday 19.12.2013, the rest interval is now coded from approximately 01:30, which is 2 hours later than the automatically coded rest interval. This is an example of the use of light illumination to determine the onset of rest interval when the activity level is low, late in the evening. All manually coded rest intervals in this example correspond to the participant's use of event marker for bedtimes.

Figure 4. Manually Coded Actogram

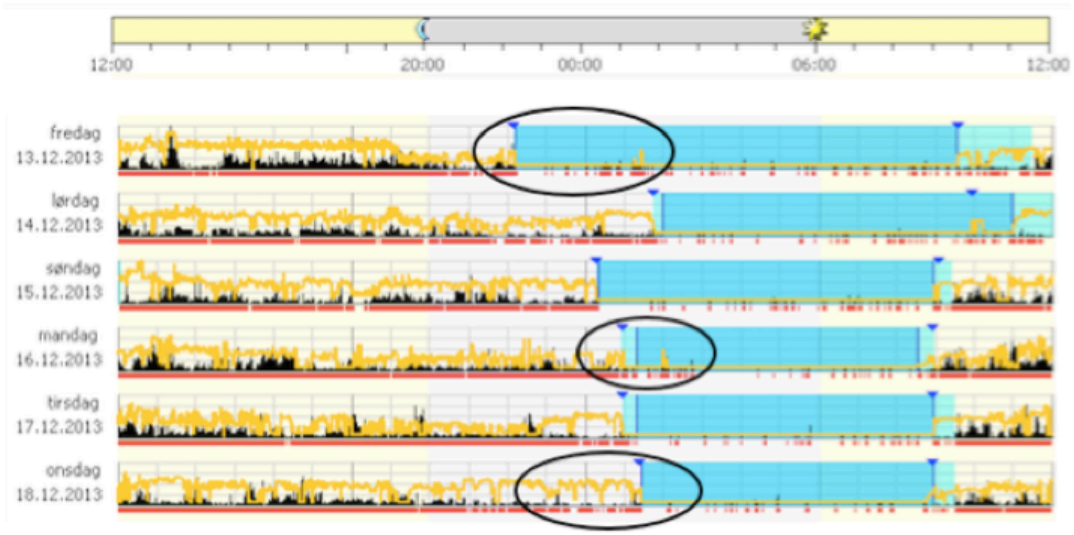

Figure 4. Actogram of participant 109 with manually coded rest interval. Ten minutes inactivity threshold is set for sleep interval.

We decided to do a preliminary analysis to compare automatic and manual coding, with two distinct settings for the sleep interval: 1) Automatically coded rest interval with both 5 and 10 minutes inactivity threshold for sleep onset and end; 2) Manually coded rest interval with both 5 and 10 minutes inactivity threshold for sleep onset and end. We chose two different settings for the sleep onset and end to explore whether there were any notable differences between automatic and manual coding when using different inactivity thresholds.

Statistical Analyses

All statistical analyses were conducted using Statistica, version 10 (StatSoft Inc). A 2 (group) x 2 (automatic/manual coding) factorial analysis of variance (ANOVA) was conducted for the preliminary analysis. To examine the impact of using automatic versus manual coding on sleep parameters among Utøya survivors and control subjects, variance in standard deviations of all selected sleep parameters (see Appendix for review) were analysed. The analyses were conducted for both 5 minutes and 10 minutes inactivity threshold for sleep onset and end, and on weekdays and weekends separately. Sleep parameters were dependent variables, and Utøya survivors/control subjects and automatic/manual coding were independent variables.

Further, a 2 (group) x 2 (weekday/weekend) ANOVA was conducted for the averages of each sleep parameter, to examine differences between Utøya survivors and control subject, and differences between weekdays and weekends. Data from manually coded rest intervals, with 10 minutes inactivity thresholds was applied. Sleep parameters were dependent variables, and Utøya survivors/control subjects as well as weekday/weekend were independent variables. Significant effects in all analyses were further analyzed using post-hoc Fisher LSD (least significant difference) comparisons.

A significance level of p< .05 was set. In addition, descriptive analyses were conducted to retrieve the means and standard errors for each sleep parameter, divided in groups and weekdays/weekends. See attachment for illustrations of Tables and Figures.

Results

Preliminary Analyses of Automatically Versus Manually Coded Rest Intervals

Standard deviations of sleep parameters were used to assess the differences in automatically and manually coded rest intervals.

Five minutes of inactivity threshold for sleep interval. First, we compared automatically and manually coded rest intervals with 5 minutes as inactivity threshold for sleep onset and end. During weekdays, manual coding reduced the standard deviation in several sleep parameters (e.g. Graph 1 and 2), manifested by significant main effects of coding. The parameters were bedtime, sleep onset, get up time, WASO and TIB ($F_{1,83}$'s > 5,7, p<0.02, all). See Table 1 for more details.

Notably, the decreases in standard deviations were only evident in the control group (bedtime: p=0.015 vs =0.283; sleep onset: p= 0.034 vs p = 0.139; get up time: p=0.037 vs p= 0.201; WASO: p=0.029 vs p= 0.237; TIB: p=0.002 vs p= 0.292, control vs survivors respectively). Among Utøya survivors the sleep parameters did not change according to the use of coding. SOL showed an interaction effect between coding and group ($F_{1,83}$=5,9, p= 0.017). The post-hoc test shows that manual coding decreased the standard deviations of SOL among controls (p= 0.006), however the standard deviations among Utøya survivors were not changed (p= 0.482).

During weekends, there was a main effect of coding on SOL only, being more regular when manually coded then automatically coded, evident among both Utøya

survivors (p=0.016) and control subjects (p=0.021). No other main effects of other sleep parameters or interaction effects were significant in the weekend data.

Ten minutes of inactivity threshold for sleep interval. Afterwards, we compared automatically and manually coded rest intervals with 10 minutes inactivity threshold for sleep onset and end. During weekdays, there were main effects of coding on SOL and SE ($F_{1,82}$'s >8,0, p<0.006, e.g. Graph 3 and Graph 4). The post-hoc test shows that manual coding decreased standard deviations only among control subjects (SOL p=0.004 vs p=0.294; SE p=0.002 vs p=0.319, controls vs survivors, respectively). During weekends, main effect of coding was evident on SOL. This time, the post-hoc test revealed that manual coding reduced standard deviations only among Utøya survivors (p=0.011, control: p= 0.209). No other main effects or interaction effects were significant when using 10 minutes inactivity threshold.

Choice of Coding and Inactivity Thresholds: A Preliminary Discussion

Several automatically coded actograms showed miscalculations as described and illustrated in the method section. Our preliminary analysis revealed that manually coded rest intervals reduced the standard deviations in several sleep parameters. This was especially evident when using 5 minutes inactivity threshold for sleep onset and during weekdays. Furthermore, manual coding appears to be distinctly sensitive regarding subjects; control subjects versus Utøya survivors as well as weekdays versus weekends. As illustrated by Table 2, manual coding increases the F value in affected sleep parameters such as bedtime, sleep onset, SOL and WASO. These findings, together with previous literature describing stronger correlations between actigraphy and PSG on sleep parameters when the rest interval is coded manually (Boyne, et al., 2013), favour the use of manual coding.

The two different settings of 5 and 10 minutes inactivity threshold were used to visually explore differences between automatic and manually coded rest intervals. As illustrated by Table 2, the increase in F values by manual coding was especially evident when using 10 minutes threshold for sleep onset (see Table 2 for exploration of F values). It is reported a strong correlation between actigraphy and PSG on sleep onset when using 10 minutes of inactivity threshold, both for automatic (r=0.681) and manually (r=0.999) coded rest intervals (Boyne, et al., 2013). When using 5 minutes of inactivity threshold the correlations between actigraphy and PSG on sleep onset are reported to be negative (manual r= -0.682; automatic r=-0.151) (Boyne, et al., 2013). Similar tendencies are found for wake up time (Boyne, et al., 2013).

Taken all information into consideration, we decided to report results regarding sleep quality and circadian rhythm of sleep and wakefulness with manually coded rest intervals, and with 10 minutes inactivity threshold for sleep onset and end.

The Effect of Trauma on Sleep Quality and The Circadian Rhythm of Sleep and Wakefulness

Comparison of Utøya survivors and control subjects were conducted to explore long-term effect of trauma on sleep quality and the circadian rhythm of sleep and wakefulness. There was a main effect of group on parameters related to circadian rhythm: bedtime, sleep onset, wake up time and get up time ($F_{1,161}$'s >=5,7, p<0.018, see Table 3). All parameters showed later times in Utøya survivors than for control subjects (see Table 4).

As illustrated by Table 4, post-hoc tests reveal that the later bedtime and sleep onset seen among Utøya survivors compared to control subjects were evident during weekdays only. There were no differences in wake up time and get up time (Table 4).

There was no significant effect of group on parameters related to sleep quality: TST, SOL, SE and WASO ($F_{1,162}$'s <1,1, p>0.29). See Table 3 and 4 for more statistics.

The Effect of Weekend on Circadian Rhythm, TIB and TST: Social Jet Lag Among All Participants

To find out more about social jet lag among adolescents, we compared sleep pattern during weekdays and weekends for all participants. There was a main effect of weekend/weekday on all four parameters of circadian rhythm of sleep and wakefulness: average bedtime, sleep onset, wake up time and get up time ($F_{1,161}$'s >22,5, p<0.001, see Table 3) were all later during weekends than during weekdays, as illustrated by Table 5. No interaction effects were found.

Bedtime, sleep onset, wake up time and get up time were later during weekends in both Utøya survivors and control subjects (Table 5).

Further, there was a main effect of weekday/weekend on TIB and TST ($F_{1,161}$>4,9, p<0.028, see Table 3). As illustrated by Table 5, the TST and TIB were longer during weekends than during weekdays. The post-hoc test revealed no significant difference for TIB. TST was longer during weekends in Utøya survivors only (p=0.011, see Table 5).

Discussion

The main aim of the current study was to explore the long-term effects of traumatic exposure on sleep quality and circadian rhythm of sleep and wakefulness. Our hypothesis about circadian rhythm was that Utøya survivors would have later bedtimes and sleep onset, but no later get up times than control subjects during weekdays. However, we expected that Utøya survivors would wake up earlier than control subjects. In regards to sleep quality, we expected to find more sleep disturbances among

Utøya survivors compared to control subjects, shown as increased SOL and WASO, and decreased SE and TST. An additional aim was to examine social jet lag among adolescents in general. Here, our hypothesis was to find a social jet lag among all participants, displayed as later bedtimes and sleep onset, and later wake up times and get up times during weekends compared to weekdays.

Our results indicate that trauma affects the circadian rhythm of sleep and wakefulness, shown by later bedtimes and sleep onset among Utøya survivors compared to control subjects. The differences in bedtimes and sleep onset were only prominent during weekdays, which support our hypothesis. Inconsistent with our expectations, Utøya survivors woke up and got up later than control subjects. Together, our hypothesis on circadian rhythm of sleep and wakefulness is partly supported. Surprisingly, our results indicate that the trauma did not notably affect the quality of sleep. The expectation of a social jet lag among all participants was confirmed.

One explanation of the lack of findings on sleep quality might be that Utøya survivors in our study actually did not have any sleep difficulties at the time of measurement. The study took place 18-30 months after the attack. Several reactions of traumatic experiences normally fade after some time (Rubin, et al., 2007; Silver, et al., 2002). Thus, it might be possible that potential sleep difficulties have faded prior to the current study, or never occurred. However, when comparing our results with the survivors' subjective sleep- and psychiatric reports presented in the introduction, they tell a different story. These subjective results were collected at the same time and by the same participants as in our actigraphy study. Here, the survivors report significantly more sleep disturbances compared to control subjects (53.1% vs 13.6%) 18-30 months after the terror attack (Grønli, Ousdal, Melinder, et al., 2014; Milde, et al., 2014). We

will interpret and discuss our results, both independently and in the light of the subjective reports about sleep- and psychiatric disorders. Some considerations for clinical purpose and further research are outlined.

The Effect of Trauma on Sleep Quality and the Circadian Rhythm of Sleep and Wakefulness

Bedtime and sleep onset. Survivors after terror attacks may try to escape the intrusive memories by distracting themselves. Such distractions may include watching movies at night, drinking alcohol, being with friends as long as possible etc, thus exposing bedtime (Paasivirta et al., 2010). Later bedtimes and sleep onset among Utøya survivors give an impression of how Utøya survivors may struggle in the evening, not being able to settle down and go to bed. Thus, the later bedtimes among Utøya survivors registered by the actigraph may reflect an avoidance of going to bed. As reported by subjective data on this sample, the prevalence of PTSD among the survivors was 30% (Milde, et al., 2014), making symptoms of avoidance likely. In addition, 80% of the survivors with PTSD also reported symptoms of insomnia (Grønli, Ousdal, Melinder, et al., 2014). The combination of the objective actigraphy data and the subjective data support the relation between sleep disturbances, and presence of psychiatric disorders among the survivors.

Experiencing nightmares are common after trauma (Krakow, Schrader, et al., 2002), especially after being victims to acts of violence (Ohayon & Shapiro, 2000). The frequency of nightmares increases among individuals with psychiatric disorders such as PTSD, anxiety and depression (Inman, et al., 1990; Levin & Fireman, 2002; Zadra & Donderi, 2000). Later bedtimes among the survivors may be explained by fear of experiencing nightmares, which is found to be frequent among Utøya survivors. All

survivors with PTSD and/or panic disorder, and 83% of those reporting previous major depressive episodes, report experiencing nightmares (Grønli, Ousdal, Melinder, et al., 2014). This illustrates the close relationship between psychiatric disorders related to trauma and nightmares, which further may affect sleep.

Traumatic experience may also lead to the occurrence of several stressors which can trigger high anxiety levels among survivors with PTSD (Inman, et al., 1990). These stressors often reflect aspects of the event and remind the survivor of the event. These can for instance include becoming afraid of the dark, of being alone, for closing their eyes or re-experiencing the event etc. (Inman, et al., 1990). These are all probable stressors among Utøya survivors with PTSD. Thus, symptoms of avoidance, fear of experiencing nightmares, and the occurrence of anxiety provoking stressors are all likely to affect sleep and make it harder for the survivors to settle down and go to bed at night.

Sleep onset latency. Restlessness during the evening and difficulties initiating sleep is found to be prominent after experiencing trauma (Waldrop, et al., 2008). Our results of later bedtimes and sleep onset among Utøya survivors support these findings. However, an increased SOL is not evident in the actigraphy data, which may indicate no notable problems initiating sleep when they finally go to bed. On the other hand, actigraphy is found to be less reliable at detecting SOL (de Souza, et al., 2003), which may have affected the results from actigraphy measurements on this specific sleep parameter. Subjective data collected from the same sample reveal that the survivors report significantly longer SOL than controls ($p<0.001$) (Grønli, Ousdal, Melinder, et al., 2014). The differences between actigraphy and subjective reports may emphasize the actigraph's limitation in detecting SOL. The survivors report that it takes over 60

minutes for them to fall asleep, while the control subjects report using only 20 minutes on average (Grønli, Ousdal, Melinder, et al., 2014). These considerable differences should not be ignored. When settling for the night, and the quietness occurs, negative thoughts and intrusive memories of earlier experiences or worries about future uncertainties may be more prominent than during the day (World Health Organization, 1992), thus making it difficult for the survivors to fall asleep.

Wake after sleep onset. Frequent awakenings are common among PTSD patients (Pillar, et al., 2000). However, neither actigraphic recording nor subjective reports show more awakenings among the survivors. Actigraphy is considered less reliable at detecting WASO (Edinger, et al., 2004; Meltzer, et al., 2012), which may have affected the actigraphy results. One could assume that the survivors with PTSD or panic disorder have higher activity levels due to nightmares and anxiety, and that this activity more easily would have been registered as awakenings by the actigraph. In addition, several Utøya survivors reported symptoms of RLS, which would make them move frequently during sleep (Grønli, Ousdal, Milde, et al., 2014). They also reported more OSA (Grønli, Ousdal, Milde, et al., 2014), which is associated with poor sleep quality (Krakow, et al., 2004). Nonetheless, these putative movements and breathing disorders were not registered by the actigraph in this study. However, the survivors report having more periods of wakefulness that lasts for more than 30 minutes than control subjects ($p<0.001$) (Grønli, Ousdal, Melinder, et al., 2014). The combination of these results indicate that the survivors may not experience frequent awakenings, but that they struggle going back to sleep quickly if they do wake up during the night.

Sleep efficiency and total sleep time. Actigraphy sometimes overestimate SE and TST (Kushida, et al., 2001), which may explain the lack of significant results on

actigraphic measurements on these sleep parameters. However, subjective reports confirm that the Utøya survivors do not differ from control subjects in perceived duration of sleep (Grønli, Ousdal, Melinder, et al., 2014). On the other hand, subjective reports from this sample indicate that the survivors are overall less satisfied with their sleep ($p<0.05$) and experience the sleep as less restorative ($p<0.05$) than controls (Grønli, Ousdal, Melinder, et al., 2014). As the N3 sleep has a restorative effect on the brain (Grønli, et al., 2013), the reported unsatisfying sleep among the survivors may be due to less amount of N3 sleep. Heightened alertness and jumpiness seen among PTSD patients (World Health Organization, 1992) may also increase their sensitivity to sounds during night, which may prevent them from entering the deep sleep stage. Thus, the survivors may in fact not experience more awakenings and less sleep than the controls, but still perceive their sleep as unsatisfactory and less restorative due to less N3 sleep. Nevertheless, the survivors experience sleep impairments (Grønli, Ousdal, Melinder, et al., 2014), which is a central criteria for several sleep disorders such as insomnia and DSPD (American Academy of Sleep Medicine, 2005).

Wake up and get up time. Lastly, the actigraph reveal that the survivors wake up and get out of the bed later than controls in the morning. This is supported by the corresponding times subjectively reported by the survivors (Grønli, Ousdal, Melinder, et al., 2014). Traumatized individuals often experience early morning awakenings (Waldrop, et al., 2008), which is inconsistent with our results of later wake up times among Utøya survivors. Subjective reports on sleep disturbances and psychiatric disorders reveal high prevalence and comorbidity of these disorders (Grønli, Ousdal, Milde, et al., 2014; Milde, et al., 2014), thus sick leaves might be prominent among the survivors. Not having chores or activities to wake up to can affect the get up time. The

presence of depression found among Utøya survivors (Milde, et al., 2014) may also cause them to stay longer in bed during the day, delaying get up times. In sum, the finding of both later bedtimes and later get up times, in both objective and subjective data, may indicate that Utøya survivors have an exaggerated delayed circadian rhythm.

Possible DSPD Among Utøya Survivors

Utøya survivors in this study display an exaggerated sleep phase delay, which may be interpreted as a symptom of DSPD (American Academy of Sleep Medicine, 2005). In accordance with AASM's description of DSPD (American Academy of Sleep Medicine, 2005), the duration and quality of sleep measured by actigraphy are not affected negatively among Utøya survivors, supporting the possibility of DSPD. In line with descriptions of DSPD patients (Thorpy, et al., 1988), TST increases among Utøya survivors during weekends. However, to fulfill a DSPD diagnosis the individuals have to experience the sleep phase delay as a problem (American Academy of Sleep Medicine, 2005). Thus, we need to take the subjective findings into consideration. The subjective data reveal that sleep disturbances in fact are reported by as many as 53.1% of the survivors, compared to 13.6% of the control subjects ($p<0.001$) (Grønli, Ousdal, Melinder, et al., 2014; Grønli, Ousdal, Milde, et al., 2014). In addition, the survivors describe their sleep as less satisfying and restorative than controls (Grønli, Ousdal, Melinder, et al., 2014; Grønli, Ousdal, Milde, et al., 2014). This supports the possibility of the presence of DSPD among Utøya survivors.

It is not possible to determine if an exaggerated delayed sleep phase was evident before the terror attack, by looking solely at the objective data from the actigraphy recordings. However, the subjective reports reveal that 46.9% of sleep disturbances reported by Utøya survivors, compared to 4.6% of the sleep disturbances reported by

controls, developed 1-3 years ago ($p<0.001$). The differences between the groups in amount of sleep disturbances developed either last year or over 3 years ago, is not significant (Grønli, Ousdal, Melinder, et al., 2014). This indicates a close relationship between the sleep disturbances reported among the survivors, and the terror attack on Utøya. Thus, the findings on time of development contradict the possibility that the exaggerated sleep phase delay among Utøya survivors were evident before the terror attack.

The actigraphy results indicate a sleep cycle of eveningness among all participants, which is common among adolescents (Adan, et al., 2012; Tonetti, et al., 2008). However, this eveningness is exaggerated among Utøya survivors. During adolescence there are biological and behavioral changes in the circadian rhythmicity of sleep and wakefulness (Crowley, et al., 2007). Taking our findings into consideration, it seems like experiencing trauma during this specific age period may increase the risk for developing a further sleep delay, and thus increase the risk of developing DSPD. If a possible increased risk for developing DSPD after trauma is present among adolescents only, and not in other age periods, has not been investigated as far as we know. However, due to the developmental changes that occurs naturally during these years, adolescents might be at greater risk of developing DSPD after trauma than other age groups.

The exaggerated sleep phase delay among Utøya survivors may be perceived as maladaptive behavior because it may increase an already present circadian sleep delay. Behaviors that delay the bedtimes and sleep onset such as watching TV, hanging out with friends during the night, use of electronic devises past bedtime etc might be seen as maladaptive especially when in risk for an exaggerated sleep delay. The delayed sleep

phase may again function as a maintaining factor for other negative difficulties, such as sleepiness during the day or excessive daytime impairments. A delayed circadian rhythm is associated with sleep loss during weekdays, and this is again associated with an increased risk for developing mood and behavior disturbances, alcohol and substance abuse (Carskadon, 1990). To capture these behaviors when measuring the circadian rhythm of sleep and wakefulness after trauma may make it possible to prevent further implications.

Possible Risk- and Protective Factors

The prevalence of PTSD subjectively reported by Utøya survivors (30%) (Milde, et al., 2014) 18-30 months after the attack is similar to prevalence reported after other terror attacks. The prevalence of PTSD after the 9/11 and the Oklahoma City bombing terror attacks, range from 20-34.3% (Galea, et al., 2002; North, et al., 1999). However, these studies were conducted within 6 months after the events, thus the prevalence would probably be lower at the time of 18-30 months. The prevalence of earlier depressive episodes (33%), panic disorders (29%) and sleep disturbances (53.1%) (Grønli, Ousdal, Melinder, et al., 2014; Milde, et al., 2014) among Utøya survivors are relatively high compared to other terror attacks (Galea, et al., 2002; North, et al., 1999). Within 6 months after 9/11 and the Oklahoma City bombing the prevalence of panic disorders were less than 10% and the prevalence of depression ranged from 9.7-22.5%, while sleep difficulties were primarily described as results of stress and hyperarousal (Galea, et al., 2002; North, et al., 1999; Schuster, et al., 2001). These discrepancies in prevalence might be an effect of the characteristics of the terror attack that happened on Utøya.

The attack at Utøya was an event of mass violence, characterized by exceptional threat and violent images, lasting for more than 70 minutes (Dyb, Jensen, Nygaard, et al., 2014). Further, all of the survivors knew others at the island which also were in great danger. Combined, over ¾ of the survivors lost a friend, a relative, a girlfriend or a boyfriend in the attack (Dyb, Jensen, Nygaard, et al., 2014). These aspects reflect high levels of psychological proximity and loss. In addition, the terror attack on Utøya took place at a small geographic area, especially compared to previous described terror attacks in cities such as New York, London and Oklahoma City. This reflects high levels of geographical proximity. Due to the small geographical area, the levels of exposure to extreme violence were also high among the survivors (Dyb, Jensen, Nygaard, et al., 2014). In addition, most of the survivors were younger than 25 years of age (>90%) (Dyb, Jensen, Nygaard, et al., 2014). Combined, young age (Bramsen, et al., 2000), geographical and psychological proximity (Galea, et al., 2002; Hansen, et al., 2013; Thoresen, et al., 2012), high level of exposure (Neria, et al., 2008) and events of mass violence (Goenjian, et al., 2001; Norris, et al., 2002) put the survivors after the attack on Utøya at great risk for developing psychopathology, possibly explaining the registered prevalence of PTSD, depression, panic disorder and sleep disturbances among the survivors. In the light of the brutality, duration, geographical and psychological proximity and young age, one might even have expected the prevalence of psychopathology to be higher.

There are some aspects that might have served as protective factors, impeding a higher incidence of psychiatric- and sleep disorders among the survivors. First, there were high levels of social support among Utøya survivors (Dyb, Jensen, Glad, Nygaard, & Thoresen, 2014). The Workers Youth League (AUF) had several meetings after the

attack, which gave the survivors the possibility to get support among friends and acquaintances that had experienced the same trauma. Survivors of terror attacks often come from different backgrounds, only having the traumatic event in common. The common interest for politics among the survivors may have been of great importance for the further solidarity and social support among the survivors, and thus reduce the risk of negative long-term reactions.

Second, all Utøya survivors were offered professional help following the attack. Each municipality in Norway became responsible for its affected residents after the attack, offering necessary health care for all survivors living in the municipality. Later follow-ups have shown that 87% of the survivors received early and proactive outreach, 84% had a contact person regarding professional help after the event, and 73% received professional help from a psychiatrist or a psychologist (Dyb, Jensen, Glad, et al., 2014). As coping strategies are found to affect the risk of psychopathology (Cohen & Roth, 1987; Silver, et al., 2002), these interventions may have reduced the survivors' risk of negative long-term reactions.

The Use of Actigraphy to Measure Sleep

The observation of several coded actograms considered unreliable due to oddly codings of primarily bedtimes and some get up times, led to the wish to examine manual coding. The automatic coding do not take into consideration the time of the day, light illumination or event markers. Major rest intervals are solely defined as rest of more than 3 hours. Further, the calculation of the automatic onset of the rest interval is proprietary and unclear for the users of Actiware. However, it appears that one of the weaknesses of the automatic coding is the postponing of rest onset as long as the criteria for onset is not fulfilled, thus making it look like restless people never go to bed. The

actogram of one participant in our sample did not register any rest interval for a whole week when automatically coded, which seems to be unlikely.

Another weakness in the automatic coding is the actigraphs interpretation of low activity. Several actograms showed low, but regular activity for hours during the evening before a sustained decrease in activity was evident at night. The actograms often coded these hours as rest, thus making the sleep onset latency last for several hours. By looking at the light illumination, it is more likely that this low activity was reflecting people resting on the couch, watching TV, and not intending to go to sleep as interpreted by the actigraph. Using the benefit of the information about light illumination and event markers trough manually coding might probably give a more reliable picture of the rest interval.

The low activity levels during the evening might be a sign of a sleep- or psychiatric disorder in the participants. Previous findings show that actigraphy is better to precisely estimate sleep in healthy participants than clinical populations (Hauri & Wisbey, 1992). This may have affected the results as the prevalence of psychopathology among the survivors is relatively high (Milde, et al., 2014). For instance, people with depression often display a reduction in motoric speed (World Health Organization, 1992), which may make them less active in bed, even though they are not sleeping. Thus, the actigraph might have misinterpreted quiet wakefulness among patients with disorders such as depression as sleep. Perhaps a different activity threshold for wakefulness would have captured a more accurate impression of the adolescents sleep quality. However, this would acquire comparisons of actigraphy to other sleep measurements such as PSG.

Previous studies fail to find the same sleep disturbances by the use of objective measurements as reported by subjective measures (Klein, Koren, Arnon, & Lavie, 2003; Pillar, et al., 2000). The lack of objective findings on sleep quality measured by actigraphy in this study might reflect the same tendency. The systematic review of the objective data compared to the subjective data found on this sample support the notion that subjective complaints after trauma are hard to replicate by objective measurements. It has been suggested that the lack of objective findings of sleep disturbances is due to an alteration of sleep perception rather than actual sleep quality (Klein, et al., 2003). Thus, the discrepancies between the subjective and objective findings might reflect an alteration of sleep perception rather than actual poorer sleep quality.

However, one meta-analysis showed that PSG detected sleep disturbances among PTSD patients when controlling for mediators such as comorbid depression or age differences (Kobayashi, Boarts, & Delahanty, 2007). As there are high prevalences of both depression and PTSD among Utøya survivors (Milde, et al., 2014), the sleep difficulties might not be visible, because of the comorbidity of these disorders. To control for both age and possible comorbidity might be important for detection of sleep difficulties after traumatic experiences.

Sleep Habits Among Adolescents

All adolescents in this study had a later bedtime and get up time during weekends than weekdays, consistent with previous research (Carskadon, et al., 1993; Wittmann, et al., 2006). This is probably due to lack of fixed schedules the next day, increasing the opportunities to enjoy social activities during the night. Later get up times may be because of a rebound-sleep after lack of sufficient sleep during the week (Wittmann, et al., 2006), and a natural consequence of later bedtimes. The effect of both lack of sleep

and social activities in weekends support both social and biological impact on social jet lag.

Both groups showed average bedtimes after midnight and get up times (Table 4) not compatible with early school schedules, which usually start between 08:00 and 09:00. This may reflect several aspects of adolescents sleep pattern presented in the introduction (Calamaro, et al., 2009; Carskadon, 1990). Use of mobile phones and computers, part time work and social activities in the evening are all probable explanations for generally late bedtimes seen in both groups (Calamaro, et al., 2009). Information given to all adolescents about how mobile phones and other electronic devices (with blue light illumination) increase brain activation and thus might impede sleep at night, may help them do behavioral changes that are compatible with good sleep health. An understanding of how behavior may influence sleep and further how this affect their mental health academic performance, might increase the likeliness of changing sleep behavior that may have positive impact on their circadian rhythm of sleep and wakefulness.

Sleep might be affected by circadian and homeostatic changes during adolescence (Crowley, et al., 2007; Jenni, et al., 2005). This may explain the delayed sleep phase pattern observed in our sample. This is an important finding because eveningness-types report more negative consequences associated with the sleep pattern than morningness-types (Díaz-Morales & Sánchez-Lopez, 2008; Giannotti, Cortesi, Sebastiani, & Ottaviano, 2002; Mecacci & Rocchetti, 1998), and both eveningness-types and DSPD patients report several symptoms of psychiatric disorders (Reid, et al., 2012). The mutual influence of both voluntary and involuntary delayed sleep pattern and the risk of psychiatric disorders need to be taken seriously. To reduce the risk of

developing negative consequences among eveningness-types, it may be important that the school system in particular consider adjusting schedules based on individual sleep preferences. Several studies have supported that later start times at school have multiple positive outcomes for the adolescents in general (Owens, Belon, & Moss, 2010; Vedaa, Saxvig, Wilhelmsen-Langeland, Bjorvatn, & Pallesen, 2012).

Clinical Relevance

There are several aspects of this study that may have clinical relevance. Our results underline the importance of addressing sleep difficulties after experiencing trauma, regardless of any psychiatric disturbances. This may contribute to earlier interventions and prevent further development of any sleep- or psychiatric disorders. After experiencing trauma, treatment of circadian rhythm of sleep and wakefulness might not be prioritized. However, treatment of a circadian rhythm disorder among adolescents exposed to trauma may have additional positive consequences on their psychological well-being in long-term. Treatment of sleep disorders is found to lead to an improvement of, or even a prevention of the development of PTSD and depression (Koren, et al., 2002; Krakow, et al., 2001). In addition, sleep disturbances are no longer regarded as just a secondary symptom of a psychiatric disorder (Spoormaker & Montgomery, 2008) supporting the importance of early assessment and treatment of sleep difficulties.

If Utøya survivors in fact experience subjective sleep difficulties, information to the survivors about not finding objectively measured sleep difficulties may reduce their personal distress of worrying about their sleep. Worrying about sleep can often lead to difficulties falling asleep at night (World Health Organization, 2004), and thus create a

vicious circle. Further, if the sleep quality is not affected, the treatment of sleep difficulties experienced after trauma may benefit from focusing on sleep perception.

There may also be the case that Utøya survivors are less exposed to daylight than others, due to the exaggerated sleep phase delay observed. In addition, they may be exposed to more light during the night, as use of mobile phones and computers are common among adolescents. Everyday light helps regulate melatonin production, and thus affecting the circadian rhythmicity (Grønli & Ursin, 2009). As people with exaggerated sleep phase delay might be light-sensitive (Aoki, et al., 2001), information about the importance of light exposure in the morning and light avoidance in the night might be beneficial. Even though all of the Utøya survivors have been offered professional help after the attack (Dyb, Jensen, Glad, et al., 2014), this study show that the sleep phase has an exaggerated delay up to 30 months after the traumatic event. This may reflect lack of focus on the sleep difficulties, which may have caused the observed sleep phase delay and possibly maintenance of the psychiatric disorders found in Milde and colleagues' research (2014).

As psychiatric- and sleep disorders after trauma are closely intertwined and tend to maintain each other (Krakow, et al., 2000), clinical professionals treating adolescents exposed to trauma should note that psychological treatment should be multidimensional. This study underline the importance of addressing both the sleep and psychiatric difficulties.

Strengths and Limitations

There are multiple strengths with this study. This study investigate long-term effects on sleep quality and the circadian rhythm of sleep and wakefulness using actigraphy as measurement among Utøya survivors, which gives important information about trauma

and sleep. There are few studies investigating how sleep is affected among adolescents after trauma, which makes this contribution important. The group was exposed to the same trauma in a small geographical area. Everyone were at risk of being shot and killed, and all experienced some kind of loss (Dyb, Jensen, Glad, et al., 2014). Earlier research papers investigating effects of trauma after terror attacks have mainly investigated traumatic events where people have differed in proximity and exposure, often with large age spans (Galea, et al., 2002; Miguel‐Tobal, et al., 2006; North, et al., 1999; Rubin, et al., 2005). Adolescents at Utøya have a small age span, and they are a unique population in terms of investigating the trauma's influence, eliminating effects of differences in age, proximity and exposure. This study also display how sleep is affected among survivors regardless of any sleep-or psychiatric disorder in long term, which to our knowledge is limited in the research field of sleep and trauma.

Further, this is one of the first studies investigating the actigraph's settings, regarding differences between automatically and manually coded rest intervals. A close look at the actograms revealed the necessity to complete additional codings and criteria for analysing if there were any major differences on the sleep parameters. This study investigate a limited part of what has been desired by AASM, regarding validation of the actigraphs' settings. However, it provides useful information about what to consider when using an actigraph to measure sleep quality and sleep pattern. It gives examples of obviously misinterpreted actograms, and new criteria for registering rest intervals manually, which can be used by other researchers in the future.

However, there are some limitations with the study that have to be considered. In spite of a small age span and matching on age and gender among the participants, they may have very different weekdays regarding daily activities. During this age period

some are high school students, some work, some have a year off after finishing school, and some may study at the university with a flexible schedule. The participants' form of daily activities are not controlled for in this study. These differences may have affected the overall results. Further, this study did not differentiate between weekdays and vacation days, and some data were collected during the summer. This may have affected their sleep duration, bedtime and get up time as they do not have to get up at the same time as during school- or work days.

Due to the scope of this study and ethical limitations, it did not include information about the amount and type of psychological or medical health care, or the use of medications. Several psychotropic drugs have drowsiness as a side effect, and some participants may be using hypnotics, which may have affected the results in this study. However, Grønli and colleagues' study (2014) which include all of the same participants as in our study, report that only one participant used hypnotics, even with subjectively major sleep difficulties. Thus, it is reasonable to assume that the use of hypnotics will not have a major impact on our results.

This thesis is part of a larger project, including fMRI and structural interviews. The measurements may have been perceived as numerous and time consuming, causing some to withstand participation. Although, the study attempted to limit the number of measurements the adolescents had to go through, the information about the scope of this project may have prevented the most deprived Utøya survivors from participating. If this is true, it would make the sample less representative by only including survivors with a certain minimum level of psychological functioning.

This study include 88 participants in total. Because of the small sample size it might have missed important associations due to low observed statistical power,

especially in measures of TST, SE, SOL and WASO (see Table 3; power < 0.18, no p-values below 0.05). It is possible that a type II error has encountered, which is an underestimation of potential effects. In our study, increasing sample size was difficult; the number of Utøya survivors living close to Oslo and Bergen was limited, and it took a while to recruit the participants. There were only 495 Utøya survivors in total living all over the country, which also naturally reduced our possibility to increase the sample size. Nevertheless, a larger sample size could increase the statistical power, and thus maybe increase the possibility of finding significant results on sleep quality measured by the actigraph.

The participants were asked to wear the Actiwatch for at least one week. However, registrations varied from 2 days to 2 weeks. Some of these varieties were due to failures in the registrations by the Actiwatch itself, some removed the Actiwatch too early, while others took the watch on and off during the week. Some may have perceived the watch as uncomfortable, and thus took it off. We do not know if the adolescents who removed the Actiwatch early in the registration experienced more sleep difficulties and thus affected the results. Further, the number of weekend nights varied among the participants. Several participants only used the Actiwatch for one weekend night or none at all, while others had two weekend registrations or more. These differences may have affected the average scores derived for weekend sleep, and thus affected the results.

Further Research

There are several suggestions for further research derived from our results. Sleep difficulties may appear before psychiatric episodes and are one of the strongest predictors for developing psychiatric disorders (T. H. Brown, et al., 2011; Koren, et al.,

2002). Our results suggest that further research should focus on changes in the circadian rhythm of sleep and wakefulness after trauma and how this is related to the development of psychiatric disorders, especially among adolescents. Also an investigation of the relation between circadian rhythm disorders such as DSPD and trauma would be interesting to see if adolescents are in greater risk for developing this disorder.

When looking at the actigraphy data, many Utøya survivors seemed to be resting a lot during the day, shown as low activity levels. Future research could benefit from including daytime activity level to see if there are any relation between activity levels during the day and nighttime sleep. Including minor sleep intervals could also give a clearer image of sleep-wake patterns among adolescents after traumatic experiences.

We also have some suggestions for further research regarding the use of actigraphy. The validation of automatically and manually coded rest intervals are almost not existing. Studies investigating how it is most appropriate to find exact estimations of sleep and wakefulness using actigraphy should be conducted. How the Actiware actually decides where to set the rest interval is not described by the software. To get a clearer understanding of how this is set automatically would be helpful for further researchers who want to compare the differences in codings of the rest interval. To establish whether manually coding is more accurate at determining rest intervals than automatic coding, it is necessary to compare the intervals in actigraphy to PSG intervals. Using the same actigraphy settings will reduce possible faults when comparing studies on automatic and manual coding. Further research on coding should ideally involve similar populations and use the same actiwatch, so that the influence of

differences between groups and measuring devices is limited, and the differences found are due to actual differences between the codings.

In addition, there is a further need of validation of the use of actigraphy in different populations. Further research on different actigraph settings in different populations, both clinical and healthy, is important. Level of movement during sleep is likely to differ in various populations based on their characteristics. Some clinical populations such as patients with depression might need a lower waking threshold than patients with PTSD. Further, younger people might need a higher waking threshold than elderly. To establish appropriate settings thus is important to find appropriate settings for the populations of interest.

Conclusion

The results shows how the terror attack on Utøya delayed the survivors' circadian rhythm of sleep and wakefulness. Notably, there are no significant differences between the groups regarding sleep quality measured by actigraphy. However, subjective reports reflect high levels of sleep disturbances in this sample, thus the lack of findings might be due to incorrect measures by the actigraph or perhaps an altered sleep perception rather than poorer sleep quality per se. In addition, the study support previous research on sleep habits among adolescents, showing a social jet lag during weekends. This finding is an important contribution to the understanding of sleep among adolescents, and how further psychopathology might be prevented. Lastly, this study provide new information regarding the use of manual coding of rest intervals in Actiware, which might look like a better way to measure sleep by actigraphy.

This study provides an additional contribution to the research field of both trauma and sleep. It illustrates the importance of addressing sleep quality and the

circadian rhythm of sleep and wakefulness after trauma, regardless of any major sleep or psychiatric disorders reported by the survivor. The sleep pattern as a possible symptom, as well as an important contributing factor to maintain other difficulties for the survivor may be of huge importance for both research, choice of treatment and the affected victim.

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Tables

Table 1
Main effects of coding (manual/automatic) on the standard deviations in selected sleep parameters.

	Manual / automatic coding							
	5 minutes, weekday		***5 minutes, weekend***		***10 minutes, weekday***		***10 minutes, weekend***	
	F	*p*	*F*	*p*	*F*	*p*	*F*	*p*
Bedtime	6.1	0.015	2.51	0.117	0.00	0.998	0.67	0.414
Sleep onset	6.5	0.013	1.88	0.174	2.98	0.088	1.27	0.264
TIB	8.4	0.005	3.43	0.068	0.00	0.954	0.08	0.776
TST	1.16	0.284	0.86	0.356	0.08	0.777	0.4	0.53
SOL	1.93	0.168 *	11.7	0.001	8.0	0.006	7.6	0.008
SE%	3.37	0.07	1.24	0.268	8.2	0.005	3.62	0.061
WASO	5.7	0.02	2.47	0.121	1.24	0.269	1.06	0.306
Wake up time	3.8	0.053	0.92	0.34	1.55	0.216	0.68	0.412
Get up time	5.7	0.02	1.01	0.317	0.02	0.904	0.07	0.792

Note. TIB = time in bed, TST = total sleep time, SOL = sleep onset latency, SE% = sleep efficiency, WASO = wake after sleep onset.
* Interaction effect (group/coding) (F=5,9, *p*= 0.017)

Table 1. Main effects of coding manual vs automatic on standard deviation in selected sleep parameters. Divided in 5 and 10 minutes inactivity threshold and weekdays/weekends.

Table 2
Main effects of group and main effects of weekday/weekend on standard deviations on selected sleep parameters, using manual and automatic coding, with 5 and 10 minutes of inactivity threshold.

		Group				**Weekday/weekend**			
		5 minutes		10 minutes		5 minutes		10 minutes	
		F value	*p value*	*F value*	*p value*	*F value*	*p value*	*F value*	*p value*
Bedtime	Auto	3.9	0.05	3.96	0.05	0.21	0.65	0.21	0.65
	Manu	4.62 *m*	0.03	3.63	0.06	0.36 *m*	0.07	3.93 *m*	0.05
Sleep onset	Auto	3.83	0.05	2.19	0.14	0.51	0.48	0.61	0.44
	Manu	5.53 *m*	0.02	5.15 *m*	0.02	4.4 *m*	0.04	3.48 *m*	0.06
TST	Auto	1.15	0.28	0.14	0.71	1.66	0.2	4.45	0.04
	Manu	0.01	0.93	0.07	0.8	1.47	0.23	1.33	0.25
TIB	Auto	0.95	0.33	0.61	0.44	2.59	0.12	2.36	0.13
	Manu	0.42	0.52	0.52	0.47	1.4	0.24	3.2 *m*	0.08
SE%	Auto	1.69	0.2	0.00	0.99	0.94	0.33	3.75	0.05
	Manu	0.14	0.71	0.88 *m*	0.35	0.57	0.45	6.44 *m*	0.01
SOL	Auto	1.68	0.2	0.17	0.68	1.04	0.31	5.19	0.02
	Manu	1.62	0.2	2.19 *m*	0.14	4.79 *m*	0.03	14.55 *m*	<0.001
WASO	Auto	0.06	0.82	0.36	0.55	0.42	0.52	0.01	0.91
	Manu	1.28 *m*	0.26	0.02	0.87	3.79 *m*	0.05	0.63 *m*	0.43
Wake up time	Auto	0.83	0.36	0.29	0.59	0.59	0.45	1.93	0.17
	Manu	0.05	0.83	0.1	0.75	0.07	0.79	0.05	0.83
Get up time	Auto	0.54	0.47	0.43	0.51	1.43	0.23	1.36	0.25
	Manu	0.37	0.54	0.04	0.84	0.08	0.78	0.08	0.78

Note. TST = total sleep time, TIB = time in bed, SE% = sleep efficiency, SOL = sleep onset latency, WASO = wake after sleep onset
m Manual coding increase *F*

Table 2. Main effects of group and weekday/weekend on the STDV of selected sleep parameters, using manual and automatic coding, with 5 and 10 minutes of inactivity threshold.

Table 3
Main effects of group (Utøya survivors/control subjects) and of weekdays/weekends on selected sleep parameters.

	Utøya survivors/control group			**Weekday/weekend**		
	F value	*p* value	Observed power	*F* value	*p* value	Observed power
Bedtime	5.86	0.017	0.67	25.86	<0.001	0.99
Sleep onset	7.2	0.008	0.76	22.51	<0.001	0.99
TIB	0.28	0.6	0.08	4.9	0.028	0.6
TST	0.18	0.67	0.07	7.67	0.006	0.79
SOL	1.11	0.29	0.18	0.39	0.53	0.1
SE%	0.0	0.98	0.05	0.97	0.33	0.17
WASO	0.13	0.72	0.065	0.18	0.66	0.07
Wake up time	6.33	0.012	0.71	35.37	<0.001	0.99
Get up time	5.69	0.018	0.66	33.58	<0.001	0.99

Note. TIB = time in bed, TST = total sleep time, SOL = sleep onset latency, SE% = sleep efficiency, WASO = wake after sleep onset. 10 minutes of inactivity threshold applied.

Table 3. Main effects of group and weekday/weekend on sleep parameters derived from manual coded rest interval with 10 minutes inactivity threshold. TIB=time in bed, TST= total sleep time, SOL= sleep onset latency, SE= sleep efficiency, WASO= wake after sleep onset.

Table 4

Descriptive statistics for Utøya survivors and control subjects during weekdays and weekends, and post hoc results for significant group effect.

		Utøya survivors		Control subjects		
		Mean	SE	Mean	SE	*p*
Weekday	Bedtime	01:17	0,009	00:38	0,01	0.045
	Sleep onset	01:42	0,01	00:54	0,008	0.018
	TST	410,45	10,28	416,28	8,48	NC
	SOL	22,92	3,43	16,93	2,87	NC
	SE%	83,53	1,08	84,83	1,16	NC
	WASO	40,52	2,49	40,92	2,37	NC
	Wake up time	09:12	0,01	08:31	0,009	0.073
	Get up time	09:33	0,01	08:49	0,009	0.069
Weekend	Bedtime	02:24	0,01	01:55	0,01	0.161
	Sleep onset	02:39	0,01	02:11	0,01	0.162
	TST	452,1	10,06	436,72	14,95	NC
	SOL	18,58	5,18	16,46	3,84	NC
	SE%	86,01	1,05	84,77	1,54	NC
	WASO	41,11	2,77	42,8	3,68	NC
	Wake up time	10:53	0,01	10:10	0,01	0.08
	Get up time	11:08	0,01	10:30	0,01	0.123

Note. TST = total sleep time, SOL = sleep onset latency, SE% = sleep efficiency, WASO= wake after sleep onset.
SE% numbers are presented as %, all other values are minutes unless presented as hh:mm.
NC: post hoc not conducted due to no significant effects found in ANOVA.

Table 4. Descriptive statistics and post hoc comparisons for Utøya survivors and control subjects, during weekdays and weekends.

Table 5

Descriptive statistic for weekday and weekend in Utøya survivors and control subjects, and post hoc results for significant weekday/weekend effect.

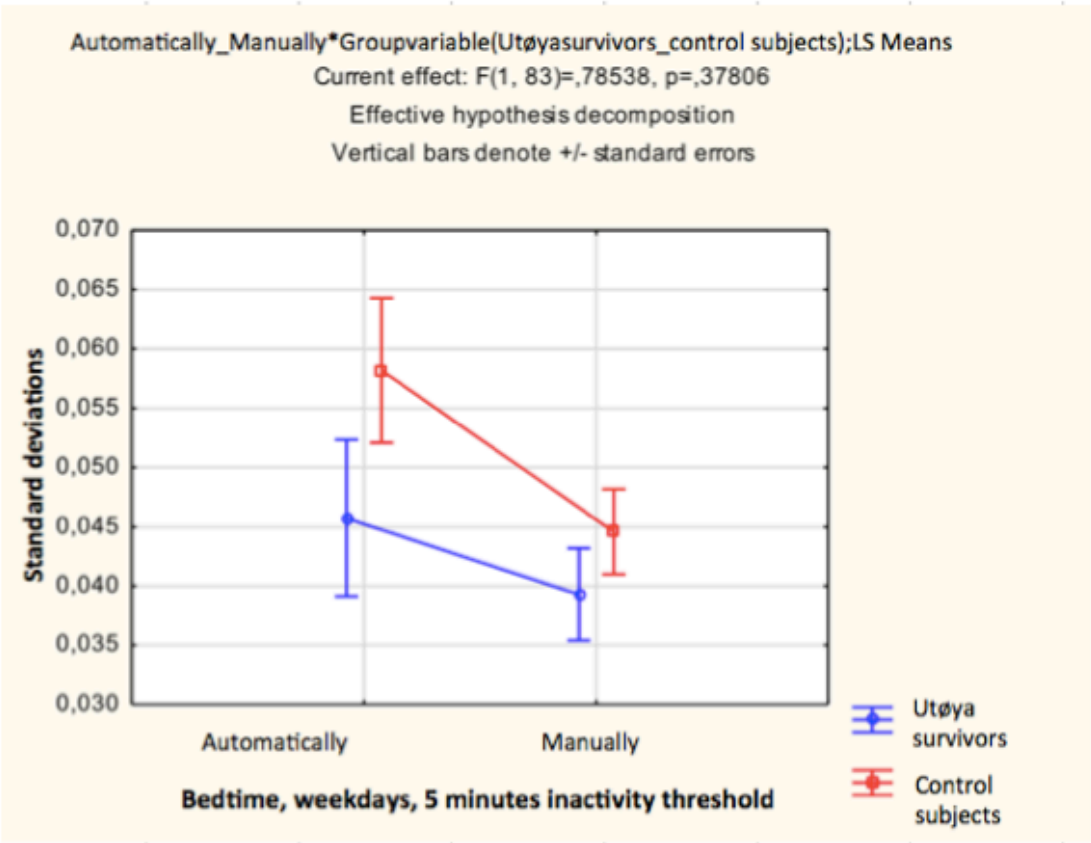
		Weekday		**Weekend**		
		Mean	SE	Mean	SE	*p*
Utøya survivors	Bedtime	01:17	0,009	02:24	0,01	0.002
	Sleep onset	01:42	0,01	02:39	0,01	0.006
	TIB	495,24	11,05	524,84	12,23	0.09
	TST	410,45	10,28	452,1	10,06	0.011
	Wake up time	09:12	0,01	10:53	0,01	<0.001
	Get up time	09:33	0,01	11:08	0,01	<0.001
Control subjects	Bedtime	00:38	0,01	01:55	0,01	<0.001
	Sleep onset	00:54	0,008	02:11	0,01	<0.001
	TIB	491,68	8,84	515,57	15,41	0.15
	TST	416,28	8,48	436,72	14,95	0.188
	Wake up time	08:31	0,009	10:10	0,01	<0.001
	Get up time	08:49	0,009	10:30	0,01	<0.001

Note. TIB = time in bed, TST = total sleep time.
All numbers not presented as hh:mm are number of minutes.

Table 5. Descriptive statistics and post hoc comparisons of weekdays and weekends, for Utøya survivors and control subject

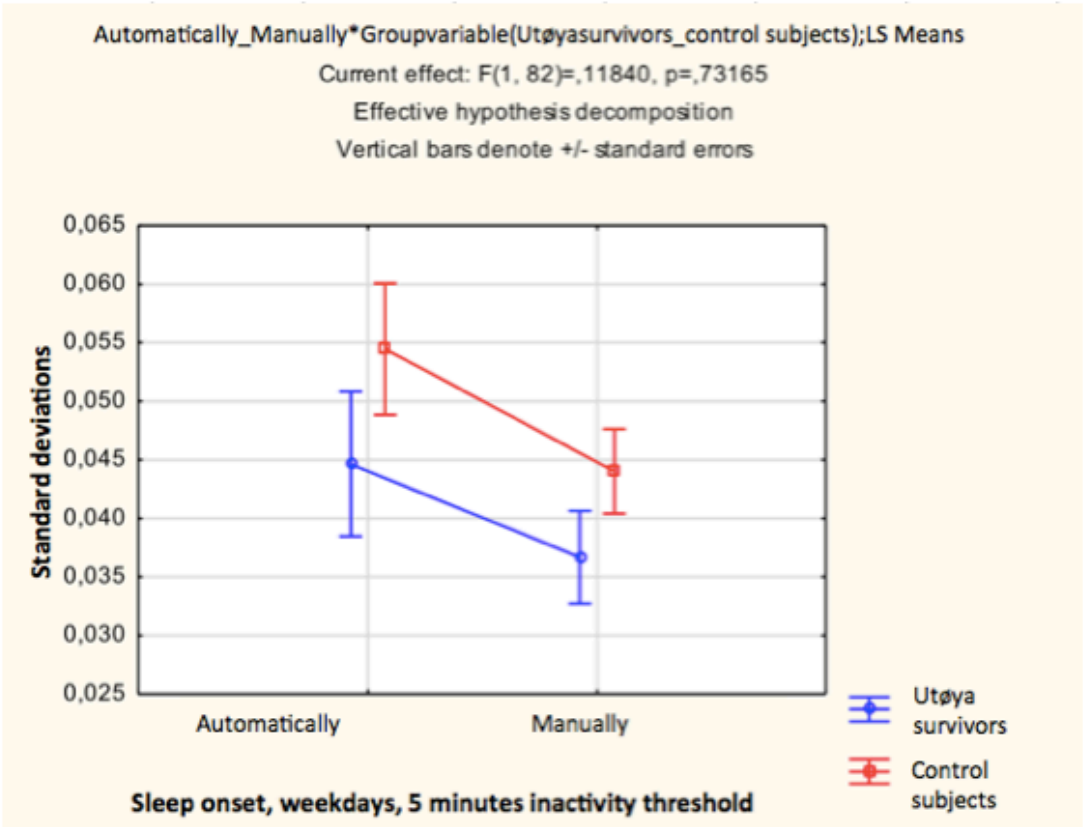
Graphs

Graph 1. Automatically Versus Manually Coded Rest Intervals, 5 Minutes Inactivity Threshold for Bedtime During Weekdays



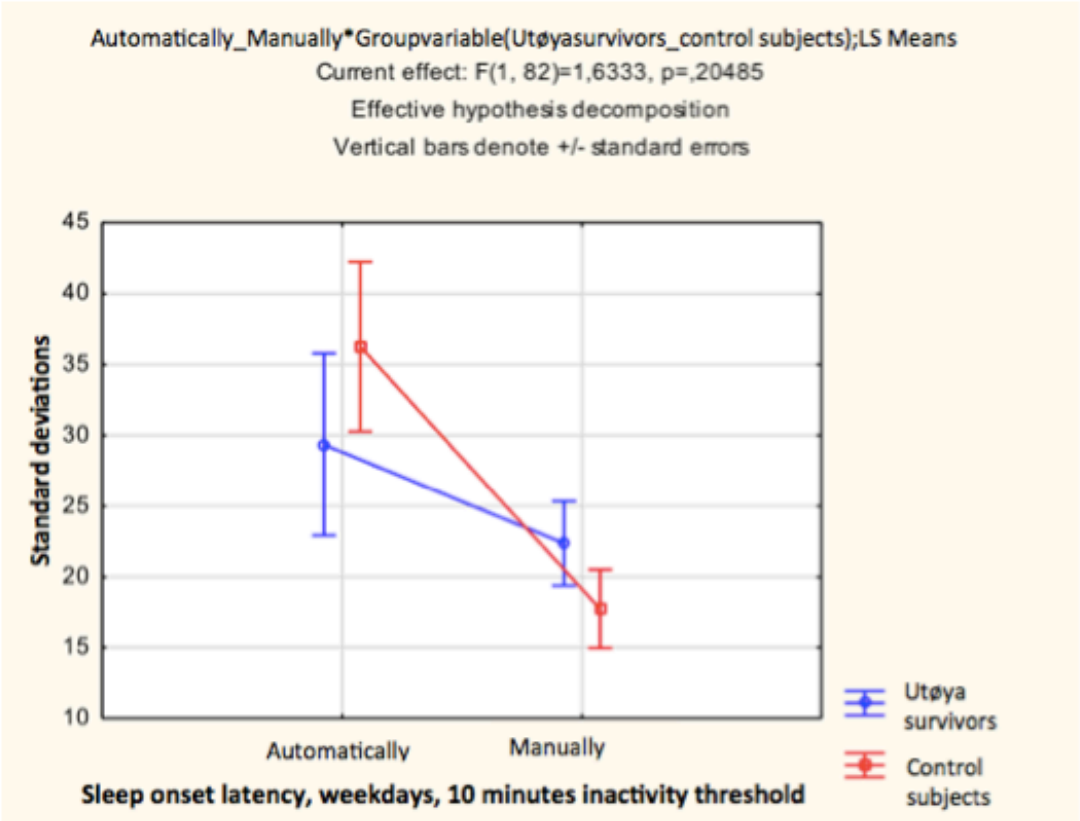
Graph 1. Illustrates standard deviations between automatically and manually coded rest intervals on bedtime for weekdays. Five minutes inactivity threshold is set for sleep onset. Comparisons of Utøya survivors and control subjects.

Graph 2. Automatically Versus Manually Coded Rest Intervals, 5 Minutes Inactivity Threshold for Sleep Onset During Weekdays



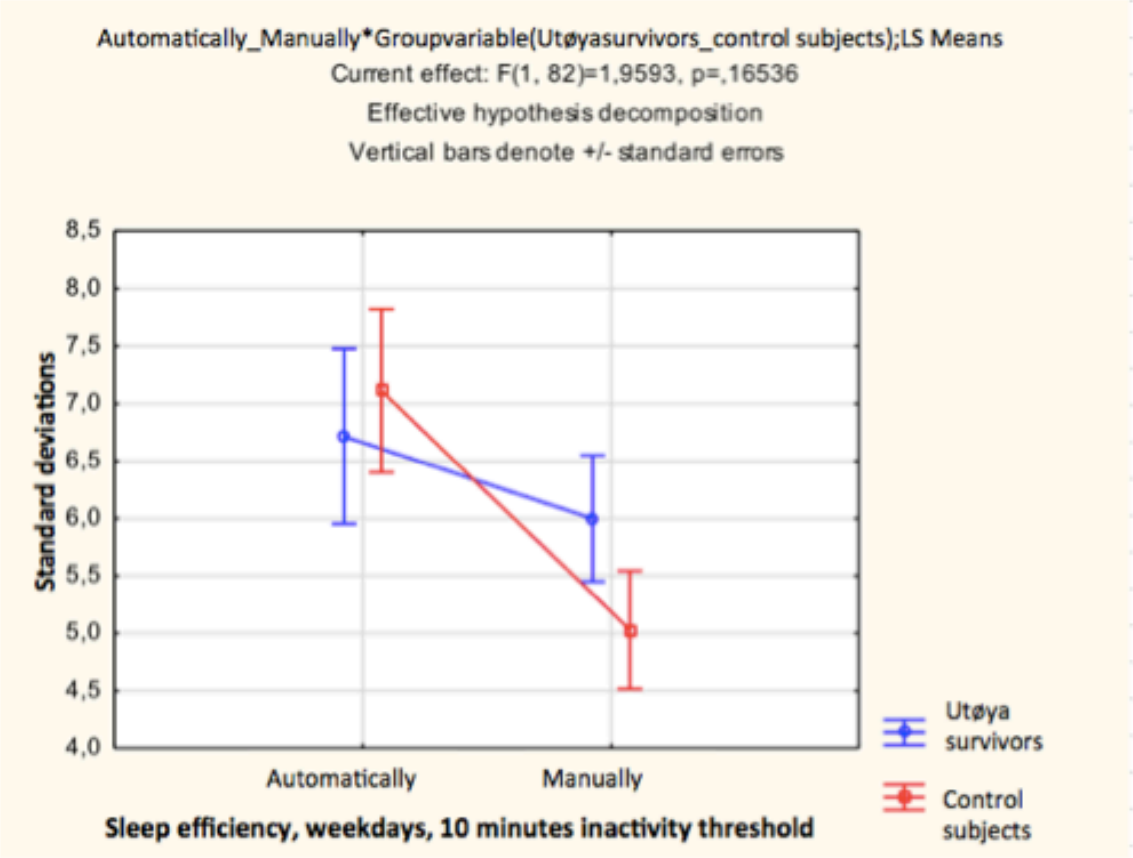
Graph 2. Illustrates standard deviations between automatically and manually coded rest intervals on sleep onset for weekdays. Five minutes inactivity threshold is set for sleep onset. Comparison of Utøya survivors and control subjects.

Graph 3. Automatically Versus Manually Coded Rest Intervals, 10 Minutes Inactivity Threshold for Sleep Onset Latency During Weekdays



Graph 3. Illustrates standard deviations between automatically and manually coded rest intervals on sleep onset latency for weekdays. Ten minutes inactivity threshold is set for sleep onset. Utøya survivors and control subjects are group variables.

Graph 4. Automatically Versus Manually Coded Rest Intervals, 10 Minutes Inactivity Threshold for Sleep Efficiency During Weekdays



Graph 4. Illustrates standard deviations between automatically and manually coded rest intervals on sleep efficiency for weekdays. Ten minutes inactivity threshold is set for sleep onset. Utøya survivors and control subjects are group variables.

Appendix

Bedtime: start time of the rest interval, when the participant is presumed to go to bed.

Sleep onset: start of sleep interval, when the participant is presumed to go to sleep.

Wake up time: end of sleep interval, when the participant is presumed to wake up.

Get up time: end time of the rest interval, when the participant is presumed to leave the bed.

TIB: time in bed, duration of the rest interval, scored in minutes.

TST: total sleep time, total minutes scored as asleep during the sleep interval.

SOL: sleep onset latency, the time required for sleep to start after initiating the intent to sleep, registered as the time between bedtime and sleep start

SE: sleep efficiency, the percentage of time in bed spent asleep, the total sleep time divided by time in bed multiplied by 100.

WASO: wake after sleep onset, the total number of minutes scored as awake within the sleep interval