ROLE OF TRAUMA EXPOSURE, EMOTION REGULATION AND NEUROTICISM ON SLEEP QUALITY AMONG POSTGRADUATE STUDENTS.

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## TITLE PAGE

# ROLE OF TRAUMA EXPOSURE, EMOTION REGULATION AND NEUROTICISM ON SLEEP QUALITY AMONG POSTGRADUATE STUDENTS. 

## CERTIFICATION

This is to certify that this research is the original work of HARRY, JANE AKPAN, a postgraduate student of the Department of Psychology, Faculty of the Social Sciences, University of Nigeria, Nsukka, with registration number PG/M.Sc./13/66962, for theaward of the Master of Science (M.Sc.) degree in Clinical Psychology. The work has not been submitted for the award of any degree in any other institution, nor published anywhere in part or full.

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## DEDICATION

I dedicate this work to the Almighty God whose mercy and grace saw me through. To my parents Deacon and Deaconess A. H. Etukudoh, my brother and sisters for their support.

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#### Abstract

The current study investigated the role of trauma exposure, emotion regulation, and neuroticism on sleep quality among postgraduate students. Four hundred and four postgraduate students (males=228, females=176) participated in the study. The studentsôages ranged from 22 years to 51 years, with a mean age of30.60 years ( $\mathrm{SD}=6.84$ ).Participants completed the Harvard trauma Questionnaire (HTQ), Difficulties in Emotion Regulation Scale (DERS), Neuroticism Subscale of Big Five Inventory (NSBFI), and Pittsburgh Sleep Quality Index (PSQI). Multiple regression was used for data analysis.Result showed that trauma exposure significantly predicted sleep quality ( $\mathrm{K}=.12$, $\mathrm{P}<.05$ ). Difficulties in emotion regulation significantly predicted sleep quality ( $\mathrm{B}=.38, \mathrm{P}<.001$ ). Neuroticism was a significant predictor of sleep quality ( $\mathrm{B}=.11, \mathrm{P}<.05$ ). Findings were discussed in relation to the literature.It was suggested that there is need to consider traumatic events, emotion regulation difficulties and neurotic traits in attending to individuals that have reduced quality of sleep. Limitations of the study highlighted and suggestionsfor further study were made.


## CHAPTER ONE

## Introduction

Sleep is an essential and inevitable part of human life that influences optimal functioning. In the literature, it is difficult to determine the underlying function of sleep because there are likely many confounding factors with regard to causality (Hale, 2005). Various happenings throughout the day may have an effect on the quality of sleep and the quality of sleep one has the night before may have an effect on the many activities and interactions during the day. How refreshed and awake an individual feels after a night $\hat{\varrho}$ sleep is in part determined by the sleep quality an individual experiences. The bodyố main biological restorative process is sleep. It is considered to be both physically and psychologically restorative to an individual. Sleep is a time that the body physically grows and repairs organs and tissues from normal wear-and-tear (Robles \& Carroll, 2011).

Sleep is also one of the essential components in the overall mosaic of health and it profoundly affects the subjective sense of physical and mental well-being (Guilleminault, 2001). It is one of the things that exemplify the direct link between the physical and the psychological state, which are mutually inseparable. Sleep is therefore a relevant issue for public health and public policy (Williams, Meadows \& Arber, 2010). Sleep appears necessary for every system of the body to work properly (Odetola \& Adejumo, 2014), and it is required to provide energy for the physical and mental activities of all humans. Sleep is one of the most essential needs of the body and mind, required by both the healthy and the ill individuals, but for sleep to actually be beneficial to health, it must be adequate in quantity, quality and latency (Richardson, Thompson, Chambers \& Turnock, 2009). An old Chinese proverb states: ñit is only when one cannot sleep, that one knows how long the night isò. This explains the importance of sleep for
human functioning. Just as water and food are essential for human well-being, sleep is judged essential for human survival.

Sleep is essential to the healthy development of adolescents and adults, as well as their success at school and in the workplace. Sleep experts believe sleep affords the neurons that were used while awake an opportunity to be repaired and restored. Without sleep, neurons may become so depleted in energy or so polluted with bye-products of cellular activities that they begin to malfunction (Salo, Vahtera \& Hall, 2012). Sleep also gives the brain a choice to exercise important neuronal connections that might otherwise deteriorate from lack of activity. Thus, the need for sleep in human beings is universal and basic because sleep serves as a restorative function for the body and mind. Sleep has a protective role in that it helps prepare people for coping with daily hassles and life stressors (Walker \& Vander Helm, 2009).

Though there is little medical basis for this belief, the amount of sleep that many people believe they should get is about eight (8) hours (Goodman, 2014). Kripke, Garfinkel, Wingard, Klauber and Marlerốs (2002) study of more than 1.1 million adult participants examined the amount of sleep each night, the number of nights with insomnia, and other controlling factors such as medication use, demographics, health and habit. Participants who slept 6.5hrs ï 7.4hrs had the least risk for all-cause mortality in both males and females. Tamakoshi and Ohnô̂s (2004) study of over 100,000 Japanese subjects also supported this notion that sleep duration of about 7 hours has the lowest mortality risk. In a study with middle-aged and older adults in Australia, short sleep durations (<6hrs) and not long durations (>9hrs) were associated with lower self-reported health and quality of life (Magee, Caputi \& Iverson, 2011). However, sleep duration is not always enough when talking about sleep quality. Sleep habits are important for a restful night and sleep quality remains an important habit of sleep (Augner, 2011).

Quality sleep is important in all individuals. Sleep quality is defined as oneô satisfaction of the sleep experience, integrating aspects of sleep initiation, sleep maintenance, sleep quantity and refreshment upon awakening (Krystal \& Edinger, 2008). Although the construct of sleep quality is widely used, a review of the empirical literature suggests that it is not yet fully understood. Indeed, Akerstedt, Hume, Minors and Waterhouse (1994) noted that there seems to be very little systematic knowledge as to what actually constitutes subjectively good sleep and how this should be measured, thereby suggesting the relevance of increasing research on this construct.

The quality of sleep is a measure of both the quantitative and qualitative components of sleep. The quantitative component includes the duration of sleep, sleep latency or number of arousals, while the qualitative component is a subjective measure of the depth and feeling of restfulness upon awakening (Lavie, Pillar \& Malhotra, 2002). The quantity of sleep an individual gets is important, but it is the quality of sleep that they really have to pay attention to. Some people sleep eight or nine hours a night but do not feel well-rested when they wake up because the quality of their sleep is poor. The best way to figure out if adults meet their sleep need is to evaluate how they feel as they go about their day. If an individual sleeps for long hours, he/she feels energetic and alert all day long, from the moment he/she wakes up until his/her regular bedtime.

Sleep quality is an important indicator of adultsôhealth and well-being (Tynjälä, Kannas, Levälahti \& Välimaa, 1999). Good sleep quality is associated with a wide range of positive outcomes such as better health, less daytime sleepiness, greater well-being and better psychological functioning, while poor sleep quality is one of the defining features of chronic insomnia, (Edinger, Bonnet \& Bootzin, 2004). Poor sleep quality has been associated with
increased tension, irritability, depression, confusion and generally lower life satisfaction (Pilcher, Ginter \& Sadowsky, 1997). Poor sleep quality contributes to poor daytime coping with daily stress, and may increase fatigue, moodiness, or memory impairments and anxiety (Harvey, Jones \& Schmidt, 2003; Joo, Shin, Kim, Yi, Ahn \& Park, 2005) and thus not functioning at optimal levels (Gaultney, 2010). The belief that one has had high quality sleep regardless of actual sleep quality may also be a factor in determining restoration from sleep. When a person does not get an adequate amount of sleep, the loss in sleep contributes to health problems because the body is unable to replenish the lost resources that are consumed during waking hours.

In Nigeria, Odetola and Adejumo (2014) conducted a research on the effect of nursing hospital routine on the pattern, quantity and quality of sleep among hospitalized patients in Ibadan and the result revealed that there is a significant difference in sleep pattern before and during hospitalization. This study provided an opportunity for nurses to appreciate the importance of sleep (both in quality and quantity) especially for hospitalized patients as a vital contribution to recovery and the need for a thorough assessment of patientsôsleep pattern which is an important indicator of improving health in the general population.

Aloba, Adewuya, Ola and Mapayi (2007) acknowledged that the extent of sleep-related problems remains largely an unidentified public health issue, particularly so in Africa where there is inadequate personnel to assess sleep problems. Reid and Baker (2008) reported that awareness of sleep and sleep disorders was low in the general population in South Africa. Indeed, this observation can easily be generalized to other parts of the sub-Saharan Africa, in that by comparison, South Africa is by far better developed in its education, health and all areas of its economy and yet sleep and sleep disorders awareness is low (Reid \& Baker, 2008). A wider
implication of this lack of awareness about sleep, is that in sub-Saharan Africa it is likely that most sleep and sleep-related issues may be presented to primary care health workers as physical problems.

Students have been identified as a population group particularly affected by problems with sleep (Pallos, Yamada, Doi \& Okawa, 2004; Carney, Edinger, Meyer, Lindman \& Istre, 2006). A number of factors can influence sleep quality. Factors of interest to the researcher are trauma exposure, emotion regulation and neuroticism.Majority of university students are in early and middle stages of adulthood. This age group may have experiences of various types of traumatic events (Davidson, Hughes, Blazer \& George, 1991). Those with traumatic experiences have frequent sleep disturbances interfering with their sleep quality. Due to the trauma they have experienced, their sleep patterns and related problems could differ from students without trauma (Fukuda \& Ishihara, 2001).

A significant factor that may affect the sleep patterns of adults is the trauma they experience like the major life events (e.g., domestic violence, divorce, rape, earthquake, accident, war, death of a family member), and more minor but daily stressors (e.g., difficulties with interpersonal relationships, work-related stress) can affect sleep patterns in otherwise healthy individuals by heightening arousal before falling asleep and during nocturnal awakenings. Nonetheless, the importance of sleep disturbance in victims of trauma has been that it has prognostic significance (Harvey, et al., 2003), and therefore worthy to investigate in sleep research.

Trauma exposure is a construct that has and is still being studied among highly and multiply traumatized populations including but not restricted to adolescents and adults in
communities, schools, and public health settings in different cultures; as well as among torture survivors, prison inmates, minorities, refugees, and mental health patients ((Lambert, Ialongo, Boyd \& Cooley, 2005; Finkelhor, Turner, Ormrod, Hamby\& Kracke, 2009; Stein, Jaycox, Kataoka, Rhodes\& Vestal, 2003). Trauma is a sudden, unpredictable, life-threatening event that is out of the ordinary life experience, and may result from natural or accidental disasters (earthquake, fire), large-scale catastrophes (war), medical trauma (stroke), and personal catastrophes (rape, assault, witnessing violence) (Keane, Marshall \& Taft, 2006). Experience such as these often are considered extreme stressors.

The Diagnostic and Statistical Manual of Mental Disorder Fifth Edition (DSM-5, 2013) describes trauma as direct personal experience of an event that involves actual or threatened death or serious injury, or other threat to one $\hat{\beta}$ integrity; or witnessing an event that involves death, injury, or a threat to the physical integrity of another person; or learning about unexpected or violent death, serious harm or threat of death or injury experienced by a family member or other close associate. Trauma not only entails physical injuries, but also includes psychological effects from stressful events (Frommberger, Angenendt \& Berger, 2014). Immediate threats during such experiences may result in death or injury.

Exposure of adults to trauma is pervasive in most parts of the world (Finkelhor, et al., 2009; Clever \& Bruck, 2010; Adebajo \& Kolawole, 2013). Nearly $80 \%$ of adults seen in community mental health clinics have experienced at least one incident of trauma during their lifetime, representing roughly five out of every six adults (Breslau \& Kessler, 2001).Exposure to a traumatic event includes individuals either experiencing or witnessing an event during which they experience intense fear, helplessness, or horror (APA, 2000). Further, exposure to trauma can occur through various means, including direct victimization, witnessing or hearing violent
acts, and media violence (Buka, Stichick, Birdthistle \& Earls, 2001). Exposure to traumatic events is quite common, with estimated lifetime rates ranging from $26 \%$ to $92.2 \%$ in men and from $17.7 \%$ to $87.1 \%$ in women (Creamer, Burgess, \& McFarlane, 2001).Traumatic events that may trigger PTSD include violent personal assaults, natural or unnatural disasters, accidents, or military combat, Boko Haram insurgency, terrorism or mass disaster, violence or war, or may involve a move to a new location, death of a friend, family member, or pet, divorce or separation or jilting, ssfear, anxiety, hospitalization, loss of trust, pain, physical injury or illness, separation from parents, or perceived abandonment. Of course, violence and mortality rates have been estimated at 3.2 to 9.5 per 10,000 per day, with the majority of these caused by attacks which caused people to flee their home (Depoortere, Checchi \& Broillet, 2004; Grandesso, Sanderson, Kruijt, Koene, \& Brown, 2004).

At the time of a traumatic exposure, the victim might feel numb and incapable of appropriate response. Thereafter, memories of the trauma engender feelings of helplessness, fear, or horror, akin to re-experiencing the trauma time and again. In children, signs of anxiety might include an increased need for physical and emotional closeness, fear of separation, difficulties sleeping, loss of appetite, bedwetting, or changes in interactions with others. Thus, it is imperative to discuss the events and feelings that accompany them in order to resolve the feeling and move forward.

The emotional aftermath of traumatic events can be devastating as any physical damage. Whether trauma stems from a personal tragedy, a natural disaster, or other overwhelming life experiences, it can shatter one $\hat{Q}$ sense of security, making one feel vulnerable, helpless, and even numb. There is no right or wrong way to feel after traumatic exposure. But there are many
strategies that can help a victim work through feelings of pain, fear, and grief and regain his/her emotional equilibrium. Whether the traumatic event happened years ago or yesterday, the person can be healed and he or she moves on. Therefore, after a traumatic exposure, the victim needs time, support, and a sense of safety to re-establish trust.

Most research about trauma exposure in adults have examined the association between adverse outcomes from specific trauma exposures such as sexual abuse (Berliner \& Elliott, 2002), domestic violence (Adebayo \& Kolawole, 2013), physical abuse (Christian, 2015), or community violence (Gaylord-Harden, Dickson \& Pierre, 2015). However, exposure to trauma is not necessarily a one-time occurrence, and students who are exposed to a single trauma are at far greater risk of experiencing multiple exposures of trauma and violence (Finkelhor, et al., 2009).

Research (Daigre, Rodriguez-Cintas, Rodriguez-Martos, Grau-Lopez, Berenguer, Casas \& Roncero, 2015; Sneed, 2015; Stanley, 2011) showed that multiple trauma exposures such as sexual and physical abuse, neglect, exposure to domestic violence, and assaults by peers, have been associated with a wide range of later psychological symptoms. One of such symptoms may be sleep-related disorders or sleep disturbance as a symptom of another disorder. Trauma can interfere with sleep onset, maintenance and quality of sleep. Sleep disturbances are common in trauma survivors, and they can exacerbate depression and PTSD, increase symptomatology, and have a negative effect on health (Krakow, Artar, Warner, Melendez, Johnston, Hollifield, Gemain \& Koss, 2000). Several studies have suggested possible links between traumatic experiences and sleep quality such as women who experienced perinatal and postpartum outcomes, sexual and physical abuse, and exposure to natural disaster(Noll, Trickett, Susman \&

Putnam, 2006; Tempesta, Curcio, De Gennaro \& Ferrara, 2013; Swanson, Hamilton \& Muzik, 2014; Hauff, 2015).

Reactions to traumatic events vary considerably, ranging from the relatively mild, which create minor disruptions in the personôs sleep quality, to the severe and debilitating. It is common for those who are exposed to trauma to experience intrusive thoughts and images, accompanied by attempts at avoidance, emotional numbing (such as sleeping difficulties or anger) (Joseph, 2010). Highly stressful and traumatic events typically produce a variety of cognitive, emotional, behavioural, and social elements that influence the eventual outcome. A common theme in models that focus on the aftermath of these significant life events is the impact of rumination that occurs (Calhoun, Cann, Tedeschi \& McMillan, 2000). The ruminative thinking that goes on after a traumatic or stressful event has been often considered to be negative, depressogenic, and intrusive thinking that may dominate the survivorsôexperience as they focus on the harm they have experienced. Therefore, trauma exposure could be enormous scientific value in studying sleep quality among graduate students who are potentially exposed to trauma.

The second predictor of sleep quality to be examined in the study is emotion regulation. The most frequently cited reason why people do not have a good quality of sleep is the negative emotion associated with life events they experience (Watson \& Clark, 1991). The need to regulate these emotions to positively impact sleep quality becomes imperative.Emotion regulation has been defined as a set of emotional, cognitive, behavioural and interpersonal skills which regulate and moderate the experience and expression of human emotions (Posner \& Rothbart, 2002). Similarly, Forbes and Dahl (2005) describe emotion regulation as "... the internal and external processes involved in the initiation, maintenance, or modification of the
quality, intensity, or chronometry of emotional responses" (Forbes \& Dahl, 2005, p. 5). Perhaps a more colloquial description of the construct is one reported by Gross (1998) who states that people regulate their emotions deliberately through their thought and behaviours or automatically outside of awareness in an attempt to modulate which emotions they have, when they have them, and how they experience and express these emotions.

It 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). 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. It seeks to meet psychological functions such as the satisfaction of hedonic needs, furthering goal pursuits, and facilitation of the global personality system (Gross \& Thompson, 2009). Combining the previous definitions, a construct emerges that involves both positive and negatively-valenced emotions, and the processes that may serve to enhance, suppress, and sustain them, or even to replace them with other emotions (Butler \& Gross, 2004).

So emotionregulation may be automatic or controlled, conscious orunconscious, and may involve the up or down-regulation(i.e., increase or decrease) of various aspects of negative orpositive emotions (Parrott 1993). Cole, Michel and Teti (1994) posited that regulation of emotion is the ability to respond to the ongoing demands of experience with the range of emotions in a manner that is socially tolerable and sufficiently flexible to permit spontaneous reactions as well as the ability to delay spontaneous reactions as needed.

Emotion regulation (ER) involves awareness, understanding, and acceptance of emotions, the ability to control impulsive behaviors related to negative emotions, and the ability to use
flexible emotions in order to meet individual goals and situational demands. The absence of any of these components would signify the presence of difficulties in emotion regulation, (otherwise known as emotion dysregulation, ED) (Gratz \& Roemer, 2004). As such, these dysregulated processes may not be optimal in meeting long-term goals and environmental demands (e.g., under- regulation or insufficient regulation of the amount and intensity of expressed emotion, and over-regulation).

Emotion dysregulation has been defined as difficulties in controlling the influence of emotional arousal on the organization and quality of thoughts, actions, and interactions. Individuals who are emotionally dysregulated exhibit patterns of responding in which there is a mismatch between their goals, responses, and/or modes of expression, and the demands of the social environment (Zeman, Cassano, Perry-Parrish \& Stegall, 2006).According to Beauchaine, Gatzke-Kopp and Mead (2007), emotion dysregulation refers to an emotional response that is poorly modulated, and does not fall within the conventionally accepted range of emotive response. It may be referred to as labile mood (marked fluctuation of mood or mood swings). People vary significantly in the quality and intensity of their emotional responding to similar stimuli and situations (Davidson 1998).

According to Gross (2015), the terms ámotion regulationôand ámotion dysregulationô refer to processes by which individuals change or maintain the intensity or the valence of an emotional experience in order to appropriately respond to environmental demands. The regulation of emotions may be conscious/overt or non-conscious/covert. The ability to adaptively regulate emotions is crucial for healthy functioning. Both negative and positive emotions may be regulated. Over the years many authors have focused mostly on strategies used to influence and modify negative emotions. In fact, negative emotions and dysregulation of negative affect have
been shown to be related to psychopathology (Aldao, et al., 2010; Aldao \& Nolen-Hoeksema, 2012).

Moreover, different theoretical models have highlighted different specific strategies that are adaptive or maladaptive, and the latter has been correlated with different types of disorders. Maladaptive strategies which are generally considered to be associated with negative outcome are: avoidance, rumination and suppression (namely the suppression of the emotional display or of the emotional experience), whereas adaptive strategies are problem solving, acceptance and reappraisal (Aldao, et al., 2010).

Possible manifestations of emotional dysregulation include affective or emotional instability, intense efforts to avoid real or perceived abandonment, unstable interpersonal relationships, angry outbursts or behavior outbursts such as destroying or throwing objects, aggression towards self or others, crying, accusing, creation of chaos or conflict, and threats to kill oneself. These variations usually occur in seconds to minutes or hours. Nonetheless, most of the extant scientific literature addressed the relationship between the use of maladaptive strategies evidencing that their use is associated to psychopathological disorders such as depression (Ehring, Tuschen-Caffier, Schnülle, Fischer \& Gross, 2010; Joormann \& Gotlib, 2010; Brockmeyer, Bents, Holtforth, Pfeiffer \& Herzog, 2012; Berking, Wirtz, Svaldi \& Hofmann, 2014), anxiety, (Aldao, et al., 2010; Aldao \& Nolen-Hoeksema, 2012) and other mental disorders (Berking \& Wupperman, 2012). However, results suggest that difficulties in emotion regulation may be markers of cognitive impairment in other psychological diseases (Gul \& Ahmad, 2014).Studies(Baglioni, Spiegelhalder, Lombardo \& Riemann, 2010; Mauss, Troy \& LeBourgeois, 2013; Sandru \& Voinescu, 2014; Tsypes, Aldao \& Mennin, 2015) found that emotion regulation has been associated with self-reported poor sleep quality. This effect could
have important consequences for individual $\hat{\$}$ healthy functioning as poor sleep may affect mental health both directly and indirectly as a mediating factor.

It is also possible that the personality traits of an individual will determine one $\hat{\widehat{s}}$ sleep quality.Personality traits are aggregate characteristics and qualities displayed by an individual over time and across different situations. It is believed that individual differences in psychological distress and mental health are partly rooted in personality characteristics (Friedman, 1990), and among the primary dimensions of personality, neuroticism is the chief determinant of mental health outcomes (Watson \& Clark, 1984).

Neuroticism (negative emotionality, trait anxiety, negative affectivity) is a personality trait, or emotional disposition used in personality research as a gauge of emotional stability (Lahey, 2009). It is a construct which has been defined in numerous ways. Some researchers have defined neuroticism as the tendency to experience negative distressing emotions and physical symptoms (Merkelbach, König \& Sittinger, 2003), the general disposition to develop psychopathological symptoms such as anger, anxiety and depression as it is sometimes called emotional instability (Jeronimus, Riese,Sanderman \& Ormel, 2014), negative affect (Wilson \& Gullone, 1999), and a psychological tendency to perceive threat (Schneider, 2004). Costa and McCrae (1992) defined neuroticism as a dimension of maladjustment or negative emotionality versus adjustment and emotional stability. Differences among definitions have been reconciled in the late 1990s with the consensus definition that, at its core, neuroticism is the propensity to experience negative emotions (Widiger, 2009). Mathew and Deary (1998) maintain that a neurotic person has an enduring tendency to experience negative emotional states and feeling such as guilt, envy, anger, anxiety and depressed mood.

Neuroticism is defined as the proneness of the individual to experience negative affective states, and may also be associated with increased exposure to stressful life events (Bolger \& Zuckerman, 1995), and greater susceptibility to the adverse effects of stress (Ormel, Oldehinkel \& Brilman, 2001; Kendler, Kuhn \& Prescott, 2004) The correlates of this trait are profound including low subjective well-being, perceptions of low physical health, maladaptive reactions to illness, higher levels of psychopathology, less mature levels of identity achievement and a lower quality of social and romantic relationships (Ozer \& Benet-Martēnez, 2006).

Eysenck and Eysenck's $(1964,1975,1985)$ personality proposition considers neuroticism as one of the three central behavioural attributes which is found in various degrees in a normal population. In Eysenck's view, neuroticism is based on activation thresholds in the sympathetic nervous system or visceral brain. 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, (Lahey, 2009).

Neurotic people, who have low activation thresholds, and who are unable to inhibit or control their emotional reactions, experience negative affect (fight-or-flight) in the face of very minor stressors - they are easily nervous or upset. By implication, individuals who score high on measures of neuroticism may be more likely to experience such feelings as anxiety, anger, envy, hostility, guilt and depressed mood. They may be prone to interpreting ordinary situations as threatening and minor frustrations as hopelessly difficult; often self-conscious and shy, and may
have trouble with self-regulation. Generally, neuroticism reflects individual differences in behaviour which are thought to be pervasive across different situations. Researchers (Eysenck \& Eysenck, 1985; Eid \& Diener, 1999) have also observed that neuroticism is associated with more variability in behaviour and experience. Consequently, it is important to consider the role of individual, developmental and social factors in neuroticism because of neuroticismô significance as a risk factor in psychopathology (Costa \& McCrae, 1980; Malouff, Thorsteinsson \& Schutte, 2005; Griffith, Zinbarg, Craske, Mineka, Rose, Waters \& Sutton, 2009).Neuroticism is related to and a robust predictor of many mental and physical health problems, (Lahey, 2009).

The public health and educational relevance of neuroticism have also been demonstrated by studies on the personality-psychopathology association within a network of traits and symptoms (Gainey, 2011; Lahey, 2012, Magee, Patrick \& Leonie, 2012). Specifically, high levels of neuroticism have been found to be associated with higher risk of major depression (Fanous, Neale, Aggen \& Kendler, 2007), increased risks of psychiatric morbidity among individuals with anxiety and mood disorders (Griffith, et al., 2009), memory problems (Neupert, Mroczek \& Spiro, 2008) and poor problem solving (Owen, 2007). A relatively recent study (Cuijpers, Smit, Penninx, de Graaf, ten Have \& Beekman, 2010) indicated that the economic costs of neuroticism are more than the costs of mental and physical illnesses not only because neuroticism is associated with those disorders but also due to its contributions to general psychopathology.

Research has revealed some relations between neuroticism and sleep indicating that neuroticism and self-criticism were negatively related to sleep length, even after controlling for depression and anxiety (Vincent, Cox \& Clara, 2009).Recent studies (Soehner, Kennedy \& Monk, 2007; Calkins, Hearon, Capozzoli \& Otto, 2013;Duggan, Friedman, McDevitt \&

Mednick, 2014) reported that high neuroticism were the best predictors of poor sleep (i.e., poor sleep hygiene, low sleep quality, and increased sleepiness). However, there is little research of sizable samples to identify neuroticism traits are related with sleep quality in adults particularly university graduate students. There is a need to assess the relationship between sleep quality and neuroticism for effective preventive mental health interventions in graduate students.

## Statement of the Problem

Sleep quality and its consequences is asignificant public health issue - an issue that has caused health organizations investing a lot of money into sleep education (Sateia \& Nowell, 2004).Of the various psychological contributing factors to poor sleep quality, dysfunctional thoughts and beliefs have been researched in recent times. Morin (1993) postulated that how one thinks about his/her sleep is likely to contribute to one $\hat{\alpha}$ ability to initiate and maintain sleep. It is an important indicator of adultsô health and well-being (Tynjälä, Kannas, Levälahti \& Välimaa, 1999). Good sleep quality is associated with a wide range of positive outcomes such as better health, less daytime sleepiness, greater well-being and better psychological functioning, while poor sleep quality is one of the defining features of chronic insomnia (Edinger, Bonnet \& Bootzin, 2004). Good sleep quality and adequate amount of sleep are important in order to have better cognitive performance and avoid health problems and psychological disorders.

Lack of sleep has been found to have significant effects on concentration, memory, and other neuropsychological deficits (Sadock \& Sadock, 2005), physical health and well-being (Nunes, Jean-Louis, Zizi, Casimir, Gizycki \& Brown, 2008), as well as psychological health (Hamilton, Nelson, Stevens \& Kitzman, 2007). Sleep problems have been the subject of
empirical inquiry by researchers for decades and the most common problems investigated include delay of sleep onset, difficulty staying asleep, awakening too early, as well as inadequate quantity and quality of sleep (Mendelson, 1987). Numerous studies have been conducted (e.g. Ellis, Hampson\& Cropley, 2007; LeBourgeois, Giannotti, Cortes \& Wolfson, 2005; Paine, Gander, Harris \& Reid, 2005) to unravel the etiological complexities of sleep quality in an attempt to search for effective prevention programme. However, it is evident from studies done that varieties of factors prevent a good quality of sleep in our society, particularly in Nigeria, (Odetola \&Adejumo, 2014).

Poor sleep quality is a distressing consequences of trauma exposure. Some of these include loss of loved ones, divorce, severe accident, physical and sexual abuse, emotional and physical neglect, loss of property, terminal illness, exposure to war, domestic violence and personality trait.Given that sleep disturbances is a common experience following trauma exposure and may contribute to the development and maintenance of PTSD, sleep disturbances is an important factor to investigate.

Emotion regulation may bea contributing to poor sleep quality, specifically in the form of emotion regulation difficulties, associated with trauma exposure and PTSD symptoms through the effects that emotional processes have on sleep onset and restfulness. Sleep problems may be aggravated by neuroticism, predisposing people to react to life stressors with negative emotions, thoughts, and behaviours. Heightenedneuroticism and dysregulated emotions may aggravate the regulation of sleep and mood through catastrophic worry (Nanette, 2013).

The present research focuses on exposure to trauma, emotion regulation and neuroticism in explaining sleep quality. Nigerian adults are too often exposed to trauma and this is becoming
a global concern. Student population group have come to be greatly affected by sleep difficulties.The impact of sleep disorders in postgraduate students could be severe and can affect both academic and personal activities. Most students may have gone through one traumatic event or another in which they may become preoccupied with thoughts about their action during the event, often experiencing guilt or shame over what they did or did not do. Some may withdraw, subdued or even mute after a traumatic event which may cause them to have difficulty falling or staying asleep which will later lead to performance impairment, lack of concentration, bad behavior, and poor relationship. It is in this view that this research is being conducted with a view to understanding studentsôemotional and physical challenges.

This study is imperative because more information is needed to better understand sleep quality and to give room for appropriate intervention. The research problems that informed this study could be stated as follows: (a) need assessment for psychological interventions among post-graduate students have not considered the psychosocial health of the graduate students, (b) there is dearth of empirical knowledge on the unique individual contributions of trauma exposure on sleep quality among postgraduate students, (c) the insufficient understanding of the role played by emotion regulation and neuroticism on sleep quality.

Thus, the present study specifically sought to provide answers to the following questions:

1. Will trauma exposure predict sleep quality among postgraduate students?
2. Will emotion regulation predict sleep quality among postgraduate students?
3. Will neuroticism predict sleep quality among postgraduate students?

## Purpose of the Study

Aloba, Adewuya, Ola and Mapayi (2007) acknowledged that the extent of sleep-related problems remains largely an unidentified public health issue, particularly so in Africa where there is inadequate personnel to assess sleep problem. Studies focusing research investigation on the variables under study are lacking. This study is therefore aimed at investigatingwhether trauma exposure,emotion regulation and neuroticismwill significantly predictsleep quality among postgraduate students.

## Operational Definition of Terms

Sleep Quality:Thisrefers to quantitative (i.e., sleep duration, sleep latency, sleep maintenance, number of arousals) and subjective aspects (i.e., depth, restfulness) of sleep and refreshment upon awakening as measured by Pittsburgh Sleep Quality Index (Buysse, Reynolds, Monk, Berman \& Kupfer, 1989).

Trauma Exposure:TraumaExposure is a traumatic event which includes individuals either experiencing or witnessing an event during which they experienced intense fear, helplessness, or horror, as measured by part A of Harvard Trauma Questionnaire (Mollica, Caspi-Yavin, Bollini, Truong, Tor \& Lavelle, 1992).

Emotion Regulation: This refers to the personô manner of influencing which emotions he/she has, when he/she has them, and how these emotions are experienced and expressed, as measured by Difficulties in Emotion Regulation Scale (Gratz \& Roemer, 2004).

Neuroticism:This is operationally defined by items referring to irritability, anger, sadness, anxiety, worry, hostility, self-consciousness and vulnerability that have been found to be substantially correlated with one another in factor analysis as measured by Neuroticism subscale of Big Five Inventory (John, Donahue \& Kentle, 1991).

Postgraduate students: This refers to students of the University of Nigeria, Nsukka who are enrolled in postgraduate diploma, masterốs degree and PhD programmes.

## CHAPTER TWO

## Literature Review

The review of relevant literature on trauma exposure, emotional regulation, and neuroticism was discussed under two perspectives- theoretical and empirical.

## Theoretical Review

The theoretical review in this study is as follows: The 3P (Predisposing, Precipitating, Perpetuating) model of sleep; Lundh and Broman̂̂ model of sleep, Cognitive model of sleep, Espieف̂ Psychological inhibition model of sleep; Stress response theory of trauma;The Emotion dysregulation model and Digmanô theory of Personality.

The 3Ps (predisposing, precipitating, perpetuating) model of sleep (Spielman, Caruso \& Glouinsky, 1987)

According to 3P model developed by Spielman, Caruso and Glouinsky (1987), poor sleep is often induced by stressful events by predisposing, precipitating and perpetuating factors. This model is widely used to explain the onset mechanism of poor sleep. It contends that factors leading to the onset and worsening of poor sleep are multidimensional in nature, and many life events and life stresses can result in poor sleep disturbance. Inadequate stress coping behaviour also precipitates poor sleep, and heightens uneasiness and tension around being unable to sleep, thereby perpetuating the sleeplessness (Abe \& Mishima, 2008). Spielmanand Colleagues (1987) incorporate the impact of various traits (predisposing factors) and life stresses (precipitating factors) in the development of poor sleep. It also recognizes that poor sleep is maintained (unintentionally) by maladaptive coping strategies (perpetuating factors). Thus, a person may be prone to sleep disturbance due to trait characteristics, may experience a trenchant or short-term poor sleep as a consequence of precipitating stresses, and may develop a persistent and chronic insomnia as a consequence of pathologic coping strategies and poor sleep quality. The 3P factors of sleep are as follows:

Predisposing factors of sleep: Psychiatric disorders may predispose to development of poor sleep. The disruption of sleep seen in association with depression, anxiety disorders, bipolar disorder, obsessive-compulsive disorder, or psychotic disorders may interact with the personality traits of these patients to increase anxiety and concern about sleep (Stein \& Mallman, 2005; Benka, 2005). Social factors may also be contributory to this process. Parental demands of caregiving for a spouse or parents, or arousal related to a bed-partner̂̂ sleep tendencies, schedule, or habits may all contribute to increased risk of developing poor sleep. Biological components may also play a role in precipitating component of poor sleep. The model of đ́yperarousalôcould be a precipitating component of poor sleep. It has also been speculated that
some patients may be congenitally or áonstitutionallyô poor sleepers, on the basis of reduced homeostatic (intrinsic) sleep drive. Patients with idiopathic poor sleep report such a history, complaining of onset of poor sleep in infancy or childhood without other cause, (International Classification of Sleep Disorders, Diagnostic and Coding Manual, 2005).

Precipitating factors of poor sleep: poor sleep can clearly be provoked in virtually everyone by the effects of stress. An example of a traumatic event that could be relatively universal to traumatic experience is flood which had destroyed properties and lives and which occurred in eleven states along riverine area in Nigeria between June and September 2012, (Mmon \& Aifesehi, 2013). Susceptible individuals have an increased risk for developing poor sleep quality, even in association with stimuli that would not be problematic for ñbetter sleepersò. These individuals are also at risk for the recurrence of poor sleep episodes whenever levels of stress are increased. A wide range of events and stimuli may precipitate poor sleep episode in susceptible individuals. Personal or family illness, death of a spouse, child, or parent; and separation, divorce, or đ́reakupôof a relationship are all obvious and significant interpersonal disturbances which may provoke poor sleep event. Social factors, such as conflict at work or school, loss of a job, or economic/financial problems such as debt or bankruptcy may be signal events for the development of poor sleep. Psychiatric and medical disorders may precipitate and predispose a person to poor sleep. Psychiatric issues such as anxiety, depression, bipolar disorder, and medical conditions- especially those causing pain, disturbed sleep, or central nervous system arousal- may provoke the onset of sleep disturbance. Examples of disorders that could precipitate poor sleep include arthritis flares, angina, prostatism, hyperthyroidism, and irritable bowel syndrome.

Perpetuating factors of sleep: As a consequence of the development of poor sleep, patients may make various changes in their habits and routines to try to compensate for their sleep loss and greater difficulties falling asleep. They may try to fall asleep at an earlier hour not appreciating that their level of alertness at that hour, controlled by their circadian sleep rhythms, will prevent premature entry into sleep. They will try harder to đ̛all asleep,ônot recognizing that sleep occurs when levels of alertness and arousal are diminished and that the effort to try to fall asleep raises levels of arousal and further interferes with the capacity to fall asleep. They may nap in the day time, not appreciating that their daytime hours of sleep will reduce sleep drive at bedtime, further interfering with their capacity to fall asleep at their desire hour. A state of conditioned arousal to the bed and bedroom may then develop. As sleep becomes more elusive, efforts to fall asleep may become more intensified, as is the level of arousal and anxiety, the continued failure to sleep generates. The bed and bedroom, which ideally are associated with comfort, pleasure, and relaxation, become increasingly associated with pain, arousal, and anxiety. Other efforts or strategies to promote sleep may ensue, further disrupting normal sleep patterns and habits. Alcohol or over- the counter sleep aids may be used. When they fail, or lead to side effects or residual morning sleepiness, sleep is further physiologically disrupted and the intensity of distress is increased (Gillin, Drummond, Clark \& Moore, 2005).

Thus, the main assumption of this model is that sleep disturbance may, over time, become dislocated from the precipitating trigger (Ebben \& Spielman, 2009). Such a model is intuitively appealing because it suggests that treatment should target, specifically, perpetuating factors involved in the maintenance of insomnia. Indeed, this is exactly what cognitive behavioural therapy for insomnia (CBT-I) attempts to do; with an emphasis on correcting
maladaptive coping strategies, behaviours and sleep-related dysfunctional beliefs and attitudes (Espie \& Kyle, 2009).

Spielman and Glovinsky (1991) provide a hypothetical example of the working model in a young man who has a tendency towards eveningness (i.e. exhibiting a preference for evening activities as opposed to morning activities). In the example the stressor is that the young man receives a job promotion that results in a higher level of scrutiny and provokes the development of an anxiety state, which pushes him over the threshold for insomnia. He tries to cope by recouping sleep at the weekend but worries about the impact of his working schedule on sleep loss. By the time the individual seeks treatment for the sleep problems, he has adapted to the increased work demands but still has problems sleeping, reflecting the chronic insomnia stage. The model has been tested to an extent, for example, in the case of 48 stress-induction studies within good sleepers, however a limitation is that it is difficult to test the model as no specific circumstances or characteristics are provided at each stage of the model (Ellis, Espie, Rieman \& Perlis, 2012; Perlis, Shaw, Cano \& Espie, 2011).

## Lundh and Broman model of sleep (2000)

Lundh and Bromanô (2000) model explains how óleep-interferingô and óleep interpretingôprocesses combine to produce poor sleep. Sleep-interfering processes refer to those that create physiological, emotional or cognitive arousal. Within the model, physiological and psychological arousal are both potential causes of poor sleep and Lundh and Broman suggest that in order to understand the cause of poor sleep, the causes of arousal must also be understood. Within the framework of the model, the causes of arousal may result from interactions between a particular event and individual factors. These may include basic arousal levels, árousibilityô
shown in response to the arousing event and slow levels of habituation, referring to the return to the basic arousal level following the event. The second part of the model focuses on intraindividual differences in sleep-interpreting processes, referring to the psychological processes involved in each individualô personal definition of poor sleep in relation to their current state. This integrative model also takes into account psychological factors where individuals may have a predisposition to both the sleep-interfering processes and the sleep-interpreting processes. These would include having higher basal levels of arousal and arousability, which can include personality traits such as having a high level of emotional sensitivity, being easily hurt and having a slow recovery from stress. Other psychological variables could include having a personal disposition to worry, experiencing emotional conflicts in personal relationships and emotional involvement in the problems of others.

Lundh and Bromanôs model involves four categories of vulnerability factors for sleepinterfering arousal processes (arousability, stimulus-arousal associations, behavioural and cognitive strategies with regard to the sleep situation and emotional aspects of interpersonal relations) and three categories of vulnerability for sleep interfering processes (high personal standards, dysfunctional beliefs, and attributions). The fact that this model does take into account intra-individual differences in baseline arousal levels, intra-individual differences in stress responses and intra-individual levels in habituation is a particular strength of the model (Ellis, Espie, Rieman \& Perlis, 2012).

Support for the model comes from a study that assessed whether perfectionism was related to pre-existing poor sleep and future poor sleep, at a baseline and at a one-year follow-up time point (Jansson-Fröjmark \& Linton, 2007) and an association was shown between perfectionism and insomnia, although this association was weak. Those with poor sleep showed
high levels of neuroticism and displayed traits related to perfectionism. These include where individuals are over-concerned (lacking- in- self-confidence and where they have greater doubts about action). However, the role of personality within insomnia is complex and longitudinal studies are needed (Van de Laar, Verbeek, Pevernagie, Aldenkamo \& Overeem, 2010). The main drawback of this model is that it does not specifically explain how the sleep-interfering processes and sleep-interpreting processes interact with each other. A further drawback is that the model cannot be used to determine a causal relationship between these two processes and requires further detail and development (Ellis, et al., 2012; Lundh \& Broman, 2000).

## Cognitive model of sleep (Harvey, 2002)

The cognitive model of sleep proposed by Harvey (2002) is based on the notion that negative thoughts, beliefs, and attitudes about sleep and the consequences of poor sleep lead to insomnia by provoking cognitive arousal and subsequent maladaptive sleep behaviors.Cognitive theory of sleep posits to linked groups of cognitions accounting for the disorder. One group relates to the individualsô beliefs surrounding poor sleep, the other relates to cognitions like worry and ruminating thoughts. According to the conceptualization put forward in the model, poor sleep is maintained by a flow of cognitive processes that are activated at night and during the day. The cognitive model posits that worry activates the sympathetic nervous system (the đ̛́ight or flight responseô thereby triggering physiological arousal, and distress. This combination of worry, arousal, and distress plunges the individual into an anxiety state, a state which results in difficulty falling asleep and maintaining sleep (Espie, 2002). Unhelpful beliefs regarding sleep may increase the potential for worry, for instance, if an individual believes one needs more than eight (8) hours of unbroken sleep each and every night to function adequately
during the day, it is likely that the individual will worry about daytime functioning (because most people find that getting eight hours of unbroken sleep is impossible to achieve).

Harvey (2002) describes a (maintenance) model of sleep which focuses primarily on dysfunctional cognitive processes, based upon a large body of work from the anxiety disorders literature. Because it is arguably the first model to give equal attention to both daytime and night-time factors, it is worth outlining some of its main features. Harvey argues that individuals with poor sleep tend to excessively worry about sleep and catastrophize about the consequences of not getting adequate sleep (in relation to impact on health and daytime functioning). Ensuing negatively toned cognitive activity, typically about sleep but also other (negatively valence) life issues, coupled with the application of maladaptive safety behaviours, results in elevated autonomic arousal and emotional distress. As a consequence of this heightened stress state, individuals with poor sleep tend to monitor for sleep-related threat cues (internal and external) to confirm that they have not slept and that functioning is adversely affected. Pre-existing dysfunctional beliefs exacerbate the situation. Otherwise innocuous cues are subsequently misinterpreted as evidence for sleep and daytime deficits, árickingô the individual into overestimating both the level of sleep and daytime impairment. This serves to cause further worry and concern about not sleeping, which may, through feedback mechanisms, increase anxiety and cognitive load, leading to the enhanced possibility of a ớealôdeficit occurring in both sleep and daytime functioning. The main assumption of this model is that cognitive processes have a causal role in the maintenance of an insomnia state. However, Harvey does acknowledge that precipitants to insomnia may include stressors such as life stress, an accident, or illness.

A central tenet of cognitive accounts of poor sleep is that individuals suffering from sleep disturbances tend to adopt counterproductive ©́afety behaviorsô in an attempt to avoid unwanted intrusive thoughts at bedtime (Espie, Broomfield, MacMahon, Macphee \& Taylor, 2006; Harvey, 2002). In support of this contention, self-report studies have revealed that insomniacs rely on thought-control strategies such as aggressive suppression and worry more often than good sleepers do, and that the use of these techniques predicts poorer sleep (Ellis \& Cropley, 2002; Harvey, 2001; Ree,Harvey, Blake, Tang \& Shawe-Taylor,2005; Schmidt, Gay, Van der Linden, 2009). Further evidence for sleep-interfering effects of certain thought-control strategies has come from experimental studies. For instance, thought suppression has been shown to entail a rebound of target-thought frequency at sleep onset (Schmidt \& Gendolla, 2008), to delay the moment of falling asleep and to worsen sleep quality (Harvey, 2003). In a similar vein, experimental induction of worrisome cognitive activity in the presleep period, for example by telling participants that they would have to give a speech after sleep, has generally been found to increase sleep-onset latency (Tang \& Harvey, 2004). Given that ñthe literature has focused primarily on presumed factors maintaining insomniaò (Espie, 2002, p. 219), comparatively little is known about predisposing factors and their associations with precipitating and perpetuating factors. Regarding the potentially predisposing role of personality traits, associations have been found, for example, between poor sleep and anxiety sensitivity (Vincent \& Walker, 2001), internalization of psychological disturbances and neuroticism (Morgan, Healey \& Healey, 1989) Despite the clinically observed comorbidity between sleep disturbances and impulsivity-related disorders such as attention deficit hyperactivity disorder (ADHD), (Boonstra,Kooij, Oosterlaan,Sergeant, Buitelaar\& Van Someren, 2007) and borderline personality disorder (BPD), (Bastien, Guimond, St-Jean \&

Lemelin,2008), the precise relations between the personality construct of neuroticism and poor sleep has yet remained largely unexplored.

## Espie's psychobiological inhibition model of sleep

The psychobiological inhibition model of sleep (Espie, 2003) focuses on the idea that poor sleep is a failure of automatic sleep activation and of sleep maintenance. In the psychobiological inhibition model, good sleep is the default state. Under normal circumstances both homeostatic and circadian processes are set to have sleep as the default setting rather than insomnia. The psychobiological inhibition model focuses on normalcy rather than on the pathology of poor sleep (Espie, Broomfield, MacMahon, Macphee \& Taylor, 2006). The model is framed as being a neurobehavioural, biological model, where good sleep has functional properties of both ̣́qlasticity ónd óautomaticityô Plasticity refers to the capability of the system to react to stressors (situational and personal factors) where the sleep-wake system can tolerate and minimise the effects of night-to-night variability in sleep patterns (Espie, 2002). Automaticity refers to the involuntary nature of a well-adjusted sleep schedule, to habitual associations forming part of a stimulus control paradigm and to the implicit expectations a good sleeper has regarding sleep continuity and quality (Espie, 2002), where this refers to the involuntary regulation of sleep under normal circumstances (Ellis, Espie, Rieman \& Perlis, 2012). Automaticity can be disrupted through selective attention to sleep, the explicit intention to sleep and also through the introduction of effort in the sleep process, which Espie terms the áttention-intention-effortô(A-I-E) pathway (Espie, et al., 2006). At the core of the model is an involuntary process of interaction between the sleep homeostatic and the circadian timer, associated with the self-perception of good sleep quality. The role of plasticity and automaticity is to defend this core. Espie (2002) notes that both endogenous (internal cues) sleep-related cues
such as physical and mental fatigue interact with exogenous, (external cues) in the environment. Essentially, good sleepers are passive as both these internal and external cues allow conditions for sleep to be set automatically and without effort.

In the model, the defensive properties of the plasticity and automaticity are maintained by four sub-systems that interact with one other, which are a mixture of behavioural, cognitive and biological processes. The subsystems are sleep-stimulus control, physiological de-arousal, cognitive de-arousal, and daytime facilitation. These interactions maintain sleep homeostasis, circadian timing and sleep quality, through action and interaction among these subsystems. A good sleeper is able to accurately interpret both physiological and mental signs of readiness for sleep and the stimulus environment of the bedroom reinforces de-arousal, thus affording the optimal circumstances for sleep to be initiated. Both the process of physiological and cognitive de-arousal is assumed to occur simultaneously in the model in a parallel manner. The model states that dysregulation would occur as a result of strong negative or positive emotions, which cause arousal. Whilst the focus of this model is on the inhibition of normal sleep, it does acknowledge poor sleep as mediating from the activation of the nervous system, psychological or environmental arousal (Espie, 2002).

However, the model does not provide information with regards to the timing and does not discuss circumstances in which an individual would recover from the stress or would continue towards the AI-E aspect of the model (Ellis, et al., 2012). Support for the model comes from the fact that evidence for selective attention to salient cues (i.e. the áttentional biasồ is strong enough to suggest it has a casual role in the majority of anxiety disorders and that the experimental evidence is strong when applied to sleep (MacMahon, Broomfield\& Espie, 2006; Spiegelhalder, Espie, Nissen \& Riemann, 2008; Woods, Marchetti, Biello \& Espie, 2009).

However, inconsistencies have been shown in attentional bias studies even when using the same Emotional Stroop Task, although this may be potentially explained by poor sleepers showing an attentional bias towards sleep-related cues and also showing an increased monitoring or avoidance for anxiety-provoking cues. Limitations include the fact that most of the model and of the A-I-E pathway component remains to be validated (Perlis, Shaw, Cano \& Espie, 2011).

## Stress response theory of trauma (Horowitz, 1976, 1986)

Horowitz, is a pioneer in the PTSD field due to his long-standing interest in the processing of thoughts, images, and moods related to loss and trauma. His theory has roots in psychodynamically informed observations of normal and abnormal bereavement reactions, and in a long tradition emphasizing peopleô development of individual assumptive worlds. Horowitz argued that when faced with trauma, people $\hat{\alpha}$ initial response is outcry at the realization of the trauma. A second response is to try to assimilate the new trauma information with prior knowledge. At this point, many individuals experience a period of information overload during which they are unable to match their thoughts and memories of the trauma with the way that they represented meaning before the trauma. In response to this tension, psychological defense mechanisms are brought into play to avoid memories of the trauma and pace the extent to which it is recalled. For example, the individual may be in denial about the trauma, feel numb, or avoid reminders of it.

However, the fundamental psychological need to reconcile new and old information means that trauma memories will actively break into consciousness in the form of intrusions, flashbacks, and nightmares. These consciously experienced trauma memories provide the individual with an opportunity to try to reconcile them with pre-trauma representations. (Brewin
\& Holmes, 2003). It becomes apparent that, according to Horowitz, there are now two opposing processes at work: One to defend the individual by the suppression of trauma information and the other one to promote the working through of the traumatic material by bringing it to mind. Therefore, the individual oscillates between avoidance and intrusions of the trauma. This oscillation allows the traumatic information to be worked through, and as this happens, the intensity of each phase decreases. In particular, longer term structures in memory representing the self or future goals can be adjusted so that they are consistent with the new data, at which point, trauma processing is considered to be completed. Failure to process the trauma information is proposed to lead to persistent posttraumatic reactions as the information remains in active memory and continues to intrude and be avoided.

Horowitz $\hat{Q}$ work contains numerous important observations and has rightly been very influential. In particular, he was one of the first theorists to emphasize the impact of trauma on wider beliefs about the self, the world, and the future and to consider how recovery might involve far-reaching cognitive change. Recognizing this broader perspective and its ability to explain the breadth of beliefs and emotions encountered in PTSD, his theory was described as ósocial-cognitive $\hat{\propto} \hat{\text { and }}$ Brewin, Dalgleish \& Joseph, 1996). Furthermore, areas not treated in any depth by the stress response theory include the difference between flashbacks and ordinary memories of trauma, individual variations in trauma response, peri-traumatic reactions, the role of environmental factors such as trauma cues and social support, and how to distinguish remission of symptoms due to successful recovery from remission due to successful avoidance (Litz, 1992).

## Emotion Dysregulation Model (Mennin, Heimberg, Turk \& Fresco, 2005)

Problems in regulating emotions has been proposed as a central feature in anxiety disorder. Adults with anxiety have difficulty understanding emotional experiences and have little skill or ability to modulate their intense emotions (Mennin, 2006). Not only do they experience more intense emotions than their peers, they are more negative, are less able to calm themselves, and have more physiological symptoms after an anxiety producing experience (Mennin, Heimberg, Turk, and Fresco 2005). Anxious ambivalent adults have difficulty regulating emotions so that their arousal is too intense for the situation, and they both misidentify and misdirect their emotions. Adults with anxiety disorders have difficulty handling worries, sadness, and anger. This may be due to the intense degree to which they experience negative emotions. They generally have little confidence in their ability to deal with intensely aroused negative emotions (Suveg \& Zeman, 2004).

The emotion dysregulation model states that emotional dysfunction may be categorized by four main dysregulation processes: (1) heightened emotional intensity, (2) poor emotional understanding,(3) negative reactivity to emotions (e.g., fear of emotions), and (4) maladaptive management attempts. Regarding the first process, the model proposes that individuals with anxiety disorder experience their emotions more easily, quickly, and intensely than those without anxiety disorder, and that this can also lead to inappropriate emotional expression (such as over-expression of negative affect). Secondly, these individuals may experience difficulty differentiating between primary emotions, and therefore cannot use important affective information due to its overwhelming nature. According to the functionalist perspective within the emotion literature (e.g., Davison, 1965; Frijda, 1986), this can interfere with the adaptive information provided by emotional experiences, including goals and action tendencies. The model then proposes that difficulties associated with the first two processes can result in
emotions being perceived as more threatening. The combination of components one and two is hypothesized to lead to the third component, which stipulates that individuals will become overwhelmed, anxious, or uncomfortable when strong emotions occur, thereby creating a feedback loop. Individuals with anxiety disorder are also hypothesized to show extreme hypervigilance for threatening information and increased attention either toward or away from emotions and pertinent negative beliefs Finally, this sequence culminates in the fourth component, which specifies that individuals with anxiety disorder make unsuccessful or maladaptive attempts to either minimize emotions or over-control emotions, or inappropriately express emotional arousal (e.g., excessive worry, suppression of emotions, emotional outbursts). As such, worry plays a fundamental role in this model as an ineffective strategy to cope with emotions. According to Mennin and colleagues, (2005), however, this succession of events can also proceed in the opposite direction (i.e., maladaptive emotion regulation strategies leading to increased negative emotion), thereby giving rise to a bi-directional cycle of emotion dysregulation and negative affect.

Empirical studies support the notion that individuals with anxiety disorder experience negative but not positive emotions more intensely than do healthy controls (Mennin, et al., 2005; Turk, Heimberg, Luterek, Mennin \& Fresco, 2005; Salters-Pedneault, Roemer, Tull, Rucker \& Mennin, 2006;) and those with other psychopathology including depression (Mennin, Holaway, Fresco, Moore \& Heimberg, 2007) and social anxiety disorder (Mennin, et al., 2007; Turk, et al., 2005). In addition, prior research suggests that the undergraduates with anxiety disorder have increased difficulty identifying, describing, and understanding their emotions compared to healthy undergraduates (Mennin, et al., 2005; 2007). Evidence also supports the notion that individuals with anxiety disorder exhibit increased fear of intense emotions compared to healthy
controls (Mennin, et al., 2005; Turk, et al., 2005 Salters-Pedneault, et al., 2006). Finally, results suggest that individuals with anxiety disorder engage in more emotional coping strategies (i.e., excessive worry, emotional outbursts, emotional suppression) compared to healthy controls (Mennin, et al., 2007) and individuals with other psychopathology including depression and social anxiety (Mennin, et al., 2007). The emotion dysregulation model posits that individuals with anxiety disorder may demonstrate under control (e.g., inappropriate expression) negative affective states, over control (e.g., avoidance or suppression) of those states, or a combination thereof.

## Digman's theory of personality (1990)

Contemporary psychological researchers have managed to come up with a more comprehensive and empirical model of analysing personality trait known as áhe big five theoryô otherwise known as the five factor theory (Digman, 1990). The five factor theory is a theory with broad characteristics associated with broad categories of personality traits, each category having its distinct behavioural characteristics associated with them. The óbig fiveô is a model of five factors including: neuroticism, agreeableness, extraversion-introversion, openness, and conscientiousness (Ewen, 1998). The researcher will only concentrate on the main variable personality trait which is neuroticism.

Neuroticism has an inherent negative denotation (Bradshaw, 1997) although sometimes reversed and called Emotional stability, an enduring tendency to experience negative emotional states and such feelings such as anxiety, anger, guilt, and depressed mood (Matthew \& Deary, 1998). Similarly, Goleman (1997) found that neurotic individuals respond more poorly to environmental stress, are more likely to interpret ordinary situations as threatening and minor
frustrations as hopelessly difficult. They are often self-conscious and shy, and they may have trouble controlling urges and delaying gratification. Hettema, Meale, Myers, Prescott and Kendler (2006), maintained that Neuroticism is associated with low emotional intelligence, which involves emotion regulation, motivation, and interpersonal skills. It is also a risk factor for ñinternalizingò mental disorders such as phobia, depression, panic disorder, and other anxiety disorders traditionally called neuroses. High scores on this dimension are indicative of emotional liability and over reactivity. High scoring individuals tends to be emotionally over responsive and to have difficulties in returning to a normal state of affair after emotional experience (Eysenck \& Eysenck, 1968). This dimension is sometimes referred to as stable - unstable. Howard \& Howard (1998) were of the view that the people with a tendency towards neuroticism are more worried, temperamental and prone to sadness. Boeree (2005) said that the name óneuroticismô does not refer to any psychiatric defect, McCrae and John (1992) argued that a more proper term could be negative affectivity or nervousness. Individuals who are high in neuroticism may show more emotional reactions whenever confronted with stressful situations (Van Heck, 1997).

Moreover, they seem to use avoiding and distracting coping strategies, such as denying, wishing, thinking, and self-criticism, rather than more approaching strategies (Bolger, 1990; Heppner, Cook, Wright \& Johnson, 1995). Ineffective coping with stressful situations in the work environment makes individuals who are high in neuroticism more vulnerable to the symptoms that are typically associated with burnout (Bakker, Van der Zee, Lewig \& Dollard, 2006). These problems in emotional regulation can diminish the ability of a person scoring high on neuroticism to think clearly, make decisions, and cope effectively with stress. Moreover, individuals high on neuroticism tend to experience more negative life event, (Jeronimus, Ormel,

Aleman, Pennix \& Riese, 2013; Jeronimus, Riese, Sanderman \& Ormel, 2014). At the other end of the scale, individuals who score low in neuroticism are less easily upset and are less emotionally reactive and they tend to be calm, emotionally stable, and free from persistent negative feelings and also freedom from negative feeling does not mean that low scorers experience a lot of positive feelings (Doland, Garcia, Cabezas \& Tzafrir, 2008).

## Empirical Review

The review of relevant empirical studies is organized under three sub headings: trauma exposure and sleep quality, emotion regulation and sleep quality; and neuroticism and sleep quality.

## Trauma Exposure and Sleep Quality

Hauff (2015) examined the relationships among cumulative trauma, posttraumatic stress, and sleep in African-American women and perinatal outcomes. A sample of 150 essentially healthy African-American women who had given birth were recruited at Hutzel Women's Hospital and St. John Providence Hospital Southfield, and asked to complete the Cumulative Trauma Scale, Community Safety Questionnaire, Conflict Tactics Scale, Clinician-administered PTSD Scale, Pittsburgh Sleep Quality Index, Edinburgh Depression Inventory, and Demographic Data Form. The prenatal and hospital medical records were reviewed for multiple maternal and neonatal outcomes. Regression analysis was used to determine if relationships between the variables exist, and the strength of those relationships. The findings of this study showed: (1) African- American women experience traumatic events. (2) Cumulative trauma is associated poor sleep quality. It was suggested that more research is needed to better understand mothers' use of over the counter and other medications to help them sleep.

Swanson, Hamilton and Muzik (2014) examined history of childhood trauma and sleep complaints in postpartum women. Participants $(N=173)$ completed questionnaires by telephone at 4-months postpartum. After adjusting for nuisance variables, there were significantly higher rates of sleep disturbance (falling asleep and staying asleep) for women with a past history of neglect $(O R=4.84, p=.036$ and $5.78, p=.006$, respectively $)$ physical abuse $(O R=9.20, p=.002$ and $3.84, p=.044$, respectively), and physical abuse with sexual abuse $(O R=5.95, p=.011$ and $3.56, p=.045$, respectively). Current PTSD was significantly associated with trouble staying asleep $(O R=4.21, p=.032)$ whereas recovery from PTSD was associated with trouble falling $(O R=4.19, p=.015)$ and staying asleep ( $O R=3.69, p=.011$ ). Findings affirm the contribution of trauma exposure to postpartum sleep.

Tempesta, Curcio, De Gennaroand Ferrara (2013) investigated the impact of the 6.3 magnitude 2009 LôAquila (Italy) earthquake on standardized self-report measures of sleep quality (Pittsburgh Sleep Quality Index, PSQI) and frequency of disruptive nocturnal behaviours (Pittsburgh Sleep Quality Index-Addendum, PSQI-A) two years after the natural disaster. Selfreported sleep quality was assessed in 665 LôAquila citizens exposed to the earthquake compared with a different sample ( $\mathrm{n} \$ 54$ ) of L'Aquila citizens tested 24 months before the earthquake. In addition, sleep quality and disruptive nocturnal behaviours (DNB) of people exposed to the traumatic experience were compared with people that in the same period lived in different areas ranging between 40 and 115 km from the earthquake epicenter ( $\mathrm{n} \sqrt{2} 74$ ). The comparison between LôAquila citizens before and after the earthquake showed a significant deterioration of sleep quality after the exposure to the trauma. In addition, two years after the earthquake L'Aquila citizens showed the highest PSQI scores and the highest incidence of DNB compared to subjects living in the surroundings. Interestingly, above-the-threshold PSQI scores were found in
the participants living within 70 km from the epicenter, while trauma-related DNBs were found in people living in a range of 40 km . Multiple regressions confirmed that proximity to the epicenter is predictive of sleep disturbances and DNB, also suggesting a possible mediating effect of depression on PSQI scores.

Brown, Mellman, Alfano and Weems (2011) studied the role of posttraumatic stress symptoms in maintenance of sleep problems in youths who experienced Hurricane Katrina. The sample consisted of 191 children and adolescents in New Orleans who had complete data for Time 1 (24 months post-Katrina) and Time 2 ( 30 months post-Katrina). The children were between 8 and 15 years old, mostly African American, with boys accounting for 55\% of the sample. The youths came from a neighbourhood that had encountered massive damage. For example, many children reported having thought that someone might die in the hurricane, that their homes were badly damaged, that they witnessed someone else being hurt, or that they had a pet hurt or die. The study used self-report questionnaires. The children filled out the Posttraumatic Stress Reaction Index for children and a measure to assess the exposure to the hurricane and its aftermath. Sleep disturbance and fear of sleeping alone were assessed via two items of the Revised Child Anxiety and Depression Scales: $\tilde{n} H a v i n g ~ t r o u b l e ~ s l e e p i n g o ̀ ~ a n d ~ \tilde{n}$ feel scared if I have to sleep on my ownò. The analyses consisted of $t$ - tests and multivariate linear regression models. In their findings, two fifths of the sample had moderate to very severe symptoms of posttraumatic stress. An important number of children reported sleep disturbance ( $46 \%$ and $50 \%$ at T 1 and T 2 respectively), while a somewhat smaller number reported fear of sleeping alone ( $25 \%$ and $16 \%$ ). Children with sleep disturbance or fear of sleeping alone reported more posttraumatic stress symptoms than children who did not report sleep problems. General sleep disturbance at T 1 predicted posttraumatic stress at T 2 , also after controlling for
age, gender, continued home damage, and posttraumatic stress at T1.The researchers suggest that sleep disturbances are common in the aftermath of childhood trauma and persist for an important number of children.

Llabre and Hadi (2009) investigated war-related exposure and psychological distress as predictors of health and sleep in Kuwaiti children. A sample of 120 preadolescent Kuwaiti boys and girls (9-12 years old) who were initially assessed for exposure to war-related trauma during the Iraqi occupation and Gulf war of 1990 were reassessed for general health and sleep, using the Pittsburgh Sleep Quality Index (PSQI), approximately 10 years later. Findings showed direct effects between exposure to war related trauma and sleep difficulties, even after controlling for stressful life events experienced in the interim.

Trauma from sexual abuse can cause long-term effects, including sleep disturbances and nightmares.Hence, Noll, Trickett, Susman and Putnam (2006) tested the role of childhood traumatic event exposure predicting subsequent sleep problems. Seventy-eight sexually abused females were compared on measures of sleep, PTSD, depression, and re-victimization, to 69 agematched females without a history of childhood sexual abuse. Sexually abused females were identified, and information regarding the abuse and psychological symptom levels (i.e., sleep problems, PTSD) were obtained through protective service agencies of Washington D.C. All females were contacted 10 years post-disclosure of the abuse, and completed an interview including a PTSD diagnostic interview, and measures of sleep and re-victimization. Participants in the comparison group also completed the interviews and measures. Results indicated sexually abused females (who reported elevated re-experiencing, arousal, and avoidance symptoms of PTSD, and greater levels of re-victimization relative to control participants) experienced significantly greater sleep problems compared to non-traumatic event-exposed females. Results
indicated childhood sexual abuse was a unique predictor of sleep problems 10 years posttraumatic event, even after controlling for symptoms of depression and PTSD.

Kaminer and Lavie (1991) also examined the role of surviving the Holocaust on sleep. Three groups were created as follows: (1) well-adjusted survivors of the Holocaust ( $n=12$ ), (2) poorly adjusted survivors of the Holocaust $(n=11)$, and (3) a control group present during the Holocaust but not involved in concentration camps ( $n=10$ ). All participants underwent four nights of polysomnography (a study done while an individual is fully asleep). Results indicated poorly adjusted survivors experienced longer latency to sleep onset and lower sleep efficiency in comparison to well-adjusted survivors and controls.

Rosen, Reynolds, Yeager, Houck and Hurwitz (1991) compared the sleep of Holocaust survivors ( $n=42$ ) to a control group of healthy elderly individuals ( $n=54$ ), and a group of depressed elderly individuals $(n=37)$. Participants completed questionnaires and a structured clinical interview pertaining to sleep. Holocaust survivors also completed specific information about their Holocaust experience (e.g., time spent in concentration camps). Results indicated Holocaust survivors reported significantly greater sleep problems than healthy control participants. Furthermore, there was a significant positive correlation between the number of years spent in the concentration camp and the total number of sleep problems. Results suggest survivors of the Holocaust experienced significantly more sleep problems compared to healthy controls and depressed controls even 45 years after the traumatic event(s) occurred. Furthermore, those who spent more time in the concentration camps experienced greater levels of sleep problems defined as lesser self-reported quality of sleep.

Following a playground sniper attack in Los Angeles, Nader, Pynoos, Fairbanks and Frederick (1990) examined Children $\hat{Q}$ PTSD reactions and sleep disturbance one year after a
sniper attack at their school and found that $77.1 \%$ of children on the playground reported sleep disturbances one month following the trauma. Nineteen children who were on the playground were followed longitudinally. At 14-month follow-up, $57.9 \%$ of this sample still reported sleep disturbances.

Hefez, Metz and Lavie (1987) examined the role of different types of traumatic event exposure on sleep problems. A total of 11 traumatic event survivors (5 Holocaust survivors, 3 combat veterans, and 3 sea disaster survivors) and 9 age- and gender-matched controls without a history of traumatic event exposure participated. Holocaust survivors were assessed 45 years post-traumatic event, combat veterans were assessed either 6 or 14 years post-traumatic event, and survivors of the sea disaster were assessed 6 and 12 months post-traumatic event. Polysomnography was conducted over two to five consecutive nights. All traumatic eventexposed participants had lower sleep efficiency, increased sleep latency, shorter REM time, and longer REM latency in comparison to control participants. Interestingly, time since traumatic event exposure was related to sleep problems. Within 18 months post-traumatic event, participants displayed increased REM motor activity, fragmented REM, and increased awakenings. In comparison, those assessed multiple years after traumatic event exposure did not display these characteristics; however they did demonstrate shorter REM time and increased latency to REM. These results suggest the effects of traumatic event exposure on sleep architecture may depend, at least to a certain degree, on time since traumatic event exposure.

## Emotion Regulation and Sleep Quality

Tsypes, Aldao and Mennin (2015) assessed emotion regulation as a potential contributor to sleep problems in generalized anxiety disorder patients (GAD). Participants in the study
comprised two groups, fifty-nine (59) individuals diagnosed with GAD and sixty-six (66) healthy controls. They were assessed for the presence of mood and anxiety disorders and then completed self-report questionnaires assessing problems with sleep and emotion regulation difficulties. Participants in the GAD group scored significantly higher on a number of sleep outcomes than did the control group. Importantly, emotion dysregulation statistically mediated the relationship between GAD and a wide range of outcomes of sleep dysfunction independently of the effects of depression and secondary anxiety diagnoses. Emotion dysregulation that characterize GAD mediate the relationship between symptoms of this disorder and a wide range of sleep problems.

Pickett, Barbaro and Mello (2015)examined the relationship between sleep disturbance, poor sleep quality, and emotion regulationdifficulties. In a sample of college students reporting exposure to at least 1 traumatic event, online surveymethodology was used to assess PTSD symptom severity (PTSS), sleep disturbances, including PTSD-specificsleep disturbances, and emotion regulation difficulties. After controlling for PTSS, sleepdisturbance and poor sleep quality domains were related to both global and specific difficulties inemotion regulation domains.

Sandru and Voinescu (2014) explored the links between sleep quality and dysfunctional beliefs, on one side, and emotion regulation on the other hand. The researchers also examined whether poor sleepers have more difficulties in regulating their emotions compared to good sleepers. One hundred and thirty-three adults, aged between 19 and $63(\mathrm{M}=26.14, \mathrm{SD}=7.61)$ years, recruited from the general community, completed a battery of scales hosted online. Their results indicate that individuals complaining of poorer sleep have problems in accepting their own emotions and engaging in goal-directed behaviours when they experience negative
emotions. They also show greater difficulties in controlling their impulse when experiencing negative emotions and in understanding their own emotions. Sleep quality was significantly, negatively and moderately linked with difficulties in emotion regulation.

Ođ̃eary, Bylsma and Rottenberg (2014) conducted a study to examined whether emotion regulation mediates the link between poor sleep quality and depression symptoms, using one hundred and forty-three(143) community-recruited participants which met screening criteria for major depressive disorder (MDD), remitted MDD, and healthy controls. Participants completed self-report scales of depression, sleep quality, and emotion regulation; ninety-five returned after six months to complete depression follow-up scales. Two mediation models were tested: the first utilized a cross-sectional design; the second utilized a longitudinal design (predicting depression at 6 months). Three emotion regulatory scales were tested: difficulties in emotion regulation (DERS; Gratz \& Roemer, 2004), worry (PSWQ; Meyer, Miller, Metzger \& Borkovec, 1994), and rumination (RSQ; Butler \& Nolen-Hoeksema, 1994) were used in the study. The indirect effect for all models was tested using bootstrapping.

For the cross-sectional model, all three emotion regulatory scales (DERS; PSWQ; RSQ) significantly mediated the relationship between sleep quality and depression. Each ER mediator was significant and accounted for $43 \%, 29 \%$, and $39 \%$ of the variance in the relationship, accordingly. In the longitudinal model, two emotion regulatory scales (DERS; PSWQ) significantly mediated the relationship between sleep quality and six month depression, accounting for $27 \%$ and $13 \%$ of the variance in the relationship, respectively. The longitudinal model, however, did not hold when controlling for Time 1 depression symptoms. This study was the first to examine emotion-based pathways by which sleep relates to depression. Their longitudinal results largely replicated cross-sectional models by showing that ER scales
accounted for a large portion of the variance within the sleep-depression relationship. Sleep disturbance is a clinically relevant target that may produce depression symptoms via degradation of emotion regulation.

Mauss, Troy and LeBourgeois (2013) examined the relationship between sleep quality and the ability to implement a type of emotion regulation that has particularly important implications for psychological health: cognitive reappraisal. One hundred and fifty- six (156) participants ( 86 male) reported on their past week's sleep quality. Their ability to implement cognitive reappraisal (CRA) was then measured with a standardized laboratory challenge. Participants with poorer self-reported sleep quality exhibited lower CRA, even after controlling for fourteen potential key confounds (e.g., age, negative affect, mood disorder symptoms, stress). This finding is consistent with the idea that poorer sleep quality impairs individuals' ability to engage in the crucial task of regulating negative emotions.

Markarian, Pickett, Daveson and Kanona (2013) investigated the relationship between emotion regulation (ER), and Sleep Quality (SQ) in an undergraduate sample ( $n=459$ ). Analyses revealed differential relationships between emotion regulation difficulties and stress symptoms among good quality and poor quality sleepers. The findings were discussed within the context of personality dimensions and self-regulatory mechanisms, along with implications for the treatment of anxiety and sleep difficulties. The findings suggest that emotion regulation difficulties has been linked to sleep quality and psychopathology.

Another finding on how emotions affect sleep was reported by Talbot, Stone, Gruber, Hairston, Eidelman and Harvey (2011). Participants completed seven consecutive days of sleep diary and mood measurements. Negative evening mood was associated with subsequent increased wake time for participants with insomnia and participants with bipolar disorder, while
positive evening mood was associated with subsequent increased wake time only for participants with insomnia. The finding concerning positive evening mood is at odds with findings presented by Talbot, Hairston, Eidelman, Gruber and Harvey (2009) in which a ñhappy mood inductionò resulted in longer sleep on latency in a group with bipolar disorder, this compared to a control group and baseline measurements. Talbot and Colleagues (2011) suggest that the more naturalistic aspects of the later study may have contributed to less intense positive mood, compared to the positive mood induction, and thereby a lower likelihood of affecting the inter episode bipolar participants' wake time.

Baglioni, Spiegelhalder, Lombardo and Riemann (2010) in their review focused entirely on sleep and emotions and tried to summarize the field of research within this area with a focus on emotional reactivity. Based on their literature overview, they concluded that there is empirical evidence that dysfunctional emotional reactivity may mediate an interaction between cognitive and automatic hyperarousal and that this maintains insomnia. Furthermore they suggest that dysfunctions in neural circuitries regulating the sleep-wake system are able to reinforce emotional disturbances with subsequent emotional dysregulation. It is plausible that this emotional dysregulation modulates the associations between insomnia and depression and/or anxiety.

Brummet, Babyak, Siegler, Vitaliano, Ballard, Gwyther and Williams (2006) found that negative affect possibly mediates the association between having a role as a caregiver (e.g. taking care of a relative or a spouse) and poor sleep quality, compared to not being a caregiver. The authors emphasizes that there may be an inverse association between negative affect and social support that may partially explain this mediating role of negative affect.

## Neuroticism and sleep quality

Duggan, Friedman, McDevitt and Mednick (2014) examined Big Five personality traits and a range of factors related to sleep health in 436 university students ( $M_{\text {age }} .88, S D=0$, $50 \%$ Male). Valid self-report measures of personality, sleep hygiene, sleep quality, and sleepiness were analyzed. To remove multicollinearity between personality factors, each sleep domain was regressed on relevant demographic and principal component-derived personality factors in multiple line regressions. Results showed that high neuroticism was the best predictor of poor sleep. Neuroticism was found to relate to insomnia severity through the mediating effects of sleep-related cognitive distortions, pre-sleep arousal, and to a much lesser degree negative affect. The results further support the notion of neuroticism being a predisposing factor for insomnia.

Calkins, Hearon, Capozzoli and Otto (2013) examined the relationship of neuroticism and sleep disturbance. They completed an Internet survey of 149 undergraduate student participants, a population with elevated risk for disturbed sleep. Participants completed a demographics questionnaire, the NEO Five-Factor Inventory, and the Pittsburgh Sleep Quality Index (PSQI). Results revealed a significant association between PSQI total score and neuroticism. In a stepwise regression, neuroticism was the statistically most important predictor of sleep disturbance. Their findings highlight the continued value of higher order concepts like neuroticism in the development of disorder-specific measures as well as indicate that distress in response to cognitive symptoms may play a role in maintaining sleep dysfunction.

Ramsawh, Ancoli-Israel, Sullivan, Hitchcock and Stein (2011) examined whether neuroticism significantly mediated the association relationship of childhood adversity and adult sleep quality in 327 college students ( 91 males), with a mean age of 18.9 years $(S D=2.1$ ).

Regression findings indicate that the relationship between childhood adversity and adult sleep quality is significant, and that there is a stronger association in men. Furthermore, a bootstrapping approach to testing the significance of the indirect effect (i.e., mediation) indicated that neuroticism mediated this relationship in both men and women. These data suggest that otherwise healthy young adults with a history of childhood adversity are at increased risk for sleep disturbance. Neuroticism may represent a potential target for change in future insomnia interventions, particularly in adults with a history of childhood adversity.

Williams and Moroz (2009) examined the associations of neuroticism (N) and sleep quality in the context of a life transition. Seventy-seven college freshmen (mean age $=19.5$ ) completed personality measures at the beginning of the academic year, and measures of sleep quality approximately two months later. N was negatively related to sleep quality.

A mail-in questionnaire study and two confirmatory archival analyses were described by Soehner and Kennedy (2007). Variables related to personality and measures of sleep timing, sleep quality, and sleep duration were initially assessed by self-report in a sample of 54 working adults ( $31.5 \%$ male, $23 i ̈ 48$ years). Neuroticism was measured by the Eysenck Personality Inventory (EPI). The quality of sleep was measured by the Pittsburgh Sleep Quality Index (PSQI) and by questions relating to habitual sleep latency and minutes awake after sleep onset from the Sleep Timing Questionnaire (STQ). The duration and timing of sleep was assessed using the STQ separately for work-week nights (Sundayï Thursday) and for weekend nights (Friday and Saturday). Morningness-eveningness was assessed using the Composite Scale of Morningness (CSM). Two confirmatory analyses using separate archival samples (Study A:
 confirm specific correlations of interest. Higher neuroticism was associated with poorer sleep as
indicated by higher PSQI scores. In contrast, no significant correlations emerged between neuroticism and any of the sleep duration variables. Personality appears to affect certain aspects of the timing and subjective quality of sleep, but not necessarily its duration.

Gau (2000) investigated the relationship between neurotic personality characteristics and sleep habits/problems using a population-based cross-sectional study. The participants were nine hundred and sixty five students from two junior high schools in Taipei and their parents were randomly selected in December 1993 for inclusion in the study. The response rate was $96.4 \%$ (930) for students and $88.6 \%$ (855) for parents. Students were administered a sleep habit questionnaire and the Junior Eysenck Personality Inventory (JEPI) at both junior high schools in Taipei. For the JEPI, high and low neuroticism was operationally defined as scores that were one or more standard deviation above or below the sample mean, respectively. This yielded two extreme groups: high neuroticism group ( $\mathrm{n}=183$ ) and low neuroticism group ( $\mathrm{n}=163$ ). The high neuroticism group went to bed later and slept less than did the low neuroticism group. Using logistic regression, adjusting for sex and years at junior high school, the high neuroticism group had significantly different sleep habits and school performance from the low neuroticism group. Subjects in the high neuroticism group had significant risk of feelings of sleep insufficiency, tiredness, moodiness, and difficulty waking up in the morning. They also had more daytime sleepiness than did those in the low neuroticism group. The high neuroticism group also suffered from more sleep-related problems than did the low neuroticism group. The findings support the hypothesis that a high neuroticism score is associated with going to bed late on school days, short sleep duration, different sleep habits, sleep problems, and impaired daytime function in comparison with a low neuroticism score.

## Summary of Review

The researcher reviewed theories of sleep quality which indicate that some people experienced poor sleep due to a number of factors. TheSpielman 3Ps model of sleep is a model of progression from normal sleep to poor sleep via predisposing, precipitating, and perpetuating factors. This model is useful for describing the course of the poor sleep and focuses treatment on those behaviours that are identified as perpetuating sleeplessness. Lundh and Broman differentiated between interfering factors generally hyper arousal related, and interpreting factors with cognitive and subjective appraisal components. This model points to assessment of psychological factors such as having a high level of emotional sensitivity, being easily hurt, and having a slow recovery from stress and worry in order to improve sleep. The cognitive theory of sleep is based on the notion that negative thoughts, beliefs, and attitudes about sleep and the consequences of sleep lead to insomnia by provoking cognitive arousal and subsequent maladaptive sleep behaviours. Moreover, Espie $\hat{\beta}$ model focuses on poor sleep as a failure to inhibit wakefulness, primarily due to the body $\hat{Q}$ failure to adapt to actual or perceived stressors.

Stress response theory of PTSD argued that when people have faced with trauma, their initial response is outcry at the reality of the trauma and they try to assimilate the new trauma information with prior knowledge, and by responding to this tension, psychological defense mechanisms are brought into play to avoid memories of the trauma and pace the extent to which it is recalled.Emotion dysregulation theory suggested that individuals with anxiety have difficulty understanding their emotional experiences and have little ability to modulate their intense emotions. Digman's personality theory viewed a neurotic person as being worried, temperamental and prone to sadness. They also show more emotional reactions whenever confronted with stressful situations.

The theory of sleep quality in which the work is anchored on is Lundh \& Broman model of sleep because ittakes into account the physiological and psychological factors where individuals may have a predisposition to both the sleep-interfering processes and the sleepinterpreting processes. These would include having higher basal levels of arousal and arousability, which can include personality traits such as having a high level of emotional sensitivity, being easily hurt and having a slow recovery from stress. Other psychological variables could include having a personal disposition to worry, experiencing emotional conflicts in personal relationships and emotional involvement in the problems of others.

Related empirical works were also reviewed. Exposure to trauma have been found to impair the quality of sleep. Evidence of studies are found in the samples such as perinatal outcomes of African American women (Hauff, 2015), postpartum women (Swanson, et al., 2014), LôAquila citizens exposed to earthquake (Tempest, et al., 2013), youths who experienced hurricane Katrina (Brown, et al., 2011), children exposed to war (Llabre \& Hadi, 2009; Pynoos, et al., 1990), women who are sexually abuse (Noll, et al., 2006), survivors of Holocaust, combat veterans, and sea disaster (Hefez, et al., 1995; Kaminer \& Lavie, 1991; Rosen, et al.,). Trauma is a predisposing factor for poor sleep quality but research on graduate students who may be traumatized by negative life stressors are scarce.

Emotion regulation has been found to impair the quality of sleep. Evidence of this proposition abounds in numerous studies using various samples such as normative adult sample in the community (Mauss, et al., 2013; Sandrus \& Voinescu, 2014), patients with generalized anxiety disorders (Tsypes, et al., 2015), undergraduate students (Markarian, et al., 2013; Pickett, et al., 2015) and depressed patients (Harvey, et al., 2014). Although Mararian, et al., (2013)
studied sleep quality in a student sample, there was no effort to consider traumatic experiences of the students.

The association of high neuroticism with poor quality of sleep has been shown in several studies of some populations such as high school students (Gau, 2000), working adults (Soehner \& Kennedy, 2007), college freshmen (Willaims \& Moroz, 2009), college students (Ramsawh , et al., 2011) and university undergraduates (Calkins, et al., 2013; Duggan, et al., 2014). Generally, neuroticism is a predisposing factor for poor sleep quality but research on students who may be traumatized by negative life stressors are scarce.

Taken together, it could be ascertained from the review of empirical literature that studies on sleep quality have incorporated the idea of preventive mental health. Populations in the community are considered to be critical targets of consistent research efforts. This study takes a cue from the studies on students (e.g., Calkins, et al., 2013, Duggan, et al., 2014; Pickett, et al., 2015)in attempt to fill the existing gaps in Nigerian studies on sleep quality. Pickett, et al. (2015) made a very useful suggestion that previous research with a collegestudent sample suggests that trauma-related symptoms increase inseverity as the number of traumatic experiences increases, it may be interesting for futureresearch to examine the association between the number and/or frequencyof trauma experiences and sleep problems in nonclinical samples.Of particular interest for the present study is Pickett, et al.仑̂ (2015) recent observation that their own sample was comprisedprimarily of White female college students and therefore thefindings may not generalize to other populations. They suggested that it would beimportant to replicate the emotion regulation-sleep quality hypotheses in samples among other cultures to determine if the findings generalize to otherpopulations or are unique to theirown sample.Although previous research has shownthat personality and sleep are each substantial predictors of health throughout
the lifespan, little is known about links between personality and healthy sleep patterns of postgraduate students. In addition, no available study has simultaneously considered trauma exposure, emotion regulation, and neuroticism as factors in sleep quality of postgraduate students. Such an investigation is considered worthwhile in order to determine their relative potency in predicting sleep quality.

## Hypotheses

The following hypotheses were proposed;
(1) Trauma exposure will significantly predict sleep quality among postgraduate students.
(2) Emotion regulation will significantly predict sleep quality among postgraduate students.
(3) Neuroticismwill significantly predict sleep quality among postgraduate students.

## CHAPTER THREE

## Method

## Participants

Participants in this study were four hundred and four (404) trauma exposedpostgraduate students of theUniversity of Nigeria, Nsukka (male=228, female=176). They were drawn from the two postgraduate hostels in UNN: UNN-Odili Hostel $(\mathrm{n}=230)$ and Kwame Nkrumah Hall ( $\mathrm{n}=174$ ). There are 178 rooms in the UNN-Odili hostel, and 3 students are officially assigned to live in a room. Based on the records obtained from the hostelôs officials,the eligible students in UNNOdili hostel were 534. Using single-stage systematic random sampling technique, students odd number rooms were selected as participants. Postgraduate students live in the ground floor and first floor of Nkrumah hall, and there are 70 rooms for the postgraduate students. Eligible students in the hall were 210 and because of this small number, participants were drawn from all the rooms. In this study, the average respondentô age was 30.60 ( $\mathrm{SD}=6.84$, ranging from 22 to 55 years). With respect to marital status, $75.7 \%$ were single, $23.3 \%$ were married, and $1 \%$ were widows. Majority ( $96.0 \%$ ) were Christians, $3.2 \%$ were Muslims, $0.5 \%$ were adherents of African

Traditional Religion, and $0.2 \%$ were members of other religious groups. The composition of the sample with regard to educational programme of study were as follows: Postgraduate Diploma (PGD) (9.7\%), Masterôdegree (86.1\%), and Doctor of Philosophy (PhD) (4.2\%).

## Instruments

A questionnaire form comprising two sections was used for the study. Section A contains some demographic characteristics. Section B comprises four measures intended to assess different constructs and domains of the respondents, they were: Harvard Trauma Questionnaire Part A,(Mollica, Caspi-Yavin, Bollini, Truong, Tor \& Lavelle, 1992), Difficulties in Emotion Regulation Scale, (Gratz \& Roemer, 2004), Neuroticism Scale of Big Five Inventory, (John, Donahue \& Kentle, 1991), and Pittsburgh Sleep Quality Index, (Buysse, Reynolds, Monk, Berman and Kupfer, 1989).

## Harvard Trauma Questionnaire(HTQ) (Part A)

The Harvard Trauma Questionnaire, Part A (HTQ-A), developed by Mollica, CaspiYavin, Bollini, Truong, Tor \& Lavelle (1992), measures the intensity of traumatic events.Part I of HTQ consists of 17 items, which are scored on a 5-point response format of 1 (never), 2 (rarely), 3 (sometimes), 4 (often) and 5 (always). Examples of the traumatic events presented in the HTQ part I include: lack of shelter, serious injury, forced isolation/separation from others, unnatural death of family member/friend, etc. Inter-rater reliability was found to be .93 while internal consistency reliability (Cronbachôs alpha) was .90 . The average individual item-total correlation was .56 with a range of .30 to .70 (Mollica, et al., 1992). A previous research in a Nigerian sample (Ebulum, 2014)in a study with flood victims in Ochuche community in Ogbaru L.G.A. of Anambra State, obtained internal consistency reliability estimate (Cronbachôs alpha) of .71 for 13 items, excluding items 7 (Brain washing), 13 (Unnatural death of family or friend),

14 (Murder of stranger or strangers) and 16 (Torture) and most of the items of the instrument had item-total correlations of .30 and above. A Cronbach $\hat{\propto}$ alpha reliability of .71 was reported for the scale, indicating acceptable reliability value.

## Difficulties in Emotion Regulation Scale (DERS)

The DERS is a 36-item, self-report measure that assesses difficulties in emotion regulation across six domains, which includes: Non-acceptance of emotional experiences (six items), (e.g., "When I'm upset, I feel guilty for feeling that way");Difficulty engaging in goaldirected behaviour (five items), (e.g., "When I'm upset, I have difficulty getting work done"); Limited access to strategies for emotion regulation (eight items), (e.g., "When Iôm upset, I start to feel very bad about myself."); Lack of emotional clarity(five items), (e.g., "I am confused about how I feel");Lack of awareness of emotions (six items) (e.g., "I am attentive to my feelings"); and Impulse control difficulties (six items) (e.g. "When I'm upset, I feel out of control".).For each item, participants rate how often each item applies to their experiences when upset using a five-point Likert-type response format ranging from (1) Almost Never, to (5)Almost Always. A total score could becalculated by summing the item responses after reverse scoring ofappropriate items. A higher score indicates greater difficulties with emotion regulation. The DERS has demonstrated excellent psychometric properties in previous research (Gratz \& Roemer, 2004).

The DERS has shown excellent internal consistency with Cronbachô alpha of .93 for the total, and .80 to .89 for the subscales. Research also shows that DERS has test-retest reliability of .80 for the total score over a period of 4-8 weeks. It has also demonstrated good construct and predictive validity as a whole as well as within the individual subscales (Gratz \& Roemer, 2004). Support for the construct and predictive validity of DERS scores within clinical
and nonclinical populations in the US have also been found (Fox, Axelrod,Paliwal, Sleeper \& Sinha, 2007; Gratz \& Roemer, 2004; Gratz, Rosenthal, Tull, Lejuez \& Gunderson, 2006).

## The Neuroticism Subscale of Big Five Inventory (NSBFI)

Neuroticism was measured using the neuroticism subscale of the 44-item Big Five Inventory (BFI) developed by John, Donahue \& Kentle (1991). Neuroticism subscale consists of eight items, which is a short descriptive statement, and respondents are asked to rate howmuch the characteristics described applies to them, using a 5-point Likert-type response scale $(1=$ disagree strongly to $5=$ agree strongly). A sample item for neuroticism (reversed item is "Is relaxed, handles stress well", "is emotionally stable, not easily upset", Remains calm in tense situations". Once reverse scored items are being corrected, a high score indicate a high level in the personality trait. The coefficient of reliability provided by John, et al., (1991) for neuroticism subscale of the BFI demonstrated adequate reliability across items, with Cronbachôs alpha scores of .84 . Separate norms have been reported by Umeh (2004) for male and female Nigerian sample and the divergent validity coefficient obtained with university Maladjusted Scale (Kleinmuntz, 1961) is .39 for neuroticism.

## Pittsburgh Sleep Quality Index (PSQI)

The Pittsburgh Sleep Quality Index, developed by Buysse, Reynolds, Monk, Berman and Kupfer, (1989) was designed to provide a standardized and psychometrically sound measure of sleep quality. The PSQI is a 19 -item self-rated questionnaire which assesses sleep quality and sleep disturbances over the past month. The PSQI consists of seven components, measuring subjective sleep quality, sleep latency, sleep duration, sleep efficiency and sleep disturbances, the use of any sleeping medication and the occurrence of any related daytime dysfunction (e.g. ñduring the past month, how would you rate your sleep quality overall?ò). Each item score
ranges from 0 (no difficulty) to 3 (severe difficulty). Adding the seven component scores together results in the PSQI global score (range of 0-21). A higher PSQI global score means that the participant has poor sleep quality. The cut-off point for poor sleepers is a score of five, which provides a sensitivity of $89.6 \%$ and a specificity of $86.5 \%$ ( $\mathrm{kappa}=0.75, \mathrm{p}<0.001$ ) to correctly distinguish between good and poor sleepers.

According to Buysse and Colleagues (1989), the Pittsburgh Sleep Quality Index has a good internal consistency, with Cronbach alpha coefficient reported of between .82 to .89 and validity .80. Aloba, Adewuya, Ola and Mapayi, (2007) validated the instrument using 520 Nigerian University students with the best cut-off score at 5 (sensitivity 0.72 , specificity 0.55 , overall correct classification rate 0.55 ). The psychometric value of PSQI in screening for insomnia among Nigerian students was moderate compared to what has been obtained in Western culture (Aloba, et al. 2007). Backhaus, Junghanns, Broocks, Riemann and Hohagen (2002) reported a Cronbachôs Ǔ of 0.85 , a testï retest reliability coefficient of 0.87 , a sensitivity ranging from $80 \%$ to $100 \%$ and a specificity ranging from 80 to $83 \%$ in German patients with primary insomnia.

## Validation Study for Instruments

For the validation study, the questionnaires comprising the four measures were completed by 100 (hundred)postgraduate students of the University of Uyo, Akwa Ibom State. The researcher administered the measures for the pilot study to graduate students in theUniversity of Uyo in Post-graduate hostel,Akwa Ibom State, Nigeria. Reliability and validity analysis was carried out for each of the scales using SPSS version 20. The HTQ Part A yielded a very high internal consistency reliability ( Cronbachô alpha $=.94$ ). Inter-item correlations of the 17 items ranged from .12 to .69 with majority of the coefficients having moderate to high correlation
values. The overall mean score of the validation sample on the HTQ-A was 25.88 (SD $=$ 10.13)(See Appendix B1). Difficulties in Emotion Regulation Scale had an internal consistency reliability estimate of Cronbachố alpha of .91 (See Appendix B2).

Neuroticism Scale of the Big Five had an internal consistency reliability estimate of Cronbach $\hat{@}$ alpha of .69 (See Appendix B3).Kaiser-Meyer-Olkin Measure of Sampling Adequacy was .607 and Bartlette $\hat{O}$ Test of Sphericity was 146.663 ( $\mathrm{p}<.000$ ), indicating that the data could be tested for factorial validity. Principal axis factoring with Maximum Likelihood Method showed that the scale is unifactorial. The scale displayed acceptable goodness-of-fit.

Pittsburgh Sleep Quality Index had an internal consistency reliability estimate of Cronbachố alpha of .73, which is relatively high (See Appendix B4).

## Procedure

The researcherand twotrained research assistants approached the students in the selected rooms of UNN-Odili Hostel and Kwame Nkrumah hall. The aim of the study was made known to the participants. Participants were assured that all their responses will be treated confidentially. Each of the students that was willing to participate in the study weregiven a questionnaire form containing an introductory page for demographic information and the four measures for the study (Harvard Trauma Questionnaire, Part A;the Difficulties in Emotion Regulation Scale; Neuroticism Scale of Big Five Inventory; and Pittsburgh Sleep Quality Index). The researcher and the assistants distributed and collected some ofthe questionnaires on the spot to ensure high rate of return, and some of the questionnaires were returned the following day. Four hundred and fifty (450) copies of the questionnaires were administered to the postgraduate students. Of the 450 copies questionnaires distributed, four hundred and forty-two (442) were returned, but eight (8) got missing in the process, only four hundred and four (404) were
appropriately filled while thirty-nine (39) were not properly completed and were discarded. The properly filled copies of the questionnaires were scored and data were used for analysis.

## Design/statistics

The study adopted a cross- sectional survey design. Pearsonô correlation (r) analysis was conducted among the study $\hat{O}$ predictors and dependent variables while multiple regression was used for hypothesis testing. The choice of the correlation is based on Urbinâ̂ (2004) emphasis of its utility as a major tool in demonstrating linkages between (a) scores on different tests, and (b) test scores and non-test (demographic) variables. It was used to ascertain if sleep quality will be bivariately related to trauma exposure, emotion regulation and neuroticism. Furthermore, it wasused to determine the relationships of sleep quality with the demographic variables (e.g., gender, age, etc.).

Multiple regression analysis allow researchers to simultaneously use several independent (or predictor) variables. By using more than one independent variable, one should do a better job of explaining the variation in the criterion and hence be able to make more accurate predictions (Mendenhall, Beaver \&Beaver, 2009). Hence, hierarchical multiple regressions, using step-wise method, were conducted to test the hypotheses for the study.

## CHAPTER FOUR

## Results

Table 1: Mean and Standard deviation of demographic, independent and dependent variables ( $\mathrm{N}=404$ ).

## Variables Mean (SD)

| Age (years), M(SD) | Range $=22-58$ | $30.60(6.84)$ |
| :--- | :--- | :--- |
| Gender | Males | $228(56.6)$ |
|  | Females | $176(43.4)$ |
| Marital status | Married | $94(23.3)$ |
|  | Single | $306(75.7)$ |
|  | Widowed | $4(1)$ |
| Educational status | PGD | $39(9.7)$ |
|  | Mastersôdegree | $347(86.1)$ |
| Religion | PhD | $18(4.2)$ |
|  | Christian | $387(96.0)$ |
|  | Moslem | $13(3.2)$ |
|  | ATR | $2(3.2)$ |


|  | Others | $2(0.2)$ |
| :--- | :--- | :--- |
| Trauma exposure M(SD) | $26.38(9.52)$ |  |
| Emotion regulation M(SD) | $70.56(14.59)$ |  |
| Neuroticism M(SD) | $19.20(5.13)$ |  |
| Sleep quality M(SD) | $13.95(5.86)$ |  |

Note: $M=$ Mean; $S D=$ Standard Deviation

Table 1 showed the means and standard deviation of participantsôscores on the independent and dependent variables. The average score on the variables of interest in the study were as follows: trauma exposure $=26.38(\mathrm{SD}=9.52)$, emotion regulation $=70.56(\mathrm{SD}=14.59)$, neuroticism $=$ $19.20(\mathrm{SD}=5.13)$ and sleep quality $=13.95(\mathrm{SD}=5.86)$.

Table 2: Correlations of demographic variables, trauma exposure, emotion regulation, neuroticism and sleep quality

|  | Variables | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| 1 | Gender | - |  |  |  |  |  |  |  |  |
| 2 | Age | -. $16^{* * *}$ | - |  |  |  |  |  |  |  |
| 3 | Marital_Status | . 05 | . 59 *** | - |  |  |  |  |  |  |
| 4 | Education | -. 03 | . $28^{* * *}$ | . $22^{* * *}$ | - |  |  |  |  |  |
| 5 | Religion | -. $16^{* *}$ | . 05 | . 07 | . 05 | - |  |  |  |  |
| 6 | Trauma exposure | -. 06 | . 07 | -. 02 | . 05 | -. 04 | - |  |  |  |
| 7 | Emotion regulation | -. 01 | . 02 | . 07 | . 08 | . 07 | . $12{ }^{*}$ | - |  |  |
| 8 | Neuroticism | . 03 | -. $15^{* *}$ | -. 06 | . 03 | -. 02 | . 08 | . $34^{* * *}$ | ${ }^{-}$ |  |
| 9 | Sleep Quality | . 02 | -. 12 * | -. 09 | . 05 | . 09 | . $12{ }^{*}$ | . 40 *** | . 23 ** | - |

Correlations in Table 2 showed that gender was not significantly related to sleep quality ( $\mathrm{r}=.02$ ). Age had a negative association with sleep quality $(\mathrm{r}=-.12, \mathrm{p}<.05)$, showing that older students had lower scores on PSQI (better quality of sleep). Marital status was not significantly
associated with sleep quality ( $\mathrm{r}=-.09$ ). Educational level was not significantly related to sleep quality $(\mathrm{r}=.05)$. The relationship between religion and sleep quality was not significant $(\mathrm{r}=.09)$. Trauma exposure had a positive association with quality of sleep ( $\mathrm{r}=.12, \mathrm{p}<.05$ ), indicating that those who had more exposure to traumatic events reported poorer quality of sleep. Emotion regulation was positively related to sleep quality $(\mathrm{r}=.40, \mathrm{p}<.001)$, which showed that students who had more emotion regulation difficulties had poorer sleep quality. Neuroticism was positively related to sleep quality $(\mathrm{r}=.23, \mathrm{p}<.01)$, showing that those who had more neurotic traits reported poorer quality of sleep.

Table 3: Hierarchical multiple regression (step-wise method) of Trauma exposure, emotion regulation and neuroticism predicting sleep quality of postgraduate students

| Step | Predictors | $\mathbf{B}$ | SE | $\mathbf{B}$ | $\mathbf{R}^{2}$ | $\Delta \mathbf{R}^{2}$ |
| :--- | :--- | :--- | :--- | :--- | :--- | :--- |
| $\mathbf{1}$ | Trauma exposure | .07 | .03 | $.12^{*}$ | $\mathbf{. 0 1}$ | $\mathbf{. 0 1}^{*}$ |
| $\mathbf{2}$ | Emotion regulation | .15 | .02 | $.38^{* *}$ | $\mathbf{. 1 6}$ | $\mathbf{. 1 4}^{* *}$ |
| $\mathbf{3}$ | Neuroticism | .13 | .06 | $.11^{*}$ | $\mathbf{. 1 7}$ | $\mathbf{. 0 1 *}^{*}$ |

Dependent Variable: Sleep Quality; $\not \mathbb{R}^{2}=$ Change in $\mathrm{R}^{2} ; * * \mathrm{p}<.001 ;$ *p<.05.
Table 3 showed that trauma exposure positively predicted sleep quality ( $\bar{\sigma}=.12, p<.05$ ), indicating that those students who were exposed to more traumatic events had poorer sleep quality. About $1 \%$ of the variance in sleep quality was explained on account of trauma exposure $\left(\not \mathbb{R}^{2}=.01\right)$ (See step 1 in $t$ able 3). In step 2, when emotion regulation was added to the regression model, it was found to be a strong positive predictor of sleep quality $(\bar{\sigma}=.38, \mathrm{p}<$ .001), as it was shown that those with greater difficulties in emotion regulation had poorer quality of sleep. The $\not \mathscr{R}^{2}(.14)$ indicated that emotion regulation explained $14 \%$ of the variance in sleep quality. In step 3 of the regression model, neuroticism was added and it positively
predicted sleep quality ( $\mathrm{K}=.11, \mathrm{p}<.05$ ), which indicated that students who had more neurotic traits reported poorer sleep quality. As shown by the $\not \mathscr{R}^{2}(.01)$, a marginal $1 \%$ of the variance in sleep quality was explained on account of neuroticism. Of all the predictors, emotion regulation was found to be the strongest predictor of sleep quality $(\mathrm{K}=.38)$, and the entire model explained $17 \%$ of the variance in sleep quality $\left(R^{2}=17\right)$.

## Summary of Findings

1. Trauma exposure was positively related to sleep quality and it positively predicted sleep quality ( $\mathrm{B}=.12, \mathrm{p}<.05$ ).
2. Emotion regulation was positively related to sleep quality and it positively predicted sleep quality $(\mathrm{B}=.38, \mathrm{p}<.001$ ).
3. Neuroticism was positively related to sleep quality and it positively predicted sleep quality $(\mathrm{B}=.11, \mathrm{p}<.05)$.

## CHAPTER FIVE

## Discussion

The major goal of this study was to examine the role of trauma exposure, emotion regulation, and neuroticism on sleep quality among postgraduate students. Three hypotheses were tested in the study and all the hypothesis were confirmed. The result showed that trauma exposure was a significantpositive predictor of sleep quality among postgraduate students. It indicates thatthose who had moretrauma exposure had higher scores on PSQI, indicating that they had poorer sleep quality outcomes. Thus, the first hypothesis which stated that trauma exposure will significantly predict sleep quality among postgraduate students was supported. The findings supports the views of relevant theories such as stress response theory (Horowitz, 1976, 1986) which upholds that when people are faced with trauma, their initial response is outcry at the reality of the trauma and they try to assimilate the new trauma information with prior knowledge, and by responding to this tension, may interfere with their quality of sleep. The finding is in agreement with the results in the literature reviewed which suggest that exposure to trauma negativelyinfluences sleep quality(Daige, et al., 2015; Sneed, 2015; Hauff, 2015; Swanson, et al., 2014; Tempest, et al., 2013; Brown, et al., 2011; Finkelhor, et al., 2009).Although the participants in the researches
reviewed in the empirical were not postgraduate students, the relationships between trauma exposure and sleep quality were adequately established in these studies. Multiple traumatic exposure was associated with poor sleep quality, and even ñjustò witnessing traumatic situations could result in significant poor sleep quality.

In the same vein, the result also supported the second hypothesis which stated that emotion regulation will significantly predict sleep quality among postgraduate students. The result showed that emotion regulation was a significant positive predictor of sleep quality among postgraduate students. It indicates that those with greater difficulties in emotion regulation had poorer quality of sleep. This implies that individuals complaining of poorer sleep had greater difficulties in emotion regulation. They had problems in accepting their own emotions and in engaging in goal-directed behaviours when experiencing negative emotions. They also showed greater difficulties both in controlling their impulse when under negative emotions and in understanding their own emotions (Sandru, et al., 2014). Inability to regulate arousal in individuals with difficulties in emotion regulation might explain the higher levels of poor sleep found among poor sleepers, given the role of the hyper-arousal in poor sleep (Harvey, 2002). In accordance with the emotion dysregulation model (Mennin, et al., 2005), individuals who have predispositions for anxiety which can occur after traumatic experiences have difficulty in understanding their emotional experiences and have little ability to modulate their intense emotions. They may have various sleep problems. The result is consistent with some previous studies (Pickett, et al., 2015; Mauss, et al., 2013;Markarian, et al., 2013; Baglioni, et al., 2010)which found a significant association between emotion regulation and studentsô sleep quality.

The third hypothesis which stated that neuroticism will significantly predict sleep quality was also supported. The result showed that neuroticism significantly predicted sleep quality among postgraduate students. In support of the 3Ps model of sleep (Spielman, et al., 1987), neuroticism has been found to be one important predisposing factor for poor sleep quality. The result indicates that neuroticism is an important determinant of sleep quality and that students who had more neurotic traits reported poorer sleep quality. This implies that a person high in neuroticism may likely experience such feeling as anxiety, anger, envy, hostility, guilt, and depressed mood. They may have strong negative reactions in the response to threats, frustration, or loss, and they may also experience heightened neuroticism during times of trauma, while someone low in neuroticism may just brush it off (Lahey, 2009).The result is consistent with some previous research focusing on sleep deprivation (Gau, 2000; Gray \& Watson, 2002; Soehner and Kennedy, 2007; William \& Morez, 2009; Calkins, et al., 2013) that reported neuroticism as factor that predict sleep quality among university students.

## Implications of the findings

The results of the study indicated that trauma exposure, emotion regulation, and neuroticism significantly predicted sleep quality among postgraduate students. In essence, these have some implications as it may likely influence the studentsô welfare, and their academic performance. In Nigeria, professional care is limited on sleep quality and this calls for an urgent need and application of psychological services to students, as students who are experiencing sleep disturbances are prone to be depressed and anxious, aggressive, irritable, have memory impairment, inability to take decision, feeling ñout of controlò, and other neuropsychological deficits. People who are otherwise healthy sometimes have sleep disturbances due to health, stress, or lifestyle. It implies that sleep disturbances are not entirely benign and could pose risks
to emotional health. Many people occasionally struggle to fall asleep at night, lie awake in bed sleepless because of the trauma they were exposed to, or wake up exhausted in the morning.

Health workers in schools settings are in a prime position to ask specific questions about possible trauma exposures. The act of obtaining such information from students in a private and safe location, such as the clinic, is an important first step to detection of and intervention for trauma. In addition, if students have experienced a traumatic event, they should be asked about other victimizations and trauma occurrences, because, as this study emphasized, trauma exposure is usually not one isolated event. Health workers who listen non-judgmentally are able to help the students begin to express their feelings and may help the students become more amenable to counselling from a professional therapist and they can also identify which students may need follow-up counselling to deal with traumatic experiences. Health workers can use developmentally-appropriate screening tools, which assess community violence and cumulative trauma exposure. Incorporating trauma assessments as standard practice would improve comprehensive care for the students. Health workers can be resources for students by knowing where they can turn to for help with PTSD or other emotional problems. Developmentally appropriate individual and group therapies for PTSD are available and helpful for students to identify their emotions, develop coping skills, and improve their overall functioning and quality of life.

Psychological intervention aimed at reducing difficulties in emotion regulation might be useful in alleviating sleep disturbances. Gross (2008) showed that participants who learned how to redirect their attention through mindfulness, an emotion regulation strategy, show a significant reduction in poor sleep symptoms. It is a common belief that a good night sleep enhances positive emotions and well-being during a day, while a poor night sleep increases negative
emotions and irritability. The finding of the current study suggests that successful intervention with students should attempt to address these more dysfunctional beliefs particularly about the causes and consequences of poor sleep. From available intervention, there is evidence showing the efficacy of cognitive-behavioural strategies such as psycho-education, cognitive restructuring and behavioural experiments in treating sleep-related beliefs (Harvey, 2002; Morin, Blais \& Savard, 2002). Clinical psychologists need to advocate for the inclusion of this vital need into the interagency framework to assist vulnerable populations in the country.

Neuroticism in students may have a negative long term impact on sleep quality. Prevention and intervention research may benefit from taking a developmental look at sleeping problems. The impact of neuroticism on poor sleep quality is likely through cognitive, physiological, psychological and behavioural processes. Parents, teachers, counselors and all those involved in education of students should try and study on the different personality traits. The knowledge of these personality traits is very important. It will help the parents, teachers, counselors and others to be in the position to give a student the right counsel that will help him/her in his/her educational pursuit. A neurotic student should also be taking care of in the school. He/she should be given necessary assistance and encouragement.

Early detection and treatment of sleep problems may have a positive influence on developmental trajectories of students. Awareness about sleep and sleep quality may be low in Nigeria and revelations about lack of qualified personnel in sleep and limited knowledge about sleep disorders raise speculation about the understandings of psychological phenomena in subSaharan Africa. It is likely that some phenomena with psychological components, like poor sleep, are presented primarily (or solely) as physical health problems at African primary care health centres. Research evidence suggests that some students "somatise" psychological
problems (Vega \& Rumbaut, 1991). Therefore, one goal of the education program would be to address somatisation of psychological problems, as well as the effects of medication use on sleep.

Furthermore, quality sleep is essential for studentôs growth and development and the skill of good sleep need to be pass to them in counselling centres in collaboration with health services by providing psycho-educational information aimed at educating students about the importance of good sleep quality to academic success.

## Limitations of the study

A limitation of the study was that it used a cross-sectional study design. A crosssectional design can give a snapshot of the studentô current perceptions at one time, and cannot provide data about how student $\hat{\Theta}$ perceptions may change over time. In addition, cross-sectional designs cannot show that one variable causes a change to occur in another variable.

The sample comprised primarily of postgraduate students and therefore the findings may not generalize to other populations. The reliance on self-report questionnairesis an inherent measurement constraint that also does not allow forthe confirmation of sleep problems or confirmation from informantsregarding the nature of the traumatic experience or thevarious types and numbers of traumatic experiences.

## Suggestions for further studies

It is through longitudinal and prospective studies that one can depict the causal relation between trauma exposure, emotion regulation and neuroticism on sleep quality. Future research should employ longitudinal designs in order to examine the development of sleep disturbances
following trauma and the relationship that these disturbances may have on the development of emotion regulation and neuroticism. Researchers in their subsequent studies should endeavour to use larger sample size in order to improve generalizability and applicability of the findings. It will also be important to replicate the current hypotheses in a community or clinical sample to determine if the finding generalize to other populations or are unique to the current samples. Similarly, it should not be assumed that the sample represents a clinical sample with clinically significant psychopathology given that the trauma exposure diagnosis was not confirmed. Although, previous research with college studentsô sample suggests that trauma-related symptoms increase in severity as the number of traumatic experiences increases, there was no such finding in the current research. It may be interesting for future research to examine the association between the number or frequency of trauma experiences and clinically significant symptoms in clinical samples.Future researchshould replicate the current study in a sample in which aPTSD diagnosis can be corroborated with other measures, such asa diagnostic interview for sleep problems and PTSD.

Meanwhile more studies should be conducted using Nigerian sample as it will help to provide clear insight and understanding about sleep quality and its intervention among postgraduate students in Nigeria. Researchers in the future should make effort to connect empirical findings in this study to clinical practice which will aim at improving therapeutic outcomes among graduate students through intervention studies and implementation research. In this case, the gap between psychological science and practice (see Chukwuorji, Mefoh \& Ezeanolue, 2014) will be bridged in this context. Lastly, the construct of sleep quality merits further research interests and attention in relation to psychopathological traits by researchers in Nigeria.

## Summary

The present study examined trauma exposure, emotion regulation, and neuroticism as predictors of sleep predictors of sleep quality among postgraduate students. The researcher reviewed the following theories of sleep quality: the 3 P (predisposing, precipitating, perpetuating) model of sleep, Lundh and Bromanô model of sleep, Cognitive model of sleep, and Espiê̂ psychological inhibition model of sleep. The relevant theories for the predictors in this study were Stress response theory of trauma, Emotion dysregulation model, and Digmans theory of personality. It was hypothesized as follows: (a) trauma exposure will significantly predict sleep quality among postgraduate students (b) Emotion regulation will significantly predict sleep quality among postgraduate students (c) Neuroticism will significantly predict sleep quality among postgraduate students.

A sample of 404 postgraduate students from University of Nigeria Nsukka (UNN) participated in the study. They were selected using a single-stage systematic random sampling method. A questionnaire form comprising four measures was given to the participants for completion. The measures are Harvard Trauma Questionnaire (HTQ-Part A), Difficulties in Emotion Regulation Scale (DERS), Neuroticism subscale of Big Five Inventory (BFI), and Pittsburgh Sleep Quality Index (PSQI). The scales were initially validated in the pilot study, indicated that they were reliable and valid for the current study. The questionnaires was administered to the students in their hostel by the researcher and a trained research assistance.

The hypotheses were tested using a cross sectional design and multiple regression. The result of the analysis confirmed the three hypotheses as: (1) trauma exposure significantly predicted sleep quality among postgraduate students. (2) Emotion regulation significantly
predicted sleep quality among postgraduate students, and (3) Neuroticism will significantly predicted sleep quality among postgraduate students.

## Conclusion

To the knowledge of the researcher, no previous studies have examined the role of trauma exposure, emotion regulation, and neuroticism on sleep quality among postgraduate students. Trauma exposure, emotion regulation, and neuroticism in various forms pose risk for predisposition, precipitation, and perpetuation of poor sleep quality as a bridge between neurophysiological processes and psychopathology such as depression. Treatment of an individual after an experience of stress or trauma may be enhanced by considering the role of the trauma exposure to the personô identity and identifying the extent to which the person is relying on the emotion regulation strategies.

Future research will need to continue to investigate the relationships in this study in order to improve theoretical understanding of sleep quality development and maintenance, which may lead to improvements or modifications of existing treatment for those expose to trauma. Improved sleep quality will likely benefit university students in their mental health status, daily activities and academic performance. Educational campaigns focused on helping university avoiding the build-up of a chronic sleep debt may be important in enhancing the academic performance and in reducing the development of psychiatric disorders later in life.By so doing, Nigeria may be better positioned to reap the benefits of their efforts of expanding access to higher education to Nigerian youths and adults.

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## APPENDIX A1

## RESEARCH QUESTIONNAIRE

Department of Psychology,
Faculty of the Social Sciences,
University of Nigeria,
Nsukka.

## Dear Respondent,

The researcher is a postgraduate student of the above mentioned institution. She is undertaking a research report on factors affecting sleep, in partial fulfilment of the award of Master of Science (M.Sc.) degree in Psychology. This is purely an academic exercise. Please kindly respond to the questionnaire as truthfully and sincerely as possible by ticking the appropriate box of your choice. There is no right or wrong answer. Your response will be treated with utmost confidentiality.

## SECTION A

Please give the needed information about yourself, by ticking the appropriate box or filling the blank spaces.

1. Gender: Male ( ) Female ( ) 2. Age: é é years 3. Marital statusé é é é é é
2. Educational Qualificationé é é é é é é . 5 Religioné é é é é é é é ..

## APPENDIX A2

## Harvard Trauma Questionnaire (Part A)

Instructions:Below are some questions about events that you and your family and community members may have experienced or witnessed. Family refers to extended family. Please indicate your degree of agreement or disagreement with these items as they apply to you in the last six months. At the right hand side of the questionnaire are possible options for you to use in making your responses by ticking (ã) any one of the following: Never, Rarely, Sometimes, Often and Always.

|  | ITEMS | Never | Rarely | Sometimes | Often | Always |
| :--- | :--- | :--- | :--- | :--- | :--- | :--- |
| 1 | Lack of food or water |  |  |  |  |  |
| 2 | Ill health without access to medical care |  |  |  |  |  |
| 3 | Lack of shelter |  |  |  |  |  |
| 4 | Imprisonment/detention |  |  |  |  |  |
| 5 | Serious injury |  |  |  |  |  |
| 6 | Combat situation |  |  |  |  |  |
| 7 | Brain washing |  |  |  |  |  |
| 8 | Rape or sexual abuse |  |  |  |  |  |
| 9 | Forced isolation from others |  |  |  |  |  |
| 10 | Being close to death |  |  |  |  |  |
| 11 | Forced separation from family members |  |  |  |  |  |
| 12 | Murder of family or friend |  |  |  |  |  |
| 13 | Unnatural death of family or friend |  |  |  |  |  |
| 14 | Murder of stranger or strangers |  |  |  |  |  |


| 15 | Lost or kidnapped |  |  |  |  |  |
| :---: | :--- | :--- | :--- | :--- | :--- | :--- |
| 16 | Torture |  |  |  |  |  |
| 17 | Threatened by dangerous animals |  |  |  |  |  |

## APPENDIX A3

Difficulties in Emotion Regulation Scale
INSTRUCTIONS: Please read the following statements carefully. Each one describes a way that you might (or might not) feel about your emotions. You are expected to indicate how often each statement applies to you using the following scales: (1) Almost never, (2) Sometimes, (3) About half the time, (4) Most of the time, (5) AlmostAlways. For each statement, circle one number from 1 to 5 , to indicate how much you agree or disagree with it. Please circle one and only one number for every statement.

| S/N | Statements | Almost Never (1) | Sometimes <br> (2) | About Half the Time (3) | Most of the Time <br> (4) | Almost Always (5) |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| 1. | I am clear about my feelings. | 1 | 2 | 3 | 4 | 5 |
| 2. | I pay attention to how I feel. | 1 | 2 | 3 | 4 | 5 |
| 3. | I experience my emotions as overwhelming and out of control. | 1 | 2 | 3 | 4 | 5 |
| 4. | I have no idea how I am feeling. | 1 | 2 | 3 |  | 5 |
| 5. | I have difficulty making sense out of my feelings. | 1 | 2 | 3 | 4 | 5 |
| 6. | I am attentive to my feelings. | 1 | 2 | 3 | 4 | 5 |
| 7. | I know exactly how I am feeling. | 1 | 2 | 3 | 4 | 5 |
| 8. | I care about what I am feeling. | 1 | 2 | 3 | 4 | 5 |
| 9. | I am confused about how I feel. | 1 | 2 | 3 | 4 | 5 |
| 10. | When I am upset, I acknowledge my emotions. | 1 | 2 | 3 | 4 | 5 |
| 11. | When I am upset, I become angry with myself for feeling that way. | 1 | 2 | 3 | 4 | 5 |
| 12. | When I am upset, I become embarrassed for feeling that way. | 1 | 2 | 3 | 4 | 5 |
| 13. | When I am upset, I have difficulty getting work done | 1 | 2 | 3 | 4 | 5 |
| 14. | When I am upset, I become out of control. | 1 | 2 | 3 | 4 | 5 |
| 15. | When I am upset, I believe that I will remain that way for a long time. | 1 | 2 | 3 | 4 | 5 |
| 16. | When I am upset, I believe that I will end up feeling very depressed | 1 | 2 | 3 | 4 | 5 |
| 17. | When I am upset, I believe that my feelings are valid and important. | 1 | 2 | 3 | 4 | 5 |
| 18. | When I am upset, I have difficulty focusing on other things. | 1 | 2 | 3 | 4 | 5 |
| 19. | When I am upset, I feel out of control. | 1 | 2 | 3 | 4 | 5 |
| 20. | When I am upset, I can still get things done. | 1 | 2 | 3 | 4 | 5 |
| 21. | When I am upset, I feel ashamed at myself for feeling that way. | 1 | 2 | 3 | 4 | 5 |
| 22. | When I am upset, I know that I can find a way to eventually feel better. | 1 | 2 | 3 | 4 | 5 |
| 23. | When I am upset, I feel like I am | 1 | 2 | 3 | 4 | 5 |


|  | weak. |  |  |  |  |  |
| :--- | :--- | :---: | :---: | :---: | :---: | :---: |
| 24. | When I am upset, I feel like I can <br> remain in control of my behaviours. | 1 | 2 | 3 | 4 | 5 |
| 25. | When I am upset, I feel guilty for <br> feeling that way. | 1 | 2 | 3 | 4 | 5 |
| 26. | When I am upset, I have difficulty <br> concentrating. | 1 | 2 | 3 | 4 | 5 |
| 27. | When I am upset, I have difficulty <br> controlling my behaviours. | 1 | 2 | 3 | 4 | 5 |
| 28. | When I am upset, I believe there is <br> nothing I can do to make myself <br> feel better. | 1 | 2 | 3 | 4 | 5 |
| 29. | When I am upset, I become irritated <br> at myself for feeling that way. | 1 | 2 | 3 | 4 | 5 |
| 30. | When I am upset, I start to feel very <br> bad about myself. | 1 | 2 | 3 | 4 | 5 |
| 31. | When I am upset, I believe that <br> wallowing in it is all I can do. | 1 | 2 | 3 | 4 | 5 |
| 32. | When I am upset, I lose control <br> over my behaviour. | 1 | 2 | 3 | 4 | 5 |
| 33. | When I am upset, I have difficulty <br> thinking about anything else. | 1 | 2 | 3 | 4 | 5 |
| 34. | When I am upset, I take time to <br> figure out what I am really feeling. | 1 | 2 | 3 | 4 | 5 |
| 35. | When I am upset, it takes me a long <br> time to feel better. | 1 | 2 | 3 | 4 | 5 |
| 36. | When I am upset, my emotions feel <br> overwhelming. | 1 | 2 | 3 | 4 | 5 |

## APPENDIX A4

## Neuroticism Subscale of Big Five Inventory

Here are number of characteristics that may or may not apply to you. For example, do you agree that you are someone who likes to spend time with others? Please circle a number to indicate the extent to which you agree or disagree with that statement.

| S/N | Items <br> I am someone who... | Disagree | Disagree a <br> little <br> $\mathbf{( 1 )}$ | Neither <br> agree nor <br> disagree <br> $(\mathbf{3})$ | Agree a <br> little | Agree <br> Strongly |
| :---: | :--- | :---: | :---: | :---: | :---: | :---: |
| $\mathbf{( 4 )}$ | $\mathbf{( 5 )}$ |  |  |  |  |  |
| 1. | Is depressed, blue | 1 | 2 | 3 | 4 | 5 |
| 2. | Is relaxed, handles stress well | 1 | 2 | 3 | 4 | 5 |
| 3. | Can be tense | 1 | 2 | 3 | 4 | 5 |
| 4. | Worries a lot | 1 | 2 | 3 | 4 | 5 |
| 5. | Is emotionally stable, not easily | 1 | 2 | 3 | 4 | 5 |
| upset. |  | 1 | 2 | 3 | 4 | 5 |
| 6. | Can be moody | 1 | 2 | 3 | 4 | 5 |
| 7. | Remains calm in tense situations | 1 | 2 | 3 | 4 | 5 |
| 8. | Gets nervous easily |  |  |  |  |  |

## APPENDIX A5

## Pittsburgh Sleep Quality Index (PSQI)

INSTRUCTION: The following questions relate to your usual sleep habits during the past months. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions by circling the appropriate answer.

## During the past months

1. When do you usually go to bed?
2. How long (in minutes) does it take you to fall asleep every night?
3. When do you usually wake up in the morning? $\qquad$
4. How many hours of actual sleep do you get at night?

| N/S | During the past month, how often have you had trouble sleeping because you... | Not during the past month (0) | Less than once a week (1) | Once or twice a week (2) | Three or more times a week (3) |
| :---: | :---: | :---: | :---: | :---: | :---: |
| a. | Cannot get to sleep within 30 minutes | 0 | , | 2 | 3 |
| b. | Wake up in the middle of the night or early morning | 0 | 1 | 2 | 3 |
| c. | Have to get up to use the bathroom | 0 | 1 | 2 | 3 |
| d. | Cannot breath comfortably | 0 | 1 | 2 | 3 |
| e. | Cough or snore loudly | 0 | 1 | 2 | 3 |
| f. | Feel too cold | 0 | 1 | 2 | 3 |
| g . | Feel too hot | 0 | 1 | 2 | 3 |
| h. | Have bad dreams | 0 | 1 | 2 | 3 |
| I | Have pain | 0 | 1 | 2 | 3 |
| J | Other reason(s), please describe, including how often you have had trouble sleeping because of this reason(s): |  |  |  |  |
| 6. | During the past month, how often have you taken medicine, (prescribed or ñover the counterò) to help you sleep? | 0 | 1 | 2 | 3 |
| 7. | During the past month, how often have you had trouble staying awake while driving, eating, meals, or engaging in social activity? | 0 | 1 | 2 | 3 |
| 8. | During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done? | 0 | 1 | 2 | 3 |
|  |  | Very good <br> (0) | Fairly good <br> (1) | Fairly bad <br> (2) | Very bad <br> (3) |
|  | During the last month, how would you rate your sleep quality overall? | 0 | 1 | , | 3 |

## APPENDIX B

## SPSS-20 Results: Reliability and Validity of Instruments

## Frequencies

## Gender

## Statistics

Gender

| N | Valid | 100 |
| :--- | :--- | ---: |
|  | Missing | 0 |


| Gender |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | Frequency | Percent | Valid Percent | Cumulative <br> Percent |
| Valid | Male | 52 | 52.0 | 52.0 | 52.0 |
|  | Female | 48 | 48.0 | 48.0 | 100.0 |
|  | Total | 100 | 100.0 | 100.0 |  |

## Marital Status

Statistics


| Marital_status |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | Frequency | Percent | Valid Percent | Cumulative <br> Percent |
|  | . 00 | 1 | 1.0 | 1.0 | 1.0 |
| id | Single | 76 | 76.0 | 79.2 | 80.2 |
| Valid | Married | 19 | 19.0 | 19.8 | 100.0 |
|  | Total | 96 | 96.0 | 100.0 |  |
| Missing | System | 4 | 4.0 |  |  |
| Total |  | 100 | 100.0 |  |  |

## Religion

## Statistics

Religion



Religion

|  | Frequency | Percent | Valid Percent | Cumulative <br> Percent |
| :--- | ---: | ---: | ---: | ---: |
|  | Christian | 94 | 94.0 | 96.9 |

## Event

## Statistics

Event

| N | Valid | 100 |
| :--- | :--- | ---: |
|  | Missing | 0 |

Event

|  |  | Frequency | Percent | Valid Percent | Cumulative <br> Percent |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Valid | 1.00 | 15 | 15.0 | 15.0 | 15.0 |
|  | 2.00 | 5 | 5.0 | 5.0 | 20.0 |
|  | 3.00 | 3 | 3.0 | 3.0 | 23.0 |
|  | 4.00 | 12 | 12.0 | 12.0 | 35.0 |
|  | 5.00 | 5 | 5.0 | 5.0 | 40.0 |
|  | 6.00 | 33 | 33.0 | 33.0 | 73.0 |
|  | 7.00 | 2 | 2.0 | 2.0 | 75.0 |
|  | 8.00 | 4 | 4.0 | 4.0 | 79.0 |
|  | 9.00 | 2 | 2.0 | 2.0 | 81.0 |
|  | 10.00 | 1 | 1.0 | 1.0 | 82.0 |
|  | 11.00 | 11 | 11.0 | 11.0 | 93.0 |
|  | 12.00 | 1 | 1.0 | 1.0 | 94.0 |
|  | 13.00 | 5 | 5.0 | 5.0 | 99.0 |


| 14.00 | 1 | 1.0 | 1.0 | 100.0 |
| ---: | ---: | ---: | ---: | ---: |
| Total | 100 | 100.0 | 100.0 |  |

## APPENDIX B1

## Reliability

Scale: HAVARD TRAUMA QUESTIONNAIRE (HTQ) PART A Reliability

Scale: HTQ1

|  |  | N | \% |
| :---: | :---: | :---: | :---: |
| Cases | Valid | 100 | 100.0 |
|  | Excluded ${ }^{\text {a }}$ | 0 | . 0 |
|  | Total | 100 | 100.0 |

a. Listwise deletion based on all variables in the procedure.

Reliability Statistics

| Cronbach's Alpha | Cronbach's Alpha <br> Based on <br> Standardized Items | N of Items |
| ---: | ---: | ---: |
| .937 | .938 | 17 |


| Item Statistics |
| :--- |
|  Mean Std. Deviation N <br> VAR00001 1.9200 .91762 100 <br> VAR00002 1.7600 .97566 100 <br> VAR00003 1.4200 .80629 100 <br> VAR00004 1.2400 .58810 100 <br> VAR00005 1.6600 .90140 100 <br> VAR00006 1.5800 .75452 100 <br> VAR00007 1.5100 .81023 100 <br> VAR00008 1.3300 .79207 100 <br> VAR00009 1.3800 .72167 100 <br> VAR00010 1.5600 .89126 100 <br> VAR00011 1.4900 .82260 100 <br> VAR00012 1.4400 .90252 100 <br> VAR00013 1.7400 .96001 100 |


| VAR00014 | 1.4800 | .81004 | 100 |
| :--- | :--- | :--- | :--- |
| VAR00015 | 1.4600 | .94730 | 100 |
| VAR00016 | 1.4500 | .85723 | 100 |
| VAR00017 | 1.4600 | .80929 | 100 |

Inter-Item Correlation Matrix

|  | $\begin{aligned} & \text { VARO } \\ & 0001 \end{aligned}$ | $\begin{gathered} \text { VAR } \\ 000 \\ 02 \\ \hline \end{gathered}$ | $\begin{gathered} \text { VAR } \\ 000 \\ 03 \end{gathered}$ | $\begin{gathered} \text { VAR } \\ 000 \\ 04 \\ \hline \end{gathered}$ | $\begin{gathered} \text { VAR } \\ 000 \\ 05 \end{gathered}$ | $\begin{gathered} \text { VAR } \\ 000 \\ 06 \end{gathered}$ | $\begin{gathered} \text { VAR } \\ 000 \\ 07 \\ \hline \end{gathered}$ | $\begin{gathered} \text { VAR } \\ 000 \\ 08 \end{gathered}$ | $\begin{gathered} \text { VAR } \\ 000 \\ 09 \\ \hline \end{gathered}$ | $\begin{gathered} \text { VAR } \\ 000 \\ 10 \end{gathered}$ | $\begin{gathered} \text { VAR } \\ 0001 \\ 1 \\ \hline \end{gathered}$ | VARO $0012$ | $\begin{aligned} & \text { VAR0 } \\ & 0013 \end{aligned}$ | VARO $0014$ | $\begin{aligned} & \text { VAR0 } \\ & 0015 \end{aligned}$ | VAR0 0016 | VARO 0017 |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| VAR0 0001 | 1.000 | . 588 | . 606 | . 298 | . 504 | . 214 | . 123 | . 315 | . 275 | . 339 | . 387 | . 409 | . 515 | . 365 | . 298 | . 303 | . 322 |
| VAR0 0002 | . 588 | 1.00 0 | . 707 | . 453 | . 458 | . 246 | . 220 | . 443 | . 461 | . 458 | . 437 | . 546 | . 483 | . 377 | . 427 | . 469 | . 333 |
| VAR0 0003 | . 606 | . 707 | 1.00 0 | . 595 | . 713 | . 276 | . 318 | . 587 | . 487 | . 527 | . 600 | . 660 | . 547 | . 431 | . 419 | . 425 | . 382 |
| VAR0 0004 | . 298 | . 453 | . 595 | 1.00 0 | . 594 | . 412 | . 461 | . 631 | . 426 | . 473 | . 569 | . 370 | . 362 | . 328 | . 434 | . 485 | . 360 |
| VAR0 0005 | . 504 | . 458 | . 713 | . 594 | 1.00 0 | . 367 | . 420 | . 583 | . 356 | . 441 | . 445 | . 484 | . 480 | . 364 | . 422 | . 461 | . 410 |
| $\begin{aligned} & \text { VARO } \\ & 0006 \end{aligned}$ | . 214 | . 246 | . 276 | . 412 | . 367 | 1.00 0 | . 503 | . 420 | . 333 | . 248 | . 400 | . 230 | . 294 | . 251 | . 428 | . 342 | . 369 |
| VAR0 0007 | . 123 | . 220 | . 318 | . 461 | . 420 | . 503 | 1.00 0 | . 617 | . 443 | . 398 | . 409 | . 298 | . 406 | . 424 | . 402 | . 437 | . 363 |
| VAR0 0008 | . 315 | . 443 | . 587 | . 631 | . 583 | . 420 | . 617 | 1.00 0 | . 627 | . 637 | . 648 | . 586 | . 512 | . 585 | . 657 | . 627 | . 501 |
| VAR0 0009 | . 275 | . 461 | . 487 | . 426 | . 356 | . 333 | . 443 | . 627 | 1.00 0 | . 545 | . 653 | . 563 | . 421 | . 497 | . 525 | . 684 | . 459 |
| VARO 0010 | . 339 | . 458 | . 527 | . 473 | . 441 | . 248 | . 398 | . 637 | . 545 | 1.00 0 | . 559 | . 658 | . 550 | . 533 | . 565 | . 606 | . 423 |
| VARO 0011 | . 387 | . 437 | . 600 | . 569 | . 445 | . 400 | . 409 | . 648 | . 653 | . 559 | $\begin{array}{r} 1.00 \\ 0 \end{array}$ | . 537 | . 508 | . 417 | . 602 | . 644 | . 401 |
| VARO 0012 | . 409 | . 546 | . 660 | . 370 | . 484 | . 230 | . 298 | . 586 | . 563 | . 658 | . 537 | 1.000 | . 611 | . 579 | . 612 | . 629 | . 467 |
| $\begin{aligned} & \text { VARO } \\ & 0013 \end{aligned}$ | . 515 | . 483 | . 547 | . 362 | . 480 | . 294 | . 406 | . 512 | . 421 | . 550 | . 508 | . 611 | 1.000 | . 604 | . 522 | . 536 | . 572 |
| VAR0 0014 | . 365 | . 377 | . 431 | . 328 | . 364 | . 251 | . 424 | . 585 | . 497 | . 533 | . 417 | . 579 | . 604 | 1.000 | . 578 | . 573 | . 507 |
| VARO 0015 | . 298 | . 427 | . 419 | . 434 | . 422 | . 428 | . 402 | . 657 | . 525 | . 565 | . 602 | . 612 | . 522 | . 578 | 1.000 | . 800 | . 657 |


| VARO 0016 | . 303 | . 469 | . 425 | . 485 | . 461 | . 342 | . 437 | . 627 | . 684 | . 606 | . 644 | . 629 | . 536 | . 573 | . 800 | 1.000 | . 616 |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| VAR0 0017 | . 322 | . 333 | . 382 | . 360 | . 410 | . 369 | . 363 | . 501 | . 459 | . 423 | . 401 | . 467 | . 572 | . 507 | . 657 | . 616 | 1.000 |

Scale Statistics

| Mean | Variance | Std. Deviation | N of Items |
| :---: | ---: | ---: | ---: |
| 25.8800 | 102.733 | 10.13573 | 17 |

## APPENDIX B2

Reliability
Scale: DIFFICULTIES IN EMOTION REGULATION SCALE (DERS)
Case Processing Summary

|  |  | N | $\%$ |
| :--- | :--- | ---: | ---: |
| Cases | Valid | 74 | 74.0 |
|  | Excluded |  |  |
|  | a | 26 | 26.0 |
|  | Total | 100 | 100.0 |

a. Listwise deletion based on all variables in the procedure.

Reliability Statistics

| Cronbach's Alpha | N of Items |
| ---: | ---: |
| .912 | 36 |

## APPENDIX B3

## Reliability

Scale: Neuroticism

|  |  | N | \% |
| :---: | :---: | :---: | :---: |
| Cases | Valid | 90 | 90.0 |
|  | Excluded ${ }^{\text {a }}$ | 10 | 10.0 |
|  | Total | 100 | 100.0 |

a. Listwise deletion based on all variables in the procedure.

Reliability Statistics
Cronbach's Alpha N of Items


## Factor Analysis

| KMO and Bartlett's Test |  |  |
| :--- | ---: | ---: |
| Kaiser-Meyer-Olkin Measure of Sampling Adequacy. | .607 |  |
| Bartlett's Test of Sphericity | Approx. Chi-Square | 146.663 |
|  | Df | 28 |
|  | Sig. | .000 |

Communalities

|  | Initial |  |
| :--- | ---: | ---: |
| Extraction |  |  |
| VAR00103 | .152 | .099 |
| VAR00104 | .296 | .063 |
| VAR00105 | .376 | .302 |
| VAR00106 | .459 | .521 |
| VAR00107 | .329 | .120 |
| VAR00108 | .406 | .412 |
| VAR00109 | .130 | .000 |
| VAR00110 | .403 | .489 |

Extraction Method: Maximum Likelihood.

Total Variance Explained

| Factor | Initial Eigenvalues |  |  |  | Extraction Sums of Squared Loadings |  |  |
| :--- | ---: | ---: | ---: | ---: | ---: | ---: | :---: |
|  | Total | \% of Variance | Cumulative \% | Total | \% of Variance | Cumulative \% |  |
| 1 | 2.635 | 32.943 | 32.943 | 2.006 | 25.079 | 25.079 |  |
| 2 | 1.468 | 18.354 | 51.297 |  |  |  |  |
| 3 | .957 | 11.957 | 63.255 |  |  |  |  |
| 4 | .860 | 10.753 | 74.007 |  |  |  |  |
| 5 | .735 | 9.183 | 83.190 |  |  |  |  |
| 6 | .646 | 8.072 | 91.262 |  |  |  |  |
| 8 | .405 | 5.062 | 96.324 |  |  |  |  |

Extraction Method: Maximum Likelihood.

Factor Matrix ${ }^{\text {a }}$

|  | Factor |
| :--- | ---: |
|  | 1 |
| VAR00103 | .314 |
| VAR00104 | .251 |
| VAR00105 | .550 |
| VAR00106 | .722 |
| VAR00107 | .346 |
| VAR00108 | .642 |
| VAR00109 | .019 |
| VAR00110 | .699 |

Goodness-of-fit Test

| Chi-Square | Df | Sig. |
| ---: | ---: | ---: |
| 52.765 | 20 | .000 |

## APPENDIX B4

Reliability
Scale: PSQI

| Case Processing Summary |  |  |  |
| :--- | :--- | ---: | ---: |
| Cases | N | $\%$ |  |
|  | Valid | 82 | 82.0 |
|  | Excluded |  |  |
|  | Total | 18 | 18.0 |
|  |  | 100 | 100.0 |

a. Listwise deletion based on all variables in the procedure.

Reliability Statistics

| Cronbach's Alpha | N of Items |
| ---: | ---: |
| .726 | 17 |

## APPENDIX C-MAIN RESULT

C1:Frequencies

## Statistics

|  | Gender | Marital_Status | Educ | Religion |
| :--- | :--- | :--- | :--- | :--- |


| N | Valid | 403 | 403 | 403 | 403 |
| :--- | :--- | ---: | ---: | ---: | ---: |
|  | Missing | 1 | 1 | 1 | 1 |
| Range | 1.00 | 3.00 | 2.00 | 3.00 |  |
| Minimum | 1.00 | 1.00 | 1.00 | 1.00 |  |
| Maximum | 2.00 | 4.00 | 3.00 | 4.00 |  |

## Frequency Table

| Gender |  |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: |
|  |  | Frequency | Percent | Valid Percent | Cumulative <br> Percent |
|  | Male | 228 | 56.4 | 56.6 | 56.6 |
| Valid | Female | 176 | 43.5 | 43.4 | 100.0 |
|  | Total | 404 | 100 | 100.0 |  |
| Total |  | 404 | 100.0 |  |  |

Marital_Status

|  |  | Frequency | Percent | Valid Percent | Cumulative <br> Percent |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Valid | Single | 306 | 75.7 | 75.9 | 75.9 |
|  | Married | 94 | 23.3 | 23.3 | 99.3 |
|  | Widow | 4 | . 9 | . 7 | 100.0 |
|  | Total | 404 | 100 | 100.0 |  |
| Total |  | 404 | 100.0 |  |  |

Educ

|  | Frequency | Percent | Valid Percent | Cumulative <br> Percent |  |
| :--- | :--- | ---: | ---: | ---: | ---: |
| Valid | PGD | 39 | 9.7 | 9.7 | 9.7 |
|  | MSc | 347 | 85.9 | 86.1 | 95.8 |
|  | PhD | 18 | 4.4 | 4.2 | 100.0 |
| Total | Total | 404 | 100 | 100.0 |  |

Religion

|  | Frequency | Percent | Valid Percent | Cumulative <br> Percent |
| :--- | :--- | :--- | :--- | :---: |


|  | Xtian | 387 | 95.8 | 96.0 | 96.0 |
| :--- | :--- | ---: | ---: | ---: | ---: |
|  | Moslem | 13 | 3.2 | 3.2 | 99.3 |
| Valid | ATR | 2 | .5 | .5 | 99.8 |
|  | Others | 2 | .4 | .2 | 100.0 |
|  | Total | 404 | 100 | 100.0 |  |

## Descriptives

| Descriptive Statistics |  |  |  |  |  |
| :--- | ---: | ---: | ---: | ---: | ---: |
| Age | N | Minimum | Maximum | Mean | Std. Deviation |
| Valid N (listwise) | 403 | 22.00 | 58.00 | 30.6030 | 6.84267 |

## C2: Correlations

| Descriptive Statistics |  |  |  |
| :--- | ---: | ---: | ---: |
| Mean | Std. Deviation | N |  |
| Gender | 1.4342 | .49627 | 404 |
| Age | 30.6030 | 6.84267 | 404 |
| Marital_Status | 1.2556 | .48529 | 404 |
| Educ | 1.9454 | .36921 | 404 |
| Religion | 1.0496 | .26862 | 404 |
| HTQ1 | 26.3896 | 9.52293 | 404 |
| DERS | 70.5633 | 14.59070 | 404 |
| Neuroticism | 19.2015 | 5.13185 | 404 |
| Sleep_Quality | 13.9525 | 5.85585 | 404 |

Correlations

|  | Gender | Age | Marital_Status | Educ | Religion | HTQ1 | DERS | Neuroticism | Sleep_Quality |
| :--- | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |


**. Correlation is significant at the 0.01 level ( 2 -tailed).
*. Correlation is significant at the 0.05 level ( 2 -tailed).

## C3: Regression

| Variables Entered/Removed $^{\text {a }}$ |  |  |  |
| :--- | :--- | :--- | :--- |
| Model | Variables Entered | Variables <br> Removed | Method |
| 1 | HTQ1 $^{\text {b }}$ |  |  |
| DERS |  |  |  |

a. Dependent Variable: Sleep_Quality
b. All requested variables entered.

Model Summary

| Model | R | R <br> Square | Adjusted R Square | Std. Error of the Estimate | Change Statistics |  |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  |  |  |  |  | R Square Change | F Change | df1 | df2 | Sig. F <br> Change |
| 1 | . $119^{\text {a }}$ | . 014 | . 012 | 5.77617 | . 014 | 5.676 | 1 | 396 | . 018 |
| 2 | . $394{ }^{\text {b }}$ | . 155 | . 151 | 5.35428 | . 141 | 65.865 | 1 | 395 | . 000 |
| 3 | . $407^{\text {c }}$ | . 166 | . 160 | 5.32645 | . 011 | 5.138 | 1 | 394 | . 024 |

a. Predictors: (Constant), HTQ1
b. Predictors: (Constant), HTQ1, DERS
c. Predictors: (Constant), HTQ1, DERS, Neuroticism

ANOVA ${ }^{\text {a }}$

| Model | Sum of Squares | df | Mean Square | F | Sig. |
| :--- | :---: | :---: | :---: | :---: | :---: |


a. Dependent Variable: Sleep_Quality
b. Predictors: (Constant), HTQ1
c. Predictors: (Constant), HTQ1, DERS
d. Predictors: (Constant), HTQ1, DERS, Neuroticism

Coefficients ${ }^{\text {a }}$

| Model | Unstandardized Coefficients |  | Standardized <br> Coefficients <br> Beta | t | Sig. | 95.0\% Confidence Interval for B |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | B | Std. Error |  |  |  | Lower Bound | Upper Bound |
| (Constant) | 11.991 | . 850 |  | 14.110 | . 000 | 10.320 | 13.662 |
| HTQ1 | . 072 | . 030 | . 119 | 2.382 | . 018 | . 013 | . 132 |
| (Constant) | 2.029 | 1.459 |  | 1.391 | . 165 | -. 838 | 4.897 |
| 2 HTQ1 | . 043 | . 028 | . 071 | 1.517 | . 130 | -. 013 | . 098 |
| DERS | . 152 | . 019 | . 378 | 8.116 | . 000 | . 116 | . 189 |
| (Constant) | . 736 | 1.559 |  | . 472 | . 637 | -2.329 | 3.801 |
| HTQ1 | . 040 | . 028 | . 067 | 1.438 | . 151 | -. 015 | . 096 |
| 3 DERS | . 138 | . 020 | . 342 | 6.956 | . 000 | . 099 | . 177 |
| Neuroticism | . 125 | . 055 | . 111 | 2.267 | . 024 | . 017 | . 233 |

a. Dependent Variable: Sleep_Quality

Excluded Variables ${ }^{\text {a }}$

| Model | Beta $\ln$ | t | Sig. | Partial Correlation | Collinearity <br> Statistics |
| :--- | :---: | :---: | :---: | :---: | :---: |


|  |  |  |  |  |  | Tolerance |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | DERS | . $378{ }^{\text {b }}$ | 8.116 | . 000 | . 378 | . 984 |
| 1 | Neuroticism | . $223{ }^{\text {b }}$ | 4.561 | . 000 | . 224 | . 994 |
| 2 | Neuroticism | . $111^{\text {c }}$ | 2.267 | . 024 | . 113 | . 886 |

a. Dependent Variable: Sleep_Quality
b. Predictors in the Model: (Constant), HTQ1
c. Predictors in the Model: (Constant), HTQ1, DERS

