

## Disturbed Sleep and Emotion: A developmental perspective

*This dissertation is dedicated to my forebearers, relatives, and friends who endowed me with deep roots and a strong sense of who I am; to my parents who have always had an undying faith in me and for providing me with a strong foundation to stand on; to Peter for being my rock, compass, and my best friend; to Adrian who is my greatest joy, inspiration, and who gives life a deeper meaning*

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A Developmental Perspective**

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## Abstract

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Sleep disturbances are not only defining features, but also diagnostic criteria for most psychiatric disorders. Recently, researchers have proposed a theoretic role for sleep disturbances in emotion dysregulation, subsequently linking neurobiological processes and psychopathology. Most prior research examining the potential role for sleep disturbance in emotion dysregulation is from a neurophysiological or clinical perspective, or primarily focused on maintaining processes. Less well understood are how sleep disturbances may be involved at the levels of predisposition, precipitation, and perpetuation of emotion dysregulation concurrently and over time.

This dissertation presents findings from three studies that were designed to expand on what is known about sleep disturbance in the predisposition, precipitation, and perpetuation of emotion dysregulation. Study 1 examined the long-term relation between sleep-onset problems and neuroticism over twenty-years. Adolescent sleep-onset posed risk (predisposition) for neuroticism in midlife, not vice versa. Study 2 investigated the effects of 3-nights partial sleep deprivation (5-hours total time in bed) on the positive and negative affect and emotions of otherwise healthy adults. Following partial sleep deprivation, people reported significant reductions in positive affect and emotions compared to rested people (precipitation). The only impact on negative emotions was on the discrete level. Sleep deprived people reported significantly more irritability, loathing, hostility, and shakiness compared to controls. Study 3 measured adolescent sleep disturbances, depressive symptoms, and catastrophic worry. In addition to direct risk, sleep disturbances posed a non-gender specific risk for depressive symptoms one-year later through catastrophic worry (perpetuation). Overall, the results provide support for the role of sleep disturbances in the predisposition, precipitation, and perpetuation of emotion dysregulation. An implication is that sleep disturbances and catastrophic worry are two potentially modifiable markers of risk for emotion dysregulation.

*Keywords:* Sleep disturbance, emotion dysregulation, catastrophic worry, sleep-onset, partial sleep deprivation, neuroticism, depressive symptoms.

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## List of studies

This dissertation is based on the following studies, which hereafter will be referred to in the text by the number of the study.

- Study 1** Danielsson, N. S., Jansson-Fröjmark, M., Linton, S. J., Jutengren, G., & Stattin, H. (2010). Neuroticism and sleep-onset: What is the long-term connection? *Personality and Individual Differences*, 48, 463-468. doi: 10.1016/j.paid.2009.11.023.
- Study 2** Danielsson, N. S., Linton, S. J., Jansson-Fröjmark, M. Effects of partial sleep deprivation on subjective emotion experience and implicit emotion regulation. *Submitted for publication*.
- Study 3** Danielsson, N. S., Harvey, A. G., MacDonald, S., Jansson-Fröjmark, M., Linton, S. J. (2012). Sleep disturbance and depressive symptoms in adolescence: The role of catastrophic worry. *Journal of Youth and Adolescence*. (Advance online publication). doi: 10.1007/s10964-012-9811-6.

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# Introduction

“The best bridge between despair and hope is a good night’s sleep.”

~ E. Joseph Cossman

Many people have known the discomfort of twisting and turning in bed, or forcing themselves up in the morning after having retired to bed 6-hours earlier. Falling or staying asleep is not always easy, especially during times of stress. Sleep disturbances are relatively common, often long lasting, and at times impairing. Complicating issues, however, is the self-imposed sleep deprivation that many in modern society inflict on themselves and their families. People are working longer hours and the lines between work and free time are blurring. This is partly due to modern technological advances such as the 24-hour society, the internet, laptops, and smartphones. The number of people sleeping less than 6-hours per week has greatly increased in the past 20-years (see, Luyster, Strollo, Zee, & Walsh, 2012). Many people seem to gauge getting enough sleep, as the bare minimum needed for at least a minimum of next day functioning.

Probably everyone has experienced a sleep disturbance sometime. According to recent polls, roughly 75% of Americans want to increase their sleep quantity and improve sleep quality. Globally, sleep deprivation is estimated to affect 45% of the population (World Sleep Day Committee, 2012). Alarmingly, 50 to 70 million Americans, in all age groups, are estimated to undergo sleep disturbances (National Heart Lung and Blood Institute, 2003). Disturbingly, two-thirds of women report regular sleep disturbances (National Sleep Foundation, 2012). Perhaps the experiences behind the statistics account for the increasing broad general interest in sleep.

On a large scale, people are starting to understand that sleep is as vital for health as diet and exercise. Many physical and mental health problems are associated with sleep disturbances (Stein, Belik, Jacobi, & Sareen, 2008). Sleep disturbances are associated with weight gain, high blood pressure, and lowered immune system function, as well as, stress, anxiety (see, Luyster et al., 2012), depression (Wiebe, Cassoff, & Gruber, 2012), and suicide (Pigeon, Piquart, & Conner, 2012). Just as sleep disturbances pose risk to health, sleep also has a protective role. Sleep helps prepare people for coping with daily hassles and life-stressors (Walker & van der Helm, 2009). Beyond survival and physical health maintenance, a new frontier in developmental sleep research is opening up. Researchers are finding that there may be long-term consequences of sleep disturbances for emotional health.

My interest in learning more about the relation between sleep disturbance and emotion grew from a seed planted while reviewing the literature on sleep disturbance in general. The area of sleep research covers a vast array of areas including epidemiological, cognitive, neurological, and psychological. While reviewing the literature, I noticed a pattern where sleep disturbances were intimately linked with emotion, and mood related psychopathology. A lot is known about the genetic influences on neurobiological processes involved in sleep disturbances and emotion dysregulation. Likewise, a lot of research exists on clinical aspects of sleep disturbances and emotion dysregulation. Researchers and theorists suggest that interactions between sleep disturbances and emotion dysregulation, at the phenomenological level, are a critical link in the relationship between neurobiology and psychopathology. There are theoretical grounds for suggesting that common underlying genetic, neurophysiological and environmental stress factors may impact both sleep disturbances and emotion dysregulation immediately and over time (Harvey, Murray, Chandler, & Soehner, 2011; van der Helm & Walker, 2011). Less is known about the phenomenological level of the relationship between sleep disturbance and emotion dysregulation. The current dissertation examines the phenomenological aspects of sleep disturbance and emotion dysregulation with a focus how sleep disturbances influence emotion dysregulation.

The many rapid physiological, social, and cognitive changes occurring during the transition from adolescence into adulthood may leave adolescents especially vulnerable to the effects of disturbed sleep and emotions. Sleep disturbances potentially act as a motor in the development and perpetuation of problems with emotion dysregulation. In this dissertation, the developmental association examined was how sleep disturbances influence changes in emotional processes concurrently and over time. It is well established that sleep disturbances are associated with the development and maintenance of a host of psychiatric disorders, especially mood disorders (Benca, Obermeyer, Thisted, & Gillin, 1992; Harvey, 2008a, 2011). So much so, that sleep disturbances are a defining characteristic and diagnostic symptom in most mental health disorders (DSM-IV-TR; American Psychiatric Association, 2000). Disturbed sleep is a potent risk factor for depression (Perlis et al., 2006), problems with bipolar disorder (Harvey, Mullin, & Hinshaw, 2006) and attention deficit-hyperactivity disorder (Dahl & Harvey, 2007), anxiety (Breslau, Roth, Rosenthal, & Andreski, 1996), and post-traumatic stress disorder (Krakow et al., 2001). All of which make the developmental relationship between sleep disturbances and emotion dysregulation a crucial research domain.

People who experience sleep disturbances often report negative emotions (e.g., worry, irritability, feeling blue). The relationship between sleep disturbance and emotion is of importance because sleep disturbances are relatively common (i.e., reported by 30% of the population), often long lasting, and associated with a host of debilitating consequences. The overarching aim of this thesis is to expand our understanding of the ways in which sleep disturbances negatively influence the emotions of otherwise healthy adolescents and adults. Examining the link between sleep disturbances and emotion may aid in identifying markers of risk for the development and maintenance of emotional health problems, as well as providing targets for prevention and intervention.

Finally, determining the developmental role of sleep disturbances for emotion would add to the growing multidisciplinary knowledge base of the functions of sleep in emotions. There is accumulating evidence that sleep plays an important role in various processes related to emotion (see, Walker & van der Helm, 2009). These include nighttime emotional memory processing and resetting of next day reactivity (Walker & van der Helm, 2009). Developmental research into psychological aspects of the relationship between disturbed sleep and emotion are sparse and mainly at the theoretic level. The research presented in this dissertation are some of the first developmental sleep disturbance and emotion studies in a new field of inquiry that is rapidly gaining interest.

This dissertation is based on epidemiological and experimental research examining potential risks posed by sleep disturbance to emotional health. The field of sleep and emotion is complex, interdisciplinary, and vast. The people included in the studies were healthy adolescents and adults in the normal population. Longitudinal and experimental methods were used to examine risks posed by sleep disturbances to emotional health. Due to the various specializations and niches within sleep and emotion research, a brief overview is warranted.

## **Theoretical framework**

There is a growing interest in discovering common psychological, biological, and social processes involved in a host of comorbid disorders (Cuthbert & Insel, 2010; Sanislow et al., 2010). Many modern holistic models, such as the psychobiosocial model (Engel, 1977), view psychological, biological, and social process as interrelated, dynamic, and reciprocal in human development. The ideas that nature and nurture are interdependent in the development of psychopathology are not new. Instead, these ideas are being used to develop new ways of thinking about psychopathology. There is a growing trend toward multidisciplinary research including

the fields of neurology and clinical research. In the field of psychology, some researchers and clinicians are looking for common, or transdiagnostic, processes and mechanisms cutting across traditional psychiatric disorder categories (Harvey, Watkins, Mansell, & Shafran, 2004).

Definitions of relevant terminology will be presented in order to aid understanding of the theoretical framework. Mechanisms or mechanistic processes, are defined as unconscious mental and emotional patterns that shape behavior in response to given situations or environments. Psychological processes are defined as underlying mechanisms, such as cognitive schemata, that influence patterns of thought, emotions, and behaviors. Transdiagnostic processes are common to and underlie multiple disorders. An example of cognitive and behavioral processes that may be common to insomnia and emotional disorders are an interactive maladaptive feedback loop fed by anxious apprehension (Barlow, 2002). Anxious people may perceive real or imagined threats in the environment. Their attention may shift to the monitoring for cues of threat. In turn, arousal may increase. Attentional, interpretive, and memory biases for threat may be activated. Performance difficulties may arise from inability to concentrate. Worrying and avoidance behaviors may develop as a form of coping (Barlow, 2002; Harvey, 2002). Normal adaptive processes, whether physiological, psychological, cognitive or emotional, likely become maladaptive when dysregulated or exaggerated.

Harvey and colleagues (2011) introduced the construct mechanistic transdiagnostic process to explain disorder comorbidity. In mechanistic transdiagnostic processes, comorbidity is due to casual interrelationships between processes (e.g., disturbed sleep and emotion dysregulation) and the development of psychopathology. For the most part, prior research has examined perpetuating processes, less is known about mechanistic processes that may predispose, precipitate, and potentially perpetuate disorders. If sleep were involved in emotion dysregulation at these three levels, this would further support sleep disturbances as a mechanistic transdiagnostic processes.

Sleep disturbances may be related to emotions in various ways in imparting risk for psychopathology. The theoretical background behind the ideas for this dissertation were adapted from a model for the development of insomnia, see Figure 1. In this model, some people have dormant underlying predisposing factors. Life-stressors may then act to fuel sleep disturbances in the acute phase. Thereafter, cognitive or behavioral perpetuating processes may kick in. Overtime, as sleep disturbances become chronic, such as in insomnia, then as the importance of the initial stressor wanes,



perpetuating processes gain in magnitude and maintain disorder (see, Erman, 2007; Spielman, Caruso, & Glovinsky, 1987).

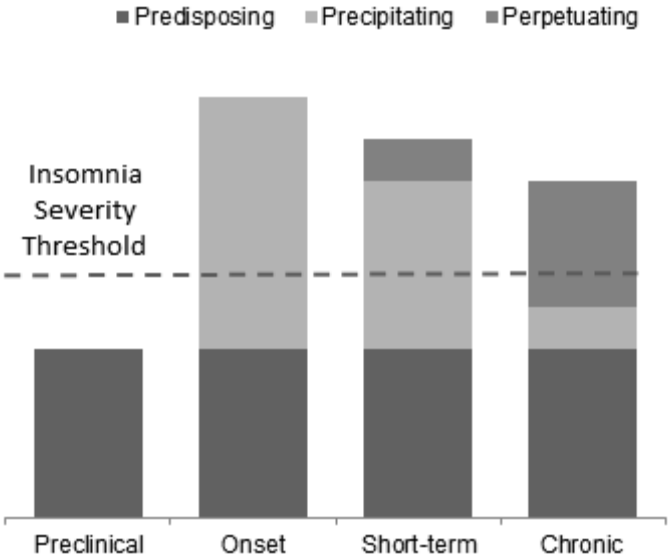


Figure 1. Spielman's 3 P model of the course of insomnia. Adapted from "Spielman A.J, Caruso L.S., Glovinsky P.B. (1987). A behavioral perspective on insomnia treatment. *Psychiatric Clinics of North America*. (10), 541-553" with kind permission from the American Psychological Association.

Predisposition for disorder involves genetic or diathesis interactions with environmental stressors in triggering psychopathology. Diatheses for disorder are not necessarily innate, but may also arise through stressors of a psychobiosocial nature. Precipitation involves internal or external stressors that trigger predisposition in the development of psychopathology. Perpetuating process are psychobiosocial processes that maintain psychopathology (for review see, Mayou & Farmer, 2002). Sleep related examples from the literature are sleep habits that are non-conducive to sleep or dysfunctional cognitive processes such as worrying.

Sleep disturbances are associated with a wide variety of psychological health problems, such as, stress (Jansson & Linton, 2006a; Linton, 2004), anxiety (Jansson & Linton, 2006b; Taylor, L., & Durrence, 2003), depression (Cole & Dendukuri, 2003; Ohayon, 2002; Riemann & Voderholzer, 2002; Taylor et al., 2003) and catastrophic worry (Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010). Furthermore, sleep disturbances are implicated in the development and maintenance of many psychiatric disorder-

ders, especially mood disorders (Benca et al., 1992; Harvey, 2008a; Harvey et al., 2011). All of which suggest that sleep disturbances may be a mechanistic transdiagnostic process in emotion related psychopathology.

Sleep disturbances may be brought on by life-stressors originating within the individual or in the contexts in which they live. Regardless, sleep disturbances may become potent life-stressors, potentially fuelling aggravating emotion dysregulation through worry, anxiety, and depression. Sleep disturbances may influence emotion dysregulation in several ways. Theoretically, people with diatheses for disorders are predisposed to react in a manner which will likely lead to disorders (Monroe & Harkness, 2005). Bouts with disorders may then aggravate and even change initial vulnerabilities such as in personality, thus increasing the likelihood of subsequent bouts of disorders (Tackett, 2006). Prior episodes of disorders may then render people vulnerable to subsequent bouts of disorders at lower stress thresholds (Drake & Roth, 2006; Post, 1992, 2002). Disturbed sleep and emotion have been proposed as mutually maintaining processes linking neurophysiological processes and psychopathology (Harvey et al., 2011). Likely, the relation is bidirectional with sleep disturbances aggravating emotional dysregulation, and vice versa.

## **Sleep**

### **Sleep disturbances**

Normal or good sleep is defined as acquiring enough sleep for optimal daytime functioning. A gauge for how much sleep is enough depends on what time that an individual would spontaneously awaken and whether they feel rested and alert upon awakening. Sleep disturbance is defined as a disruption of normal sleep patterns followed by distress and subsequent disruption of next day functioning. It is an overriding concept including multiple sleep problems and disorders affecting the quality of sleep, such as insomnia, sleep-onset problems, and sleep deprivation. In this dissertation, three types of sleep disturbances related to emotion are included, ranging from the specific to the broad. These are sleep-onset problems, sleep deprivation, and sleep disturbances in general.

#### **Sleep-onset problems**

Sleep-onset problems can be acute or chronic, found in normal healthy populations, or people with a clinically diagnosed sleep disorders. Often sleep-onset problems are a response to life stressors. Sleep-onset problems are characterized by difficulty falling asleep when desired, or when taking longer to fall asleep than anticipated. The research criteria generally used is

when people experience difficulty initiating sleep and sleep-onset takes longer than 30-minutes (DSM-IV-TR; American Psychiatric Association, 2000; National Heart Lung and Blood Institute, 2004; Ringdahl, Pereira, & Delzell, 2004). Due to strong environmental influences in childhood, much of the research on sleep-onset problems examines pediatric populations.

Sleep-onset problems are one of the most common sleep disturbances reported in childhood and show commonalities with adolescent behavior. Sleep-onset problems associated with behavior are divided into limit setting and association subtypes. Examples of the limit-setting type are refusal to go to bed, or using stalling tactics to avoid going to bed at an appropriate time. The association type is characterized by inappropriate behaviors non-conducive to falling asleep. Examples of these behaviors are late-night TV watching or listening to the radio (for a review see, Ivanenko & Patwari, 2009). In adolescence, there are many behavioral influences on sleep. Many teenagers have cellphones, gaming consoles, computers, and televisions in their rooms. Many activities such as playing console or computer games or surfing the internet are without set end-times. Behavior plays a very large role in sleep-onset problems.

Research into sleep-onset problems in adolescence are of interest both due to risks posed for chronicity of continued sleep-onset problems, but also potential risks to emotional health in adulthood (Roane & Taylor, 2008). This may occur in two ways. Physiology in the brain is still malleable in adolescence and may be vulnerable to physiological and neurological changes brought on by behaviors incompatible with falling asleep (Susman, Reiter, Ford, & Dorn, 2002), and maladaptive sleep behaviors learned in adolescence may crystalize and carryover into adulthood.

### Partial sleep deprivation

Many people in modern society suffer from chronic partial sleep deprivation. Despite this, the precise prevalence is unknown, as sleep deprivation is largely underreported. Estimates are that sleep deprivation is rapidly increasing. Sleep duration is drastically reduced with more people staying awake for longer periods of time (National Center on Sleep Disorders Research, 2003).

Partial sleep deprivation is defined as the failure to obtain adequate sleep over an extended time for maintaining physical and mental health. In consequence, people may experience physical or psychiatric symptoms and daytime deficits in performance of routine tasks. On average, people sleep 6.9 hours per night during the week and 7.5 on weekends. Roughly, a quarter of a million Americans judge their sleep quality as fair or poor

(National Sleep Foundation, 2002). Sleep deprivation is characterized by not acquiring enough sleep for maintaining physical, mental, and emotional health and may be either acute or chronic. Healthy populations as well as clinical populations suffering from diagnosed sleep and other medical disorders may experience sleep deprivation. Causes range from physical and mental health problems, to stress, and prioritizing lifestyle choices such as family, work, free time or other activities over sleep. Resulting negative effects are due to sleeping fewer hours than the body and mind require. The minimum sleep requirements needed are individual and vary dependent on individual differences. Chronic short-term partial sleep deprivation is associated with fatigue, daytime sleepiness, clumsiness, moodiness, and problems with weight gain or loss (Babson, Trainor, Feldner, & Blumenthal, 2010; Ikegami et al., 2009; Taheri, Lin, Austin, Young, & Mignot, 2004). There is a large behavioral component to sleep deprivation due to people not prioritizing and scheduling sufficient time for sleep.

### Sleep disturbances in general

Healthy people in the general population, as well as people with sleep or psychological disorders experience disturbed sleep. The difference with sleep disturbance from that of sleep-onset problems, or sleep deprivation is that it is an overriding concept including multiple sleep problems and disorders such as insomnia, all of which affect the quality of sleep. Included in this concept is over 70 specific diagnoses within eight categories, and other sleep disorders related to medical or psychiatric disorders (ICSD-2, Sateia, 2005). Some of the difficulties associated with disturbed sleep are sleep-onset problems, frequent awakenings, and waking too early. Very often sleep disturbances are related to environmental factors such as room temperature, light, noise, etc. These factors singly or in combination may interact with a person's individual reactivity in shaping the degree to which the sleep disturbance is experienced (Muzet, 2004). Other factors may be due to lifestyle, health behaviors, and time constraints.

### Developmental sleep issues

Two of the studies in this dissertation deal with adolescent populations, and one is based on a largely young adult population. Adolescents and young adults, ages 12 to 25-years, are an especially vulnerable group and are considered a high-risk population for problems associated with sleepiness (National Heart Lung and Blood Institute, 1997). Not getting enough sleep appears to place adolescents and young adults at higher risk for cognitive difficulties, low academic performance, emotional problems, as well as injuries (Carskadon, 2002; Dahl & Lewin, 2002). Distal risks,

such as personality may keep adolescents on particular trajectories. Other examples are individual chronotypes, such as morningness or eveningness. Proximal factors, such as life-events, values held by friends, or parental attitudes about bedtimes may alter trajectories (Schulenberg, Sameroff, & Cicchetti, 2004). A particular distal risk for sleep problems and mental health is the influence of genes on problem stability. In a longitudinal study of twins, ages 8 to 10, genes appeared to govern 46% of the stability of sleep disturbances, with a hint of a genetic association between sleep problems and depression over time (Gregory, Rijdsdijk, Lau, Dahl, & Eley, 2009). Due to the close association between sleep disturbances and psychopathology, looking at sleep disturbances as a process involved in emotion dysregulation may be informative for prevention and intervention research.

Adolescent sleep differs biologically, socially, and behaviorally from that of other developmental periods. The differences are reflected in the prevalence and character of sleep disturbances during this period. Child and adolescent sleep disturbance prevalence estimates are quite high, falling between 25% and 40% depending on the study (see, Meltzer & Mindell, 2006). Roughly, 69% of American parents report that their children have difficulty falling or remaining asleep, several times a week. Approximately 51% of adolescents report sleep-onset problems at least once a week (National Sleep Foundation, 2006; Roane & Taylor, 2008). An epidemiological study with 1,125 15 to 18 year olds, found that 25% had insomnia symptoms and 4% experienced clinical insomnia (Ohayon, Roberts, Zulley, Smirne, & Priest, 2000). Another longitudinal study followed 943 teens from ages 13 through 15. One third of the youths experienced sleep disturbances at age 13. The prevalence of disturbed sleep at age 15 increased to 33%. Sleep disturbances in adolescence showed persistence with 48.5% experiencing disturbed sleep from ages 13 to 15 (Morrison, McGee, & Stanton, 1992). Many psychological disorders associated with sleep originate in adolescence with full onset occurring during the transition to adulthood (Merikangas et al., 2010). As such, adolescence may be a stress-sensitive developmental period for sleep related emotional health processes. Sleep in adolescence appears to be especially vulnerable to disruptions and to be a risk factor for a variety of concurrent and later health outcomes.

### Decreases in sleep quantity

It is relatively common that adolescent sleep quantities decrease in the transition moving from childhood toward adult sleep patterns. In a study comparing sleep duration for 3,120 students, ages 13 through 19, total sleep time decreased by 40 to 50 minutes across ages. Overall, decreases in

sleep were largely due to later bedtimes as youths became older (Wolfson & Carskadon, 1998). In addition to normative changes, sleep quantities also appear to be changing between cohorts over time. Researchers, looking at school day sleep trends, compared sleep duration for adolescents ages 10 to 15 in 1985, with those in 2004. They found that the overall sleep time declined between surveys by roughly 30 minutes (Dollman, Ridley, Olds, & Lowe, 2007). Biological and behavioral factors such as sleep-wake schedules influence the amounts of sleep that adolescents acquire.

#### Behavioral influence of biology

The onset of adolescence exerts changes to sleep patterns in several ways. A number of biological processes related to sleep regulation begin with the physical onset of puberty. One change is that many adolescents experience a biologically driven preference to stay up later and sleep later (Dahl & Lewin, 2002). This preference is a shift in adolescents' internal clocks called *circadian phase preference* (Crowley, Acebo, & Carskadon, 2007). Many adolescents' circadian clocks would have them wake up later than the early wake up time required by school systems. When at school some teens are not fully awake until their biological clocks would have awoken them. This leaves many teenagers sleep deprived and drowsy during the day (Carskadon, Wolfson, Acebo, Tzischinsky, & Seifer, 1998). The mismatch between adolescent changes in circadian rhythmicity, an increased need for sleep and earlier school schedules is called the *sleep paradox* (Wolfson, 1996). In addition to biological processes, behavioral factors can also exert influence on adolescent sleep patterns.

#### Life-style behavioral influences

Sleep schedule discrepancies between sleep-wake schedules on weekdays and weekends are a particular behavioral factor influencing adolescent sleep cycles. In a longitudinal study with 1,146 youths ages 10 through 13, sleep habits changed during the shift from childhood to adolescence. For a majority of youths, total sleep time decreased, bedtimes were delayed, and the difference between weekend and weekday schedules increased with age (Larberg, Petit, Simard, Vitaro, & Tremblay, 2001). Many adolescents have a shift in bedtime from weekday to weekend. However, those who experience sleep disturbances tend to have greater shifts (Kirmil-Gray, Eagleston, Gibson, & Thoresen, 1984). Sleep-onset problems are one type of sleep disturbance particularly associated with sleep schedule discrepancies between weekdays and weekends (Larberg et al., 2001). Perhaps sleep schedule discrepancies contribute to disturbances in sleep cycles and con-

tribute to sleep deprivation. In a diary study with 750 14 and 15 year olds, youths who slept less, slept less on both weeknights and weekdays. Those with varied sleep times throughout the week slept less overall (Fuligni & Hardway, 2006). In one study, annual and weekly changes in sleep-wake cycles were recorded in daily diaries by 64 youths ages 10 to 14. Indications were that youths may be sleep deprived during the school week and therefore sleep more during the weekend to compensate. In this study, the weekday sleep discrepancy disappeared during breaks and when on vacation (Szymczak, Jasinska, Pawlak, & Zwierzykowska, 1993).

There are other behavioral factors influencing adolescent sleep. After school activities, such as sports, socializing, hobbies, jobs, or homework also delay bedtimes. In a longitudinal study with 2,546 adolescents ages 13 through 16, having a television or computer in the bedroom was associated with later bedtimes. Adolescents participating in unstructured activities at night without end-times, such as surfing the internet, computer and console gaming, or watching TV, reported later bedtimes during the week and weekend, sleeping later on weekends, obtaining less sleep than peers, and more daytime drowsiness (Van den Bulck, 2004). Perhaps adolescent sleep behaviors contribute to the development of sleep disturbances and adjustment problems.

#### Antecedents and consequences of maladaptive sleep behavior

Whether due to biological or behavioral processes, many teens become increasingly sleep deprived. In a cross-sectional study looking at sleep and waking behaviors, roughly 87% of 3,120 high school students, ages 13 to 19, reported that they acquired less sleep than they think that they need (Wolfson & Carskadon, 1998). From ages 13 to 19, total sleep time successively decreased by 40-50 minutes. The gradual reduction in total sleep time was associated with later and later bedtimes as the teens became older. Adolescents with poorer grades had 25 minutes less sleep and went to bed on average 40 minutes later than those with good grades did. Having less sleep during the week and having 2 hour later bedtimes during the weekend was associated with more daytime sleepiness, depressed mood, and problems falling asleep and waking up (Wolfson & Carskadon, 1998), likening jetlag. In a longitudinal study with 2,259 early adolescents, ages 11 to 14, sleep deprivation posed a risk for depression and lower self-esteem (Fredriksen, Rhodes, Reddy, & Way, 2004). It appears clear that sleep deprivation has an effect on mental health.

Sleep disturbances are problematic during the day as well as at night. Sleep is important for cognitive functions such as memory (Susman et al., 2002), influences attention and behaviors affecting academic achievement

(Dahl, 1996; Dahl & Lewin, 2002). Results from longitudinal research suggest that many youths, ages 10 to 16, have difficulty adjusting to sleep reductions, sleep deprivation, and daytime drowsiness (Andrade, Benedito-Silva, Domenice, Arnhold, & Menna-Barreto, 1993). Adolescents who are sleep deprived often report daytime drowsiness (Dahl & Lewin, 2002). In two cross-sectional studies with 3,235 high school students, 70% were deemed mildly to severely sleep deprived. Roughly 41% percent of adolescents in the study reported excessive daytime sleepiness, with 58% to 68% reporting being “really sleepy” between 8 and 10 in the morning. Roughly 23% of these adolescents reported feeling that daytime drowsiness reduced their school performance (Gibson et al., 2006). A longitudinal study with 2,259 students ages 11 to 14, found that sleep loss was related to poorer grades (Fredriksen et al., 2004). Among long-term risks are reductions in life options due to low academic achievement. Sleep deprivation also has consequences for psychological health.

While adolescents may physically require more sleep, many are obtaining less. Perpetual sleep deprivation due to sleep disturbances or maladaptive sleep behaviors may influence emotion dysregulation. In a study with 242 adolescents, ages 10 through 16, obtaining less sleep during the week than on the weekend was associated with increased depressive symptoms (Pasch, Laska, Lytle, & Moe, 2010). Adolescent health behavior may lead to sleep deficits and daytime drowsiness, which in turn may influence emotion dysregulation. One consequence may be that adolescents are in a perpetual state of fatigue similar to jetlag.

### Neurophysiology, sleep, and psychopathology

In addition to biological and behavioral processes, physiological processes related to sleep exert pubertal maturational changes in hormone production from childhood into adolescence. Therefore, adolescence may be a critical period for physiological changes due to health factors such as sleep disturbances and depression (Susman et al., 2002). Stress sensitization for disorders may occur at the gene level (Monroe & Harkness, 2005; Post, 1992), by potentially aggravating or giving rise to disorder diatheses. Maladaptive cognitive, emotional, and behavioral patterns may become like well-traveled paths, becoming less dependent on life-stressors for recurrence over time. Findings from animal research suggest that persistent sleep-onset problems in adolescence may increase reactivity and reduce stress thresholds through physiological changes brought on by subsequent sleep deprivation (Meerlo, Sgoifo, & Suchecki, 2008). Sleep-onset problems may become a potent stressor, leaving adolescents sleep deprived and vulnerable to heightened emotion dysregulation over time (Susman et al.,



2002). Changes in hormonal processes may be both stress induced, and stress inducing. Thus, the relation between hormonal and psychological processes may be bidirectional. Cortical arousal is a reaction to the release of cortisol, a stress hormone associated with sleep disturbances. Changes in the production of hormones such as cortisol may disturb adolescent sleep (Forbes et al., 2006). In turn, sleep disturbances may disrupt mood and emotion regulation (Susman et al., 2002). These theoretical ideas and findings from animal studies are supported by research with adolescents.

Hormonal processes, related to sleep and emotionality seem especially vulnerable to dysregulation with the onset of puberty. The hypothalamic-pituitary-adrenal (HPA) axis is responsible for the regulation of cortisol. Dysregulated HPA axis hormonal processes may trigger existing vulnerabilities for stress related psychopathology (Walker, Sabuwalla, & Huot, 2004) such as sleep disturbances, anxiety, and depression. Anxiety and depression are associated with the development of sleep disturbances (Jansson & Linton, 2006b). In turn, sleep disturbances are also precursors for depression and anxiety (Taylor et al., 2003). The link between sleep, hormonal processes, and psychopathology may be through cortisol arousal at sleep-onset.

There is some evidence to support a link between sleep-related pubertal hormone production and psychopathology. Alterations of cortisol secretion before sleep-onset have been observed in a study with 224 children and adolescents, ages 9 through 16, investigating neurobehavioral characteristics of children and adolescents with anxiety and depression in comparison to a control group. Sleep related physiological changes were associated with pubertal changes from childhood to adulthood. Sleep-onset cortisol regulation for those with anxiety and depression was different depending on whether experienced in childhood, or adolescence. Adolescents with depression had higher pre-sleep cortisol than other adolescents, or children with depression. Adolescents with anxiety experienced higher pre-sleep cortisol arousal than the adolescent control group (Forbes et al., 2006). In another study with 59 adolescents, ages 12 to 18, those who were depressed had hyper-secretion of cortisol at sleep-onset relative to controls (Dahl et al., 1991). Thus, a link between sleep and mental health problems may be through the dysregulation of cortisol at sleep-onset. Biological, behavioral, and neurophysiological influences in adolescence may all negatively influence sleep and consequently drive emotion dysregulation.

## **Emotion**

On a taxonomy level, emotions are innate and universal programs for motor and autonomic reactions in response to subjective emotional experienc-

es, and for expression. They are also complex feeling states resulting in physical and psychological changes that influence thoughts and behavior. Emotions are associated with a range of psychological phenomena including temperament, personality, mood, and motivation.

### **Emotional processes**

Emotions have a long evolutionary history, playing a critical role in the survival of the human species by influencing adaptive survival and social behaviors (see, Gainotti, 2012; Haselton & Ketelaar, 2006). There are a myriad of definitions for what constitutes emotion. There is as much disagreement as there are theories of what emotion is. There is one point of agreement however. Emotion is widely defined as a group of highly integrated psychological states including subjective experience, expressive behavior (e.g., facial, bodily, verbal), and physiological responses (e.g., heart rate, respiration; for review see, Gross & Barrett, 2011). The development of emotion on all levels of organization is thought by researchers to be epigenetic. That is, people are actively involved in their own emotional development. Emotions are continual processes (e.g., feelings, thoughts) involving interactions within a developing person and the environment (i.e., endogenous, exogenous; Mason & Capitanio, 2012). There is an abundance of emotion theories and models. Extensive descriptions of competing theories are outside of the scope of this dissertation.

Herein, emotion refers to one of two operative systems used by organisms to adapt to their environments. These are the emotional and the cognitive systems. The emotional system can be thought of as an early alert system outside of awareness. Emotions in this sense, act quickly to interrupt current activity in order to select appropriate responses. The cognitive system is more complex and more highly evolved. Therefore, the cognitive system operates much slower and secondary to the emotion system (Oatley & Johnson-Laird, 1987). The emotional system is central to this dissertation. The cognitive system plays a minor role in this dissertation as a secondary process through which cognitive vulnerability may interact with sleep disturbances and emotion dysregulation in psychopathology.

A developmental model of emotion suggests that there are three functional levels of emotion processing. These are the sensorimotor, the schematic, and the conceptual levels. The sensorimotor level is comprised of innate and universal programs for expression, motor and autonomic reactions in response to subjective emotional experience. The three processes are linked by developmental mechanisms, such as the development of the sensorimotor level through conditioned learning and individual experiences, which then develop into the emotionally laden schematic units underly-

ing the schematic level. The schematic level of emotional processing is schematically primed and automatically elicited. These are spontaneous emotions experienced as feelings, or true emotion. The third level is the conceptual level, which is explicit and involves declarative memories, abstract emotional concepts, and social rules for emotional expression (Gainotti, 2012). The sensorimotor level corresponds to subjective emotion experience. The schematic level is associated with implicit emotion automatically evoked by external stimuli through the utilization of schemata, without conscious monitoring, insight or awareness (Bargh & Williams, 2007). The first two implicit levels are those relevant to this dissertation.

### **Emotion working definition**

In this dissertation, emotion is used as an umbrella concept, encompassing a breadth of emotional phenomena from the broad to the specific. Emotion is often used interchangeably in emotion research with affect, as both a collective designation and a specific component. Emotion in the collective sense in this dissertation refers to hierarchical emotion phenomena. While acknowledged that the highest order hierarchical component of emotion is the hedonic experience of pleasure and displeasure (Russell & Carroll, 1999a, 1999b; Tellegen, Watson, & Clark, 1999a, 1999b), this level along with arousal are not within the scope of the dissertation other than as experimental manipulation checks. Instead, the broad emotion construct encompasses a widely defined hierarchy comprised of trait-like emotional dispositions and affective states at the highest levels and state-like emotions and moods at lower levels. The higher levels reflect emotional valence and the lower levels reflect specific content (Gross, 1998; Watson & Clark, 1999). An important distinction in the various components of emotion is occurrence in time. They may be long-term, dispositional and trait-like or episodic and state-like. Dispositions (e.g., neuroticism) can be thought of as omnipresent, underlying emotional tendencies influencing the experience of an internal or external event. Emotions may last for a few seconds (e.g., surprise), moods for many years (e.g., depression). Moods are typically diffuse, slow moving, long lasting, and not anchored to an object or eliciting stimulus. Emotions are valenced responses to external stimuli and/or internal representations (Gross, 1998). Emotions involve changes across multiple response systems, such as experiential, behavioral, and physiological. Multiple appraisal processes are thought to link the significance of the emotion to current goals, which depend upon various neural systems (Ochsner & Gross, 2005).

In other words, emotions involve changes across multiple response systems, such as cognitive, behavioral, and physiological. Emotion can there-

by be viewed as a process of appraisal, affective states (e.g., facial, vocal, behavioral) including subjective emotion experience, and the upward or downward regulation of the affective states and behaviors (for review see, Phillips, Drevets, Rauch, & Lane, 2003). This dissertation is limited to the subjective emotion experience and implicit emotion regulation, which are defined below. Negative emotionality, higher negative affect and emotion, lower positive affect and emotion, as well as depressive symptoms are used as markers of emotion dysregulation.

### **Emotion regulation and dysregulation**

In an enormous array of psychiatric disorders, emotion dysregulation is one common denominator that prominently stands out. Emotion dysregulation is such a common factor in disorder comorbidity that it is suggested as a transdiagnostic process (for review see, Kring, 2008). In a recent clinical review, a model of emotion dysregulation in mood and anxiety disorder was presented for how emotion dysregulation stemming from diathesis and stress may lead to mood and anxiety disorders (Hofmann, Sawyer, Fang, & Asnaani, 2012). Theoretically, processes initiating from a stressful triggering event, interact with existing diatheses in disorder development. Secondary to the initial stressor and diatheses, affect and emotions become dysregulated. Dysregulation of positive and negative affect and emotion are implicated in resulting in mood related psychopathology such as anxiety and depression.

People regulate their emotions deliberately through their thoughts and behaviors or automatically outside of awareness in an attempt to modulate which emotions they have, when they have them, and how they experience or express them. Regulation of emotion is the action of regulatory processes initiating, or altering ongoing emotional responses (Gross & Thompson, 2007; Ochsner & Gross, 2005). Emotion regulation is broadly defined as a set of processes used by people to manage all emotional states including broad affects, moods, specific emotions, and stress (Koole, 2009). In the literature emotion regulation is described in terms of targets and functions. Primary targets of emotion regulation are attention, relevant cognitive-emotion knowledge, and bodily emotional manifestations. Emotion regulation seeks to meet psychological functions such as the satisfaction of hedonic needs, furthering goal pursuits, and facilitation of the global personality system (Gross & Thompson, 2007). The emotion regulatory processes are complex involving multiple systems (e.g., cognitive, behavioral, physiological).

Emotion regulation is likely a continuum from conscious effortful control, to unconscious effortless automatic control. Emotion regulation is

both intrinsic and extrinsic. In other words, emotion regulation is both extrinsically controlled and implicitly out of conscious control, internally and externally managed (see, Gross, 1998). Implicit emotion regulatory processes occur outside of awareness, are schematically primed or automatic modulation of emotions (Gainotti, 2012). In this dissertation, these processes are not measured or investigated directly. Instead, phenomenological aspects of implicit emotion regulation were measured by changes, prior to and after strong emotion elicitation. Explicit emotion regulation, which is the conscious controlled and self-reflective upward or downward regulation of emotion in response to an emotion stimulus, is outside the scope of this dissertation.

This dissertation examines emotion regulation in adolescence, adulthood, and from adolescence into midlife. Adolescence is a time with rapid temperamental, neurobiological, conceptual, and social changes occurring. Together these changes interact with individual differences to create foundations for emotion regulation, which may follow into adulthood (Calkins & Hill, 2007; Rothbart & Sheese, 2007; Thompson & Meyer, 2007). For instance, physiological changes may be brought on by health behaviors such as sleep disturbances (Susman et al., 2002), and sleep incompatible behaviors learned in adolescence may carry into adulthood. Changes to emotion regulatory processes continuing and developing over the life-course are largely due to contextual factors (Thompson, 2006).

Dysregulated emotions are poorly modulated emotions, not falling within the conventionally accepted range of emotional responses. Various facets define emotion dysregulation, such as negative emotionality (neuroticism); reductions in positive affect and increases in negative affect; specific negative emotions; and depressed mood. The differences are temporal, either occurring within seconds as a reaction, or pervasively underlying negative experiences (Gross & Thompson, 2007). In this dissertation, several markers of emotion and emotion dysregulation are used. These markers are negative emotionality, positive and negative affect and emotion, and depressive symptoms. Changes in these emotion markers were used to gauge implicit emotion regulation.

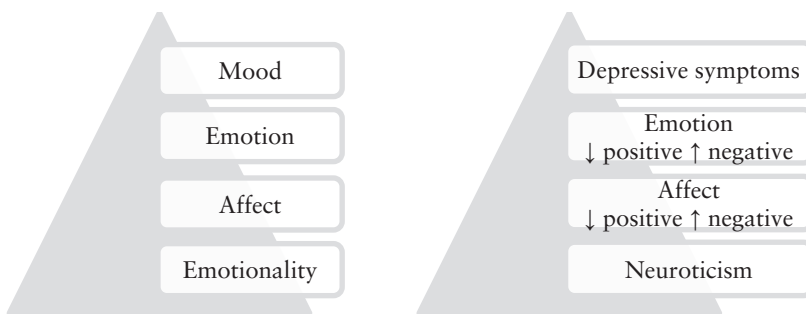
### Markers of emotion and dysregulation

Disordered emotions are measured by levels on emotion markers, ranging from negative emotionality (i.e., neuroticism) to positive and negative affect and emotions, to negative mood (i.e. depressive symptoms). See Figure 2 for a hierarchical structure of emotional phenomena used as markers in this dissertation.

## Neuroticism

Simply defined, neuroticism (a.k.a., negative emotionality, trait anxiety, negative affectivity) is a personality trait, or emotional disposition used in personality research as a gauge of emotional stability (see, Lahey, 2009). Neuroticism is characterized by negative emotionality, and a tendency to experience negative emotions in response to blocked goals. Another way of thinking of neuroticism is as a negative emotional reactivity continuum ranging from low to high. For instance, a person high in neuroticism may have strong negative reactions in the response to threats, frustration, or loss, while someone low in neuroticism may just brush it off. Another instance, people high in neuroticism but within the normal range, may experience heightened neuroticism during times of stress. Neuroticism is related to and a robust predictor of many mental and physical health problems (see, Lahey, 2009).

Figure 2. *Emotion hierarchy and hierarchical markers of emotion dysregulation.*



While personality traits are relatively stable, personality development continues through the lifespan, plateauing in midlife and declining in old age. Genetic influences on personality stability have the greatest influence during childhood through young adulthood. Stability and changes thereafter are largely due to environmental influences (for review see, Kandler, 2012) and person-situation interactions (see, Buss, 2009). From an evolutionary perspective, genes guide people in selection of situations and interactions with people who fit their personality, influence behavior which evokes behaviors from others, and manipulation of the environment in personality congruent ways (see, Buss, 2009).

Neuroticism is related to and a robust predictor of many mental and physical health problems (see, Lahey, 2009). Little is known however about the influence of health behaviors on neuroticism. Sleep-onset problems are associated with neuroticism. Unknown however is the direction of

effects of this association or the long-term developmental course from adolescence through midlife. Unknown is whether neuroticism drives sleep-onset problems or the other way around. Possibly sleep-onset problems aggravate a predisposition toward negative emotionality and perhaps this aggravation has a long-term effect. That is one of the questions that I sought to answer.

### **Positive and negative affect and emotion**

Through natural selection, evolution led the emotion system down two valenced affective paths, positive and negative. Affect is emotion valence and emotions are what people actually subjectively feel in response to the neurochemicals produced in reaction to a subjective experience (Watson & Tellegen, 1985). Rather than polar opposites, positive and negative affect are two distinct and independent aspects of neuroanatomical functions in emotion ranging on a continuum from high to low (Hamann, Ely, Hoffman, & Kilts, 2002). Reports of subjectively experienced emotions allows researchers to objectively study what emotions feel like to individuals (see, Barrett, Mesquita, Ochsner, & Gross, 2007). Love for example has ecological precedents (e.g., comfort, care, contact, pheromones) prior to antecedent neurochemical-level changes in the body (e.g., oxytocin, endorphins, serotonin, and dopamine). The resulting flood of feelings is what is generally described as subjectively felt emotions (see, Buck, 2012). In a neutral state people may feel lower levels of both positive and negative affect. There seems, however, to be an innate adaptive positivity offset, or tendency for people to feel mild positive emotions even when in neutral situations (Diener & Diener, 1996). It is also possible to feel high levels of positive and negative emotions. An example is the birth of a child occurring at the same time that a loved one has died. Humans have an amazing capacity to experience two opposing emotion valences at the same time, such as sorrow and joy. These two processes correspond with approach and avoidance motivational systems. Emotions serve adaptive functions helping people meet threats and opportunities (Nesse & Ellsworth, 2009). Positive emotions serve an appetitive function, and negative emotions serve an aversive function (see, Hamann et al., 2002). Positive and negative emotions are both important to subjective emotion experience and implicit emotion regulation.

Broaden and build theory suggests that the function of positive emotions is to promote flourishing and resiliency. Positive emotions are associated with a positive valence, are associated with opportunities, and approach behaviors (Fredrickson & Cohn, 2008). Theoretically, positive emotions facilitate the broadening of awareness and engagement with the

environment. This is through the promotion of approach and exploration of novel objects, people, and situations. Positive emotions are also associated with diverse and exploratory cognitions and behaviors. Behaviors, skills, and resources are expanded upon through experiences such as meeting new people and trying new activities (Fredrickson & Cohn, 2008). Researchers suggest that for positive emotions to surpass the strength of negative emotions, positive emotions must greatly outnumber negative emotions. Theoretically, if people experience more negative than positive, or not enough positive to offset the negative, then they may feel emotional distress and social impairment (see, Fredrickson & Cohn, 2008). In addition, positive emotions are implicated in counteracting negative emotions and aiding in recovery from anxiety and fear (see, Fredrickson & Cohn, 2008). Positive emotions may not have just restorative and protective roles, but also a dysfunctional role.

Positive, like negative emotions, are associated with disorder when extreme, long lasting, or inappropriate to the context. Examples are mania when positive emotion is too high, and depression when too low. Disordered positive emotion regulation and the inability to engage emotion related brain structures important to positive affect and reward are features of depression (Heller et al., 2009). Indeed, lowered positive emotion, or a reduced capacity to experience pleasure is a formal symptom criteria for major depression (DSM-IV-TR; American Psychiatric Association, 2000). Perhaps one way in which positive emotions are involved in psychopathology is through reductions in some of the compensatory benefits and resiliency strengths that are missing when positive emotion levels are lower (Fredrickson & Cohn, 2008).

A facet of sleep disturbance generally examined with positive and negative affect is sleep deprivation. Lower positive affect is a facet of emotion showing greater specificity for depression than other diagnostic criteria. Research has shown low positive affect to be most strongly related to disorder specific symptoms of depression (e.g., anhedonia, dysphoria, lassitude, suicidality) than to other psychopathology such as anxiety (Watson & Naragon-Gainey, 2010). Reduced positive affect is also characteristic of sleep deprivation in both adolescent (Dagys et al., 2012; Talbot et al., 2010) and adult populations (Franzen, Siegle, & Buysse, 2008; Talbot et al., 2010). In a study examining the effects of one night's sleep deprivation on facial expressiveness, an especially large inhibiting effect was found for positive affect relative to negative affect when viewing emotionally evocative film clips. The same study found similar effects on subjective measures, these however, failed to reach significance (Minkel, 2010). This suggests



that positive affect is potentially more susceptible to the influence of sleep deprivation than negative affect.

Negative emotions have a negative valence and are associated with displeasure. They are attention grabbing, more differentiated and complex than positive emotions. There are many more ways of describing negative than positive emotions (Watson & Clark, 1999). Through much of evolutionary history, there has been a greater survival value for attenuation to negative events over positive ones. Negative emotions are associated with physiological arousal that aids in acting in threatening situations, brings threats into awareness, and aids in dealing with loss (see, Fredrickson & Cohn, 2008; Garland et al., 2010; Nesse & Ellsworth, 2009). People tend to ruminate more over negative arousing events than over positive events. In addition, memory recall for negative memories is stronger than for positive memories (Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001). Negative emotions are those most associated with psychopathology. An otherwise adaptive process, negative emotions when exaggerated, frequent, or long lasting may be maladaptive. Types of problems associated with negative emotion are anxiety, aggression, depression, and suicide (Fredrickson & Cohn, 2008). Negative emotion may be adaptive, or become dysregulated.

Increases in negative affect following sleep deprivation have been reported with medical residents confronted with goal-discordant events (Zohar, Tzischinsky, Epstein, & Lavie, 2005) rather than passive exposure to sad film clips. In previous research, sleep deprivation has been demonstrated to impair inhibitory control in cognitive (Chuah, Venkatraman, Dinges, & Chee, 2006) and emotional tasks (Yoo, Gujar, Hu, Jolesz, & Walker, 2007). In comparison with those who were rested, sleep deprived people reported higher levels of anxiety, anger, and confusion in response to low levels of stress. In contrast, affective responses to higher stress levels were milder and failed to reach significance (Minkel, 2010; Minkel et al., 2012). These findings suggest that perhaps sleep deprived people over-respond to milder stressors and respond normally when faced with stronger stressors (Minkel, 2010). Negative affect is a key element common to most psychopathology.

### **Depressive symptoms**

Depressive symptoms have been demonstrated as good markers of depressed mood, in adolescent populations (see, Olsson & Von Knorring, 1997). Moods generally have a positive or negative valence, are less specific than emotion, less intense, and less likely to be triggered by a particular stimulus or event. Markers of depressive symptoms are herein defined as

emotions and behaviors such as feeling depressed or blue, lonely, sad, or crying. The more symptoms, the stronger they are, the longer they have lasted, the more likely that a person is suffering from depression.

Adolescent depression is of concern because research has shown that depression onset by age 15, and recurrent depression by age 20, may increase the risk of life-course persistent depression. Other associated risks are concurrent and long-term adjustment problems such as attention deficit hyperactivity disorder (Hammen, Brennan, Keenan Miller, & Herr, 2008; Hasler et al., 2005), smoking, substance abuse (Keenan-Miller, Hammen, & Brennan, 2007; Rao, 2006), and suicidality (Sihvola et al., 2007). Many adults with recurrent depression experienced first-onset in adolescence (Hammen et al., 2008). All of which make adolescent depression a critical research domain.

Another facet of adolescent depression warrants attention. Starting in puberty, prevalence rates for depression are higher for girls, with the greatest increase between ages 15 and 18 (Essau, Lewinsohn, Seeley, & Sasagawa, 2010; Hilt, McLaughlin, & Nolen-Hoeksema, 2010). In a study with adolescents, ages 11 to 14, a link was shown between insufficient sleep and higher levels of depression over time. It has long been established in the literature that the risk for sleep disturbances, catastrophic worry, and depression in adolescence is greater for girls than for boys (Flouri & Panourgia, 2011; Hilt et al., 2010; Johnson, Roth, Schultz, & Breslau, 2006). In addition, gender seems to predict the amount of sleep acquired (Fredriksen et al., 2004). The greater tendency for girls to experience catastrophic worry and to ruminate has been suggested to account for the gender difference in vulnerability for depression (Hilt et al., 2010). All of which suggests that the effects of sleep disturbances on depressive symptoms may be differential dependent on gender. Therefore, sleep disturbance was examined as a precipitator of adolescent depressive symptoms, and as a perpetuating process through catastrophic worry, a potentially gender specific cognitive vulnerability.

### **Cognitive vulnerability: Catastrophic worry**

Worry is an example of an adaptive cognitive emotion regulation strategy that when exaggerated may pose risk for emotion dysregulation. Worries of healthy people are generally limited to bringing potential threats into awareness, and aiding in constructive problem solving (see, Watkins 2008). Worry is less adaptive when exaggerated (Ruscio, 2002) and when becoming catastrophic (Wells & Carter, 1999). Like many psychological processes, worry ranges on a continuum from high to low. Excessive use of maladaptive emotion regulation strategies such as worry, are related to the de-

velopment and maintenance of psychopathology such as depression (see, Aldao & Nolen-Hoeksema, 2011). Furthermore, people who are depressed report significantly elevated levels of worry (see, Mathews & MacLeod, 2005). Catastrophic worry is a reappraisal process involving continuous automatic questions, such as “What if...,” that progressively escalate the worsening of imagined outcomes to specific worries. Catastrophic worry incites, maintains, and fuels feelings of uncertainty, anxiety, discomfort, and the belief that catastrophic outcomes are impending (Davey, 2006; Robichaud & Dugas, 2006; Vasey & Borkovec, 1992). Once the process of catastrophic worry is initiated, it may become cyclical and difficult to stop. Reappraisal is a cognitive emotion regulation strategy ordinarily used to down-regulate negative emotions. However, negative reappraisals, as in catastrophic worry, may serve to regulate negative emotions upwards, contributing to fear and anxiety (Ochsner, Silvers, & Buhle, 2012). This may occur in due to a rebound of negative emotion in response to emotion elicitation. Consequences may be memory difficulties, sympathetic activation, and reductions in autonomic flexibility. Catastrophic worry may be a key factor in maintaining disorder.

Perhaps catastrophic worry is a mediator, or process, bridging biological factors such as sleep disturbances with psychopathology such as depression. An example relative to this dissertation is the maintenance of insomnia by catastrophically worrying about the consequences of not obtaining enough sleep (Harvey, 2002; Harvey & Greenall, 2003). Negative moods may be maintained by repetitive focus on the states initiating and maintaining it, but perhaps once sat into motion catastrophic worry becomes over-generalized.

It is common for tired people to worry, experience negative emotions, report aches and pains, or report just having had an argument. Very often, the content that people are worrying about has little to do with what initiated the bout of worrying (see, Robichaud & Dugas, 2006). Theoretically, once worry becomes maladaptive, or catastrophic, a self-sustaining negative feedback loop may be set into motion (see, Mathews & MacLeod, 2005). Consequently, repeated catastrophic worry may become a cognitive vulnerability or diathesis, leaving people susceptible to disorders such as depression (Abela & Hankin, 2008). Perhaps sleep disturbance not only pose risk for depression, but also serve to maintain depressive symptoms through cognitive vulnerabilities such as catastrophic worry. Sleep disturbances may not only precipitate, but might also perpetuate emotion dysregulation through catastrophic worry.

## **Empirical findings: Sleep disturbances and emotion**

### **Sleep-onset and negative emotionality**

There is some evidence to support a link between sleep-onset problems and negative emotionality. In studies with college students, sleep-onset problems were associated with heightened neuroticism (Calkins, Hearon, Capozzoli, & Otto, 2013; Williams & Moroz, 2009). In comparison to a normative sample, college students with sleep-onset problems reported higher levels of neuroticism (Shealy, Lowe, & Ritzler, 1980). In a sleep laboratory, the sleep-latency of good and poor sleepers differentiated dependent on personality. People with delayed sleep-onset tended to experience more neuroticism (Freedman & Sattler, 1982). In another study, there were personality differences between university students with objective, versus subjective sleep-onset problems. Students, who subjectively experienced sleep-onset problems reported more neuroticism on average than those with objective sleep-onset problems (Dorsey & Bootzin, 1997). Sleep-onset problems is a sleep disturbance commonly associated with neuroticism. Neuroticism is related to and a robust predictor of many mental and physical health problems (see, Lahey, 2009). Perhaps this is a route through which sleep disturbances contribute to emotion dysregulation.

### **Partial sleep deprivation and emotion dysregulation**

There is evidence supporting a role for sleep dependent processes in emotion dysregulation. Mood may be especially vulnerable to the effects of sleep deprivation. In a study examining cumulative sleep debt, 16 young adults had sleep restricted to 4-5 hours per night for one week. Mood appeared vulnerable to sleep restriction with deficits increasing steadily into the last two days of restriction (Dinges et al., 1997). Researchers suggest that stress-related neurobehavioral effects of partial sleep deprivation may increase sensitivity to mood related disorders. In a study with 25 adults, looking at associations between sleep deprivation and psychopathology, people with 56 hours wakefulness reported sub-clinical increases in affective symptoms of psychopathology (Kahn-Greene, Killgore, Kamimori, Balkin, & Killgore, 2007). A meta-analysis examining the effects of sleep deprivation on performance and mood, found mood more affected by sleep deprivation than either cognitive or motor performance. In addition, partial sleep deprivation had a more profound effect on functioning than either long-term or short-term sleep deprivation (Pilcher & Huffcutt, 1996). In addition to mood, there are other reported effects of sleep deprivation, which provide insight into how sleep disturbances may influence emotion dysregulation.

Research during the past decade indicates that sleep deprivation poses risk to emotion regulatory processes normally occurring during sleep. These sleep dependent process may make a difference for how people experience emotion, and implicit emotion regulation during the following day. The field of neurobiology has laid a framework for how sleep dependent emotion regulation processes may become disrupted by sleep deprivation (Walker & van der Helm, 2009).

Two emotion regulatory processes appear to be especially sleep dependent. These are the modulation of emotional memories and regulation of emotional reactivity. Researchers suggest that sleep deprivation disrupts two sleep-dependent emotional memory processes simultaneously. These are emotional memory consolidation and attenuation of negative affective memory tone (Walker, 2009b; Walker & van der Helm, 2009). Accordingly, some people following sleep deprivation may be predisposed to negative encoding and retrieval bias in emotional memory (Sterpenich et al., 2007), strengthening of negative memories during REM sleep, and hyperlimbic reactivity in response to next day negative emotional events (Yoo, Gujar, et al., 2007; Yoo, Hu, Gujar, Jolesz, & Walker, 2007). Sleep deprivation appears to play a key role in next day both emotion experience, and emotional reactivity a component of emotion regulation.

Normally over time, the strength of a memory (hippocampal) remains the same upon re-exposure, while emotional reactivity (amygdala) declines (Walker & van der Helm, 2009). Research with animals has shown that as well as being a portent stressor, sleep deprivation alters reactivity to stress (Meerlo, Koehl, van der Borgh, & Turek, 2002; Novati et al., 2008; Sgoifo et al., 2006). Prefrontal cortex responses in brain structures, such as the hippocampus, have been found to rebound after chronic stress, but not amygdala responses (Quirk, 2007). Participants sleeping well after learning showed improved memory for emotional experiences. These memories were associated with reduced amygdala reactivity. Sleep-deprived participants had poorer recall of arousing emotion slides 72 hours later. Despite this, upon re-exposure to these pictures, there was no reduction in amygdala reactivity. People not sleeping the first night after emotional learning, despite receiving two nights full recovery sleep, had no reduction in amygdala reactivity (Sterpenich et al., 2007). This suggests that without reduction of affective tone of an emotional memory, the magnitude of the affective charge will remain in autobiographical memory. Thus, this nightly process may become cyclical, repeating night after night until resolved (Walker & van der Helm, 2009). Perhaps this is an analog for how sleep disturbances may interact with emotion dysregulation in producing symptoms of disorders such as insomnia, or depression.

Researchers have an idea how this may work. Normal sleep may act as affective therapy by the reactivation of affective experiences without associated affective chemical processes. The associated affective charge diminishes during encoding and the memory consolidates. This may enable large-scale neurological network cooperation at night, allowing for integration, and greater understanding of recent emotional events in light of existing stored semantic memories. REM sleep and perhaps dreaming may allow for emotional processing without affective chemical processes involved in stress and anxiety responses (Walker & van der Helm, 2009). Thereby people may be able to later recall incidents without stress and anxiety laden emotional reactions.

Another way that sleep disturbances may influence emotion dysregulation is through cognitive processes such as faulty beliefs about sleep and maladaptive attentional biases. Researchers investigated psychophysiological reactivity to sleep-related emotional stimuli dependent on whether or not people met the criteria for primary insomnia. Compared to controls, people meeting insomnia criteria showed an increased physiological craving response for positive sleep-related stimuli, increased physiological response to all stimuli, and increased subjective hyper-arousability for negative stimuli. Craving was defined as decreased corrugator (i.e., facial nerve or muscle) activity in response to positive sleep related stimuli, relative to all other stimuli (Baglioni et al., 2010). These findings show similarities with the attention-intention-effort theory (Espie, Broomfield, MacMahon, Macphee, & Taylor, 2006). Theoretically, people with insomnia perceive sleep requirements or deficits, which motivate sleep. Inability to sleep becomes a serious threat, sleep-related stimuli enhances arousal in order to monitor for threats to sleep (Baglioni et al., 2010). This may contribute to a negative attentional bias while awake. Looking at the day's events with a negative attentional bias during the day may potentially influence the subjective experience of positive and negative emotion by priming people to look for the negative and perhaps overlook positive events. In addition, if next day emotional reactivity is not reset during the previous night-sleep processes, then perhaps people are left more vulnerable to the emotion dysregulation in response to daily stressors.

Sleep loss in addition to amplifying negative affective reactions, is also thought to reduce positive emotional benefits related to rewarding or goal-enhancing activities (Zohar et al., 2005). In turn, positive affect is thought to encourage restorative health behaviors such as sleep, exercise, and relaxation. Positive emotions are suggested as having a protective role in modulating negative stress responses (Gruber, Oveis, Keltner, & Johnson, 2011). There appear to be several routes through which sleep deprivation

may influence emotion experience and implicit emotion regulation. It seems that sleep disturbances contribute to precipitation of emotion dysregulation through several sleep dependent processes including dysregulation of positive and negative emotions.

### **Sleep disturbance and emotion dysregulation**

Sleep disturbances are associated with a host of psychological disorders, are a diagnostic criterion for many mood disorders, as well as, a risk factor for several (Harvey, 2008a, 2011; Harvey et al., 2011). Thus, sleep disturbance is a potent threat to emotional health. Sleep disturbances are implicated in the development and maintenance of many psychiatric disorders, especially mood disorders (Benca et al., 1992; Harvey, 2008a; Harvey et al., 2011). For example, insomnia is a potent risk factor for depression (Perlis et al., 2006), problems with bipolar disorder (Harvey et al., 2006) and attention deficit hyperactivity disorder (Dahl & Harvey, 2007), and development of anxiety (Breslau et al., 1996) and post-traumatic stress disorder (Krakow et al., 2001). Sleep deprivation has been shown to be related to biased negative emotional memory encoding and retrieval (Sterpenich et al., 2007), physiological next day reactivity (Yoo, Gujar, et al., 2007; Yoo, Hu, et al., 2007), as well as, an increased physiological response to all stimuli, and increased hyperarousability for negative stimuli accompanying insomnia (Baglioni et al., 2010). These findings show similarities with the attention-intention-effort theory (Espie et al., 2006). During the day following sleep disturbances, people may develop a negative attention bias and become hypersensitive to threat cues in their environments. Perhaps there are other regulatory processes at work outside the bounds of conscious awareness.

Another way in which sleep disturbances may impart risk for emotion dysregulation may be indirect through cognitive processes, such as catastrophic worry. During the day, good sleep may enhance (Pace-Schott, Nave, Morgan, & Spencer, 2011) and sleep disturbances may hinder (Killgore, Balkin, & Wesensten, 2006) emotionally guided decision-making. The high rates of sleep disturbances in mental health disorders suggest a modulating role of sleep for emotion (Walker & van der Helm, 2009). Research suggests that in addition to processing the day's events, the brain is also prepared through sleep for next day's cognitive processes and emotional reactivity (Walker & van der Helm, 2009). Sleep is implicated in the modulation of the emotional psychological processes of extinction and habituation (Pace-Schott et al., 2009; Pace-Schott, Shepherd, et al., 2011). Perhaps sleep disturbances not only pose a direct risk for emo-

tion dysregulation, but perhaps also indirectly through ensuing cognitive vulnerabilities such as catastrophic worry.

Evidence from neurological research suggests that chronic stress may heighten amygdala ability for learning and expression of fear associations, and lessen prefrontal cortex ability to control fear. Theoretically, this could lead to a vicious circle with escalating fear and anxiety leading to additional stress and increased dysregulation (Quirk, 2007). The above scenario is called “allostatic load” and is useful for explaining chronic sleep disturbances as potent catalysts of emotion dysregulation (McEwen, 2003, 2006). Researchers have found that prefrontal changes are reversible following stress cessation (Radley et al., 2005), while changes to the amygdala are not (Vyas, Pillai, & Chattarji, 2004). In addition, chronic stress is known to create pathology in the hippocampus (McEwen, 2003). As reviewed previously the hippocampus is related to prefrontal cortex brain responses and memory. All of which suggest that chronic stress induced by sleep disturbances may potentially create permanent alterations to fear circuits thus. Thus, it appears sleep disturbances pose risk for depression not only directly, but also indirectly through subsequent cognitive vulnerabilities. Conceivably, sleep disturbances are involved in precipitation and perpetuation of emotion dysregulation. It appears that sleep disturbances may exert influence on the predisposition for, precipitation and perpetuation of emotion dysregulation, leaving people vulnerable to psychopathology.

### **Remaining problems, limitations, and unanswered questions**

The connection between sleep disturbance and emotion related psychopathology is well established. However, most of what is known about this relationship is from the fields of neurology, physiology, clinical research or based on adult populations. Less well-known are the risks posed to emotion regulation by disturbed sleep from adolescence through midlife. Regardless of the cause, sleep disturbances might subsequently become a stress factor contributing to psychopathology through emotion dysregulation. Sleep disturbances likely play several roles. It is conceivable that sleep disturbances are predisposing, precipitating, and perpetuating factors for developing emotion related psychopathology. Perhaps sleep disturbances are a contributing factor in long-term development of psychopathology by conferring risk through aggravation of predisposition for emotion dysregulation. This would suggest a mediating role of sleep disturbance for emotion dysregulation. Alternatively, sleep disturbances act as a stressor, initiating onset of emotion dysregulation. This would be indication of a precipitating role of sleep disturbances for emotion dysregulation. Additionally, sleep disturbances may perpetuate psychopathology through secondary



processes related to emotion dysregulation. This would suggest that sleep disturbances also have a perpetuating influence on emotion dysregulation through fueling of potential cognitive vulnerabilities such as catastrophic worry. Largely unknown are the mechanistic processes in the relationship between disturbed sleep and emotion dysregulation which may pose risk for developing psychopathology in otherwise normal populations.

### **Sleep-onset problems**

Missing from the literature are longitudinal studies exploring the direction of effects between sleep-onset problems and neuroticism from adolescence through midlife. People with sleep-onset problems reportedly have more neuroticism than people who sleep well (Dorsey & Bootzin, 1997; Freedman & Sattler, 1982; Shealy et al., 1980). Sleep-onset problems may aggravate predisposition for negative emotionality, or vice-versa. Which problem proceeds or precipitates the other cannot be determined with cross-sectional or retrospective reports and the latter have the added disadvantage of memory distortion. Sleep disturbances such as sleep-onset problems may predispose, or precipitate emotion dysregulation by heightening emotional reactivity and contributing to emotional instability. Looking at the relationship between sleep-onset problems and neuroticism from adolescence through midlife allows for a determination of the direction of effects, and provides a glimpse into whether early sleep disturbances may predispose people to emotion dysregulation over time.

### **Partial sleep deprivation**

More is known about the adverse effects of total short-term sleep deprivation to the brain and cognitive function (Lim & Dinges, 2010). Less is known about the consequences of continuous partial sleep deprivation, whereby people gradually lose more and more sleep over the course of days, months, or years (Zaharna & Guilleminault, 2010). What has been missing from the literature is how short-term partial sleep deprivation may negatively impact subjective emotion experience and the implicit emotion regulation of otherwise healthy adults, not only as a direct result of sleep deprivation, but also in response to a strong negative emotion elicitation. More specifically, I was interested in examining the influence of partial sleep deprivation on multiple levels of emotion to gain a better understanding of implicit emotion experience and dysregulation at various levels of the emotion hierarchy (i.e., broad affect, basic, and specific). This was in order to examine whether the effects of sleep deprivation on emotions was the same on several levels. If we take positive emotions for example, it is possible that positive affect would be dysregulated, just one or two positive

basic emotions, and only a few specific. Looking at this question provides an idea about how the lower and higher levels are affected. Of interest was a deeper understanding of the differential impact of partial sleep deprivation on the regulation emotions following sleep deprivation, and emotion elicitation.

In this dissertation, I use a randomized experimental design with a control group to examine the effects of short-term partial sleep deprivation on emotion experience and implicit emotion regulation on otherwise healthy adults. This was in order to create a proxy for how risks posed by long-term partial sleep deprivation may influence the emotional health of otherwise healthy adults. This design allows for an examination into how partial sleep deprivation may differentially influence subjectively experienced positive and negative emotions, and the implicit emotion regulation of these following emotion elicitation. Examining both subjective emotion experiences after sleep deprivation, and following implicit emotion regulation following a strong negative emotion elicitation may provide an indication of how sleep disturbances precipitate emotion dysregulation.

### **Sleep disturbance in general**

Missing from the literature are the processes through which sleep disturbances pose risk to emotional health. More specifically, I was interested in examining whether sleep disturbances in adolescence posed risk for depressive symptoms, not only directly, but also indirectly through catastrophic worry. Catastrophic worry is a potential cognitive vulnerability associated with both sleep disturbances and depression. Inclusion of sleep disturbances in general in a short-term longitudinal study examining depressive symptoms and catastrophic worry allowed for examination of a potential mediating process linking sleep disturbance and depressive symptoms. This study provides an indication of how sleep disturbances may be involved in not only risk for, but also perpetuation of depressive symptoms.

### **Conceptual model**

Disturbed sleep and emotion have both been implicated in the literature as transdiagnostic processes in psychopathology. A body of neurophysiological and clinical research suggests interaction between sleep disturbances and emotion regulation in psychiatric disorder. Furthermore, sleep disturbances have been posited as a mechanistic transdiagnostic process explaining disorder comorbidity and causal interrelationships in psychopathology (Harvey et al., 2011). The rationale behind the candidature of sleep disturbances as a mechanistic transdiagnostic process is based on purported etiological links in various psychopathological disorders. The reciprocal

relationship with emotion dysregulation is estimated to be through shared and interacting underlying neurobiological processes such as genes involved in the generation and regulation of circadian rhythms as well as dopaminergic and serotonergic function (Harvey et al., 2011). Sleep disturbances are also related to biased negative emotional memory encoding and retrieval (Sterpenich et al., 2007), and next day physiological reactivity (Yoo, Gujar, et al., 2007; Yoo, Hu, et al., 2007). Sleep disturbances are associated with a host of psychological disorders, are a diagnostic criterion for many mood disorders, as well as, a risk factor for several (Harvey, 2008a, 2011; Harvey et al., 2011). All of which make sleep disturbances a prime candidate as a mechanistic transdiagnostic processes in the mutual maintenance of emotion dysregulation, linking neurobiology to psychiatric disorders (Harvey et al., 2011; van der Helm & Walker, 2010). See Figure 3 for the biological plausibility model of sleep disturbance as a mechanistic transdiagnostic process.

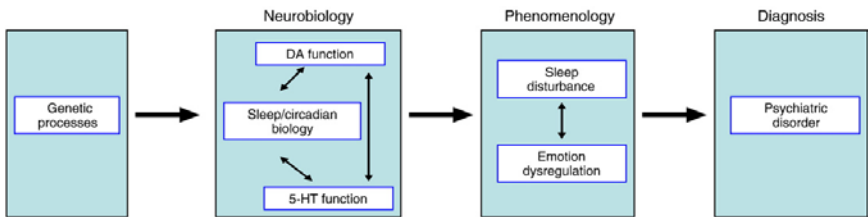


Figure 3. The biological plausibility of sleep disturbance as a transdiagnostic process in the multifactorial cause and/or maintenance of psychiatric disorder (Harvey et al., 2011).

Researchers propose that sleep plays a critical regulatory role for optimal homeostasis of emotional brain function (Armitage, 2007; Benca et al., 1992; Gujar, McDonald, Nishida, & Walker, 2011; Harvey, 2008b; Nofzinger, Buysse, Reynolds, & Kupfer, 1993; Walker, 2009a, 2009b). Therein is a suggestion that disruption of this sleep-emotion regulatory process may have a detrimental impact for clinical symptomatology in emotion related psychopathology (Turek, 2005). The conceptual model above is used in this dissertation as a framework for how sleep disturbances may interact with emotion dysregulation, in the cause and maintenance of psychiatric disorder (Harvey et al., 2011). This model differs from the model included in the theoretical framework. The theoretical model presented earlier relates specifically to processes leading to the chronicity of sleep disturbances, while the conceptual model proposes that sleep disturbances may be a mechanistic transdiagnostic process linking neurobiology

and psychopathology. This dissertation focuses solely on the phenomenological aspects of the relationships pictured in Figure 3. The three studies in the dissertation are descriptive and explore the model with a focus on the direction from sleep disturbances to emotion dysregulation. See Figure 4 for how the three studies in the dissertation are used to examine the phenomenological relationships between sleep disturbances and emotion dysregulation in potentially linking neurobiology and psychiatric disorders.

Summary

A large body of evidence suggests that sleep disturbances may be intricately involved in the experience and regulation of emotion. The studies in this dissertation were intended to examine relationship between sleep disturbances and emotion dysregulation. It is proposed that sleep disturbances are a mechanistic transdiagnostic process acting as a potent stressor, incurring

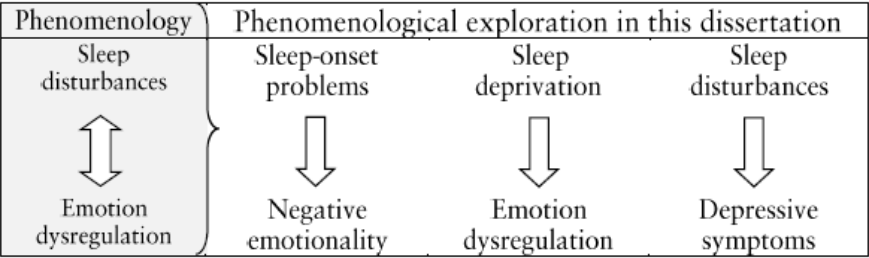


Figure 4. Phenomenological aspects of the biological plausibility model for sleep disturbances as a mechanistic transdiagnostic process influencing emotion dysregulation.

risk for psychopathology through the predisposition, precipitation, and perpetuation of emotional dysregulation. Perhaps the interrelation of sleep disturbances and emotion dysregulation leave people vulnerable to developing psychopathology.

There are several ways that sleep disturbances may influence emotion regulation and pose risk for psychopathology. To start with, sleep disturbances such as sleep-onset problems are perhaps both a diathesis and a stressor interacting with pubertal and behavioral processes in posing long-term risk for negative emotionality. In this way, sleep disturbances may drive and perpetuate emotion dysregulation. Next, sleep disturbances such as sleep deprivation may precipitate initiation of emotion dysregulation in various forms such as negative emotionality, increases in negative affect and emotions and decreases in positive affect and emotions, as well as spurring depressive symptoms. Finally, in addition to direct risks posed by

sleep disturbances for depressive symptoms, an indirect risk may be through catastrophic worry. What this may mean is that sleep disturbances not only precipitate, but also perpetuate emotion dysregulation by contributing to cognitive vulnerability. See Table 1, for an overview of the variables examined in the dissertation.

*Table 1. Disturbed sleep, emotion, and cognitive processes examined in the dissertation.*

	Study 1	Study 2	Study 3
Process	<i>Predisposition</i>	<i>Precipitation</i>	<i>Perpetuation</i>
Sleep disturbance	Sleep-onset	Sleep deprivation	Disturbed sleep in general
Emotion markers	Neuroticism	Dysregulated positive/negative affect/emotions	Depressive symptoms
Cognitive vulnerability			Catastrophic worry

**Dissertation aim**

This aim of this dissertation is to extend what is known about the relationship between sleep disturbances and emotion dysregulation, as well as to expand upon the theoretical framework for how sleep disturbances and emotion dysregulation impart risk for psychopathology. To begin with, sleep-onset problems were explored as predisposition for adolescents to experience negative emotionality over time. Next, sleep deprivation was investigated as a likely precipitator of emotion dysregulation. Finally, sleep disturbances in general were examined as a potential mechanism perpetuating depressive symptoms directly and indirectly through the mediation of catastrophic worry. More specifically, the first study explored the relationships between adolescent sleep-onset problems and midlife neuroticism over a 20-year period. The intention was to establish the direction of effects and the long-term impact of adolescent sleep-onset problems on emotional stability in midlife. The second study was intended to investigate the risk posed by partial sleep deprivation on the subjective emotion experience and implicit emotion regulation of healthy adults. The third study was intended to examine the risk posed by adolescent sleep disturbance for

depressive symptoms one-year later, directly and the indirectly through catastrophic worry, as well as to determine the relationships were gender specific. Together these three studies were intended to investigate the predisposing, precipitating, and perpetuating role of sleep disturbances for emotion dysregulation.

### **Specific aims**

The central aim or silver thread running through this dissertation was to examine the role of sleep disturbances in emotion dysregulation. This was in order to provide a greater understanding for how these two processes may be related in posing risk for psychopathology. Sleep disturbances were examined as predisposing, precipitating, and as perpetuating factors in emotion dysregulation as measured on emotion markers (i.e., negative emotionality, dysregulated emotions, and depressive symptoms).

Life-stressors may initiate a cycle whereby sleep disturbances in the forms of sleep-onset problems and sleep deprivation become predisposing and precipitating factors driving emotion dysregulation. Likely once the initial life-stressor wanes, perpetuating processes gain in magnitude. Over time, as sleep disturbances become chronic, then the importance of the original stressor is likely reduced (see, Spielman et al., 1987). Thereby, sleep-onset problems may predispose, sleep deprivation precipitate, and sleep disturbances perpetuate emotion dysregulation through secondary processes (Spielman et al., 1987). Sleep-onset problems may aggravate neuroticism, predisposing people to react to life-stressors with negative emotions, thoughts, and behaviors. Ensuing sleep deprivation may precipitate the dysregulation of emotion. In turn, heightened neuroticism and dysregulated emotions may further aggravate the regulation of sleep and mood through catastrophic worry. Together these three studies were intended to investigate potential predisposing, precipitating, and perpetuating roles of sleep disturbances for emotion dysregulation.

### **Specific research questions**

This dissertation is comprised of three studies. Study I, examines the developmental relation between adolescent sleep-onset problems and adult negative emotionality over a 20-year period. Study II, investigates risks posed by short-term partial sleep deprivation on the subjective emotional experience and implicit emotion regulation of healthy adults. Study III explores risk posed by adolescent sleep disturbance for depressed mood one-year later, directly and the indirectly through catastrophic worry. The questions addressed in this dissertation are with a focus on the developmental relationships of sleep disturbance and emotion dysregulation in adolescence

and adulthood. Following are the questions answered by the three empirical studies:

1. Does sleep disturbance in the form of adolescent sleep-onset problems predispose people to experience emotion dysregulation into midlife in the form of negative emotionality? (Study 1)
2. Does sleep disturbance, in the form of partial sleep deprivation, precipitate the emotion dysregulation of otherwise healthy adults? (Study 2)
3. Do adolescent sleep disturbances, in addition to posing direct risk, also perpetuate emotion dysregulation in the form of depressive symptoms through catastrophic worry? (Study 3)

# Short description of the studies

## STUDY I

### NEUROTICISM AND SLEEP-ONSET: WHAT IS THE LONG-TERM CONNECTION?

#### Introduction

People with sleep-onset problems often experience symptoms similar to neuroticism, otherwise known as negative emotionality. To what extent the one problem leads to the other is unknown. Traditionally, the supposed direction of effects has been for neuroticism as a risk factor for developing sleep-onset problems, not the other way around (Dorsey & Bootzin, 1997; Freedman & Sattler, 1982; Shealy et al., 1980). The few studies examining this question have been cross-sectional or retrospective, with which determination of the direction of effects cannot be established. A growing knowledge base from neurobiological and clinical research implies that sleep disturbances are a vital link in emotion dysregulation. In this study sleep disturbance in the form of sleep-onset problems is examined as a predisposing factor in emotion dysregulation as measured with levels of neuroticism. Adolescent sleep-onset problems are potentially a stressor posing vulnerability for negative emotionality over the long-term. The two aspects of the relationship between sleep-onset problems and neuroticism examined are the directions of effects and the nature of the relationship. The only way to make any prediction about the direction of effects is over time. Therefore, this study examines longitudinally the relationship between sleep-onset problems and neuroticism from adolescence through midlife.

#### Aim

The aim of the first study was to determine whether adolescent sleep-onset problems posed risk for negative emotionality in midlife. Therefore, the nature of the relationship between sleep-onset problems and neuroticism in adolescence and midlife were examined with a focus on the direction of effects. The overriding question was whether sleep disturbances, in the form of adolescent sleep-onset problems, were a predisposition for adult emotion dysregulation through aggravation of negative emotionality.



## Design

In this prospective study, three time points were used spanning over twenty-years in order to examine whether or not sleep-onset problems posed risk for not only continuance of sleep-onset problems from adolescence to midlife, but also midlife negative emotionality. Data for this study was from a Swedish longitudinal project following participants from birth through midlife. The initial data collection started in the mid-1950s by researchers at the Clinic for the Study of Children's Development and Health at Karolinska Hospital in Stockholm. The study was part of an international collaboration led by the Centre International de l'Enfance in Paris.

## Participants

A sample of 212 people, 90 female, was followed prospectively from birth to midlife by researchers at the Clinic for the Study of Development and Health at the Karolinska Hospital, Stockholm. Participants were born between April 1955 and April 1958. Sample comparisons on many relevant variables such as parents' socioeconomic status, marital status, sibling order, children's gestational age and birth weight show the sample to be representative of Swedish urban communities (see Karlberg et al., 1968; Stattin & Klackenber-Larsson, 1990). Researchers tracked participants' somatic, psychological, and social development through somatic registrations, medical examinations, interviews, inventories and ratings, objective tests, sociometric methods, and projective techniques (Karlberg et al., 1976). In adulthood, 85% of participants at age 25, and 91% of the original sample still alive at 35 years of age, participated in the data collections. Therefore, the final sample size was 185.

## Measures

All measures were self-reported on questionnaires. Sleep-onset problems were measured at ages 15 through 17, and 25 with 1-item, and at age 35 with 2-items developed for the Solna Project (Karlberg et al., 1968). Adolescent neuroticism was measured at age 15 with the High School Personality Questionnaire (HSPQ, Form A) and in midlife at age 35 with the Eysenck Personality Questionnaire (EPQ-I).

## Statistical analyses

All analyses were performed by structural regression modeling using Mplus 5.0 (Muthén & Muthén, 2007). Cross-lagged panel analyses were conducted to examine temporal relations between neuroticism and sleep-onset problems, see Figure 5. This model controls for stability and concurrent

relations. Paths included in the initial model were (a) concurrent relations between sleep-onset problems and neuroticism, (b) stability paths, (c) and cross-lagged paths between earlier and later sleep-onset and neuroticism. Non-significant paths were consecutively removed from the model with guidance from Chi-square difference tests. At each step of the way, fit indices were used to test model fit.

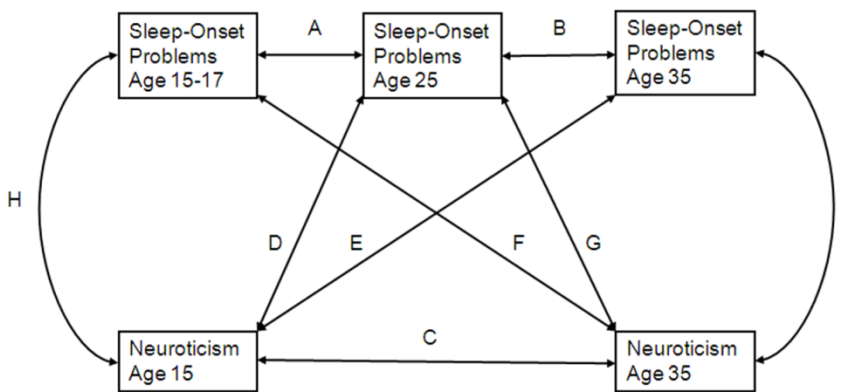


Figure 5. Cross-lagged path model for the bidirectional effects between sleep-onset problems and neuroticism from adolescence through midlife.

## Results

The full model, aside from concurrent and stability paths, included all possible cross-lagged links between these two concepts. As a first modification, the path between adolescent neuroticism and sleep-onset problems in young adulthood (path D in Figure 5,  $p = .26$ ) was removed. This modification was acceptable in terms of model fit as indicated by the non-significant change in chi-square between steps 1 and 2 (see Table 2). In step 3, the path between adolescent neuroticism and sleep-onset problems in midlife was removed (path E in Figure 5,  $p = .23$ ). Again, the chi-square difference test indicated this was an acceptable modification. All paths in this version of the model were statistically significant and therefore no further modifications were made. The final model had acceptable fit,  $\chi^2 (3, N = 185) = 7.67, p = .05$ , RMSEA = .09, CFI = .95. Sleep-onset problems were stable from adolescence to young adulthood ( $\beta = .40, p < .001$ ), and from young adulthood into midlife ( $\beta = .28, p < .001$ ). Neuroticism also showed stability from adolescence through midlife ( $\beta = .41, p < .001$ ). Neuroticism and sleep-onset problems co-occurred in both adolescence ( $\beta = .34, p < .001$ ) and midlife ( $\beta = .25, p < .01$ ). Sleep-onset problems in young adulthood were predictive of heightened neuroticism by midlife ( $\beta = .20, p$

< .01), not the other way around. When controlling for sleep-onset problems in young adulthood in step 4, adolescent sleep-onset problems appeared as an independent predictor of neuroticism in midlife ( $\beta = .19$ ,  $p < .05$ ).

*Table 2. Test of Model Fit and Chi-Square Difference, Removing Least Significant Paths, and Fixing the Supposed Mediating Path in Step 4 to Zero.*

Model	$\chi^2$	<i>df</i>	$\chi^2/df$	RMSEA	CFI	$\Delta\chi^2$	$\Delta df$
Step 1. Full model	4.97*	1	4.86	.14	.96		
Step 2.	6.23*	2	3.12	.10	.96	1.26	1
Step 3.	7.67	3	2.56	.09	.95	1.44	1
Step 4. Media- tion model	14.31**	4	3.58	.11	.89	6.64*	1

*Note:*  $\chi^2$  = Chi-square, *df* = Degrees of freedom; CFI = Comparative Fit Index; RMSEA = Root Mean Square Error of Approximation. \*  $p < .05$ . \*\*  $p < .01$ .

## Conclusions

The aim of this study was to examine the direction of effect in the relationship between sleep-onset problems and neuroticism. This was in order to determine whether sleep disturbance in the form of adolescent sleep-onset problems pose risk for negative emotionality in into midlife. Adolescent neuroticism was not predictive of sleep-onset problems in young adulthood or midlife. Instead, sleep-onset problems in adolescence and young adulthood were predictive of neuroticism in midlife. Adolescent sleep-onset problems appear to be a stressor posing vulnerability or predisposition for negative emotionality over the long-term. Life-stressors may initiate a cycle whereby sleep-onset problems become a motor fuelling emotion dysregulation. Likely once the initial life-stressor wanes, perpetuation processes gain in magnitude. Overtime, as sleep disturbances become chronic, then the importance of the original stressor is likely reduced (see, Spielman et al., 1987). Sleep-onset problems may become a predisposing factor for, a precipitator of, and perhaps a maintaining factor in emotion dysregulation

through secondary processes (Spielman et al., 1987). Sleep-onset problems may aggravate neuroticism, predisposing people to react to life-stressors with negative emotions, thoughts, and behaviors. In turn, heightened neuroticism may aggravate sleep and mood through catastrophic worry. This study provides longitudinal support for adolescent sleep-onset problems as a potent risk factor for heightened neuroticism, or negative emotionality through midlife. The implication is that sleep-onset problems in adolescence pose risk for neuroticism in midlife both directly and indirectly through the continuance of sleep-onset problems into young adulthood. This study revealed a long-term risk posed by adolescent sleep disturbance, in the form of sleep-onset problems, for long-term emotional instability. Questions remaining were how sleep disturbances may precipitate and maintain emotion dysregulation.

## STUDY II

### EFFECTS OF PARTIAL SLEEP DEPRIVATION ON SUBJECTIVE EMOTION EXPERIENCE AND IMPLICIT EMOTION REGULATION

#### Introduction

It is a common observation that people with sleep deprivation may be irritable or display other signs of emotionality. Sleep disturbances have long been held to be related to negative emotion. The nature of the relationship has been less well understood. There have been mixed results in the literature for how negative emotions (e.g., mood, affect) are influenced by sleep deprivation with some showing significant increases in total mood disturbance (Dinges et al., 1997; El-Sheikh, Buckhalt, Cummings, & Keller, 2007; Franzen et al., 2008; Novati et al., 2008) and negative affect (Zohar et al., 2005), others have not (Talbot et al., 2010). Negative emotion is no longer viewed as the only emotion facet affected by sleep deprivation.

Positive emotion, in the form of positive affect, is characteristic of sleep deprivation in adolescence (Dagys et al., 2012; Talbot et al., 2010) and adulthood (Franzen et al., 2008; Talbot et al., 2010). Early indications from a recent study suggest that positive emotion may be more susceptible to the effects of a full night's sleep deprivation than negative emotion (see, Minkel, 2010). Positive emotion is important in the regulation of negative emotions (for review see, Dillon & Pizzagalli, 2009) and is therefore a critical research domain.

#### Aim

Regardless of the reason for its occurrence, sleep deprivation may pose a risk for reduced emotion regulation for people who generally would not be considered at risk. Therefore, a greater understanding was sought for how partial sleep deprivation may influence the positive and negative emotions of otherwise healthy adults. What is known about the relationships between partial sleep deprivation and the subjective experience of emotion and on implicit emotion regulation following strong emotional events is still limited. The overriding question this study intended to answer was how sleep disturbance, in the form of sleep deprivation, may act as a potent stressor, precipitating emotion dysregulation.

The aim of this study was to examine how 3-nights partial sleep deprivation would influence the subjective experience of emotions, and implicit emotion regulation of healthy people who generally sleep well following a strong negative emotion elicitation. Previous findings have been mixed. In

this study, it was expected that sleep disturbance in the form of sleep deprivation, would precipitate emotion dysregulation in the form of reductions in positive emotions and increases in negative emotions as a response to the sleep condition and following a strong negative emotion elicitation. The first hypothesis was that people who were partially sleep deprived would experience more negative emotions and less positive emotions compared to controls following the sleep condition. The second hypothesis was that partial sleep deprivation would hamper the implicit emotion regulation of otherwise healthy people compared to rested controls when confronted with strong negative emotional stimuli. Following emotion elicitation, partially sleep deprived people were expected to report significantly greater increases in negative emotion, and decreases in positive emotion compared to those who were rested.

## Design

This study utilized a randomized design with experimentally manipulated sleep and emotions. This was in order to examine the differential influence of partial sleep deprivation on both subjective emotion experience, and on implicit emotion regulation following exposure to strong negative emotional stimuli. Participants either were randomly assigned to a partial sleep deprivation or rested condition. A 4-night baseline was established with all participants encouraged to sleep at least 8-hours. The following 3-nights, one group spent 5-hours total time in bed per night, while the other group was encouraged to sleep a minimum of 8-hours per night. Sleep condition compliance was gauged with a 1-week sleep diary and by measures of fatigue (see measures below). The effects of sleep deprivation on emotion experience and implicit emotion regulation were measured with self-report questionnaires the day following the sleep condition week with first a pre-test, and then a posttest following a strong negative emotion elicitation procedure. Emotion was elicited with melancholic classical music (MCI; Eich, Ng, Macaulay, Percy, & Grebneva, 2007), negative guided imagery, and pictures with high mean ratings for arousal and negative valence (IAPS; Bradley & Lang, 2007). See Table 3 for an overview of the experimental study design.

## Participants

Participants were eighty-one adults, 44 females,  $M_{age} = 25$ , age range: 18-50 years, recruited on campus through the newspaper, posters, and classroom presentations. The sample consisted of students, faculty, and members of the community. Selection was based on being free from sleep disorder.

ders, sleep disrupting medication, drug or alcohol abuse, or abstinence problems within the last month. Other inclusion criteria were being free from anxiety, depression, or sleep disturbances during the last month or having irregular sleep and wake times.

*Table 3. Randomized control group pretest posttest study design.*

Phase 1	Phase 2	Phase 3
Recruitment	Sleep condition week	Experiment day
Screening	Sleep diary 1-week	Pretest
Baseline measurement	Baseline sleep 4-nights	Emotion elicitation
Randomization	Sleep condition 3-nights	Posttest
	<i>Sleep <math>\leq</math> 5hrs OR sleep <math>\geq</math> 8hrs</i>	

### Measures

Subjective emotion experience was measured emotion markers gauged with levels of positive and negative affect, basic and specific emotions reported on the Positive Affect and Negative Affect Scale-Expanded form (PANAS-X; Watson & Clark, 1999). Implicit emotion regulation was measured by changes in emotion markers, gauged by levels of self-reported emotion from pretest prior to emotion elicitation, to posttest immediately after. Compliance and manipulation checks were administered at posttest to determine whether there were significant differences between groups in compliance with the sleep and emotion elicitation procedures. Sleep diaries were used as a sleep condition compliance-check. The PANAS-X fatigue subscale which tends to load significantly on positive and negative affect and is a marker of high negative and low positive affect (Watson & Clark, 1999), was also used as a sleep condition manipulation check. Levels of arousal, pleasure (i.e., valence), and emotion dominance were assessed with the Self-Assessment Manikin (SAM; Russell, Weiss, & Mendelsohn, 1989) and the Affect Grid (Russell et al., 1989) as emotion manipulation checks. Measures of social desirability, willingness, and realism were also used.

### Statistical analyses

One-way between-group multivariate analyses of variance were conducted to compare differences, dependent on whether participants were sleep deprived or rested, on emotion markers following the sleep condition and subsequently after emotion elicitation. Separate analyses were conducted for each of the emotion. In each of these analyses, there were three dependent variables. These were levels of emotion as measured by emotion markers at the three time points: baseline (Time 1), pretest following the sleep

condition (Time 2), and posttest after emotion elicitation (Time 3). In order to answer our research questions, we used sleep condition (sleep deprived, rested) as our independent variable. Scores and changes on scores on emotions markers following sleep deprivation and after emotion elicitation were our dependent variable, gauging subjective emotion experience and implicit emotion regulation. A Bonferroni correction for multiple comparisons (i.e., baseline, pretest, posttest) was applied at the .017 level in order to prevent alpha inflation.

## Results

The only significant within and between-group differences on the negative emotion markers were evidenced on a few discrete negative emotions at pretest prior to emotion elicitation. People who were sleep deprived reported significantly more irritability, loathing, hostility, and shakiness than people who were rested. After Bonferroni correction at  $p < .017$ , only irritable and shaky remained significant. See Table 4 for significant mean differences and Table 5 for one-way between-groups multivariate analyses of variance and contrasts of significant within-group time effects, and between-group contrasts from pretest to posttest. At pretest following the sleep condition prior to emotion elicitation there were few differences in negative emotion and many on positive emotion. Following sleep deprivation, compared to controls, people reported significantly less positive emotion on markers at pretest prior to emotion elicitation. All of the between-group differences on the positive emotion markers remained significant following Bonferroni correction at  $p < .017$ , except the basic positive emotion self-assurance.

After emotion elicitation, both groups reported significantly higher negative affect, emotion, arousal, displeasure, emotion dominance, and reduced positive affect and emotions. The sleep-deprived group reported feeling significantly more sluggish, tired, sleepy, and drowsy. The only significant between-group difference in emotion at posttest was for the discrete positive emotion fearless. Following emotion elicitation, sleep deprived people reported significantly more fearlessness than people who were rested. This one posttest difference no longer remained significant after applying a Bonferroni correction of  $p < .017$ .



*Table 4. Significant mean differences between the sleep deprived and rested groups on markers of positive emotion.*

Variable	Sleep deprived		Rested	
	Pre	Post	Pre	Post
Positive affect	17.35	14.16	20.73	13.91
Basic positive emotion scales				
Joviality	19.06	12.14	24.37	12.35
Self-assurance	13.33	11.33	14.95	10.72
Attentive	10.31	11.58	13.30	12.49
Discrete positive emotions				
Active	2.41	2.32	3.23	2.20
Alert	2.03	2.54	3.05	2.75
Attentive	3.00	3.51	3.77	3.84
Bold	2.51	2.49	2.93	2.20
Concentrating	2.69	2.92	3.42	3.33
Confident	3.03	2.73	3.45	2.84
Delighted	2.30	1.11	2.95	1.09
Energetic	1.95	1.97	3.00	1.84
Enthusiastic	2.22	1.51	3.11	1.64
Excited	1.84	1.68	2.35	1.53
Fearless	3.46	2.86	3.43	2.34
Inspired	2.41	1.59	3.27	1.61
Interested	3.08	2.30	3.70	2.19
Joyful	3.00	1.42	3.59	1.50
Lively	1.76	1.54	2.52	1.50
Proud	2.46	1.57	3.18	1.84
Strong	2.84	2.46	3.32	2.68

*Note: Pre = pretest; Posttest = Post.*

Table 5. One-way between-groups multivariate analyses of variance for significant within-group time effects, and between-group contrasts on markers of positive emotion from pretest to posttest.

Variable	Wilks	F	df1/ df2	$\eta p^2$	Contrasts	
	$\lambda$				(F)	$\eta p^2$
Positive affect	.72	9.32 <sup>†</sup>	3,73	.28	12.27 <sup>† a</sup>	.14
Basic positive emotion scales						
Joviality	.71	10.11 <sup>†</sup>	3,75	.29	12.88 <sup>† a</sup>	.14
Self-assurance	.84	4.61 <sup>*</sup>	3,75	.16	5.00 <sup>* a</sup>	.06
Attentive	.68	11.61 <sup>†</sup>	3,75	.26	26.53 <sup>† a</sup>	.26
Discrete positive emotions						
Active	.78	7.45 <sup>†</sup>	3,77	.23	13.64 <sup>† a</sup>	.15
Alert	.75	8.63 <sup>†</sup>	3,77	.25	21.99 <sup>† a</sup>	.22
Attentive	.80	6.56 <sup>†</sup>	3,77	.20	12.00 <sup>† a</sup>	.19
Bold	.87	3.75 <sup>†</sup>	3,77	.13		
Concentrating	.86	3.98 <sup>†</sup>	3,75	.14	11.62 <sup>† a</sup>	.13
Confident					4.28 <sup>* a</sup>	.05
Delighted	.90	2.98 <sup>*</sup>	3,77	.10	6.62 <sup>† a</sup>	.08
Energetic	.66	3.37 <sup>†</sup>	3,77	.34	25.24 <sup>† a</sup>	.24
Enthusiastic	.82	5.74 <sup>†</sup>	3,73	.18	15.10 <sup>† a</sup>	.16
Excited	.88	3.33 <sup>*</sup>	3,76	.12	4.56 <sup>* a</sup>	.06
Fearless					5.57 <sup>* b</sup>	.07
Inspired	.84	4.83 <sup>†</sup>	3,77	.16	12.23 <sup>† a</sup>	.13
Interested	.85	4.58 <sup>†</sup>	3,76	.15	9.54 <sup>† a</sup>	.11
Joyful	.87	3.89 <sup>†</sup>	3,77	.13	8.09 <sup>† a</sup>	.09
Lively	.79	6.94 <sup>†</sup>	3,77	.21	8.98 <sup>† a</sup>	.10
Proud					7.42 <sup>† a</sup>	.09
Strong					5.05 <sup>* a</sup>	.06

Note: Wilks Lambda  $\lambda$  is an index of the proportion of variance in the dependent variable accounted for by a predictor variable and estimated by an F statistic. The Wilks Lambda and the significant F value test the overall changes on the different dependent variables. Only F values of  $p < .05$  are listed. \*  $p < .05$ . †  $p < .017$  significant at the Bonferroni corrected alpha level.  $\eta p^2$  = partial eta2. (F) = Test of Between-Group contrasts. Significant contrasts from baseline to pretest = <sup>a</sup>, pretest to posttest = <sup>b</sup>.

## Conclusions

These findings extend the literature by showing that partial sleep deprivation poses risk to subjective emotion experience of otherwise healthy adults by reductions in positive affect and emotions. These findings suggest that partial sleep deprivation may be a potent stressor in emotion dysregulation through reductions in positive emotions. In addition, people who were sleep deprived exhibited a negativity bias on discrete emotion markers, scoring positive (e.g., delighted, joyful) and neutral items (e.g., concentrating, attentive) lower and negative items (e.g., irritable, hostile) higher than controls. Following confrontation with strong negative emotional stimuli, the implicit emotion regulation of healthy people appears to work equally as well regardless of sleep condition. Sleep disturbance in the form of sleep deprivation poses risk to emotion regulation of otherwise healthy adults through reductions in positive emotions and perhaps through a negativity bias. This study provided insight into how effects of sleep disturbance in the form of sleep deprivation impacts dysregulation of positive and negative affect and emotions. Still largely unknown were the maintaining processes involved in disturbed sleep and emotion, and whether there were gender differences in these relationships.

## STUDY III

### SLEEP DISTURBANCE AND DEPRESSIVE SYMPTOMS IN ADOLESCENCE: THE ROLE OF CATASTROPHIC WORRY

#### Introduction

Adolescents experiencing sleep disturbances are at greater risk for developing depression. Previous research implicates sleep disturbances as a risk factor for depression onset and recurrence. Perhaps the risk posed by sleep disturbances for depression is indirect, as well as direct. Increased reactivity due to persistent sleep disturbances may activate cognitive processes such as catastrophic worry, contributing to depressive symptoms through elicitation of physiological arousal and emotional distress contributing to both problems.

#### Aim

One aim was to investigate whether catastrophic worry mediated the relationship between adolescent sleep disturbance and depressive symptoms. Based on previous research, sleep disturbances were hypothesized to pose risk for developing depression and that catastrophic worry would mediate this relationship. According to prior research, girls have an elevated risk of experiencing sleep disturbances, catastrophic worry, and depression (Garnefski, Teerds, Kraaij, Legerstee, & van den Kommer, 2004). Therefore, another aim was to determine whether these patterns of relations were gender specific. The final hypothesis was that the relations would be stronger for girls than for boys. The central question of this study was how sleep disturbances may perpetuate emotion dysregulation in the form of depressive symptoms.

#### Design

In this study, the relationships between adolescent sleep disturbances, depressed mood, and catastrophic worry were examined longitudinally. This was in order to test whether sleep disturbances posed an indirect, as well as an indirect risk for depressive symptoms through the mediation of catastrophic worry. The regional ethics board approved this study. Participants gave informed consent, were told that participation was strictly voluntary, that they could withdraw at any time. Örebro University conducted this study with local school health services.

## Participants

Participants were from a sample of 1,760 adolescents, ages 16 to 18, in a small town in Sweden, 48% girls. Students participated in a three-year longitudinal project, from fall terms 2006 thru 2008. Data were from annual health surveys including reports of sleep disturbance, catastrophic worry, and depressive symptoms. Students completed physical and psychological health questionnaires provided by a school nurse the first year and by teachers the following years. Each year some students graduated and incoming students were recruited: 2006,  $n = 960$ ; 2007,  $n = 840$ ; 2008,  $n = 760$ . Students absent from school on the day of data collection, or missing more than 80% data, were not included in the study. Data from Time 1 was used for the correlational analyses. Ideally, data from all three waves would have been utilized for the longitudinal analyses. Unfortunately, there was only complete data for 148 students who participated in all three waves. Therefore due to statistical power considerations, two waves at a time were used in mediation analyses, Time 1 and Time 2 ( $n = 394$ ) and Time 2 and Time 3 ( $n = 374$ ).

## Measures

*Sleep disturbance* was measured with a reversed continuous sleep-quality item: "Do you sleep well?" responses ranged on a 5-point Likert from 1 (strongly disagree) to 5 (strongly agree). The average stability for the continuous measure over three years was .74. One-item sleep disturbance measures (Wong & Brower, 2012), including reversed sleep quality items (Lundh, Bjärehed, & Wångby-Lundh, 2012), are commonly used in epidemiological research with adolescents and as a screening tool in primary care (MacGregor, Funderburk, Pigeon, & Maisto, 2012). *Depressive symptoms* were measured with the Center for Epidemiology Studies-Depression Child scale (CES-DC, Olsson & Von Knorring, 1997).

*Catastrophic worry* was measured with the catastrophizing subscale from the Safety Behavior and Catastrophizing Scale (SBCS, MacDonald, Linton, & Jansson-Fröjmark, 2008).

## Statistical analyses

A mediation model was tested using a SPSS macro developed to test mediation models (Preacher & Hayes, 2008) in order to determine whether in addition to predicting depressive symptoms one year later, sleep disturbances would also predict depressive symptoms indirectly through catastrophic worry. Next, gender was controlled for in the original mediation model in order to determine whether the relation between sleep disturbances, catastrophic worry, and depressive symptoms were gender depend-



Results

Our first hypothesis was that adolescent sleep disturbance poses risk for the development of depression, and that catastrophic worry mediates this relationship. Mediation steps along with bootstrap analyses are summarized in Tables 6 and 7. In step 1, earlier sleep disturbance was related significantly to catastrophic worry at Times 1 and 2 ( $a_1$  &  $a_2$ ), as well as in step 2 with later depressive symptoms at Times 2 and 3 ( $c_1$  &  $c_2$ ). Adolescents experiencing sleep disturbance tended to report concurrent catastrophic worry and depressive symptoms one year later. In step 3, when earlier sleep disturbance and catastrophic worry were included as predictors of later depressive symptoms, catastrophic worry remained significant at Times 1 and 2 ( $b_1$  &  $b_2$ ). Controlling for earlier catastrophic worry in step 4, the direct path between earlier sleep disturbances and depressive symptoms from Time 1 to Time 2 ( $c'_1$ ) was significantly reduced indicating partial mediation, and nonsignificant from Time 2 to Time 3 ( $c'_2$ ), indicating perfect mediation.

Table 6. Bootstrap estimates of the mediation model on the relation between earlier sleep disturbance (Time 1) to later depressed mood (Time 2), and the role of earlier catastrophizing (Time 1).

Step	Path	$\beta$	Estimate (B)	SE	95% CI	
1	$c$ = total effect (X on Y)	.21***	.18	.04	.10	.26
2	$a$ = (X on M)	.18***	.16	.04	.07	.24
3	$b$ = (M on Y)	.32***	.30	.04	.21	.38
4	$c'$ = direct effect (X on Y)	.16**	.14	.04	.06	.21
a*b = indirect effect (X on Y through M)		Effect size ( $r^2$ )	Indirect/ total ratio	Sobel's $z$	SE	95% CI
		.05	.07	.05/.21 = .23	3.13**	.02 .08

Note. X = independent variable, Y = dependent variable, M = proposed mediator. N = 394. Estimates are standardized. Percentile 95% bias corrected bootstrap CI, 5,000 trials, defined using the values that mark the upper and lower 2.5% of the bootstrap distribution. There is evidence for partial mediation: significant Sobel's test, zero is not contained in the confidence interval, and the indirect/total ratio is < .80. \*\*  $p = .01$ , \*\*\* $p = .00$ .

Since perfect mediation is extremely rare in psychological science (Baron and Kenny 1986), bootstrap analyses were used to examine the total, direct, and indirect effects to determine mediation (Hayes 2009;

Rucker et al. 2011). Bootstrap results indicated partial mediation from Time 1 to Time 2 ( $a_1 \times b_1$ ) and from Time 2 to Time 3 ( $a_2 \times b_2$ ). Sobel's tests were significant from Time 1 to Time 2 ( $z_1$ ) and from Time 2 to Time 3 ( $z_2$ ). The exclusion of zero in the confidence interval coupled with the significant Sobel's test indicates mediation. The indirect/total ratios were below .80, which suggests that the mediation of catastrophic worry on the relationship between earlier sleep disturbance and later depressive symptoms is partial. The results of the bootstrap analyses indicated that earlier catastrophic worry partially mediated the relationship between earlier sleep disturbance and later depressive symptoms.

Table 7. Bootstrap estimates of the mediation model on the relation between earlier sleep disturbance (Time 2) to later depressive symptoms (Time 3), and the role of earlier catastrophizing (Time 2).

Step	Path	$\beta$	Estimate ( <i>B</i> )	<i>SE</i>	95% CI	
1	<i>c</i> = total effect (X on Y)	.15**	.13	.04	.04	.21
2	<i>a</i> = (X on M)	.19***	.18	.05	.08	.27
3	<i>b</i> = (M on Y)	.39***	.34	.04	.26	.43
4	<i>c'</i> = direct effect (X on Y)	.08	.06	.04	.01	.14
a*b = indirect effect (X on Y through M)		Effect size ( <i>r</i> <sup>2</sup> <i>r</i> )	Indirect/ total ratio	Sobel's <i>z</i>	<i>SE</i>	95% CI
		.06	.06	.06/.15 = .40	3.41***	.02 .03 .11

Note. X = independent variable, Y = dependent variable, M = proposed mediator. N = 394. Estimates are standardized. Percentile 95% bias corrected bootstrap CI, 5,000 trials, defined using the values that mark the upper and lower 2.5% of the bootstrap distribution. There is evidence for partial mediation: significant Sobel's test, zero is not contained in the confidence interval, and the indirect/total ratio is < .80. \*\*  $p = .01$ , \*\*\* $p = .00$ .

The second hypothesis was that the relationships between adolescent sleep disturbances, catastrophic worry, and depressive symptoms would be stronger for girls than for boys. Therefore, we reran the original mediation analyses with gender as a covariate to determine whether the relationships between earlier sleep disturbance and catastrophic worry, and later depressive symptoms were gender dependent. In step 1, earlier sleep disturbance was related significantly to catastrophic worry when controlling for gender at Times 1 and 2 ( $a_1$  &  $a_2$ ), as well as in step 2 with later depressive symp-



toms at Times 2 and 3 ( $c_1 \neq c_2$ ). When earlier sleep disturbance and catastrophic worry were included as predictors of depressive symptoms while controlling for gender, catastrophic worry remained significant from Time 1 to Time 2 ( $b_1$ ) and from Time 2 to Time 3 ( $b_2$ ). In step 4, there was a significant reduction in the direct effect from Time 1 to Time 2 ( $c'_1$ ) indicating partial mediation, and an insignificant effect from Time 2 to Time 3 ( $c'_2$ ) indicating full mediation. The bootstrap analyses however, indicate partial mediation from both Time 1 to Time 2 ( $a \times b_1$ ), and from Time 2 to Time 3 ( $a \times b_2$ ).

## Conclusions

This study provides support for sleep disturbances as a perpetuating process in emotion dysregulation. Adolescent sleep disturbances appear to pose a two-fold risk for depressive symptoms one-year later, directly and indirectly through fuelling of catastrophic worry. Girls report more sleep disturbances, catastrophic worry, and depressive symptoms. Despite this, indications are that catastrophic worry is a non-gender specific cognitive vulnerability linking adolescent sleep disturbances and depressive symptoms over time.

Sleep disturbances and catastrophic worry may provide school nurses, psychologists, teachers, and parents with non-gender specific early indicators of risk for depression. Clinical evidence finds that sleep disturbances add to depression severity, poor response to depression treatment, and is a common residual symptom following depression treatment, as well as a predictor of relapse and recurrence (Manber and Chambers 2009). Research also suggests that sleep disturbances are a potentially modifiable risk factor for both the prevention of depression, and for the maintenance of depression remission (Franzen and Buysse 2008). Results from the current study provide two markers of risk for depression in adolescence, as well as two potentially modifiable targets for prevention and intervention, sleep disturbances and catastrophic worry. Perhaps starting early by targeting adolescent sleep disturbances and ensuing catastrophic worry through early prevention and intervention efforts may reduce the short and long-term economic and burdens placed on individuals, families and society by depression.

# General discussion

## Answers to the overall research questions

The overarching aim of this dissertation was to investigate the role of sleep disturbances in the predisposition, precipitation, and perpetuation of emotion dysregulation. Different levels of sleep disturbances and markers of emotion dysregulation were used to answer these questions. The first research question was whether sleep disturbance in the form of adolescent sleep-onset problems was a predisposing factor for dysregulation of neuroticism in midlife. Underlying questions in study 1 were about the long-term relation between adolescent sleep-onset problems and midlife negative emotionality, as well as the direction of effects. Theoretically, neuroticism has been thought to be important for the development of sleep-onset problems. To our knowledge, this was the first study to examine the longitudinal connection between sleep-onset problems and negative emotionality. The results of this study demonstrated that the relationship goes in the opposite direction. Adolescent neuroticism failed to predict sleep-onset problems in young adulthood or in midlife. Instead, sleep-onset problems in adolescence and young adulthood, predicted neuroticism in midlife. The implications are that adolescent sleep-onset problems confer risk for midlife neuroticism, not vice versa. The risks posed by adolescent sleep-onset problems for midlife neuroticism appear to be direct, as well as indirect through continued sleep-onset problems in young adulthood. This study provided an indication that adolescent sleep disturbances in the form of sleep-onset problems are a predisposing risk for emotion dysregulation stretching as far as midlife.

The second research question was how sleep disturbance, in the form of partial sleep deprivation, may act as a potent stressor precipitating the emotion dysregulation of healthy adults, and hampering implicit emotion regulation. Underlying questions used to answer the thesis question were how 3-nights partial sleep deprivation would influence the subjective experience of emotion and implicit emotion regulation of otherwise healthy adults. The findings suggest that partial sleep deprivation is a potent stressor involved in emotion dysregulation through reductions in positive affect and emotion. Sleep disturbances and sleep incompatible behaviors underlying sleep deprivation and ensuing reductions in positive affect and emotions may provide viable targets of treatment when people seek treatment for disorders such as depression.

Following sleep deprivation, people were expected to report more negative affect and emotions, and less positive affect and emotion than those who

were rested. The findings were mixed and contrary to expectations. As expected, sleep deprived people reported less positive affect and emotion, than rested people. Unexpectedly, people reported similar levels of negative affect and emotion. The exceptions were on discrete emotions that people who were partially sleep deprived reported significantly higher levels of irritability, loathing, hostility, and shakiness, compared to controls. After Bonferroni correction, only irritability and shakiness remained significantly higher. This finding suggests that it may be important to include discrete levels of emotions as markers of subjective emotion experience in order to observe subtle nuances in emotion.

It was also expected that people who were sleep deprived would have a more difficult time implicitly regulating positive and negative emotion in response to strong negative emotion elicitation procedures. Contrary to what was expected, people responded similarly to strong emotion elicitation regardless of whether or not they were sleep deprived. All participants reported heightened negative and lowered positive emotion on markers. There was one exception. People who were partially sleep deprived reported more fearlessness than controls following the strong negative mood induction. Fearless is a discrete positive emotion included in the basic emotion self-assurance. The results suggest that in response to strong negative emotional stimuli, people will respond similarly, regardless of how they sleep.

The effects of partial sleep deprivation seem to precipitate emotion dysregulation through a few specific negative emotions: irritability, loathing, hostility, and shakiness, as well as through reductions in positive affect and emotions. Whether sleep deprived, or not, people appear to respond similarly following exposure to strong negative emotional stimuli. The primary route through which partial sleep deprivation appears to influence emotion dysregulation is through positive emotions, and a few discrete negative emotions. Overall, it appears evident that sleep disturbance, in the form of partial sleep deprivation, precipitates emotion dysregulation.

The final research question addressed how adolescent sleep disturbances in general may perpetuate emotion dysregulation in the form of depressive symptoms. To our knowledge study 3 was the first study to examine longitudinally, whether catastrophic worry, a potential cognitive vulnerability, mediates the relationship between adolescent sleep disturbances and depressive symptoms. The results showed that sleep disturbances pose not only direct risk for depressive symptoms, but also indirectly through catastrophic worry. Another goal of study 3 was to examine whether any mediation by catastrophic worry was gender dependent. Girls have a higher propensity to report more sleep disturbance, depression, and catastrophic

worry (Garnefski et al., 2004). Therefore, another intention was to examine whether any potential indirect risk through catastrophic worry was gender dependent. The results showed that adolescent sleep disturbances in addition to posing direct risk for depressive symptoms, also posed indirect risk through catastrophic worry. These results were non-gender specific. This indicates that catastrophic worry is a non-gender specific cognitive vulnerability factor for the development of depressive symptoms. This study lends credence for sleep disturbances as not only a precipitating factor, but also a perpetuating factor for the dysregulation of emotions. Overall, it appears that sleep disturbances in adolescence and adulthood are important in emotion dysregulation. Several types of sleep disturbances seem to be closely linked to various levels of emotion dysregulation concurrently and over time. To summarize, the three studies implicate sleep disturbances as important predisposing, precipitating, and perpetuating factors in emotion dysregulation.

### **The findings in relation to previous research**

Findings in the studies presented in this dissertation provide support, challenge, and extend what is known about the relationship between sleep disturbances and emotion dysregulation. Insomnia models suggest that hierarchical processes are involved in the development of sleep disturbances. In these models, biological and genetic vulnerabilities predispose, stress-full life-events precipitate, and physiological, cognitive and behavioral factors perpetuate sleep problems (Borkovec, 1979; Borkovec, 1982; Espie, 2002; Harvey, 2002; Morin, Stone, Trinkle, Mercer, & Remsberg, 1993; Perlis, Giles, Mendelson, Bootzin, & Wyatt, 1997; Spielman, 1986; Spielman, Caruso, & Glovinsky, 1987). Instead of looking at sleep disturbances solely as an outcome of life-stressors, the studies in this dissertation were designed to examine sleep disturbances as a catalyst for the predisposition, precipitation, and perpetuation of emotion dysregulation.

The field of neurobiology has laid down a framework for sleep-dependent processes involved in emotion regulation (Walker & van der Helm, 2009). Sleep disturbances may predispose some people to negative memory encoding and retrieval bias (Sterpenich et al., 2007), consolidation of negative memories during REM sleep, and strengthening of hyperlimbic reactivity in response negative emotional events the next day (Yoo, Gujar, et al., 2007; Yoo, Hu, et al., 2007). The conceptual model adopted in this dissertation proposes that sleep disturbance and emotion have a mutually maintaining role linking neurobiology and psychopathology. Further, sleep disturbances are suggested as a mechanistic transdiagnostic process in psychopathology (Harvey et al., 2011). The studies in this dissertation were

designed to examine one aspect of the conceptual model in which sleep disturbance theoretically links bio-physiological processes and psychopathology through interaction with emotion dysregulation. This one part of the model examined is how sleep disturbances may interact with and impart risk for emotion dysregulation. It appears from the findings in the studies included in this dissertation that sleep disturbances pose long-term risk for emotional instability from adolescence through midlife, initiates emotion dysregulation, and maintains emotional instability by spurring catastrophic worry. Perhaps sleep disturbances are a mechanistic transdiagnostic process in disorder comorbidity through fueling and driving the relationship with emotion dysregulation. Perhaps this is one way in which sleep disturbances are involved in risk for psychopathology.

Study 1 is the first study to examine the relationship between sleep-onset problems and neuroticism from adolescence through midlife. Previously in the literature, sleep disturbances were seen as purely a consequence of, or secondary to psychological disorders. The results from Study 1 contradict previous ideas about adolescent neuroticism as a risk factor for developing sleep-onset problems over time. Previous research into this question has been based on cross-sectional and retrospective reports (Dorsey & Bootzin, 1997; Freedman & Sattler, 1982; Shealy et al., 1980), neither of which methods allow for prediction of direction of effects. Neuroticism likely predisposes people to react to life-stressors with negative thoughts, emotions, and behaviors incompatible with falling asleep. Any risk posed for sleep-onset problems appear insufficient for the development of sleep-onset problems. Any risk posed is likely indirect through the negative thoughts, emotions, and behaviors that are associated with neuroticism. Instead, adolescent sleep onset-problems pose risk for neuroticism well into adulthood. Sleep disturbances appear to catalyze and aggravate predisposition for emotion dysregulation. Study 1 lends credence to the theory of sleep disturbance as a mechanistic transdiagnostic process involved in the predisposition for emotion dysregulation. Sleep disturbances are not just a benign symptom or comorbid problem, but instead pose a real risk for emotion dysregulation from adolescence through midlife.

The second study looked at sleep disturbance in the form of partial sleep deprivation as a precipitator of emotion dysregulation. Specifically, the effects of partial sleep deprivation on the subjective experience of emotion as measured by positive and negative affect, basic and specific emotions. Another facet of Study 2 was the examination of implicit regulation of these emotion markers following a strong negative emotion elicitation. Results from previous research on the effects of sleep deprivation on emotion, affect, and mood, have been mixed. In previous studies, increases in

negative mood in response to total sleep deprivation as measured by the Profile of Mood States (POMS; Dinges et al., 1997; Zohar et al., 2005), and visual analog scales (Franzen et al., 2008) has been reported. Results from Study 2 and others, run contrary to these findings.

Researchers using the PANAS to test the effects of total sleep deprivation on positive and negative affect reported no differences on negative affect and decreases in positive affect in the context of daily events (Franzen et al., 2008). Another sleep deprivation study had participants sleep no more than 6.5-hours the first night and only 2-hours the second night. Positive and negative affect were examined, as well as discrete emotions using the PANAS-Child scale. Sleep deprived participants reported no significant differences in negative affect compared to controls, only on two negative discrete emotions, nervous and miserable. Researchers also found that positive affect and all of the discrete emotions except calmness, decreased.

Study 2 supports these findings. People reported similar levels of negative emotion, with the exception of several discrete emotions not examined previously in relation to partial sleep deprivation. People who were partially sleep deprived reported feeling more irritability, loathing, hostility, and shakiness than controls. In line with previous research, participants reported significantly less positive affect, basic (i.e., joviality, self-assurance, attentiveness) and specific (i.e., active, alert, attentive, concentrating, confident, delighted, energetic, enthusiastic, excited, inspired, interested, joyful, lively, proud, strong) positive emotions. The wider range of discrete emotions examined in this study extends the literature.

Another aim of Study 2 was to investigate the effects of partial sleep deprivation on implicit emotion regulation in response to partial sleep deprivation. A previous study examined the impact of total sleep deprivation on subjective stress and mood of 53 healthy adults in response to low-stress and high-stress cognitive testing conditions. Mood was measured with the POMS Depression-Dejection, Tension-Anxiety, and Anger-Hostility subscales. Participants who were sleep deprived reported higher subjective stress, anxiety, and anger than controls in response the low-stress, but not the high stress condition (Minkel et al., 2012). Study 2 supports and extends previous research by finding comparable responses to strong negative emotion elicitation procedures regardless of whether participants were partially sleep deprived or not. It appears that whether people are partially or totally sleep deprived, under conditions of extreme stress, or when faced with strong negative emotional stimuli, they will respond similarly to people who are rested. Study 2 also extends the research by replicating these findings with positive and negative affect, basic and dis-

crete emotions. In response to strong negative emotion elicitation procedures, both the partially sleep deprived and rested groups reported similar levels of emotions on the emotion markers. Negative affect and emotions were heightened and positive affect and emotions were reduced. The results lend support for sleep disturbances as a transdiagnostic mechanistic process in the precipitation of emotion dysregulation.

The third study was designed to investigate whether sleep disturbances in addition to precipitation of depressive symptoms, also perpetuate emotion dysregulation through catastrophic worry. Another aspect examined was whether catastrophic worry might be a gender specific cognitive vulnerability. The findings from this study provide additional support to previous research reporting sleep disturbances in adolescence as a risk factor for the development of depression (Clarke et al., 2001; Johnson, Roth, Schultz, et al., 2006; Roberts, Roberts, & Chen, 2002). This study also supports findings of co-occurrence of sleep disturbances and catastrophic worry (Talbot et al., 2010), and between catastrophizing and depressive symptoms (Garnefski, Legerstee, Kraaij, van den Kommer, & Teerds, 2002), as well as the greater propensity of girls to experience these (Flouri & Panourgia, 2011; Hilt et al., 2010; Johnson, Roth, Schultz, et al., 2006).

What this study adds to the existing literature are the findings that adolescent sleep disturbances predict not only precipitation, but also perpetuation of depressive symptoms through catastrophic worry. This suggests that catastrophic worry is a cognitive vulnerability mediating the relationship between adolescent sleep disturbances and depressive symptoms. Furthermore, the mediating effects of catastrophic worry on the relationship between adolescent sleep disturbances and depressive symptoms do not appear to be gender dependent.

Overall, the studies included in this dissertation complement neurobiological findings (Walker & van der Helm, 2009) and theories that suggest sleep disturbances may be a mechanistic transdiagnostic process (Harvey et al., 2011) in the predisposition, precipitation, and perpetuation of emotion dysregulation. The studies included in this dissertation add to the literature by providing support for the involvement of sleep disturbances in posing risk for, initiation and maintenance of emotion dysregulation from adolescence through midlife.

## **Limitations and strengths**

There were several potential limitations in the studies included in the dissertation worth mentioning. One potential limitation is the reliance on self-report inventories in the three studies. Potential response biases may threaten internal validity (Kazdin, 2003) by under or over reporting of

sleep disturbances, or levels on emotion markers. On the other hand, the intention of this dissertation was the subjective experience of sleep disturbances and emotion. This is a standard way to capture what is commonly thought of as subjective sleep experiences or emotions. The longitudinal design offsets this limitation by using two or more time-points. In addition, the studies are strengthened by using a number of established and psychometrically sound scales.

Two other potential limitations involve measurement. In studies 1 and 3, 1 and 2-item measures of sleep-onset problems and sleep disturbances were used. This may lead to an underestimation of the relationships between sleep-onset problems and neuroticism in the former, and between sleep disturbances and depressive symptoms in the later. Stricter criteria for sleep disturbances would likely demonstrate stronger links catastrophic worry and depressive symptoms (Taylor, Lichstein, Durrence, Reidel, & Bush, 2005). It is relatively common however in epidemiological research to use 1-item sleep-onset (Morphy, Dunn, Lewis, Boardman, & Croft, 2007; Roberts, Roberts, & Duong, 2008 ) and sleep disturbance measures (Lundh et al., 2012; MacGregor et al., 2012; Wong & Brower, 2012).

Other potential threats to validity are behaviors taken out of context. The artificiality of an experiment influences how readily the findings are generalizable to other contexts. Therefore, measures of social desirability, compliance, willingness, and realism were incorporated into the design. As well as, sleep condition manipulation and emotion manipulation checks in order to control for potential bias. There were no significant differences between the experiment and control groups on these measures. Another precaution was counter balancing between morning and afternoon testing times to compensate for any influence of circadian rhythms.

There are a couple of potential limitations related to participants. In Study 2, participants were primarily undergraduate students. Therefore, the results may not generalize to other populations, such as the general adult population. In addition, the use of different age groups and ranges within the three studies also limits generalization to other age groups not represented by the age-spans. Study 1 included adolescents through midlife. Study 2 included adults, most in early adulthood. Study 3 used an adolescent population. On the other hand, this dissertation was designed from a developmental perspective spanning from adolescence through midlife. When designing study 2, it was deemed indefensible to subjugate adolescents to the risks posed by 3-nights with only 5-hours total time in bed, or to expose them to the strong explicit graphic content in the emotion elicitation procedures. Having different age group means that the results from the purely adolescent study cannot be generalized to an adult population,



and results from the adult population cannot be generalized to adolescents. Other than that, using different age groups and age-spans allows for investigation of processes (i.e., causal, mediation) and for hypothesis building (e.g., predisposition, precipitation, perpetuation).

The greatest strengths of the studies in this dissertation are the longitudinal and experimental designs. Longitudinal studies provide a developmental look at relationships between sleep disturbances and emotion markers over time as well as changes in these relationships. The sample sizes were ample. In the 20-years investigated in Study 1, there was very low attrition with 91% of those still living participating in the final data collection. Additional strengths were the long-term stability shown by both the neuroticism and sleep-onset problem measures. Neuroticism, measured at two time-points, showed high reliability and validity. Sleep-onset was measured at three time-points. Using cross-sequential longitudinal design in study 3 provided the additional benefit of replication of findings within the same study with two different sets of time-points. This demonstrated internal validity. Using an experimental design with a control group in study 2, demonstrated a cause and effect relationship between sleep deprivation and dysregulated emotions. Both groups reported robust changes on the positive and negative emotions markers, falling within the scale limits. This indicates that there were no ceiling effects. The longitudinal and experimental methods used in this dissertation allowed for a developmental look into the relationships between disturbed sleep and emotion over time, changes over time, and indications of cause and effect.

Overall, the strengths outweigh the limits. The longitudinal and experimental data presented serve to strengthen conclusions. Prospective data are essential to investigating risk, and mediating processes as in studies 1 and 3. Likewise, experimental methods are necessary for teasing out cause and effect and in this way strengthen the conclusions drawn. In line with general guidelines for these types of studies, the psychometric soundness of the measures used in the studies also serve to strengthen the conclusions drawn (Kazdin, 2003).

## **Future directions**

More longitudinal and experimental studies with adolescents and young adults are warranted in the quest to discover whether sleep disturbances are a mechanistic transdiagnostic process in emotion dysregulation. This question is one puzzle piece in the larger puzzle presented earlier in the conceptual model. What are other ways in which these two processes may link neurophysiology and psychopathology? The field would benefit from more multidisciplinary longitudinal and experimental research between

domains such as the fields of neurology and psychology, as well as use of multi-methods. For example, more structured equation modeling would aid in the quest to identify additional mediating and perhaps moderating processes involved in sleep and emotion disturbances over time. Another example is person-oriented research methods, which allow for examination of individual differences in risk for predisposing, precipitating, and perpetuating processes linking sleep disturbances and emotion dysregulation would perhaps aid prevention or intervention efforts by early identification of markers of risk.

The finding in study 1, that neuroticism failed to predict sleep-onset problems suggests that other processes may be involved (e.g., mediation). Future research into disturbed sleep and emotion as potential joint mechanistic transdiagnostic processes linking neurophysiology and psychopathology may consider exploring potential mediating and moderating processes related to neuroticism and sleep disturbances. In addition to the view of neuroticism as a personality trait, in the literature this construct is also viewed as a temperament of negative emotionality and with the neurological behavioral inhibition system (see, Hampson, 2012). Preliminary findings from physiological and neurobiological studies suggest that high neuroticism is characterized by reductions in the ability to regulate emotions. Psychological research supports these findings with reported links between neuroticism and negative bias in attention and interpretation, in retrieval processes, ineffective coping, increased reactivity, and variability in affect (Ormel et al., 2013). Together these findings suggest that there may be individual differences in emotion regulation, which in turn may fuel both amygdala hyper-arousal, which may serve to drive both neuroticism and psychopathology (Ormel et al., 2013). A person-oriented approach may provide a greater understanding of the role of neuroticism in disturbed sleep and emotion in psychopathology.

Research presented in this dissertation suggests that several processes that are activated following sleep disturbance. These are reductions in positive emotions and catastrophic worry. It may be that in addition to priming people to have a negative attentional bias during the day (Baglioni et al., 2010), sleep disturbances may also influence how people experience positive and negative events in response to daily stressors. Sleep deprivation in addition to increases in emotional reactivity are also estimated to reduce positive experience of otherwise rewarding activities (Zohar et al., 2005). Positive affect is thought to promote positive health behaviors through encouragement of restorative health behaviors such as sleep, exercise, and relaxation. Positive emotions are also considered a protective factor in the modulation of negative stress responses (Fredrickson & Cohn, 2008).

Without positive emotions, people may find it difficult to regulate negative emotions.

Findings in this dissertation provide evidence for the dysregulation of positive affect and emotion in response to sleep deprivation. Reductions in positive emotion are key features in both sleep deprivation (Talbot et al., 2010) and in depression (DSM-IV-TR; American Psychiatric Association, 2000). Perhaps this is a route through which sleep deprivation poses vulnerability for emotion dysregulation in people throughout the lifespan. Sleep disturbances may influence emotion dysregulation through processes related to reductions in positive emotion. Perhaps positive emotion mediates the relationship between sleep disturbances and psychopathology. This provides an interesting avenue for sleep and emotion researchers to investigate.

Another finding in this dissertation was that sleep disturbance poses risk for catastrophic worry in addition to depressive symptoms. Other potential mediating processes may underlie catastrophic worry. A facet of the transdiagnostic perspective is that of disrupted cognitive processes (e.g., attention, memory) found across disorders may serve to aggravate, or perpetuate disorders (Harvey et al., 2004). Study 2 showed reductions in three discrete positive emotions underlying the basic positive emotion attentiveness: alert, attentive, and concentrating. One of the diagnostic criteria for a major depressive episode is a “*diminished ability to think or concentrate, or indecisiveness, nearly every day (either by subjective account or as observed by others)*” (DSM-IV-TR; American Psychiatric Association, 2000). Perhaps reductions in concentration, alertness, and attentiveness associated with sleep disturbances are involved in the development of catastrophic worry. Having difficulty processing thoughts, events, feelings, and situations may perhaps fuel cognitive vulnerabilities such as catastrophic worry, or perhaps it is the other way around. This remains to be investigated.

Other pieces in the puzzle not addressed in this dissertation are the social and behavioral aspects that may be involved in the development of sleep and emotion disturbances. Sleep disturbances of a subclinical level in otherwise healthy populations are influenced to a large degree by social and behavioral factors. Inclusion of these in sleep and emotion research may be informative for how these aspects influence the joint development of sleep and emotional disturbances. The area of research examining sleep disturbance as mechanistic transdiagnostic process in emotion dysregulation is still largely at the theoretic level. There is still a lot to be learned about developmental processes and psychological mechanisms linking disturbed sleep and emotion dysregulation. As well as how these processes may link neurophysiological processes and psychopathology.

## Implications and applications - Why is this important?

People who are otherwise healthy sometimes have sleep disturbances whether due to health, stress, or lifestyle. The take home message from this dissertation is that sleep disturbances are not entirely benign and can pose risks to emotional health. Many people occasionally struggle to fall asleep at night, lie awake in bed sleepless, or wake up exhausted in the morning. The human sleep/wake regulatory system is generally self-correcting with homeostatic drives increasing the need for sleep exponentially in relation to how long a person has been a wake, and through circadian pressures facilitating sleep during the night and counteracting sleep during the day (for review see, Gillberg, 2011). The evidence presented herein, suggests that it is essential that people take sleep seriously rather than for granted. The intention is not to scare people, but instead to inform people that sleep has an important role in emotion regulation and is worth prioritizing.

There is a growing consensus that adolescent and adult sleep disturbances pose risk for a myriad of concurrent and long-term physical and mental health, and adjustment problems (Hammen et al., 2008; Hasler et al., 2005; Keenan-Miller et al., 2007; Rao, 2006; Sihvola et al., 2007). Sleep disturbances once established in adolescence, whether due to physiological changes or through solidification of counterproductive sleep behaviors, may follow adolescents into adulthood posing further risks to emotional health (Roane & Taylor, 2008; Susman et al., 2002). Health behavior patterns initiated in adolescence may be especially resistant to change in adulthood (Kelder, Perry, Klepp, & Lytle, 1994). Therefore, the prevention and intervention of adolescent sleep disturbances appear to be a critical research domain.

Sleep behaviors play an instrumental role in the quality and quantity of sleep that people obtain. Exercise, regular sleep and wake schedules, and bedtime routines are all examples of behaviors that are conducive to sleep (see, Carskadon, 2011). Over the longer term, sleep can become dysregulated by both poor sleep habits, and prioritizing other activities over sleep (see, Carskadon, 2011). Numerous contemporary activities do not have natural stop times (e.g., computer gaming, electronic games, internet surfing). Many adolescents have computers, gaming consoles, computer-tablets, smartphones, and televisions in their rooms. These produce light, and stimulation, which are non-conducive to falling asleep (Noland, Price, Dake, & Telljohann, 2009).

Research suggests that information alone about health benefits and the risks of poor sleep are not enough to bring about behavior change in adolescence. In a recent meta-analysis examining school based sleep education programs, adolescents reported a heightened knowledge about sleep. How-

ever, results about the effectiveness of sleep education programs on sleep parameters were mixed. Some studies reported improved sleep hygiene or sleep duration while others did not. The implication is that more research with larger samples should be conducted (Blunden, Chapman, & Rigney, 2011). School based intervention and prevention efforts with adolescents generally use sleep education. Including information and education about sleep in existing health classes undeniably plays an important role in enlightening adolescents about the importance of sleep and the risks posed by not getting enough sleep. However, sleep education alone appears to be toothless when it comes to combating sleepiness, improving sleep duration, or encouraging good sleep health behaviors.

There are other potential targets of health behavior change to incorporate into sleep disturbance prevention and intervention program designs. In addition to sleep knowledge, it may be helpful to target adolescent *attitudes* about sleep in order to prepare them for change. Likewise, it may be beneficial to identify ways for adolescents to become *invested* and *committed* to actively changing their own sleep behaviors. Perhaps one of the most important factors is the targeting of adolescents' *motivation* through stressing the importance of sleep behavior change for the adolescent (Blunden et al., 2011). Adolescent health behaviors are greatly influenced by hormones, peers, multimedia, activities, and behaviors that run counter to good sleep and are therefore perhaps very resistant to change.

The costs of adolescent sleep disturbances are not only limited to adolescence, but may also carry over into adulthood in the form of either chronicity or associated physical and mental health disorders. There is an apparent lack of effectiveness of solely targeting adolescent sleep knowledge in prevention efforts. Another broader full-scale approach would be to develop comprehensive healthy sleep promotion programs on several ecological levels (e.g., governmental, schools, family). Directives from governmental health services would perhaps send a strong signal that promoting healthy sleep habits is a crucial health priority worth promoting. Governmental services may pave the way by supporting good sleep promotion and sleep disturbance intervention research, as well as sleep health program implementation. Governmental agencies may also exert their influence through the schools and through health care services. These routes could play a pivotal role through dissemination of information and education about on the benefits of promoting good sleep, and about the risks of sleep disturbances for physical and mental health.

On another level, adults in society are models for health behaviors whether these modeled behaviors are good or bad. The trend in modern society toward sleep deprivation suggests that many see sleep quantity as

optional. Increasingly time is spent on work, family, and pleasurable pursuits at the cost of sleep quantity. More activities are packed into already strained time budgets. The desire for more time that is expendable might mean that time is taken out of the sleep budget. The idea seems to be that sleep is something that can be pushed up until later. Perhaps then, later never comes. There has been a growing focus on the importance of sleep in the media in recent years. Nevertheless, research is showing that information and education may not be enough. The model that adults in modern society are providing for adolescents is that sleep just is not that important. Adolescents learn a lot by watching what adults do rather than what they say. The findings herein, suggest that sleep deprivation has a very strong impact on emotion regulation through next day reductions in positive affect and emotions. People may experience a diminished capacity to modulate negative emotions when positive emotions are reduced (Fredrickson & Cohn, 2008). The findings in study 2 suggest that even short-term partial sleep deprivation has a negative impact on the dysregulation of positive emotions of otherwise healthy adults.

One avenue for targeting adolescent sleep disturbance under investigation is through the engagement of parents. Parent involvement in adolescent sleep behavior is showing promise in promoting change. In a study with 385 adolescents, those whose bedtimes were set by parents, slept more, reported feeling less fatigue and more alert during the day than a control group (Short et al., 2011). It may be beneficial to target other behaviors in addition to bedtimes. A few examples are having regular sleep and wake times, having stop times for multimedia use, turning off cell-phones at night. It may be possible to involve parents in sleep disturbance prevention and intervention programs, if they consider adolescents' sleep behaviors as important and within their power to control or influence. Perhaps parents can be enlisted through informing and educating them about the importance of sleep and the risks associated with sleep disturbances. Early-onset sleep disturbances incur risk not only for other comorbid early-onset disorders, but also for long-term physical and mental health problems (Roane & Taylor, 2008). Perhaps parents can be persuaded to become involved in prevention efforts if they understand that sleep disturbances pose risk for early-onset psychopathology such as depression (Clarke et al., 2001; Johnson, Roth, & Breslau, 2006; Roberts et al., 2002), as well as other mental, physical, and behavioral problems over the long-term (Roane & Taylor, 2008). Parents might be encouraged to participate actively in adolescent sleep health promotion and sleep disturbance intervention programs both through holding firm attitudes about accepta-

ble sleep behaviors, and through influencing sleep behaviors that are under their control (e.g., setting bedtimes).

Empirical findings including those found in this dissertation, suggest that sleep disturbances are important in emotion dysregulation (see, Harvey et al., 2011; van der Helm & Walker, 2010). It appears that sleep disturbances act as both the fuel and the ignition key to a motor driving emotion dysregulation. Findings in this dissertation provide markers of risk for depression in adolescence, as well as for emotion dysregulation in adolescence and adulthood. Also, provided are two potentially modifiable targets for prevention and intervention, sleep disturbances and subsequent catastrophic worry. The studies in this dissertation suggest that sleep disturbances have an important role in the predisposition and precipitation for emotion dysregulation. The findings in study 3 suggest that the risks posed by sleep disturbance for depressive symptomology in adolescence is two-fold. Sleep disturbances in adolescence appear to not only confer risk for depressive symptoms, but also perpetuate symptomology through spurring of catastrophic worry. Identifying sleep disturbances and catastrophic worry as markers of risk for developing depressive symptoms may provide parents, school counselors and nurses, teachers, patients, and psychologists with early warning signs that there is a potential problem brewing. Implementation of prevention programs in on-going health education programs in the schools with the support of parents may aid in reducing the long-term societal, financial, and emotional costs for at-risk individuals and their families in the long-term.

Clinicians report that sleep disturbances add to depression severity, are behind poor response to depression treatment, are a frequent residual symptom following depression treatment, and are a viable predictor of depression recurrence and relapse (see, Manber & Chambers, 2009). Indications are those sleep disturbances are a mechanistic transdiagnostic process involved in emotion dysregulation. Therefore, sleep disturbances and ensuing catastrophic worry may provide clinicians with targets for treatment when meeting clients with depressive symptoms or other signs of emotion dysregulation.

## Conclusions

There is a growing consensus of the importance of good sleep for mental health. The way that society thinks about sleep is changing, as evidenced by the popularity of sleep as a “hot topic” in the media and popular culture. New ways of thinking about sleep and psychological health are developing along with changed ways of thinking about disturbed sleep. There was a time in the psychological sleep literature when sleep disturbances

were considered primarily a consequence of life-stressors or a secondary outcome of some other disorder. Sleep and emotion research is moving in a new direction with neurologists and psychologists leading the way.

Sleep disturbances are a diagnostic feature and risk factor for many psychological disorders. So often, in fact that neurologists and psychologists have proposed that sleep disturbances may be a mechanistic process in emotion dysregulation. Depression is an example of a disorder sharing high comorbidity with sleep disturbances co-occurring in 70% to 75% of cases (Liu et al., 2007; Ohayon, Shapiro, & Kennedy, 2000). Perhaps sleep disturbances are a mechanistic transdiagnostic process interacting with emotion dysregulation in comorbidity of other disorders. Together, these two processes are suspected in having key mechanistic roles in the relationship between neurophysiology and psychopathology.

In this dissertation, it was argued that sleep disturbances act as a catalyst for predisposition, precipitation, and perpetuation of emotion dysregulation. In turn, these processes likely co-act in the development, triggering, and maintenance of many psychopathological disorders. Admittedly, the influence of emotion dysregulation on sleep is also of great importance in the bidirectional relationship between these two processes. However, the focus of this dissertation was primarily on the role of sleep disturbances in emotion dysregulation. The evidence from the studies presented in this dissertation support the critical role of sleep disturbance in emotion dysregulation.

This dissertation lends support for the conceptual framework that sleep disturbance may be a mechanistic transdiagnostic process in emotion dysregulation. Through which sleep disturbances and emotion dysregulation may jointly link neurophysiology and psychopathology (Harvey et al., 2011). Study 1 provided strong support for sleep disturbances in adolescence as an underlying vulnerability for emotional instability stretching as far as into midlife. Study 2 showed how even short-term partial sleep deprivation has a role in the dysregulation of positive emotions. Mechanistic psychological processes are involved in both conscious processes (e.g., catastrophic worry) and unconscious processes (e.g., implicit emotion regulation) in the generation of recurrent patterns of thinking, feeling, and behaving. Study 3 provided support for adolescence sleep disturbance as a precipitator for and a perpetuator of emotional distress in the form of depressive symptoms through fuelling of catastrophic worry.

Theoretically, mechanistic transdiagnostic processes explain comorbidity as due to a causal inter-relationship between processes and psychopathology (Harvey et al., 2011). The studies included in this dissertation suggest that sleep disturbances in various forms pose risk for predisposi-



tion, precipitation, and perpetuation of emotion dysregulation on various levels. The overriding conclusion is that sleep disturbances are a mechanistic transdiagnostic process interacting with emotion dysregulation as a bridge between neurophysiological processes and psychopathology such as depression.

It appears that sleep disturbances act as both the fuel and the ignition key to a motor driving emotion dysregulation.

~Nanette Suzanne Danielsson

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