Examining Negative Emotional States, Daytime Drowsiness & Resting-State Cognition On Resting-State EEG Patterns & Sleep Quality In Young & Older Adults

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Abstract

As individuals age, sleep quality is reported to decrease compared to earlier stages of life. These declines often coincide with an increase in the incidences of poor health outcomes, including physiological, psychological and neurodegenerative disorders. Given the critical link between sleep quality and health in ageing, addressing sleep concerns may be vital to encourage healthier ageing. However, it is important to recognise the reciprocal relationship between these factors. Poor lifestyle factors and illness can create sleep disturbances, while poor sleep quality may also exacerbate health conditions. Therefore, it is vital to consider whether poor sleep quality necessarily worsens with age, which may serve as a catalyst for age-related pathologies, or if the onset of such pathologies may exacerbate sleep disturbances, leading to a cycle of declining health and sleep quality.

Further complexity arises in the exploration of these variables, as the brain regions responsible for arousal and sleep regulation deteriorate with age. Currently, there is limited research examining whether such deterioration results in changes in sleep patterns or manifests as abnormal arousal regulation, let alone whether these factors should be a concern when understanding the development of poor sleep outcomes in older adults. While efforts exist to investigate the relationships between arousal regulation, the development of poor sleep outcomes, and affective disorders, these parameters have not been explicitly studied in older populations. Additionally, the tools used to examine changes in sleep quality are complicated by a lack of standardisation in methods, particularly concerning EEG measurements for monitoring brain activity and its associations with various forms of waking cognition and psychological health.

To begin address these complex relationships, this thesis investigates whether negative emotional states, daytime drowsiness, resting-state cognition, and specific features of resting-state EEG can explain variations in sleep quality. Moreover, it explores whether

these factors produce differential effects in young versus older adults. The need for age-group contrasts is important, as comparing these variables between younger and older adults may identify age-specific drivers. This can highlight factors unique to older age that contribute to the development of sleep deficits and their unique impact on older adults. Such comparisons would also determine if the same tools used to investigate these variables in younger adults can be robustly applied to older adults. The primary focus of this study is to discern whether there may be age-exclusive factors that impact sleep quality in the absence of large pathologies, such as sleep and psychiatric disorders.

This thesis is structured around three primary goals. Firstly, to investigate the relationships between sleep quality, negative emotional states, daytime drowsiness, and resting-state cognition. Secondly, to explore whether the arousal regulation hypothesis can serve as a model for implicating age-related changes in the brain, and how it may provide links between psychological dimensions and sleep quality. Lastly, this thesis aims to address the methodological considerations regarding resting-state EEG components, and consolidate contemporary best practices for age-related comparisons and highlight their importance in understanding the ageing brain.

The present study included 23 healthy young adults (aged 18-30) and 22 healthy older adults (over 60 years old). Participants completed the PSQI for subjective sleep quality, the DASS to measure symptoms of depression, anxiety, and stress, and the ESS to assess the severity of daytime drowsiness. They also underwent a 20-minute eyes-closed resting-state EEG recording, followed by a post-recording ARSQ questionnaire to measure their resting-state cognition. Arousal regulation measures were extracted from the resting-state EEG recordings using VIGALL 2.1. Specific features of the EEG signal, including periodic and aperiodic components, were processed via the FOOOF algorithm to enable comparisons of alpha and theta power. Further scrutiny was performed to examine the conventional approach

versus the individualised approach in bandwidth power comparisons, a consideration vital for cross age-group comparisons. Lastly, elastic net regression was performed to develop age-group models, aiming to better understand the effects of various variables on sleep quality, and to identify if there were any differentiated predictors of sleep quality between young and older adults.

The findings of this thesis challenge the notion that poor sleep quality is an inevitable consequence of ageing. Contrary to previous literature, the older adults in this sample did not exhibit significantly worse sleep compared to young adults and reported fairly good scores across most indices, consistent with the absence of large pathologies. In addition to that, no age-group differences emerged in arousal regulation patterns, despite robust age-group differences in sleep quality and negative emotional states. This suggests that changes in arousal regulation may not solely depend on the severity of negative emotional states (e.g., depression) but may be modulated only in individuals with affective disorders. This finding provides valuable insight into the utilisation of the VIGALL and enriches the understanding of the arousal regulation hypothesis. Furthermore, extensive comparisons between conventional and parameterised approaches to EEG spectral analysis underscore the importance of individualised definitions of frequency bands and the need for standardisation of methods, particularly when comparing spectral properties between young and older adults. Notably, the parameterisation and individualisation of EEG signals eliminated age-group effects observed with conventional approaches, indicating that conventional methods may conflate EEG signal properties and lead to misinterpretations. This is crucial for a better understanding of the underlying neural mechanisms that may influence sleep quality.

Overall, these results suggest that age may not be the sole driver of sleep quality differences as commonly reported. Instead, future research should explore age-related pathology in relation to sleep, rather than simply attributing sleep changes to ageing.

Additionally, this thesis demonstrates the importance of considering age-related sensitivities in EEG measures. Failure to account for such sensitivities could lead to misinterpretation of findings, making it challenging to draw meaningful conclusions, especially in the context of age and sleep quality.

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Table of Abbreviations

Abbreviation	Meaning
ADHD	Attention-Deficit / Hyperactivity Disorder
ARAS	Ascending Reticular Activating System
ARSQ	Amsterdam Resting State Questionnaire
ASS	Arousal Stability Score
BVA	BrainVision Analyzer
DASS	Depression Anxiety Stress Scale
DoM	Discontinuity of Mind
EDS	Excessive Daytime Sleepiness
EEG	Electroencephalography
ESS	Epworth Sleepiness Scale
FOOOF	Fitting Oscillations & One-Over F
IAF	Individual Alpha Frequency
IAPF	Individual Alpha Peak Frequency
ICA	Independent Component Analysis
MCI	Mild Cognitive Impairment
MDD	Major Depressive Disorder
MINMSE	Minimum Mean Squared Error
MoCA	Montreal Cognitive Assessment
MSE1SE	Mean Squared Error Minus One Standard Error
NREM	Non-Rapid Eye Movement
PSD	Power Spectral Density
PSQI	Pittsburgh Sleep Quality Index
REM	Rapid Eye Movement
rsEEG	Resting-State Electroencephalography
ToM	Theory of Mind
VIGALL	Vigilance Algorithm Leipzig

Chapter 1: Background & Literature Review

1.1. Importance of Sleep

The phenomenon of sleep is loosely defined as a period of inactivity that is accompanied by a general disengagement of perceptual processes, which include a lack of responsiveness to the external environment as well as rest-related behaviours such as eye closure, motor inhibition and the presence of dreams (Carskadon & Dement, 2005). This state of sleep produces significant effects on the body, affecting the cellular and physiological processes that are vital to the healthy functioning of the body (Coutrot et al., 2022).

On the surface level, the primary functions of sleep are restorative. That is, the period of sleep allows organisms to conserve energy and recover from fatigue or other expenditures of energy (Shapiro & Flanigan, 1993). At the physiological level, sleep modulates protein synthesis and muscle repair, which are important for recovery and energy conservation (Dattilo et al., 2011). In addition to that, sleep also regulates circadian and hormonal functions, through regulations of cortisol, growth hormones, prolactin, thyroid-stimulating hormones and melatonin secretions (Carley & Farabi, 2016; Duan et al., 2022). Besides that, sleep also provides mitigation effects against adverse health diseases such as diabetes through the regulation of hormones relating to appetite regulation and glucose metabolism (Duan et al., 2022). Furthermore, sleep also supports immunological function by modulating cytokine levels during sickness and managing systemic inflammation (Zisapel, 2007). It is also during these periods of sleep that metabolic waste, such as amyloid-b and cerebral tau are cleared from the brain (Johnson et al., 2012).

At the cognitive level, sleep is heavily involved with the memory consolidation process. For instance, the consolidation of declarative memories such as episodic and

semantic memory is linked with early stages of sleep, while rapid eye movement (REM) sleep is linked with the consolidation of emotional and procedural memories (Siegel, 2001). Good sleep is also important for healthy psychological wellbeing. For instance, having poorer sleep is associated with higher reports of dissatisfaction in life, higher psychological distress and poorer self-reported mental health (Rodrigues et al., 2022). Besides that, having poor sleep is also linked with a higher risk of developing depressive, bipolar and psychotic disorders (Scott et al., 2021). Likewise, having better sleep efficiency is also linked to faster recovery from physical stress (Eiman et al., 2019).

1.2. Structure of Sleep

The phases of sleep are conventionally divided into two categories that cycle across a period of sleep: non-rapid eye movement sleep (NREM) which includes different stages such as N1, N2, and N3; and REM sleep. A typical period of sleep consists of four to five repeating cycles going from N1, N2, N3, N2, and REM (Carskadon & Dement, 2011). NREM sleep makes up about 75-80% of sleep and is characterised by slow eye movements along with neural oscillations in the lower frequencies. N1 sleep is typically marked by the appearance of low-voltage theta waves. This progresses into N2 sleep, mainly characterised by the appearance of sleep spindles and K-complexes accompanied by a drop in heart rate and body temperature. It is also during this stage that consolidation of procedural and declarative memories is believed to occur (Antony et al., 2019). Lastly, N3 sleep also called slow-wave sleep is characterised by high amplitude delta waves and is where physiological repair and the strengthening of the immune system happens (Assefa et al., 2015). In contrast to the NREM stages, REM sleep is associated with high-frequency waves and rapid eye movements in all directions, and is usually accompanied by reduced muscle activity and is the stage where most dreams are believed to occur (Coleman, 1999, Patel et al., 2022).

Curiously, the structure of these sleep stages is dynamic across the lifespan. Newborns show no regular rhythm of sleep-wake cycles and spend about 16-18 hours of the day asleep. Unlike typical cycles of sleep, the onset of sleep in newborns starts with REM sleep and only cycles with NREM sleep once or twice in a sleep episode (Ohayon et al., 2004). Going into early childhood, the hours of sleep decrease to 11-13 hours, and their sleep cycles tend to be moderated by circadian phases (Gaudreau et al., 2001). This number then further reduces to 9-10 hours by adolescence and the time spent in N2 sleep increases. By adulthood, the structure of sleep stages tends to stabilise until senescence, where sleep patterns tend to take on slightly divergent trends, particularly with total sleep durations. In older age, slow-wave sleep tends to decrease, however, total sleep durations have been reported to increase, decrease or remain unchanged when compared to adulthood (Chaput et al., 2020, Coutrot et al., 2022; Ohayon et al., 2004). These patterns highlight an important point: the structure of sleep evolves across the lifespan, indicating that sleep patterns vary in older age. This observation holds particular significance for the premise of the present thesis.

1.3. Sleep Disorders & Age-Related Declines in Older Adults

Understanding sleep disturbances in the ageing population is of high clinical importance. According to a survey of older adults over the age of 65, Foley et al. (1995) found that 42% of older adults have difficulty initiating and maintaining sleep. Older adults tend to report fragmented sleep, longer times to fall asleep, an increase in nighttime awakenings, more daytime sleepiness and napping, and a decreased quality of sleep compared to younger adults (Gulia & Kumar, 2018).

Sleep disorders span across six major types: (i) insomnias, which are characterised by challenges with sleep initiation, sleep maintenance, daytime fatigue, mood disturbances and reduced productivity in the daytime (Sateia, 2014); (ii) circadian rhythm disorders, which are sleep problems that arise due to misalignment of the endogenous circadian system with an

individual's sleep habits (Pavlova & Latreille, 2019); (iii) sleep-disordered breathing, which include obstructive, central and complex sleep apneas (Kuźniar & Kasibowska-Kuźniar, 2011); (iv) hypersomnias, which are characterised by excessive fatigue in the day typically due to narcolepsy, which describe a class of symptoms that include sleep paralysis, hypnagogic hallucinations and cataplexy (sudden loss of muscle tone) (Dauvilliers & Barateau, 2017); (v) parasomnias, which describe a group of behaviours relating to sleep disturbances depending on the sleep stage in which they occur. For example, NREM parasomnias can include sleepwalking, confused arousals, and night terrors, while REM parasomnias include acting out dream activity (e.g. kicking, screaming, and talking while asleep; Howell, 2012); (iv) and restless leg syndrome, referring to uncomfortable sensations and urges to move the limbs while at rest, which tend to worsen at night (Leschziner & Gringras, 2012).

The incidences of these disorders are more prevalent in the ageing population. Insomnias are the most prevalent sleep problem in older adults, with about 40-50% of adults above 60 reporting symptoms of insomnia, in contrast to approximately 30% in the general adult population (Ancoli-Israel et al. 2008; Roth, 2007). While there are no direct prevalence rates of circadian rhythm disorders reported in older adults, other circadian outputs have shown deficits in older age. These include a decline in melatonin secretion in older adults, lower amplitude body temperature, and earlier phases of body temperature regulatory rhythms when compared to younger adults, all of which affect the timing of sleep (Czeisler et al., 1992; Karasek, 2004; Logan & McClung, 2019). In the case of sleep-disordered breathing, some reports roughly identify that about 32- 81% of adults aged 65-95 years old meet the diagnosis for a lower degree of sleep-disordered breathing (e.g. sleep apneas), in contrast to about 11-50% in middle-aged adults (Fell & Kaplan, 1991; Heinzer et al., 2015; Young, 2002). Similarly, hypersomnias have been identified to be more frequent in older

adults, with one report showing that 23% of adults 78-102 years old meet the diagnostic criteria for some form of hypersomnia (Miner et al., 2019). Parasomnias by themselves, such as sleep paralysis are less common in the ageing population (Iranzo, 2018). However, parasomnias are highly comorbid with certain neurodegenerative disorders, such as dementia with Lewy bodies, Parkinson's disease, and multiple system atrophy, which themselves are more prevalent in older adults (Iranzo et al., 2016). In addition to that, restless leg syndrome is also more prevalent in older adults, with reports putting prevalence rates of restless leg syndrome at about 8.6-9.8% in older adults compared to approximately 2.7–7.2% in people under the ages of 60 (Ohayon & Roth, 2002; Rothdach et al., 2000).

These sleep disorders do not pose problems only in themselves but are also a catalyst for a range of psychological, cognitive and health problems. For instance, in a large sample of 42,116 older adults, Koyanagi et al. (2014) found that older adults who report sleep problems are also more likely to suffer from a large range of chronic health issues, such as angina, arthritis, asthma, chronic lung disease, diabetes, hypertension, obesity, strokes. As a similar pattern, longitudinal observations of older adults who report sleep problems also tend to be more likely to develop neurodegenerative disorders, such as Parkinson's disease and Alzheimer's dementia and are at higher risk of all-cause mortality (Kim et al., 2022; Li & Qian, 2023; Wu et al., 2022). These findings are quite robust, with a systematic review containing over 4.4 million participants across 30 countries finding that sleep duration that is too short (< 7 hours) or too long (> 8 hours) is associated with an increase in many negative health outcomes, including cardiovascular diseases, Type 2 diabetes, mental health, cognitive decline, incident obesity and osteoporosis (Chaput et al., 2020). Furthermore, Cao et al., (2022) found in their sample of 7,924 participants that poorer sleep was also associated with lower global cognitive scores. In addition to that, Nadorff et al., (2018) also found strong

correlations between sleep disorders and the incidences of psychiatric problems, such as depression, anxiety, and suicidal ideation.

Of noteworthy aspect, the effects of poor sleep on negative health outcomes do not have a linear relationship across the lifespan. In a study by Hinz et al. (2017), who examined the subjective sleep quality of adults aged 18-80, they observed that sleep quality tends to decrease gradually from young adulthood to about 50 years old. However, beyond that point, this trend tends to level off, showing consistently poor sleep quality up to 80 years old which is worse compared to sleep quality in the first 50 years of life. A similar observation was made by Coutrot et al. (2022), who found significant shifts in sleeping habits starting at the age of 53, with adults beyond that age sleeping longer as they age. Coutrot et al. (2022) attributed these shifts to changes in lifestyle, such as the age period where child-rearing and work-related responsibilities tend to reduce, therefore allowing more time for sleep. A comparison of these two studies presents an interesting image. From early life up to about 50 years, both sleep duration and sleep quality tend to decrease similarly, however, this trend starts to diverge in senescence. Despite longer sleep durations, older adults report worse sleep quality.

As a general overview, sleep disorders and poor sleep are heavily correlated with many adverse health outcomes, ranging from physiological, psychological, and cognitive problems. Older adults tend to report more problems across all these domains, so it is of clinical importance to better understand the relationships between these factors, to be able to better treat and mitigate the deterioration in quality of life as humans age.

1.4. Complex Nature of Sleep, Ageing & Disease

The common consensus within the literature is that higher incidence rates of sleep disorders in ageing adults are due to a variety of lifestyle and ageing-related factors, rather than due to

age itself (e.g. Chaput et al., 2020). To illustrate this point more clearly, human ageing accompanies many changes, such as deterioration of the suprachiasmatic nucleus, which can result in less synchronised sleep-wake cycles (Ancoli-Israel et al., 2008). Similarly, as people age, they may become less active, which can decrease light exposure and result in shifts in hormonal, endocrinal and circadian rhythms (Li et al., 2018). In addition to that, older adults may tend to experience less social interaction, or be more likely to encounter stressful situations (e.g. loss of jobs, or the death of a friend etc.) that can contribute to developing certain psychiatric disorders (Li et al., 2018). From this interpretation, it would suggest that poor sleep occurs as a symptom of pathological ageing, rather than ageing by itself.

However, this interpretation becomes a little more complex when considering that the relationship between poor sleep and pathological ageing may also occur in the inverse direction, such that symptoms of pathological ageing may be onset by poor sleep. For instance, Keage et al. (2012) who surveyed 2,012 older adults found that insomnia symptoms precede the onset of age-related decline symptoms. In a similar recent study by Otaiku (2022) who observed 2,770 older men over a decade found that those who had reported poor sleep quality in the earlier phases of the study were also more likely to develop Parkinson's disease later in life. A similar pattern is also observed for the development of chronic health illnesses. Wang et al. (2022) compared sleep records obtained in 2011 to a follow-up of health conditions in 2018 from 7,025 middle-aged and older adults and found that those who had reported poor sleep earlier in life were also more likely to have developed chronic illnesses, such as heart disease, diabetes, high blood pressure, hypertension and kidney disease in the follow-up screenings. More recently, two studies have employed a more novel method of comparing chronological brain age with a model-estimated brain age in relation to ageing. These studies identified that in contrast to people with healthy sleep, people with disordered sleep have physically "older" brains (i.e. higher cortical thinning in functional areas) when

compared to their actual chronological age brains, which in turn might accelerate the development of age-related negative health problems (Ramduny et al., 2022; Yook et al., 2022).

Considering the evidence, there should be little contention that the relationship between sleep, ageing and negative health outcomes is reciprocal. However, there is a lack in the literature offering a synthesis of these interpretations, and it is unclear how causal these factors are in relation to each other. Mapping these relationships could be incredibly beneficial and may offer high clinical importance. Namely, in the treatment of chronic illnesses, should early interventions aim to detect and treat sleep-related pathologies first, as to prevent future related adverse health effects? Or should chronic illness be managed first, with sleep therapies being treated secondarily to mitigate a worsening of symptoms? Until more explicit endeavours are taken to outline this relationship, the treatment of chronic illness in old age cannot be targeted to tackle the root problem and may merely be treating symptoms of an underlying issue. To begin addressing these questions, the present thesis will bring attention to the primary method of investigation used in the study — electroencephalography (EEG).

1.5. What Resting-State Measurements Are & Why They Relate to Sleep

The resting state refers to brain activity that is not task or event-specific. It may also generally be referred to as the default mode network, which includes looking at network-wide brain activity and may represent a fundamental aspect of neural communication (Shen, 2015). In the context of studying sleep-related measures, the resting state coupled with the closing of the eyes can mimic the initial mental and physical conditions of waking before sleep (Diaz et al., 2016). Resting-state EEGs (rsEEG) measure the spontaneous electrical activity of the brain at rest, and these recorded signals may be used to infer certain characteristics about an

individual's brain and mental health (Khanna et al., 2015). The use of rsEEG in ageing research is increasingly popular and appears to be quite versatile. For instance, several researchers have called for the use of rsEEG as a diagnostic tool to track the progression of neurodegenerative disorders (Babiloni et al., 2016; Cassani et al., 2018). Similarly, rsEEG recordings can also reveal early signs of mild cognitive impairment, or general issues with memory and cognition (Meghdadi et al., 2021; Trammell et al., 2017).

One common method of analysing EEG data includes spectral or narrowband analysis, that is the splitting of a raw EEG signal into bandwidths of certain frequency parameters. These canonical frequency bands include delta (2–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), beta (15–30 Hz), lower gamma (30–80 Hz), and upper gamma (80–150 Hz). Within these bandwidths, the power and phases of these signals can be used to infer certain characteristics of brain activity. For example, alpha activity is associated with non-arousal while beta activity is associated with higher forms of arousal; meanwhile, theta activity can refer to a restful, drowsy state (Annarumma et al., 2018; Miller, 2007).

1.6. Exploring Drowsiness as The Link Between Sleep & Age-Related Decline

The transition from waking to sleep is usually marked by a reduction in alpha activity, simultaneous with an increase in theta activity, and this ratio is also commonly used to mark a change in waking arousal (Jiao et al., 2020; Makeig et al., 2000). While there is no clear demarcation between these states, efforts have been made to classify "drowsy" periods using observations of relative theta and alpha activity within a time window (Strijbis et al., 2022). Theta power in the resting state is an indicator of the homeostatic sleep drive, referring to the increased need for sleep accompanied by subjective feelings of drowsiness (Cajochen et al., 2010; Strijkstra et al., 2003). Theta power has been shown to increase across hours spent awake and prolonged periods of sleep deprivation, and often follows a return to baseline

levels after a period of prolonged recovery sleep (Hao et al., 2023; Snipes et al., 2023). In the context of sleep and ageing, early signs of brain pathologies can be examined by analysing the spectral properties of rsEEG recordings. For example, healthy older adults tend to show a decrease in theta and delta power, with an increase in beta activity in the resting-state when compared to young healthy adults (Barry & De Blasio, 2017). In contrast, older adults with signs of pathological ageing tend to show an increase in delta and theta activity, with a decrease in beta (Fröhlich et al., 2021; Meghdadi et al., 2021). These observed patterns also tend to show an overlap with findings from sleep studies. For instance, some insomniacs tend to show increased theta power in the resting-state (Zhao et al., 2021). In addition to that, patients who suffer from sleep-disordered breathing also tend to show higher delta and theta activity during resting-state wakefulness (Wu et al., 2020).

1.7. Examining Age-Related Differences In Alpha & Theta Power

Across the literature, theta power has both been shown to decrease and increase in old age. As an overview, “healthy” older adults tend to report lower theta activity (e.g. Barry & De Blasio, 2017; Vlahou et al., 2014), while older adults with signs of pathology tend to report higher theta activity (e.g. Babiloni et al., 2016). Until recently, these diverging patterns were not addressed but were finally given attention by Cesnaite et al. (2023). They argued that the differences in theta power as reported in the literature are due to other features of EEG signals that are often overlooked in typical EEG designs, rather than due to differences in theta power specifically.

Neural activity as measured in typical EEG recordings reflects rhythmic oscillatory (i.e. periodic; such as alpha and theta waves) and non-rhythmic (i.e. aperiodic) components, with non-rhythmic components reflecting spontaneous neural noise. The aperiodic component is characterised by the 1/f slope which reflects the ratio of excitatory and

inhibitory inputs (Donoghue et al., 2020). Specifically, the 1/f slope is flatter in older adults aged 60 and above, and this is believed to reflect higher excitability and “noisy” baseline neural activity (Voytek et al., 2015b). Cesnaite et al. (2023) propose that it is this underlying feature that creates the difference in findings in slow-wave activity in older age (i.e. theta). Due to the shape of the 1/f slope, the aperiodic components exert a larger influence on the overall power distribution at low frequencies (e.g. theta) compared to higher frequencies (e.g. alpha). As people get older, the increase in “neural noise”, reflected as a flattening of the 1/f slope will subsequently be conflated as theta power. As such, variability in theta power as reported in older age may not be due to changes in theta power per se, but due to changes in the underlying aperiodic component.

To examine this hypothesis, Cesnaite et al. (2023) looked at EEG data of over 1,700 older adults and evaluated the signals using the conventional versus the parameterised approach. The parameterised approach involves a separation of EEG signals into their periodic (i.e. narrowband parameters like alpha and theta oscillations) and aperiodic components, while the conventional approach makes no such distinction. Using the conventional approach, they found that alpha power tended to decrease with increasing age, which is consistent with previous findings (e.g. Fröhlich et al., 2021). However, after parameterising the data, the periodic components were no longer significantly different; rather, it was the non-rhythmic components (1/f slope) that showed flatter slopes with increasing age. This finding supports their initial hypothesis, showing that the age-related differences observed between slow-wave activity may be due to a change in the underlying noise rather than in the periodic oscillations themselves. Beyond parameterisation, Cesnaite et al. (2023) also included individualised approaches in defining the frequency bandwidths, contrasted with the canonical frequency ranges. Such approaches also have consequences for

the interpretation of theta power, however, this point will be further elaborated on and given focus in Chapter 4.

1.8. Exploring Arousal Regulation Systems To Examine Age-Related Changes In Sleep & Health Outcomes

As emphasised by the findings of Cesnaite et al. (2023), future examinations of age-related comparisons should take into account age-related changes in the brain. Alongside considerations regarding the treatment and analysis of neural signals, there may be merit in exploring conceptual approaches that may implicate psychological and neurobiological features that occur in ageing to assess sleep quality. One possible angle for such exploration involves the monitoring of arousal regulation systems.

As humans age, the within-network connectivity between brain regions tends to decrease, while between-network connectivity increases (Bethlehem et al., 2020; Guardia et al., 2022). Consequently, this manifests as reduced network segregation, resulting in less functional distinction between brain networks. Until recently, little evidence has explained what causes these age-related shifts in network organisation (Guardia et al., 2022). However, recent investigations by Guardia et al. (2022) propose that these network changes might be driven by age-related changes to the ascending reticular activating system (ARAS). Due to the ARAS' widespread connections across the cortex and its involvement with cognition, they suggest that changes affecting the ARAS might simultaneously affect the connectivity between cortical networks (Handra et al., 2019; Jones, 2003). In support of their hypotheses, Guardia et al. (2022) found that ARAS-cortical connections are reduced in older-aged adults, particularly in the locus coeruleus, which is an important neuromodulatory structure affecting many aspects of cognition. Notably, they also observed that weaker connections between the ARAS and cortical regions also produced worse cognitive performance (Handra et al., 2019).

Of importance to the present study, the ARAS is crucial in modulating awareness and arousal. Guardia et al. (2022) highlighted that in addition to network changes and cognition, deterioration of the ARAS also produces disruptions to the circadian and sleep-wake regulation systems.

In line with such arguments, previous research looking at the interactions between arousal regulation systems, sleep behaviour and the development of poor health outcomes also supports this interpretation. The vigilance regulation model as introduced by Hegerl and Hensch (2014) posits that affective disorders, such as depression and attention deficit hyperactivity disorder (ADHD) may be the result of poor arousal regulating mechanisms. Within the present context, vigilance is defined as neurophysiological arousal and is used interchangeably by the authors (Hegerl & Hensch, 2014; Oken et al., 2006). Succinctly, the authors describe stable vigilance regulation as a gradual descent from aroused to less aroused states under resting conditions. In contrast, unstable vigilance regulation describes rapid descends and shifts in arousal states, and hyperstable vigilance regulation describes a constant state of arousal that is resistant to changes in the environment even after a considerable amount of time. In such cases, the authors suggest that external behaviours manifest as an autoregulatory attempt to stabilise these states. These external behaviours then become characteristic of affective disorders. For instance, in response to unstable regulation, the body may attempt to stabilise arousal by creating stimulating activities, which appear as hyperactivity, talkativeness, novelty seeking, and impulsivity, which are behaviours common in ADHD and manic depression. Meanwhile, depressive symptoms such as avoidance and withdrawal may be the autoregulatory response to “destabilise” hyperstable vigilance.

Of relevance to the present study, this model as proposed by Hegerl and Hensch (2014) also implicates the systems that affect sleep-wake regulation. In the instance of unstable vigilance regulation, sleep deficits are often the earliest symptom of mania,

suggesting that the dysregulation in sleep-wake cycles may destabilise vigilance regulation and produce manic symptoms (Harvey, 2008). It was also noted by Hegerl and Hensch (2014) that ADHD and mania show similarities, as both sleep and circadian disorders are commonly reported with such conditions, where poor sleep tends to aggravate symptoms. Meanwhile, patients with depression tend to show hyperstable regulation, which also correlates with prolonged sleep onset latency along with inner restlessness and tension (Holsboer & Ising, 2010). Such reports are also corroborated by Ulke et al. (2017) who found that patients who have depression tend to suffer from more sleep disturbances, despite not feeling sleepy. Their study also found that in depressed patients, sleep disturbances were correlated with higher brain arousal stability scores. This was accompanied by increases in autonomic features, such as heart rate, skin conductance and muscle tone, all of which are characteristic of a hyperstable vigilance regulation (Carney et al., 2005)

According to Hegerl and Hensch (2014), the neurobiological basis of vigilance regulation is the central noradrenergic system, particularly the locus coeruleus. This is because the firing rate of neurons in the locus coeruleus appears to synchronise with changes across external and internally arousing events. This suggestion parallels Guardia et al. (2022)'s work, where the importance of the ARAS in affecting network connectivity and arousal regulation systems was highlighted. By considering their findings, a broader interpretation of the subject emerges. The neurobiological systems that regulate arousal regulation are vulnerable to age-related deterioration and are important in modulating sleep-wake rhythms (Guardia et al., 2022). Furthermore, dysregulation of either system is significant to the development of affective disorders, which can by themselves further aggravate other physiological conditions and reciprocally worsen the systems that maintain healthy functioning (Hegerl & Hensch, 2014).

Though combined evidence may support this interpretation, some gaps must be considered. While age-related differences in the neurobiological systems affecting arousal have been documented, it is still unexamined if the arousal regulation patterns (i.e. unstable or hyperstable regulation) reflect these age-related differences. Given that these systems shift and deteriorate in old age, do they also produce non-stable arousal regulation patterns? And if such patterns are observed, to what extent might they be explained by age-related decline? In the recent decade, it has become possible to study and differentiate states of arousal in the waking state via EEG activity using the Vigilance Algorithm Leipzig (VIGALL), a tool which was instrumental to the work relating to vigilance regulation (i.e. Hegerl & Hensch, 2014). However, few attempts have been made to use the VIGALL to compare age-related differences. Given the crucial aspect of age-related changes in these regulatory systems, it would be worth considering if the VIGALL may be robustly applied across different age groups to study sleep quality, an angle which will be further expanded on in Chapter 3.

1.9. Scope of Present Study & Thesis

To investigate the complex nature of sleep and ageing, the present thesis intends to outline possible relationships between neurobiological changes to the brain in older age and implicate them in the differences in sleep quality, along with related psychological variables that may modulate this relationship.

In this first chapter, a broad overview of the importance of sleep and intersecting findings was introduced (e.g. patterns observed in sleep studies contrasted with age-related studies). Across the literature, common features exist between changes in sleep patterns across ageing, ageing and the onset of pathology, as well as the presence of pathology and its effect on sleep. However, certain inconsistencies also need to be addressed (e.g. sleep durations reported as being unchanged, shorter and longer in older age when compared to

earlier adulthood; Chaput et al., 2022; Coutrot et al. 2022; Ohayon et al., 2004), theta power being shown to increase and decrease in older age (e.g. Babiloni et al., 2015; Barry & De Blasio, 2017; Vlahou et al., 2014). These differences are partly due to different methodological approaches and to date, few studies have looked at these interacting variables in combination with each other, which may also account for the lack of uniform findings. As such, the investigations in this thesis aim to identify a common premise that should link them together.

The scope of the thesis includes a prominent focus on EEG-related measures, tapping into age-related differences in brain activity and zooming into the arousal regulation hypothesis, which implicates several important factors, including age-related deteriorations of the ARAS and the regulation of the sleep-wake cycle. It also brings in related factors that are implicated in these systems, including sleep quality, daytime drowsiness, stress, anxiety and depression, and resting-state cognition. Given the broad nature of these relationships, the investigation scope of this study is only limited to the exploration of these relationships in healthy young (aged 18-30) and older adults (> 60 years of age) without the presence of diagnosed pathology, such as sleep or psychiatric disorders. While a comprehensive overview of pathological ageing and sleep disorders may not be possible within this thesis, it hopes that by synthesising a common premise and approach, further investigations may build upon this.

The primary research questions of this thesis are divided into three goals, each of which will be addressed in its section in each of the subsequent chapters. The following chapter (Chapter 2) will address the first goal, that being to explore the relationships between sleep quality, negative emotional states, daytime drowsiness, and resting-state cognition. This will then build into Chapter 3 to address the second goal — whether the arousal regulation hypothesis can provide a model implicating age-related changes to the brain on sleep quality.

This will be done through an evaluation of the Vigilance Algorithm Leipzig (VIGALL), including its processing steps and extracted parameters.

The third goal of this thesis, covered in Chapter 4 aims to incorporate contemporary practices in evaluating EEG components that may be sensitive to age-related changes. These considerations will involve a comparative overview of the conventional canonical approach versus the individualisation of data fit approaches in narrowband power analysis (e.g., canonical alpha band versus individualised alpha band activity). Of particular interest, parameters relevant to alpha and theta band activity will be given the spotlight. This is due to the proximity of their frequency band definitions, combined with their prominence in the literature concerning age-related studies. In addition to that, discussions relating to the parameterisation of EEG data will also be included, bringing a focus to certain qualities of cortical properties that may be sensitive to age-related shifts (i.e. 1/f slope), and whether they may be important features to consider when examining sleep quality in older age. Lastly, Chapter 5 will synthesise all the variables studied in the earlier chapters into age-group-specific models to identify the potential factors that may be crucial in understanding sleep quality across young and old adults.

Chapter 2: Sleep Quality, Negative Emotional States, Daytime Drowsiness & Resting-State Cognition

2.1. Introduction

2.1.1 Background Information

In this chapter, the focus will be directed towards the comprehensive examination of non-EEG variables employed in this study. This analysis will encompass an evaluation of age on sleep quality, negative emotional states, daytime drowsiness, and resting-state cognition. The rationale for this approach is to first inspect age-related differences in these variables and outline potential interactions. Through investigating these interactions, further context can be provided when exploring the EEG-derived data, which will be handled in more detail in the following chapters.

Sleep quality has been reported to decrease across the lifespan, with adults above the age of 50 starting to show significantly worse sleep compared to early life (Hinz et al., 2017; Ohayon et al., 2004). Considering the crucial role of sleep quality in sustaining overall health and promoting healthy ageing, numerous studies have explored potential mechanisms and related developments underlying the interplay between these variables. One particular aspect of interest is the influence of negative emotional states.

In the present context, negative emotional states encompass feelings of depression, anxiety, and stress. These aspects have been examined extensively in their progression across the lifespan, as well as their relationship to sleep disturbances. The selection of these specific dimensions is based on their alignment with the three subscales of the Depression, Anxiety and Stress Scale (DASS) by Lovibond and Lovibond (1995). The DASS, a widely employed and validated tool has been utilised across diverse age groups, clinical settings, and cultural populations (e.g. Oei et al., 2013; Page et al., 2007; Yeung et al., 2020). Given the present

study also utilised the DASS, the scope of negative emotional states in this context is limited to those three dimensions and refers to the severity of symptoms, rather than the disorders.

Depression, anxiety, and stress are often comorbid with poor sleep quality. For instance, Einar et al. (2019) found that individuals who report elevated stress, anxiety and depression symptoms tended to engage in more ruminative thinking, leading to increased sleep disturbances. Similarly, João et al. (2018) observed that in non-clinical samples, poor sleep quality predicted higher feelings of depression, anxiety, and stress. Besides that, higher feelings of stress and depression have also been linked to higher wakefulness during sleep, impairing the restful and restorative functions of sleep (Buysse et al., 2000). Moreover, chronic exposure to stress can affect the neuroendocrine regulation of sleep and result in fragmented and unstable sleep patterns (Van Reeth et al., 2000). Furthermore, depression and anxiety adversely impact daytime alertness, sleep quality, and exacerbate insomnia severity, particularly when occurring comorbidly (Oh et al., 2019).

In addition to these negative emotional states, another significant concern is daytime drowsiness. Excessive daytime drowsiness (EDS) describes the inability to remain awake and vigilant during the waking periods of the day (Medicine, 2005). This can have a detrimental impact on overall quality of life, drastically increasing the risk of road and workplace accidents, and influencing various aspects of daily functioning (Connor et al., 2002; Haavisto et al., 2010). EDS is often comorbid with other psychiatric conditions, including depression, stress, and anxiety, which exert notable effects on sleep quality (Åkerstedt et al., 2014; Maestri et al., 2020; Slater & Steier, 2012). Moreover, EDS often occurs as a clinical symptom in those experiencing sleep disorders, such as sleep apnea and restless leg syndrome (Slater & Steier, 2012).

Particularly relevant to the current subject matter, experiences of EDS tend to rise with advancing age (Zalai et al., 2017). Carvalho et al. (2017) observed that older adults with

EDS showed impaired cognition, more disturbed sleep, and were often presented with other medical comorbidities. Moreover, EDS may also reflect an early symptom of dementia and is notably common in pathological ageing populations (Maestri et al., 2020; Tsapanou et al., 2015). Alongside EDS, occurrences of depression are higher in older populations (Sivertsen et al., 2015). Regarding stress and anxiety in older adults, the evidence is mixed, with reports varying between 15-52.3% for anxiety symptoms (Lindesay et al., 2012; Mehta et al., 2003; Schaub & Linden, 2000). The manifestation of anxiety in older age is complex and nuanced, though its association with stress and depression are still quite pronounced (Bryant, 2010; Lindesay et al., 2012). Occurrences of stress are linked to different life circumstances, with stressors transitioning from occupational and child-rearing concerns to health-related, familial, or other social stressors in senescence (Pearlin et al., 2005). As such, determining estimates for the prevalence of anxiety and stress in old age proves challenging (Bryant, 2010; Lindesay et al., 2012). However, given the interconnected relationship outlined between daytime drowsiness, depression, anxiety, and stress, it may be inferred that as occurrences of daytime drowsiness and depression rise in old age, so would instances of anxiety and stress.

Exploring a related angle, another aspect of interest in this study aims to assess measures related to resting-state cognition. The cognitive activity during the resting-state is thought to encompass inner-mentation, or mind wandering which may reflect important aspects of the default-mode network (Buckner et al., 2008). Conceptually, it can be regarded as the ‘baseline’ of inner mental experiences (Delamillieure et al., 2010). Its conception was driven by the intention to establish a bridge between the physical measurements of neuroimaging studies and secondary cognitive states that could also bias neuroimaging data, such as feelings of arousal and comfort (Diaz et al., 2013; Ogedegbe et al., 2008).

The concept of ‘resting-state cognition’ also offers additional resolution when linking neurobiological activity with other mental experiences and psychological dimensions, such as depression and anxiety. These dimensions were investigated by Diaz et al. (2013), who in creating the Amsterdam Resting-State Questionnaire (ARSQ) investigated the relationships between resting-state cognition and depression, anxiety, and sleep quality — comparisons that hold particular relevance for this study.

The ARSQ 2.0 postulates ten dimensions of resting-state cognition, complemented by an ‘other’ dimension that encompasses thoughts and feelings that do not align with the main ten (Diaz et al., 2014). While the ARSQ presents many dimensions, six of these dimensions exhibit significant correlations with sleep quality, depression, and anxiety. These include (i) discontinuity of thought; (ii) theory of mind; (iii) self; (iv) planning; (v) sleepiness; and (vi) comfort. As an overview, higher scores in these dimensions are associated with poorer sleep quality and higher scores of depression and anxiety (except for comfort, where a negative correlation exists; Diaz et al., 2013). Interestingly, Diaz et al. (2014) also noted that discontinuity of mind, self and planning tends to decrease with advancing age. These findings are challenging to reconcile with previous literature — if higher scores on these dimensions are associated with poorer sleep quality (Diaz et al., 2013), and if these dimensions are reduced in older age (Diaz et al., 2014), why do older adults also report poorer sleep quality, when it should suggest otherwise (e.g. Hinz et al., 2017)? As such, closer scrutiny may also be necessary to assess the validity of the ARSQ dimensions and the intricate relationships at play.

In this study, while the primary focus is on rsEEG as the key investigative method, the inclusion of the ARSQ in the observations will facilitate a closer examination of the interplay between psychological factors and EEG features associated with the resting state. Furthermore, the incorporation of the ARSQ would also serve the purpose of validating the

construct of resting-state cognition by testing it against measures of stress and daytime drowsiness — dimensions that were not explored in the original papers by Diaz et al. (2013) & Diaz et al. (2016). The ARSQ has not been widely applied in the investigative context of the present thesis, which combines both sleep quality and ageing. As such, hypotheses relating to the dimensions of the ARSQ are mostly speculative. In line with existing evidence, it is hypothesised that older adults will have poorer sleep quality compared to young adults (i.e. Hinz et al., 2017). Consequently, a common pattern should manifest across the previously examined ARSQ dimensions linked to poor sleep quality, where older adults would show poorer outcomes on discontinuity of mind, theory of mind, self, planning, sleepiness, and comfort. Furthermore, since these dimensions have also correlated significantly with depression and anxiety (Diaz et al., 2013), it is also expected that they would correlate with stress and daytime drowsiness.

2.1.2. Aims Of The Present Chapter

This chapter is dedicated to two primary objectives. Firstly it aims to replicate the findings identified in existing literature relating to age-related differences in sleep quality, negative emotional states, and daytime drowsiness. Subsequently, an exploration of the ARSQ dimensions will be conducted to uncover potential correlations with these variables. The emphasis will be on the six specified dimensions of the ARSQ — (i) discontinuity of thought; (ii) theory of mind; (iii) self; (iv) planning; (v) sleepiness; and (vi) comfort, though exploratory examinations will also investigate the other ARSQ dimensions, those being: somatic awareness, health concern, visual thought, verbal thought, and ‘other’.

This chapter will proceed with an overview of the materials and methodology used in this study, with a brief mention of the EEG setup as an integral part of the procedure. Further details related to the treatment of EEG data (and the VIGALL) will be addressed in Chapters

3 and 4. The objectives of this chapter will be approached through the examination of six primary research questions (RQ), each accompanied by its corresponding hypotheses (H) (Refer to Table 2.1).

Table 2.1

Table of Research Questions & Corresponding Hypotheses

RQ1	How does self-reported sleep quality differ between young and older adults?
H1	Older adults will report higher scores on the PSQI in contrast to young adults, suggesting lower overall sleep quality.
RQ2	How do the experiences of negative emotional states, such as depression, anxiety and stress, vary between young and older adults?
H2	Older adults will report higher scores in depression, stress and anxiety in contrast to young adults.
RQ3	How do the levels of daytime drowsiness compare between young and older adults?
H3	Older adults will report higher scores of daytime drowsiness compared to young adults.
RQ4	How do sleep quality, negative emotional states, and daytime drowsiness correlated with each other?
H4	Poor sleep quality, depression, anxiety, and daytime drowsiness will be positively correlated with each other.
RQ5	How do the experiences of resting-state cognition differ between young and older adults?
H5a	Older adults will report higher scores on theory of mind and sleepiness compared to young adults.
H5b	Older adults will report lower scores on discontinuity of mind, self, planning, and comfort compared to young adults.
RQ6	How do various resting-state dimensions correlate with sleep quality, negative emotional states, and daytime drowsiness?
H6a	Poor sleep quality, depression, anxiety, stress and daytime drowsiness will correlate positively with discontinuity of mind, theory of mind, self, planning, and sleepiness.
H6b	Poor sleep quality, depression, anxiety, stress and daytime drowsiness will correlate negatively with comfort.

2.2. Methods

2.2.1 Participants

The minimum sample size for the present study was determined using previous EEG experimental designs as the reference for pragmatic purposes, as opposed to a priori power calculations. Based on a similar study that examined drowsiness (i.e. vigilance) via EEG signatures, a minimum sample size of 22 was required for each group (Awais et al., 2014). In this study, a total of 23 young adults (Range: 18-27 years old, Mean age: 22 (*SD* = 3), Gender: 17 Females, 6 males) and 22 older adults (Range: 60 - 71 years old, Mean age: 65 (*SD* = 3), Gender: 17 Females, 5 males) were recruited via convenience sampling for this study. Young participants were recruited via recruitment posters or through online participation platforms (i.e. SONA systems), while older adults were recruited via online recruitment messages distributed through elder-targeted hobby/activity social groups. The study was approved by the local ethics committee of the University of Nottingham Malaysia (JHCW011121).

The inclusion criteria for young adults were to be between the ages of 18-30, while older adults had to be above the age of 60 with no clinical diagnosis of neurophysiological disorders. Older adults also had to score above the cut-off score for the classification of non-MCI on the MoCA (see MoCA section below for details). The MoCA scores for all older adult participants were between 24 – 30 (*M* = 27, *SD* = 2), meeting the requirement for the present study. Participants were also excluded if they had a clinical diagnosis for psychiatric illness or sleep disorders, and if they were on sleep medication. While the present study did not have a specific criterion for participant ethnicity or nationality, the sample of participants here mostly consisted of Malaysians (88.89%), with five participants being non-Malaysian.

2.2.2. Materials

2.2.2.1. Montreal Cognitive Assessment (MoCA). The MoCA (Nasreddine et al., 2005) is a popular neuropsychological assessment tool that is primarily used for detecting mild cognitive impairments and dementia. However, it can also be used as a general measure of cognitive ability. It is scored on a 30-point assessment that covers the following range of cognitive abilities: (i) Orientation; (ii) Short-term memory; (iii) Executive functioning / visuospatial ability; (iv) Language; (v) Abstraction; (vi) Animal naming; (vii) Attention; and (viii) Clock-drawing test. The present study utilised the MoCA (v8.3) in English and was used as an initial screening tool to evaluate the cognitive suitability of older adult participants for the study.

The standard cut-off score used in the MoCA to differentiate between healthy cognitive function and mild cognitive impairment is 26. However, this cut-off tends to show a high number of false-positive classifications depending on the populations tested and the language used (Thomann et al., 2020). As most participants in this study are Malaysian, an adjusted cut-off score more appropriate to Asian samples was used instead, that being 21/22 (Yeung et al., 2014).

2.2.2.2. Pittsburgh Sleep Quality Index (PSQI). In the present study, the PSQI (Buysse et al., 1989) was used to measure overall sleep quality. The PSQI is a self-report questionnaire that assesses the quality of sleep over the past month. It contains 19 items which form 7 components, (i) Sleep Quality; (ii) Sleep Latency; (iii) Sleep Duration; (iv) Habitual Sleep Efficiency; (v) Sleep Disturbance; (vi) Use Of Sleep Medication; (vii) Daytime Dysfunction. These components included questions about the typical timing of sleep and wake, and the frequency of sleep disturbances across a week, such as coughing, experiencing pain or bad dreams and waking up to use the bathroom. It also includes

questions about the use of sleep medication and disruptions to daily functioning, such as falling asleep while driving or eating. The scores on all components are aggregated into a global score ranging from 0 to 21 that provides an index of sleep quality, with scores lower than 5 indicating good sleep. The test-retest reliability of the PSQI is good, with a Cronbach's alpha of .83 to .87 (Backhaus et al., 2002; Buysse et al., 1989).

2.2.2.3. Depression, Anxiety & Stress Scale Long Form Questionnaire (DASS-42). The DASS (Lovibond & Lovibond, 1995) was used in this study to measure the severity of negative emotional states corresponding to depression, anxiety, and stress over the past week. The DASS contains 42 statements that are divided the three subscales: depression (e.g. "I felt that I had nothing to look forward to."); anxiety (e.g. "I was aware of the dryness of my mouth"); and stress (e.g. "I found it difficult to relax."). All items are rated on a 4-point rating scale, where participants had to agree to each statement ranging from 0 to 3 (0 = Did not apply to me at all, 3 = Applied to me very much, or most of the time). The scores for each item on each subscale are summed to form interpretations of the severity of symptoms. Refer to Table 2.2.2A for the interpretation of scores for each of the subscales. The test-retest reliability of the DASS is good with a Cronbach's alpha of .89 to .96 (Akin & Çetin, 2007; Makara-Studzińska et al., 2022)

Table 2.2.2.3

Interpretation of DASS Scores

	Depression	Anxiety	Stress
Normal	0 - 9	0 - 7	0 - 14
Mild	10 - 13	8 - 9	15 - 18
Moderate	14 - 20	10 - 14	19 - 25
Severe	21 - 27	15 - 19	26 - 33
Extremely Severe	28+	20+	34+

2.2.2.4. Amsterdam Resting State Questionnaire (ARSQ). The ARSQ 2.0 (Diaz et al., 2013, 2014, 2016) is a self-report questionnaire that examines the resting-state cognition. It contains 54 statements which map onto 11 dimensions of resting-state cognition: (i) Discontinuity of Mind; (ii) Theory of Mind; (iii) Self; (iv) Planning; (v) Sleepiness; (vi) Comfort; (vii) Somatic Awareness; (viii) Health Concern; (ix) Visual Thought; (x) Verbal Thought; (xi) and Other (refer to Table 2.2.2B for descriptions of ARSQ dimensions and sample statements). All statements are rated on a 5-point scale, ranging from 1 to 5 (1 = Completely Disagree and 5 = Completely Agree). Statements belonging to each dimension are averaged to obtain a score for each dimension. Higher scores indicate a higher occurrence of the dimension during resting state cognition. ARSQ dimensions provide insight into the relationships between subjective experience during rest and have previously been linked to other aspects of interest to this study, namely sleep quality, experiences of sleepiness, depression and anxiety (Diaz et al., 2013). Diaz et al. (2014) report fairly consistent test-retest reliability with all ARSQ dimensions showing consistency up to 31.5 months, except the dimensions of sleepiness and health concern, which the authors note are expected to fluctuate as part of the variation in daily sleep obtained or health status.

Table 2.2.2.4

Description of ARSQ Dimensions

Dimension	Description	Sample Statement
Discontinuity of Mind (DoM)	Includes rapidly switching busy thoughts, feelings of being restless and lack of control over thoughts.	I had busy thoughts.
Theory of Mind (ToM)	Includes thoughts about other people and placing oneself in another person's perspective.	I thought about others.
Self	Self-referential thoughts relating to feelings and behaviour.	I thought about my feelings.
Planning	Includes thoughts about work/study, the past and future, problem solving and tasks that needed to be done.	I thought about things I need to do.
Sleepiness	Includes feelings of tiredness and drowsiness.	I felt tired.
Comfort	Includes feelings of being relaxed and happy.	I felt comfortable.
Somatic Awareness	Includes feelings about one's health, body, heartbeat and breathing.	I was conscious of my body.
Health Concern	Includes feelings about one's feelings of extreme discomfort and health status.	I felt ill.
Visual Thought	Includes thoughts in images, including events and places.	I thought in images.
Verbal Thought	Includes thoughts occuring in words, including silent conversations with oneself.	I thought in words.
Others	Includes statements referring to thoughts that do not belong to the defined ten dimensions.	I had similar thoughts throughout the session.

2.2.2.5. Epworth Sleepiness Scale. The Epworth Sleepiness Scale (ESS) is an 8-item questionnaire and was used in this study to assess the severity of general daytime sleepiness. Measures of daytime drowsiness, specifically via the ESS have been associated with arousal regulation, with participants scoring high on ESS exhibiting lowered EEG vigilance (Jawinski et al., 2017). All ratings are made on a 4-point scale from 0 to 3, where participants had to indicate their chance of falling asleep in different situations (e.g. “How likely are you to doze off or fall asleep while sitting and reading?”; 0 = Would never doze, 4 = High chance of dozing). Scores across all items are summed for final interpretation. Refer to Table 2.2.2C for the interpretation of ESS score ranges. The test-retest reliability of the ESS is good with a Cronbach’s alpha of .88 (Johns, 1992).

Table 2.2.2.5

Classification of ESS Scores

Score Range	Classification
0 - 7	Unlikely To Be Abnormally Sleepy
8 - 9	Average Amount of Daytime Sleepiness
10 - 15	Excessively Sleepy Depending On The Situation
16 - 24	Excessively Sleepy

2.2.3. Procedure

Participants were sent reminders a day before the day of experimentation where they were instructed to wash their hair and scalps before their sessions and to get at least 6 hours of sleep the night prior to the day of experimentation. Participants were also instructed to keep to their regular dose of caffeine or nicotine the morning of their sessions. While the effects of caffeine and nicotine can produce alterations in observed EEG signals (Liu et al., 2022), temporary abstinence has also been shown to affect resting-state measurements, especially in

chronic users (Imperatori et al., 2020; Teneggi et al., 2004). Since the present study aimed to observe participants as they usually are, participants were asked to keep to their routines, to ensure that differences in EEG activity would not be attributable to abstinence effects from caffeine or nicotine. This allowance is also consistent with previous studies utilising the VIGALL protocol (Huang et al., 2015).

On the day of experimentation, participants were initially provided with an information sheet outlining the study's details and asked to provide signed consent. Additionally, verbal explanations of the study were given to all participants, allowing them the opportunity to ask questions or voice any concerns they may have had. Following the completion of consent forms, participants filled in a demographic questionnaire with a summary of their medical history. This included any personal or family history of physical or psychiatric illness, as well as details of any prior surgical procedures.

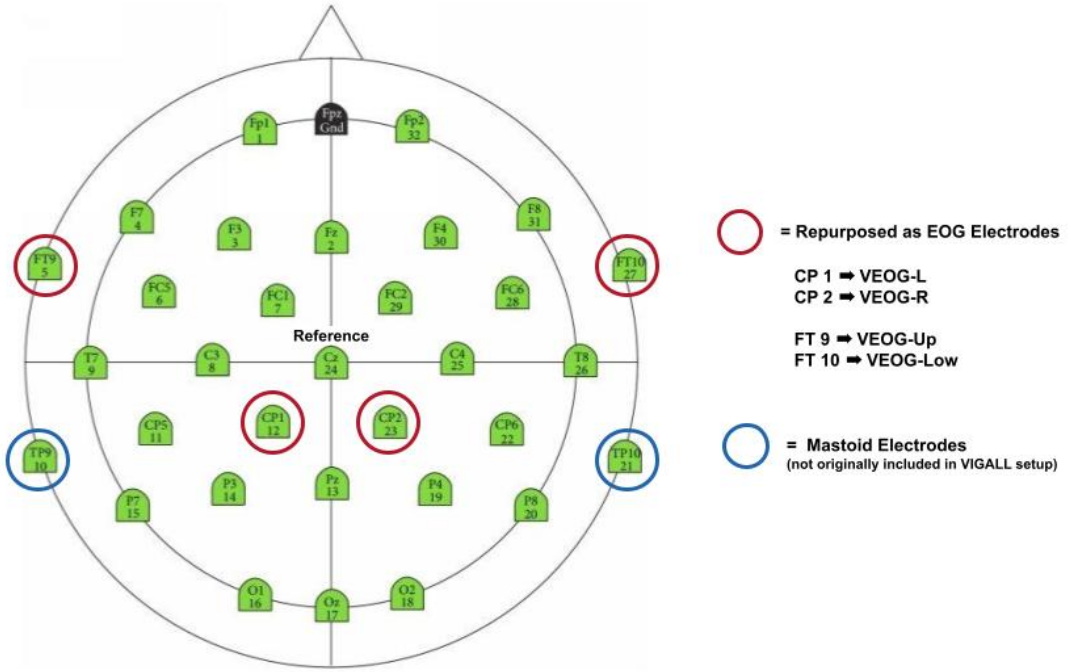
For the older adult participants, the MoCA was administered to obtain a brief profile of their cognitive health. This was also done to identify older adults who may be exhibiting symptoms of mild cognitive impairment or dementia, which would further disqualify them from the study. After that, participants were asked to fill in the DASS, PSQI and ESS. The order of these questionnaires was randomised. For young participants, all forms and questionnaires were filled in via Qualtrics, but paper equivalents were provided for older adults who were unfamiliar with online questionnaires.

Once participants were completed with the questionnaires, they were brought into the EEG recording booth. All recordings were performed between 9 a.m. to 1.00 p.m. and the recording booth was maintained at a temperature between 25-26° C. Once participants were comfortably seated, electrodes were set up according to the VIGALL protocol. Using a 32-channel EEG (QuickAmp amplifier, Brain Products GmBH, Gilching, Germany), 28 of the electrodes were used for the EEG and were set up following the international 10-20 system

using the EasyCap (EASYCAP Brain Products GmbH, Gilching, Germany). According to the VIGALL protocol, only 25 electrodes are required for the setup, but electrodes for the mastoids (Tp9 & Tp10) and reference (Cz) were additionally included in this study. Electrodes were referenced against Cz for online recordings (Refer to Figure 2.2.3A for EEG electrode setup).

Figure 2.2.3A

EEG Electrode Setup



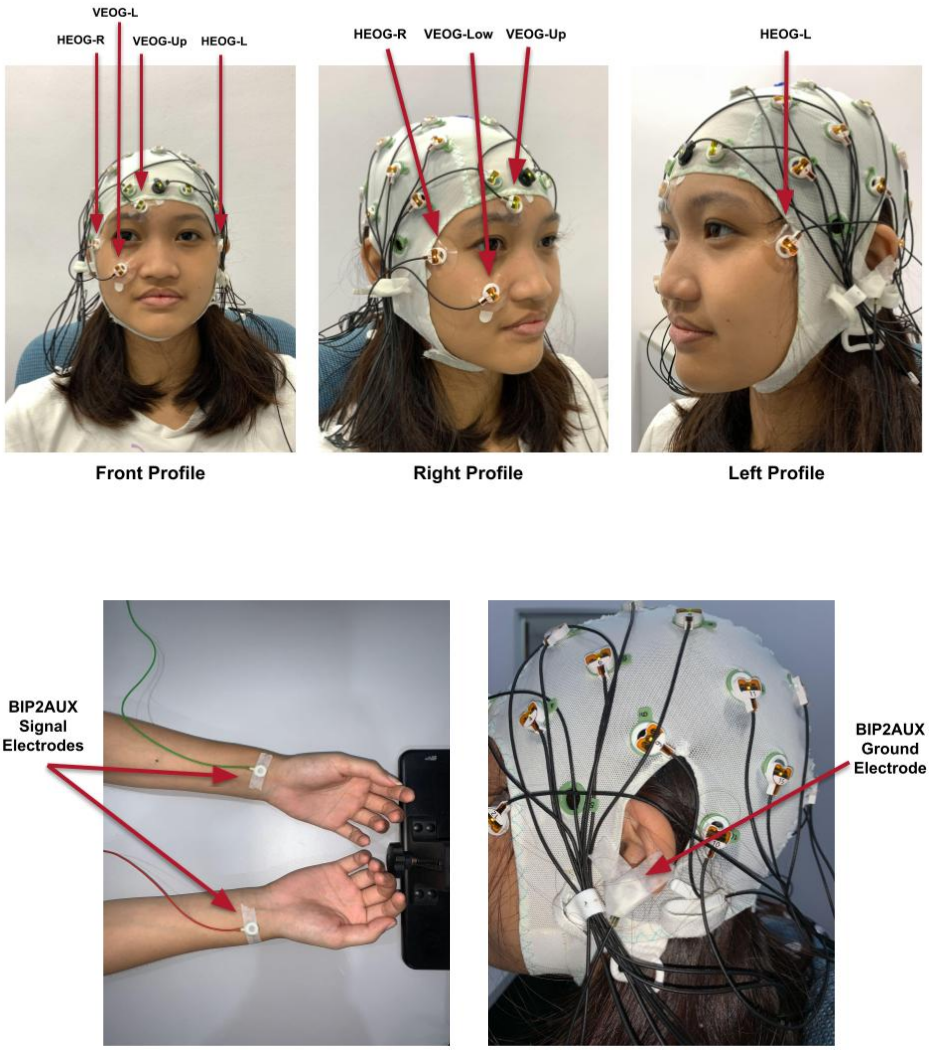
Note. Base diagram of electrode position was obtained from "Discovering Patterns in Brain Signals Using Decision Trees." By Bastos et al., 2016, Computational Intelligence and Neuroscience.

Impedances were kept below 50 kΩ and the EEG was sampled at 1000 Hz. The four remaining electrodes not used for the VIGALL protocol were used to set up bipolar EOGs, placed at the lateral of the left and right eye (horizontal EOG) and above and beneath the right eye (vertical EOG). ECG was measured using a BIP2AUX adaptor with the two signal

electrodes being placed on the wrists above the radial pulse, and the ground electrode on the left earlobe (refer to Figure 2.2.3B for sample of electrode setup).

Figure 2.2.3B

Bipolar EOG & ECG Electrode Placement



Before the commencement of the recording, participants were reclined to approximately 45° position and the lights in the recording booth were dimmed. Once settled, the Berger Manoeuvre (i.e. alternation between opening and closing of the eyes) was performed with slightly adapted instructions following the VIGALL protocol; refer to Appendix A for instructional scripts used). Once completed, participants were told to adjust themselves into a comfortable position and they were asked to relax and keep their eyes

closed for the 20-minute eyes-closed resting state recording. Participants were also instructed to move as little as possible and to follow their natural course of rest during the recording. They were also provided explicit instructions that they do not have to stay awake for the entire duration, so falling asleep was permitted. After the instructions were delivered, participants were told to close their eyes and the 20-minute recording commenced.

After 20 minutes, participants were gently woken up and they were asked to provide a verbal report of whether they had fallen asleep during the recording duration. Afterwards, participants filled in the ARSQ and were guided to clean themselves up. All participants were debriefed and compensated with either course credits or cash vouchers of MYR 20 after the full completion of the study.

2.3. Results

No data was removed prior to analysis. A loose approach to data selection was taken, where data with outliers were not removed. However, due to the relatively smaller sample size than required for the analyses performed, stricter considerations were taken to account for biases and violations (see Wilcox & Rousselet, 2023). In the instances where the data fit the assumptions of a parametric test, parametric approaches were favoured. However, if the normality or homogeneity of variances assumptions were not met, then non-parametric approaches were used instead. All comparisons were performed with an alpha criterion of 0.05 ($\alpha = .05$).

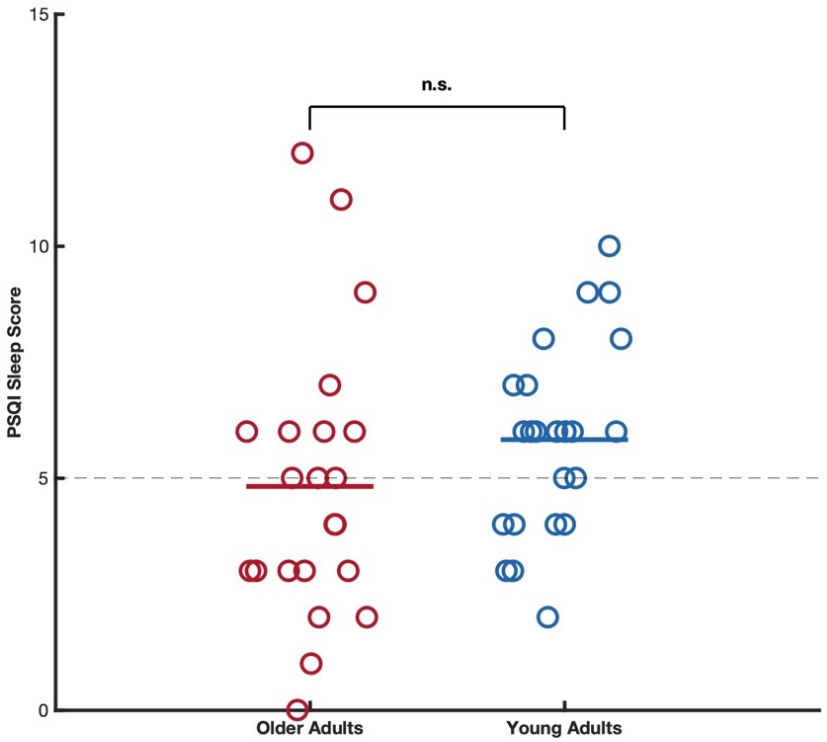
2.3.1. Sleep Quality

To evaluate differences in self-reported sleep quality, scores on the PSQI were compared between young and older adults. PSQI scores for both young and older adults met the assumptions of normality (Shapiro-Wilk's test, young $p = .55$, older $p = .11$) and homogeneity

of variances (Levene's test, $p = .10$). An independent-sample t-test found that the sleep quality of young ($M = 5.83$, $SD = 2.10$) and old adults ($M = 4.82$, $SD = 3$) were not significantly different ($t(43) = 1.31$, $p = .20$, Cohen's $d = .40$; Refer to Figure 2.3.1).

Figure 2.3.1

PSQI Score Distribution & Comparison Between Young & Older Adults



Note. PSQI Scores of > 5 indicate poor sleep quality. Horizontal lines going through points indicate mean values.

2.3.2. Negative Emotional States

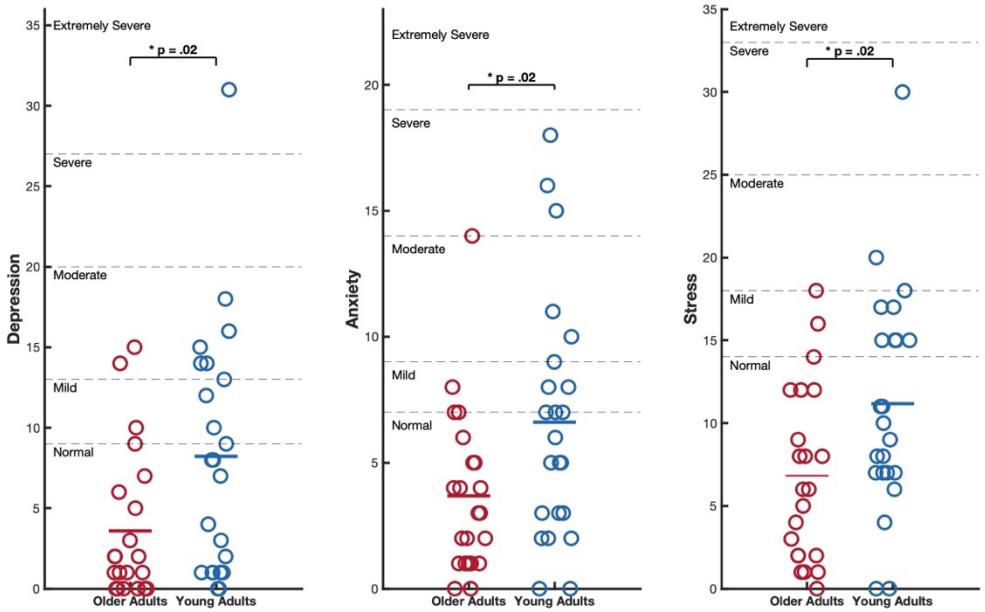
To compare levels of negative emotional states between young and older adults, cumulative scores from each dimension of the DASS were used. Three comparison tests were run to compare depression, anxiety, and stress between young and older adults. Depression scores were not normally distributed and did not meet the equal variances assumption across young

and older participants (Shapiro-Wilk's test, young $p = .01$, older $p < .001$; Levene's test, $p = .02$); anxiety scores for older participants were also not normally distributed (Shapiro-Wilk's test, young $p = .09$, older $p = .006$; Levene's test, $p = .07$). However, stress scores were normally distributed and met the equal variances assumption (Shapiro-Wilk's test, young $p = .14$, older $p = .10$; Levene's test, $p = .26$). As such, non-parametric comparisons were performed for depression and anxiety scores.

All tests revealed significant differences across all three DASS dimensions, with older adults reporting lower scores of depression ($M = 3.59$, $SD = 4.65$) (Mann-Whitney U: $U = 630$, $p = .02$, $r = .34$), anxiety ($M = 3.68$, $SD = 3.30$) (Mann-Whitney U: $U = 406$, $p = .02$, $r = .34$), and stress ($M = 6.82$, $SD = 5.32$) (Independent-t: $t(43) = 2.38$, $p = .02$, Cohen's $d = .71$) compared to young adults (Depression: $M = 8.82$, $SD = 7.72$; Anxiety: $M = 6.61$, $SD = 4.90$; Stress: $M = 11.17$, $SD = 6.84$).

Figure 2.3.2

Comparison of Depression, Anxiety & Stress Across Young & Older Adults



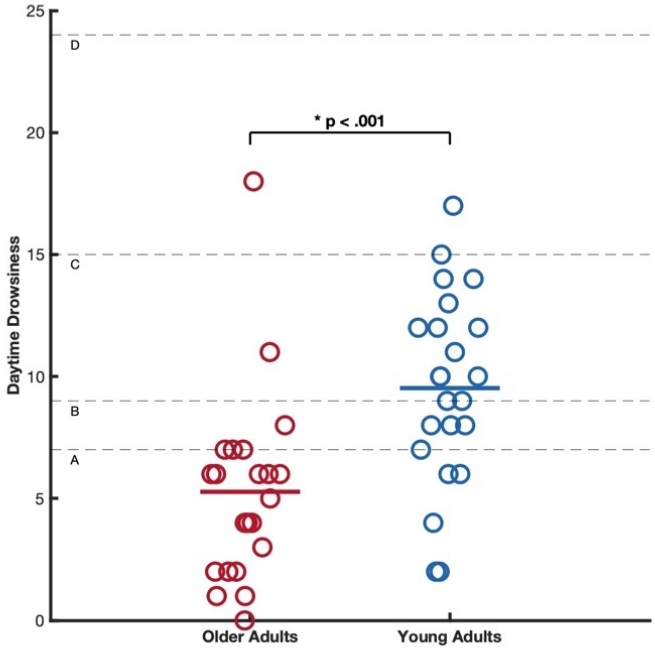
Note. Dashed lines correspond to cut-off points for classifications of negative emotional symptoms. Horizontal lines going through points indicate mean values. Asterisk (*) indicates significance ($p < .05$).

2.3.3. Daytime Drowsiness

To evaluate differences in daytime drowsiness, scores on the ESS were compared between young and older adults. ESS scores for young adults were normally distributed (Shapiro-Wilk's test, young $p = .87$), but scores for older adults were not (older $p = .004$;). The homogeneity of variances was met (Levene's test, $p = .97$). A Mann-Whitney U test indicated that the ESS scores were significantly higher in young adults ($M = 9.52$, $SD = 3.94$) compared to older adults ($M = 5.27$, $SD = 3.91$) ($U = 685$, $p < .001$, $r = .53$; refer to Figure 2.3.3), indicating that young adults have higher feelings of daytime drowsiness compared to older adults.

Figure 2.3.3A

Comparison of Daytime Drowsiness Across Young & Older Adults



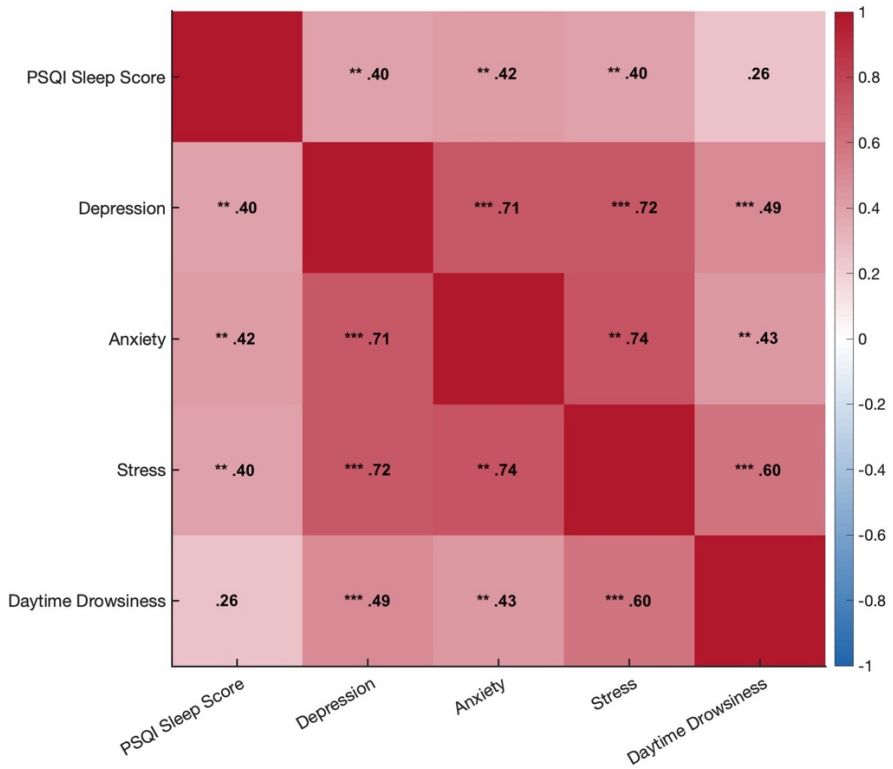
Note. Scores in each region correspond to the classification of drowsiness symptoms. A = Unlikely to be abnormally sleepy; B = Average amount of daytime sleepiness; C = Excessively sleepy depending on the situation; D = Excessively sleepy.

***** Horizontal lines going through points indicate mean values. Asterisk (*) indicates significance ($p < .05$).

To further explore the relationships between sleep quality with negative emotional states and daytime drowsiness, correlational analyses were performed across PSQI scores, DASS scores and ESS scores (Refer to Figure 2.3.3B). Note that higher PSQI scores indicate poorer quality sleep.

Figure 2.3.3B

Spearman's Correlation Across Depression, Anxiety & Stress



Note. Values within cells indicate Spearman's rho. Asterisk (*) indicates significance. * = $p <$ .05; ** = $p < .01$; *** = $p < .001$

PSQI sleep scores, depression, anxiety, stress, and daytime drowsiness are positively correlated with each other (p's ≤ .04), suggesting that poorer sleep quality is positively correlated with higher symptoms of depression, anxiety, and stress. Daytime drowsiness was positively correlated with depression, anxiety, and stress (p's ≤ .003), but was not significantly correlated with sleep quality (p = .08).

2.3.4. Resting-State Cognition

Firstly, all dimensions of the ARSQ were compared across young and older adults. For each dimension, pairwise comparisons were made between young and older adults. Refer to Table 2.3.4 for a summary of statistics and Figure 2.3.4A for visualisation of differences.

Table 2.3.4

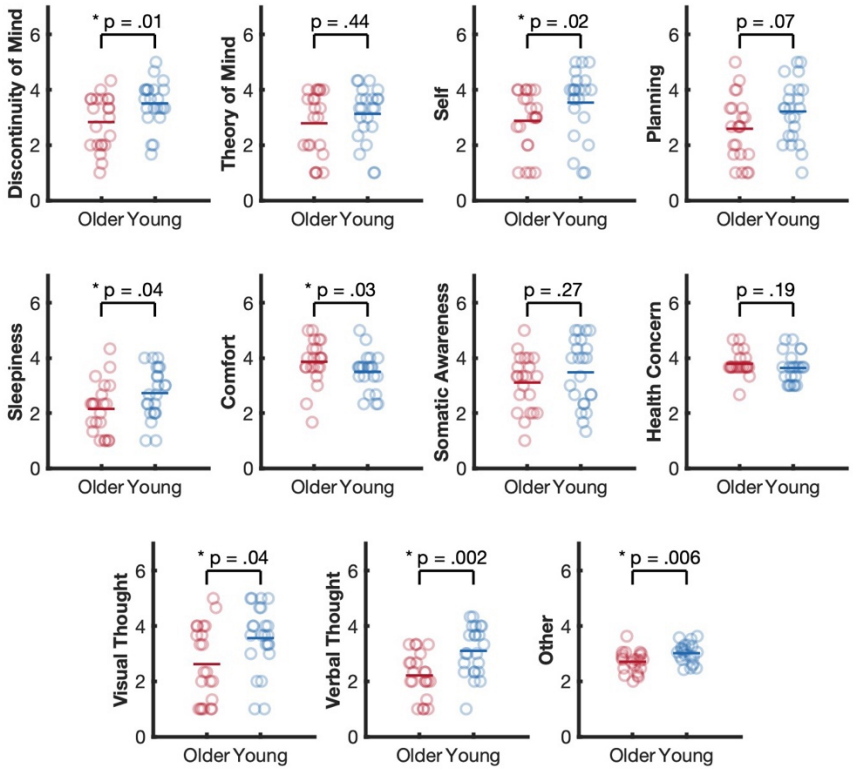
ARSQ Age-Wise Pairwise Comparisons Across ARSQ Dimension Statistics

Dimension	Statistical Test	Test Statistic	*p*	Significance	Effect Size
Discontinuity of Mind (DoM)	Independent Samples t	2.55	0.01	*	0.76
Theory of Mind (ToM)	Mann-Whitney U	563	0.44		0.11
Self	Mann-Whitney U	628	0.02	*	0.34
Planning	Independent Samples t	1.86	0.07		0.55
Sleepiness	Independent Samples t	2.09	0.04	*	0.62
Comfort	Mann-Whitney U	437	0.03	*	0.32
Somatic Awareness	Independent Samples t	1.13	0.27		0.34
Health Concern	Mann-Whitney U	474.5	0.19		0.2
Visual Thought	Mann-Whitney U	621	0.04	*	0.31
Verbal Thought	Mann-Whitney U	667.5	0.002	*	0.47
Other	Independent Samples t	2.88	0.006	*	0.86

Note. Summary of pairwise statistics conducted comparing age groups across all dimensions of the ARSQ. Test statistics of independent-sample t refer to t-statistic and Mann-Whitney U refer to U. Effect sizes correspond to Cohen's d (independent-samples t) and Mann-Whitney r (Mann-Whitney U). Asterisk (*) indicates significance (p < .05).

Figure 2.3.4A

Comparison of Scores Between Young & Older Adults Across ARSQ Dimensions



Note. Horizontal lines going through points indicate mean values. Asterisk (*) indicates significance ($p < .05$).

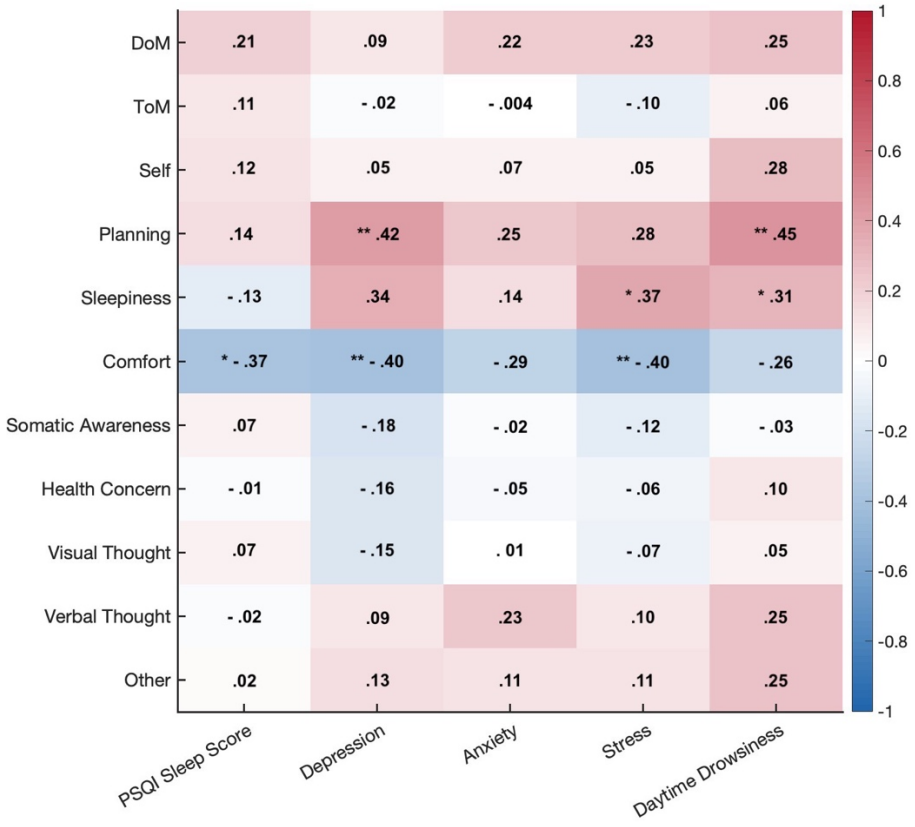
Scores on the dimensions discontinuity of mind, self, sleepiness, visual thought, verbal thought, and 'other' were higher in young adults compared to older adults (p's $\leq .04$), while comfort was higher in older adults ($p = .03$). The scores on the dimensions of theory of mind, planning, somatic awareness and health concern were not significantly different between young and older adults (p's $\geq .07$) (Refer to Table 2.3.4).

To explore relationships between resting-state cognition, sleep quality, negative emotional states and daytime drowsiness, correlational analyses were performed across all

dimensions of the ARSQ on PSQI scores, DASS scores and ESS scores (Refer to Figure 2.3.4B).

Figure 2.3.4B

Correlational Matrix of ARSQ Resting-State Dimensions on Sleep Quality, Depression, Anxiety, Stress & Daytime Drowsiness



Note. Values within cells indicate Spearman's rho. Asterisk (*) indicates significance. * = $p <$ .05; ** = $p <$ .01; *** = $p <$ .001

Planning was positively correlated with depression and daytime drowsiness (p's $\leq$ .01). Sleepiness was positively correlated with stress and daytime drowsiness (p's $\leq$ .04) and comfort was negatively correlated with sleep quality, depression and stress (p's $\leq$ .01). All other correlations were not significant (p's $\geq$ .07) (Refer to Figure 2.3.4B).

2.4. Discussion

The results obtained in this chapter slightly diverge from the expected hypotheses. Firstly, there was no significant difference in average PSQI sleep scores between young and older adults. This suggests that sleep quality did not differ significantly between the two groups, contrary to the hypothesis that older adults would report worse sleep compared to young adults. This unexpected finding contradicts the commonly reported trend in the literature, where in older age, sleep quality tends to decrease relative to younger age (e.g. Chaput et al., 2020; Hinz et al., 2017). One possible interpretation of this finding may suggest that the older adults recruited in this study may have better sleep quality in contrast to the typical ageing population, where poor sleep quality may be more common. Notably, only 36.36% of older adults in this sample reported PSQI scores above 5 (which indicates poor sleep quality). In contrast to that, 60.87% of the young adults had PSQI scores above 5. While the average sleep quality was not significantly different between the groups, it does suggest that a larger portion of the young adult sample suffers from poor sleep quality compared to the older adults.

Furthermore, the young adults in this study also report higher scores of depression, anxiety, stress and daytime drowsiness when compared to the older adults. These findings are also not as hypothesised, showing trends contrary to what is reported in the literature (Bryant, 2010; Lindesay et al., 2012; Sivertsen et al., 2015; Zalai et al., 2017). However taken together, these findings may not be too surprising. The young adult sample in this study consisted entirely of university students. It may be possible that the elevated scores of negative emotional states and poor sleep quality could be attributed to typical stressors in a university setting, such as academic or financial stress, social pressure, or concerns regarding post-graduation life (Ibrahim et al., 2013).

Looking across resting-state cognition dimensions, young adults also scored higher in discontinuity of mind, sleepiness, visual thought, verbal thought and 'other' dimensions, while scoring lower in comfort on the ARSQ. Particularly with sleepiness and comfort, the observed effects in these dimensions contradict the initial hypothesised directions. Examining these dimensions collectively would suggest that young adults experienced higher feelings of restlessness and a lack of control over their thoughts (Discontinuity of Mind, DoM), higher sensations of sleepiness, and a reduced sense of relaxation and happiness when at rest (Comfort). Besides that, the higher scores in visual thought and verbal thought suggest that young adults engage in thoughts that are richer in words and images, consistent with a previous finding by Diaz et al. (2014). The observations for theory of mind, self and planning were not significant, indicating that the young and older adults engage in similar levels of thinking about others (ToM), themselves (self), and their tasks and work that needed to be done (planning). Notably, the characterisation of ToM in the ARSQ is superficial and does not adhere to conventional definitions of theory of mind, and may instead more closely resemble empathy (Diaz et al., 2014). Taken together, the observations found across these measurements seem to place young adults in a poorer condition when compared to the older adults — which altogether places the older adults in a contrary position to what is commonly perceived.

However, while the age-related comparisons yielded contrary findings to the literature, the interactions observed between the variables support the hypotheses and are consistent with the literature (Hall et al., 2000; Oh et al., 2019; Thorsteinsson et al., 2019). Particularly noteworthy is the significant positive correlation between poorer sleep quality, depression, anxiety, stress and daytime drowsiness, suggesting that higher symptoms of depression, anxiety, stress and daytime drowsiness are associated with poorer sleep quality.

When incorporating the ARSQ dimensions, the findings would suggest that thoughts about the future and pending tasks (planning) correlate with higher levels of depression, stress and daytime drowsiness, while sleepiness is associated with stress. Finally, higher scores of comfort are associated with lower scores of depression and stress. However, while these findings from the ARSQ may provide certain insights, the findings are not particularly robust, making it challenging to formulate a sensible interpretation. A majority of the ARSQ dimensions did not show significant correlations with sleep quality, depression, anxiety or stress, suggesting that the ARSQ may not be sufficiently robust or sensitive, at least for this sample of participants to have revealed an effect. Furthermore, the 'other' dimension in the ARSQ showed significant differences in young and older adults in this study. However, since the 'Other' dimension of the ARSQ includes 19 statements that do not correspond to any of the ten defined dimensions, it is challenging to form informative interpretations of this dimension.

In summary, this chapter highlights significant relationships between poor sleep quality, negative emotional states, and daytime drowsiness, aligning with common findings in the literature. Unexpectedly, age-related comparisons revealed that young adults exhibited no significant differences in sleep quality, but reported higher symptoms of depression, anxiety, stress and daytime drowsiness when compared to old adults. Despite deviations from the initial hypotheses, the general pattern persists, indicating that higher scores in negative emotional states correlate with increased reports of daytime drowsiness and poor sleep quality. However, the resting-state cognition measures yielded less robust and consistent findings than anticipated, which may warrant further examinations to better understand the construct validity of these dimensions. In the subsequent chapter, neurobiological factors will be explored to further examine the relationships between these factors, providing a more comprehensive understanding and helping contextualise the findings in this chapter.

Chapter 3: Arousal Regulation, Negative Emotional States, Daytime Drowsiness & Sleep Quality

3.1. Introduction

3.1.1. Background Information

In the preceding chapter, the relationships between sleep quality, negative emotional states, daytime drowsiness, and resting-state cognition in both young and older adults were explored. Despite initial expectations of poorer sleep quality in older adults, the findings suggest that age-related differences in this regard were in contrast to what was hypothesised. However, the relationships among the variables, regardless of age were quite robust. This challenges the notion that age itself is a primary factor of poor sleep quality; instead, these findings hint at other variables, particularly negative emotional states playing a more pivotal role. An initial interpretation would suggest that the decline in sleep quality might not be an exclusive feature of ageing, but rather a consequence of other moderating factors, which may become more prevalent in older age.

The findings in the previous chapter were primarily derived from self-reports, reflecting mostly subjective experiences of sleep quality and emotional states. Building upon those initial findings, the current chapter will delve into more physiological features, tapping into a crucial dimension of the sleep-wake cycle — arousal regulation. The purpose of doing so is to explore what bridging evidence may explain the preliminary examinations from the previous chapter, offering a more nuanced understanding of the changes in sleep quality associated with ageing.

3.1.2. Model of Arousal Regulation & The Vigilance Algorithm Leipzig (VIGALL)

As previously outlined in the first chapter, the ascending reticular activating system (ARAS) plays an important role in modulating awareness and arousal, being involved with the

regulation of circadian and sleep-wake systems. The ARAS has widespread connections across the brain, and these connections are reduced in older adults (Guardia et al., 2022). The ARAS is the basis for vigilance (i.e. neurophysiological arousal) regulation and dysregulation of this system can impact the development of affective disorders, exacerbating physiological conditions and sleep quality. As such, exploring these arousal regulation systems may inform us about these features in ageing. To do so, the following examinations will be guided by the arousal regulation model of affective disorders by Hegerl and Hensch (2014).

The model of arousal regulation is based on three distinct patterns: stable, unstable and hyperstable. In environments without major external stimulation, arousal (i.e. vigilance) typically declines from alert wakefulness to more relaxed, drowsy states over a period of time. This decrease in arousal levels when at rest characterises a stable pattern of arousal regulation. Conversely, an unstable pattern is characterised by rapid declines to lower levels of arousal within a few seconds (in contrast to a gradual decline); and hyperstable patterns show a constant state of arousal that is resistant to change, even after 15 minutes or longer (Hegerl & Hensch, 2014).

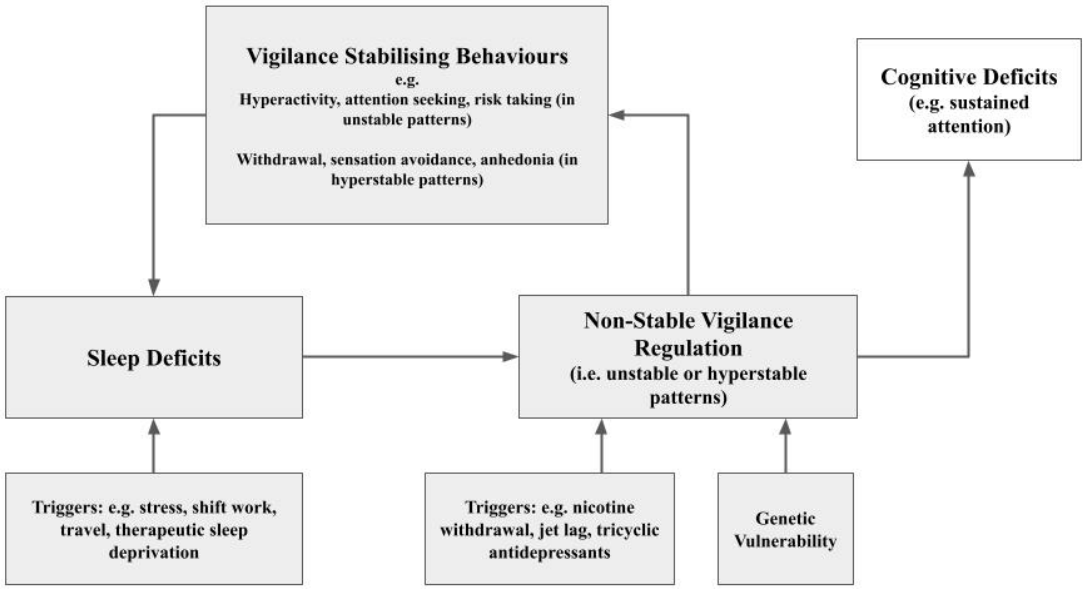
Non-stable arousal regulation can be triggered by various factors, including genetic predispositions, prolonged stress, shift work, sleep deprivation and side effects of nicotine withdrawal or antidepressants (Hegerl, Sander & Hensch et al., 2016).

The model of arousal regulation offers a mechanistic explanation for the link between affective disorders, their manifesting behaviours and sleep deficits, particularly through non-stable arousal regulation patterns. More importantly, this model captures the reciprocal relationship between these variables. However, introducing complexity to this model involves considering the impact of these mechanisms in older adults. Considering the evidence pointing to age-related deteriorations in these arousal-regulation regions (i.e. ARAS), the question arises as to how these deteriorations will affect the model. For instance, in the

ageing process, could arousal regulation systems be less sensitive to environmental stimulation, resulting in more pronounced or rapid declines in vigilance over time, resembling unstable patterns? Likewise, could the higher incidences of depression observed in older adults be indicative of hyperstable regulation, subsequently contributing to increased disruptions in sleep? Exploring such dynamics would provide not only additional credibility to the arousal regulation model of Hegerl and Hensch (2014) but also begin to address the complex relationship between ageing and sleep.

Figure 3.1.2A

Pathogenic Cycle in The Arousal Regulation Model



Note. Adaptation of the vigilance regulation model. From "Arousal regulation in affective disorders." by Hegerl, U., Sander, C. and Hensch, T., 2016, Systems Neuroscience in Depression, pp. 341–370.

To more closely study arousal regulation and its relationships to the development of affective disorders and sleep deficits, researchers from the University of Leipzig, together with the Leipzig Research Center for Civilisation Diseases (LIFE) developed the Vigilance Algorithm Leipzig (VIGALL). Similar to how sleep stages have been traditionally scored (e.g. NREM N1-N3 Sleep, REM Sleep), the VIGALL utilises EEG and EOG (electrooculogram) information to classify 1-second segments into different vigilance stages, describing the transition from fully awake states to the onset of sleep (refer to Figure 3.1.2B). The VIGALL functions as an add-in for the BrainVision Analyzer 2, requiring a 15-20 minute resting-state EEG recording prepared according to the VIGALL setup, with 25 electrodes placed on the scalp according to the extended 10-20 system, and horizontal and lateral bipolar

EOGs with ECG (refer to Chapter 2 methods for set up of electrodes). The VIGALL takes into account delta, theta, and alpha band activity, combined with cortical source localisation approaches to determine vigilance stages (refer to Appendix B for VIGALL scoring rules). Common approaches to interpreting VIGALL findings include examinations of averaged vigilance levels across time blocks (e.g. averaged vigilance across minutes 1-5), mean vigilance across total recording duration, arousal stability scores, slope indices and initial vigilance levels, parameters which will be further explained in subsequent sections of this chapter (Huang et al., 2015; Jawinski et al., 2017).

Figure 3.1.2B

Vigilance Stages According to VIGALL & EEG Characteristics

Behaviour	Vigilance Level	Stage	EEG Characteristics
Awake	7	0	Low amplitude, desynchronised non-alpha EEG in the absence of slow horizontal eye movements
	6	A1	Occipital dominant alpha activity
	5	A2	Starting shifts of alpha to central and frontal areas
	4	A3	Continued frontalisation of alpha
	3	B1	Low amplitude, desynchronised EEG with slow horizontal eye movements
	2	B2/3	Dominant delta- and theta-power
Sleep Onset	1	C	Occurrence of K-complexes and sleep spindles

Note. VIGALL Stages. From "Vigilance Algorithm Leipzig (VIGALL) Version 2.1 Manual" by Hegerl, U., Sander, C., Ulke, C. et al., 2016.

The VIGALL has been widely applied to investigate arousal regulation, successfully establishing links between patterns of arousal regulation and corresponding affective disorders. Additionally, it has also been utilised in experimental EEG designs to monitor participant arousal, specifically linking fluctuation in arousal states to performance in perceptual tasks (e.g. Bekhtereva et al., 2014; Geissler et al., 2014; Sander et al., 2015).

However, despite its extensive use, the application of the VIGALL in older populations is still limited.

Within the VIGALL literature, few studies have made dedicated efforts to examine arousal regulation in older adults. As of the present study, these include: Jawinski et al. (2017), who utilised the VIGALL 2.1 to explore the associations between subjective daytime sleepiness and brain arousal in adults aged 40 to 56, and 60 to 79 years; Olbrich et al. (2015), who conducted similar investigations in adults aged 22 to 58 years; and Ulke et al. (2016), who employed the VIGALL to compare arousal regulation patterns and sleep disturbances among older adults aged 60 to 79, with and without depressive syndromes. Beyond these studies, the VIGALL has not been widely applied to older adults, and explicit efforts to compare brain arousal patterns across different age groups are lacking. Furthermore, current examinations have not addressed whether age-related differences should be a relevant point of concern, particularly when considering potential age-related deteriorations in arousal regulation areas (Cesnaite et al., 2023).

3.1.3. Aims of The Present Chapter

This chapter serves two main objectives. Firstly, it aims to explore how arousal regulation patterns may differ between young and older adults, marking one of the first directed comparisons of the VIGALL across different age groups. The parameters of focus include the arousal stability score, slope index, initial vigilance level and mean vigilance level. Secondly, the chapter will explore potential correlations between these arousal regulation patterns and factors like sleep quality, negative emotional states, and daytime drowsiness. The scope of the analyses in this chapter (and thesis) is also limited to patterns more commonly associated with depressive states, rather than states characterised by unstable patterns (e.g. ADHD), as information relating to those states was not collected in this study.

As such, research questions and hypotheses are formulated with hyperstability as the anchor point, rather than unstable patterns.

Furthermore, due to the lack of robust findings in resting-state cognition from the previous chapter, the analyses in this chapter will deliberately exclude those dimensions, leaving it to a more focused examination in the final chapter of the thesis. The purpose of the explorations in this chapter is to identify aspects of arousal regulation that may be linked to variations in sleep quality. This process will also provide insights that will inform subsequent examinations in Chapter 4, which will primarily focus on the individualisation approach of neurobiological parameters relevant to ageing and sleep.

The following section will introduce the steps involved in processing EEG according to the VIGALL protocol. It will also provide definitions and calculation methods for the VIGALL parameters. Subsequently, the chapter will proceed with analyses aimed at exploring the research questions (RQ) and testing the hypotheses (H) outlined in Table 3.1.3. The hypotheses were formulated following the findings from Chapter 2, where young adults had reported higher scores across depression. As such, it was hypothesised that young adults would show arousal regulation patterns with hyperstable features, characterised by lower arousal stability scores, lower slope index scores and higher mean vigilance levels — arousal regulation patterns which are associated with depression. Simultaneously, it is hypothesised that these features of hyperstability would correlate significantly with sleep quality, negative emotional states, and daytime drowsiness.

Table 3.1.3

Table of Research Questions & Corresponding Hypotheses

RQ1	**How might patterns of arousal regulation differ between young and older adults?**
H1a	Younger adults will show lower arousal stability scores, indicating more hyperstable patterns compared to older adults.
H1b	Younger adults will show lower slope index scores, indicating less instability (i.e. less steep declines in arousal) compared to older adults.
H1c	Young and older adults will have similar initial vigilance levels.
H1d	Young adults will have higher mean vigilance levels, indicating higher arousal levels across time compared to older adults.
RQ2	**How might arousal regulation patterns correlate with self reported sleep quality, negative emotional states and daytime drowsiness?**
H2a	Arousal stability scores will correlate positively with depression, anxiety, stress, poorer sleep quality but correlate negatively with daytime drowsiness.
H2b	Slope index scores will correlate positively with daytime drowsiness but correlate negatively with poor sleep quality, depression, anxiety and stress.
H2c	Mean vigilance levels will correlate positively with depression, anxiety, stress and poor sleep quality, but correlate negatively with sleep quality.

3.2. EEG & VIGALL

3.2.1. EEG Cleaning Procedure

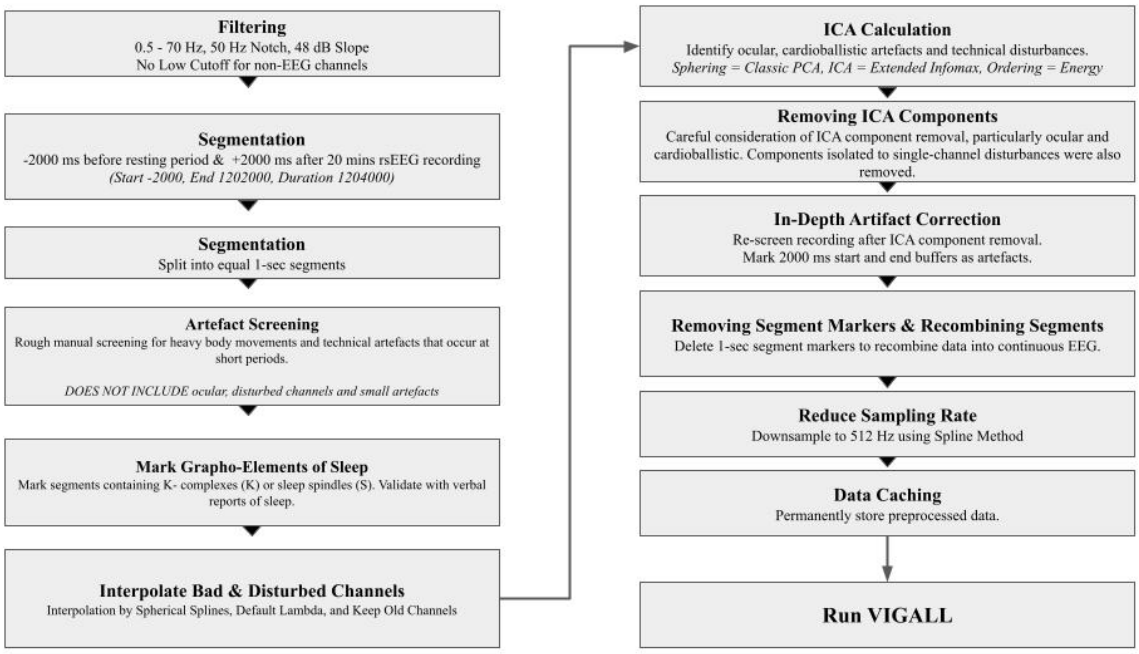
All 20-minute eyes-closed resting-state EEG recordings were recorded from 28-channel Ag/AgCl scalp electrodes (BrainProducts GmbH, Gilching, Germany) mounted in an elastic cap (EASYCAP Brain Products GmbH, Gilching, Germany) according to the extended 10-20 international system. While the VIGALL recommends only 25 EEG channels, 28 electrodes were used in the present set-up. Two of the additional electrodes were Tp9 and Tp10 and were included as mastoid electrodes to be used for mastoid re-referencing (only relevant to the data treatment procedure used in Chapter 4), and one of the electrodes at Cz was used as the reference electrode. Four additional electrodes were also used to record bipolar montages for vertical and horizontal eye movements and bipolar electrodes were attached to the wrists

as ECG. Scalp electrodes were referenced against Cz (refer to Chapter 2 methods for images of setup). Recordings were sampled at 1000 Hz with impedances kept under 50kΩ.

All recordings were preprocessed on BrainVision Analyzer (Version 2.2.2, Brain Products GmbH, Gilching, Germany) following the VIGALL processing pipeline (refer to Figure 3.2.1).

Figure 3.2.1

VIGALL Data Processing Pipeline



Note. PCA = Principle component analysis; ICA = Independent component analysis

Firstly, the data was passed through a low-pass filter of 70 Hz and a high-pass filter of 0.5 Hz, with a notch filter at 50 Hz to remove power line artefacts. Subsequently, the 20-minute eyes-closed rsEEG was isolated from the overall recording with the help of start and end markers, with a 2-second buffer included at both the start and end of the recording.

The data was then segmented into 1-second segments, creating a total of 1204 segments (consisting of the 20-minute recording and buffer segments). Each segment went through a rough, preliminary manual screening to identify and mark large artefacts. These included large movement artefacts and technical disturbances occurring briefly (e.g.

temporary disconnection of the electrode from the scalp). Ocular artefacts and disturbed channels were not marked at this stage of processing. The purpose of this initial screening was to flag segments containing large artefacts that would affect subsequent ICA calculations.

After the initial screening and marking of segments, interpolation of bad channels was conducted if necessary. Among the 45 datasets in this study, only one required the interpolation of a single C4 channel due to the electrode disconnecting during recording. Subsequently, the entire recording was screened for signs of sleep, with segments containing K-complexes or sleep spindles marked with a ‘C’ label. This manual labelling was performed to support the VIGALL classification, where segments (and the next 30 seconds after it) are labelled as ‘C’ stages if a ‘C’ marker is indicated. This process also considered verbal reports obtained from participants after their recordings to ensure the validity of sleep element identification.

After marking the graphic elements of sleep, ICA was applied using the extended Infomax algorithm across the entire rsEEG recording to identify non-cerebral components impacting the data, including ocular, cardio-ballistic and other technical disturbances. Following ICA calculations, inverse ICAs were performed to manually remove ICA components. Primarily, ocular components, indicated by large drifts or deflections in the EEG channels corresponding to eye twitching or eye-rolling movements were removed. A conservative approach to component removal was taken, retaining components that seemingly reflected cerebral activity, even if they contributed to a relatively noisy channel (low amplitude, high frequency “fuzz”). Components that were localised to one source were also removed if they showed activity that did not propagate to other channels, possibly signalling a disturbed electrode or a muscle artefact.

After careful removal of ICA components, a second more in-depth artefact screening was performed to identify “bad” segments (that remained “noisy” despite ICA correction) and

to mark the buffer segments to exclude them from VIGALL classification. The percentage of valid segments ranged from 97.25% to 100%, with the number of total "bad" segments varying from 0 to 33 across all 45 datasets. Subsequently, all 1-second segments were recombined into a continuous EEG, forming a continuous 1204s recording that was downsampled to 512 Hz using the Spline method. The processed data was stored in a persistent cache file and the VIGALL 2.1 algorithm was applied via an add-in on BrainVision Analyzer.

3.2.2. VIGALL-Extracted Parameters of Arousal Regulation

Following the execution of the VIGALL on the preprocessed 20-minute rsEEG data, each 1-second segment throughout the entire recording is assigned a vigilance stage, ranging from 0 to C (refer to Figure 3.1.2B above). These stages correspond to values ranging from 1 to 7, with 7 referring to Stage 0 (i.e. fully awake) and 1 referring to Stage C (i.e. the onset of sleep). These labelled segments allow for the computation of various parameters of interest related to arousal regulation patterns. Specifically, these parameters crucial for interpreting arousal regulation encompass the mean vigilance level over time, the arousal stability score, and the slope index.

3.2.2.1. Mean Vigilance Level Over Time. The mean vigilance over time describes an averaged value of the vigilance levels either grouped into time blocks or spanning the total rsEEG duration. A higher mean vigilance level would indicate higher awakeness.

3.2.2.2. Arousal Stability Score. The arousal stability score (ASS) is also referred to as the lability score in the literature (e.g. Huang et al., 2015). The ASS quantifies the speed and extent of vigilance decline, with lower ASS scores being associated with hyperstable

arousal regulation patterns and higher ASS scores being associated with unstable patterns. To compute the ASS, epochs of 60-second labelled segments are grouped and analysed. All epochs are passed through four-level criteria check to determine the ASS (refer to Table 3.2.2.2).

Table 3.2.2.2

Arousal Stability Score (ASS) Scoring Criteria

Arousal Stability Score	Criterion	EEG Block	Operational Definition
14	Level 1: Occurrence of C stage	1	Stage C emerged in minute 1-5
13		2	Stage C emerged in minute 6-10
12		3	Stage C emerged in minute 11-15
11		4	Stage C emerged in minute 16-20
10	Level 2: At least 1/3 of segments classified as B2/3 stage	1	Stage B2/3 emerged in minute 1-5
9		2	Stage B2/3 emerged in minute 6-10
8		3	Stage B2/3 emerged in minute 11-15
7		4	Stage B2/3 emerged in minute 16-20
6	Level 3: At least 1/3 of segments classified as B (B1 + B2/3 stage)	1	Stage B emerged in minute 1-5
5		2	Stage B emerged in minute 6-10
4		3	Stage B emerged in minute 11-15
3		4	Stage B emerged in minute 16-20
2	Level 4: More than 2/3 of segments classified as 0/A- or 0-A1 stages	1-4	Rigidity, unique appearance of 0 and A
1		1-4	Rigidity, unique appearance of 0 and A1

Note. ASS criteria adapted from Huang et al. (2015)

Succinctly, the first level checks for the occurrence of C stages (i.e. sleep). If a C stage is found, it is flagged and given an ASS score ranging from 11 to 14 depending on the time block in which the first instance occurred. If no C stage is found, the data is passed through the second and third levels to determine the proportion of B stages (B2/3 or B1 + B2/3) present in each epoch. If over 1/3 of segments in an epoch classify as a B stage, it is given an ASS of 3 to 10, depending on which minute this occurred in. Lastly, if none of the epochs are flagged in the second and third levels, it is handed to the final level, where over 2/3 of segments in all epochs would be classified as 0 or one of the A stages. An ASS of 2 is given if 2/3 of segments are flagged as 0 or one of the A-stages (A1, A2 & A3), while an ASS of 1 is given if 2/3 of segments are flagged as 0 or A1. The interpretation of ASS scores is less

intuitive, with higher ASS scores being associated with unstable patterns and lower scores being associated with hyperstable patterns. The computation of the ASS for each data set was performed with a custom MATLAB script.

3.2.2.3. Slope Index. The slope index measures the degree of arousal instability, reflecting where and how rapidly declines in arousal occur. It is calculated with the functional equation:

$$f(\mathrm{x}) = m\sqrt{\ln x} + n$$

where, *f(x)* represents the vigilance level at any given minute (e.g., the mean vigilance level at minute 2, obtained by finding the mean vigilance level from second 61 to second 120 of the resting-state recording), which is calculated by multiplying the slope index (*m*) with the square-rooted logarithmic of the respective minute (e.g., *x* = 2 for minute 2), and then adding that to the initial mean vigilance level at minute 1 (n). To derive the slope index (*m*), the equation is rearranged such that:

$$m = \frac{f(x) - n}{\sqrt{\ln \mathrm{x}}}$$

The square root logarithmic accounts for immediate and more delayed declines in arousal, ensuring that it adequately captures changes over time, regardless of the magnitude (Huang et al., 2015). In addition to that, to compensate for floor effects in participants who show low initial vigilance levels, a linear correction factor was applied (-0.33 * (7-*n*)) (Huang et al., 2015). From these calculations, nineteen possible functional equations are derived (minutes 2 through 20). From those nineteen values, arousal instability was taken as the slope (m) with the lowest value, reflecting the point of steepest decline.

3.3. Results

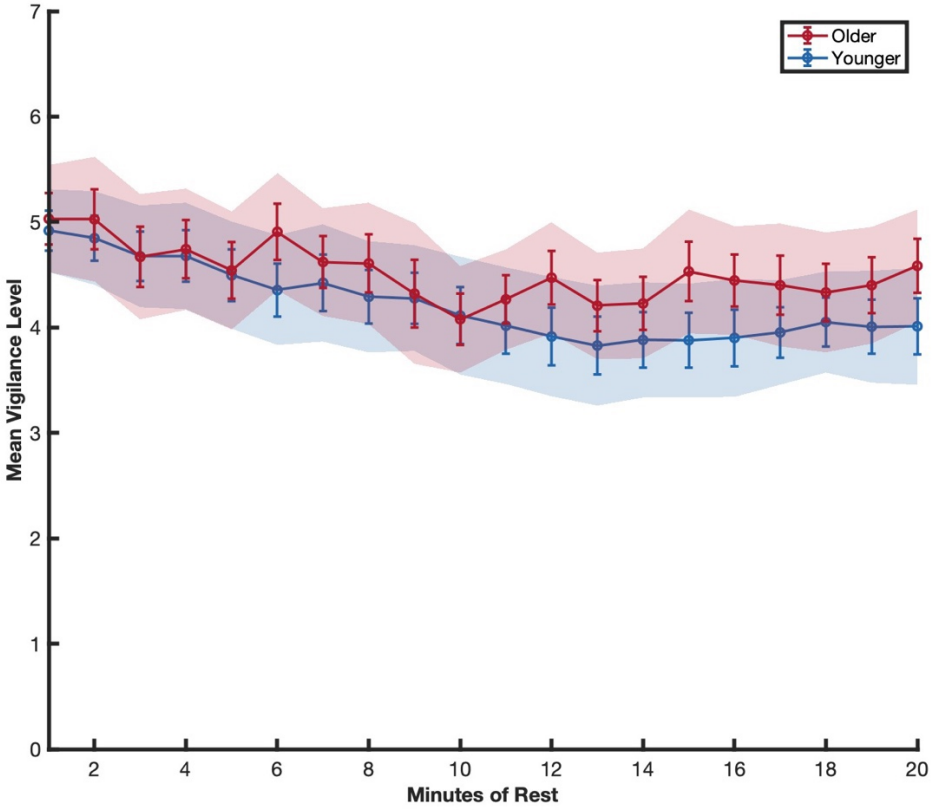
No data was removed prior to analysis. In the instances where the data fit the assumptions of a parametric test, parametric approaches were favoured. However, if the normality or homogeneity of variances assumptions were not met, then non-parametric approaches were used instead. All comparisons were performed with an alpha criterion of 0.05 ($\alpha = .05$).

3.3.1. Mean Vigilance Level Over Time

To compare mean vigilance levels over time between young and older adults, overall mean vigilance levels over 20 minutes were compared between age groups, along with mean vigilance divided into four 5-minute time blocks (Refer to Figure 3.3.1). The data was normally distributed (Shapiro-Wilk's test: $p's \geq .13$) and met the assumptions of homogeneity (Levene's Test: $p's \geq .67$). Table 3.3.1 provides a summary of test statistics.

Figure 3.3.1

Mean Vigilance Level of Young & Older Adults Across 20 Minutes of Rest



Note. Error bars indicate one standard error of the mean. Shaded regions indicate a 95% confidence interval.

Table 3.3.1

Summary Of Statistics Comparing Mean Vigilance Levels Across Age Groups & Time Blocks

	Mean Vigilance (Older) / (*SD*)	Mean Vigilance (Young) / (*SD*)	*t*	*p*	Significance	Effect Size
Total	4.52 (.97)	4.23 (1.07)	-0.98	0.34		0.29
Block						
Block 1 (Min 1 - 5)	4.8 (1.17)	4.72 (1.03)	0.24	1		0.07
Block 2 (Min 6 - 10)	4.51 (1.17)	4.29 (1.19)	0.64	1		0.19
Block 3 (Min 11 - 15)	4.34 (1.03)	3.9 (1.22)	1.3	1		0.39
Block 4 (Min 16 - 20)	4.43 (1.07)	3.98 (1.11)	1.34	1		0.4

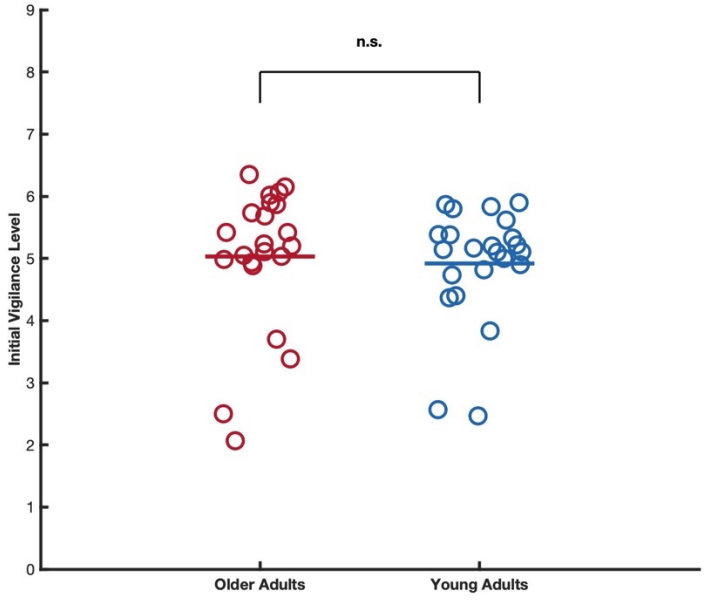
An independent t-test revealed no significant difference in mean vigilance over the 20 minutes (t (43) = -.98, p = .34). In addition to that, a 2x4 mixed-designed ANOVA between age groups and time blocks revealed a simple main effect of time blocks (F (3,129) = 12.49, p < .001), but not age groups (F (1,43) = .94, .34). There was also no significant interaction effect between age groups and time blocks (F (3,129) = 1.25, p = .30). To correct for the multiple post-hoc comparisons for time blocks, Holm corrections were performed for adjusted p-values. Post-hoc tests of time blocks revealed that vigilance levels were highest in Block 1 compared to Blocks 2, 3 and 4 (t's ≥ 3.20, p's ≤ .007, Cohen's ds' ≥ .32), and vigilance levels in Block 2 were higher than in Block 3 (t = 2.44, .049, Cohen's d = .25). All other pairwise comparisons revealed non-significant differences (p's ≥ .19, Cohen's ds' ≥ .07).

3.3.2. Initial Vigilance Level

To evaluate differences in initial vigilance levels, the averaged value of vigilance levels in the first minute of recording was compared between young and older adults. Initial vigilance level was not normally distributed (Shapiro-Wilk's test, young $p = .001$, older $p = .002$), but met the homogeneity assumption for variances (Levene's test, $p = .30$). A Mann-Whitney U test indicated that the initial vigilance levels for young ($M = 4.92$, $SD = p = .91$) and older adults ($M = 5.03$, $SD = 1.44$) were not significantly different ($U = 486.5$, $p = .34$, r = .14) (refer to Figure 3.3.2).

Figure 3.3.2

Comparison of Initial Vigilance Levels Across Young & Older Adults



Note. Horizontal lines going through points indicate mean values.

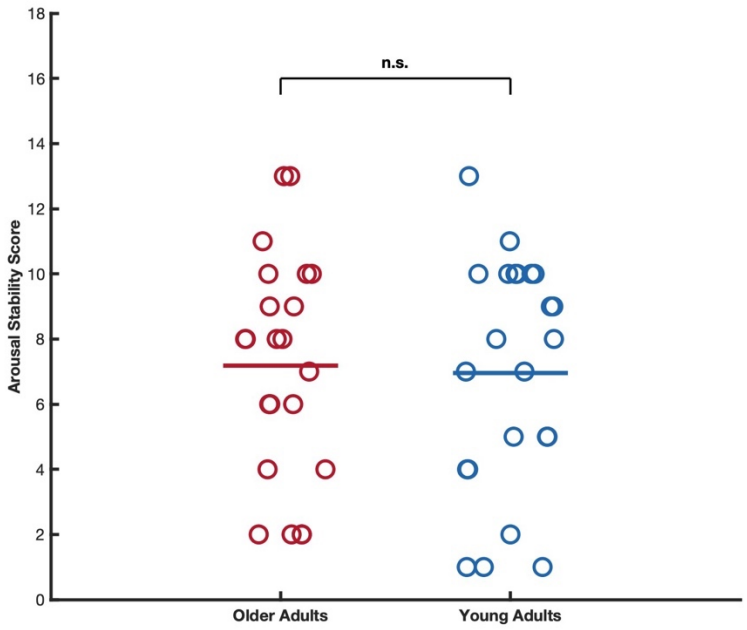
3.3.3. Arousal Stability Score

Arousal stability scores between young and older adults were normally distributed and met the homogeneity assumption of variance (Shapiro-Wilk's test, young $p = .07$, older $p = .18$,

Levene's test, $p = .87$). An independent t-test revealed that the arousal stability scores between young ($M = 6.96$, $SD = 3.56$) and older adults ($M = 7.18$, $SD = 3.43$) were not significantly different ($t(43) = -.22$, $p = .83$, Cohen's $d = .06$) (Refer to Figure 3.3.3).

Figure 3.3.3

Comparison of Arousal Stability Scores Across Young & Older Adults



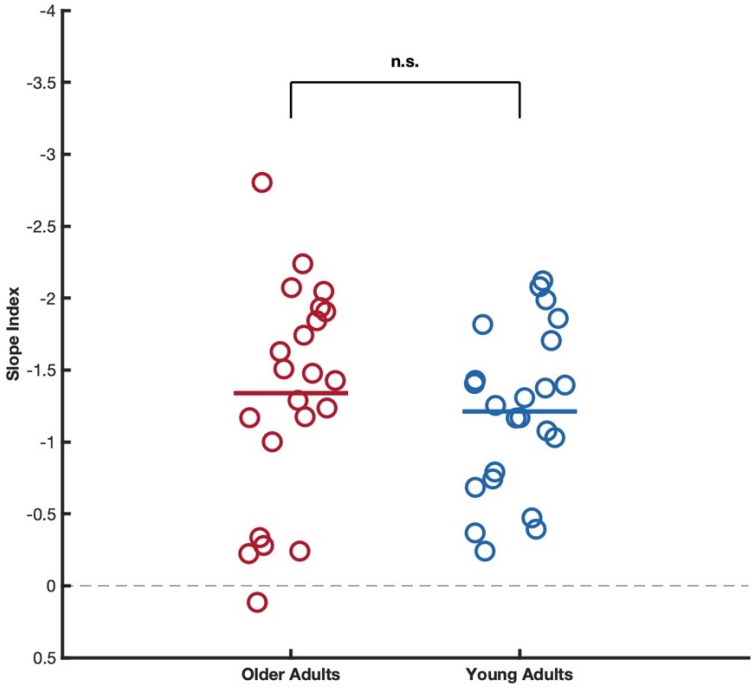
Note. Horizontal lines going through points indicate mean values. Higher arousal stability scores do not indicate 'higher stability'. Rather, higher scores are associated with unstable patterns, and lower scores are associated with hyperstable patterns.

3.3.4. Slope Index

Slope indices between young and older adults were normally distributed and met the homogeneity assumption of variance (Shapiro-Wilk's test, young $p = .45$, older $p = .35$, Levene's test, $p = .17$). An independent t-test revealed that the slope indices between young ($M = -1.21$, $SD = .56$) and older adults ($M = -1.34$, $SD = .76$) were not significantly different ($t(43) = .64$, $p = .52$, Cohen's $d = .19$) (Refer to Figure 3.3.4).

Figure 3.3.4

Comparison of Slope Indices Across Young & Older Adults



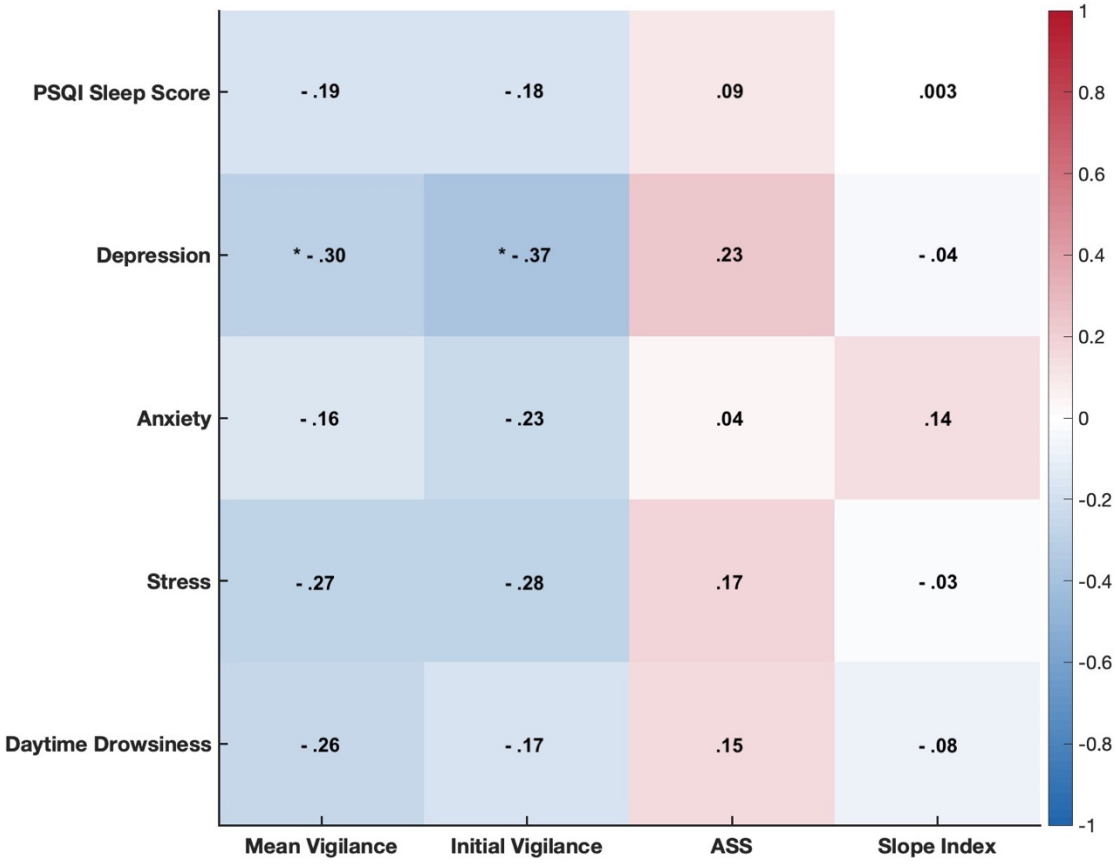
Note. Horizontal lines going through points indicate mean values. The polarity of the y-axis is inverted. Dashed line indicates 0 on the y-axis.

3.3.5. Correlations

To explore relationships between the arousal regulation parameters and sleep quality, negative emotional states, and daytime drowsiness, correlation analyses were performed across mean vigilance levels, initial vigilance levels, arousal stability scores, and slope indices on PSQI scores, DASS scores and ESS scores (refer to Chapter 2 for details of these dimensions; Refer to Figure 3.3.5).

Figure 3.3.5

Correlational Matrix of Arousal Regulation Parameters on Sleep Quality, Depression, Anxiety, Stress & Daytime Drowsiness



Note. Values within cells indicate Spearman's rho. Asterisk (*) indicates significance. * = $p < .05$

Spearman's correlations revealed that only mean vigilance (r = - .30) and initial vigilance levels (r = - .37) were negatively correlated with depression, indicating that higher scores of depression symptoms are associated with lower mean arousal levels in the first minute of rest, along with the whole duration of rest (i.e. 20 minutes). All other correlations were not significant (p's ≥ .06) (refer to Figure 3.3.5).

3.4 Discussion

In this chapter, a focus was placed on investigating arousal regulation patterns in young and older adults and examining their relationships with sleep quality, negative emotional states, and daytime drowsiness. From the analyses conducted, most findings did not align with initial hypotheses. Although mean vigilance levels decreased over time, suggesting transitions from higher to lower arousal states during rest, there were no significant differences in the patterns between young and older adults. This finding is contrary to expectations, where it was hypothesised that young adults would show higher mean vigilance scores over time. In addition to that, no significant differences were observed between young and older adults for initial vigilance levels, arousal stability scores and slope indices. The lack of group-level differences here is unexpected, given that the findings from the previous chapter identified robust group-level differences, with younger adults scoring higher on depression, anxiety, and stress symptoms, along with higher incidences of daytime drowsiness.

Besides that, the correlations performed between the arousal regulation parameters on sleep quality, negative emotional states and daytime drowsiness revealed lacklustre findings, with only mean vigilance levels and initial vigilance levels showing moderate negative correlations with depression. These findings are inconsistent with the literature, as previous investigations have found significant correlations between several of these related dimensions. For instance, studies have reported negative correlations between sleep quality and ASS (Ulke et al., 2016); negative correlations between mean vigilance levels, ASS, and slope indices with daytime drowsiness (Jawinski et al., 2017); and positive correlations between the ASS with daytime drowsiness, and negative correlations between the ASS with sleep latency (Olbrich et al., 2015). Note that daytime drowsiness was both positively and negatively correlated with ASS (Jawinski et al., 2017, Olbrich et al., 2015). However, the present findings found no significant correlations.

In addition to that, the negative correlations of mean total and initial vigilance levels with depression deviate from the expected relationship based on the arousal regulation model. According to the model, individuals with depression should exhibit hyperstable patterns, indicated by higher mean vigilance levels over time. However, in this study, the opposite relationship between depression symptoms and vigilance levels was observed. These findings are challenging to make sense of as mean vigilance levels alone are not an indicator of arousal regulation patterns. A more comprehensive account of arousal regulation patterns should also consider the other parameters, such as the ASS and slope indices. However, the ASS and slope indices did not show significant correlations with depression, which make it challenging to interpret the precise mechanisms by which mean vigilance levels may be related to depression.

In reference to the arousal regulation model (and the VIGALL by extension), the lack of group-level differences is unexpected. In this study, young adults had higher depressive symptoms compared to older adults (refer to Chapter 2). According to the model, this would predict that young adults would show arousal regulation patterns that resemble hyperstable patterns, indicated by higher mean vigilance levels, lower ASS, and lower slope indices. If age-related differences were observed in these depressive symptoms, it raises the question of why these were not reflected in the arousal regulation parameters. Several possible interpretations may explain this.

Firstly, since the measure of depressive symptoms in this study was recorded via the DASS, that might not be emblematic of the disorder itself (e.g. major depressive disorder, MDD). As depressive disorders tend to occur dimensionally, rather than on a spectrum of severity, higher depressive symptoms do not necessarily indicate the condition or disorder (Prisciandaro & Roberts, 2005). Hyperstable patterns may be a unique feature of clinical forms of depression or reflect very specific characteristics of depressive disorders that may

not have been adequately captured through the DASS in this study. For instance, previous studies detailing arousal regulation patterns in depression were conducted in patients suffering from major depressive disorder (Schmidt et al., 2016; Ulke et al., 2017). As participants in this study were specifically excluded from having a psychiatric diagnosis, it may be the case that symptoms of depression (as measured in this study) may not be pathological enough to manifest as an effect. This may explain the lack of correlations across the other negative emotional state dimensions as well (i.e. anxiety & stress). Perhaps non-stable arousal regulations may only affect certain characteristics of affective disorders. However, such examinations would be outside the immediate scope of this study, as it would involve looking into the taxonomy of depression and other affective disorders, along with examining the psychometric properties of the tools examining these conditions.

A second possibility for the non-significant differences in these observations may be due to a lack of account in exploring if these variables produce symmetric effects across young and older adults. As a theoretical argument, it may be possible that arousal regulation in older age could be modulated by different processes compared to young age (i.e. refer to previous sections discussing deteriorations of arousal-regulation regions in older age). As such, the effect of modulating variables vital to the model may present itself differently in older age. When grouped and analysed (such as in the correlation analyses performed in this chapter), possible age-group level effects are conflated and evened out, leading to non-significant associations. This might partially explain the absence of significant findings in this study when previous studies found robust associations between arousal regulation patterns and sleep measures (e.g. Ulke et al., 2016). In addition to this, this chapter examined the combined data of participants across a large age range, unlike previous studies that predominantly focused on specific age groups (e.g. 60-79 years; Jawinski et al., 2017; Ulke et al., 2016). Perhaps an isolation of these effects within narrower age ranges may enhance their

visibility. This possibility will be addressed in the final chapter of this thesis, where the effects of all variables examined in this thesis will be examined within their respective age groups.

In relation to the interpretation of the arousal regulation parameters themselves, it is worth noting that the demarcation between different arousal patterns is not very well defined. For instance, normative scores of mean vigilance levels classifying hyperstable, stable or unstable patterns are not clear. In previous studies utilising the VIGALL, no clear procedure has defined the boundary between the three conditions. Olbrich et al. (2015) attempted this by creating three clusters referring to the three patterns in their investigations but did not detail specifically how those clusters were defined. Secondly, arousal stability score scales tend to differ across protocols: for example, Jawinski et al. (2017) and Huang (2015) had an ASS range of 1 to 14, while Ulke et al. (2016) and Olbrich et al. (2015) only had an ASS range of 1-11. As such, the cutoff between those of different patterns may be a little arbitrary as there are no normative cut-off points, which makes cross-study comparisons slightly challenging. While native comparisons are possible within those designs, cross-study findings are difficult to interpret.

Another issue that is worth further discussion is the application of the VIGALL in its current state to examine arousal regulation, particularly in older adults. This specific point here relates to a limitation included in the VIGALL protocol, where it is noted that the VIGALL may have reduced accuracy when dealing with EEG containing alpha variant rhythms (i.e. alpha centre frequencies [individual alpha frequency, IAF] below 8.5 or above 12.5 Hz). This is crucial to the present thesis as the IAF has been shown to decrease in older age (Cesnaite et al., 2023), which may subsequently shift the alpha power range of older adults, an aspect that is vital to VIGALL classification. This might suggest that specific

considerations and adjustments must be accounted for when examining the EEG activity of older adults.

As key takeaways, the current chapter did not find significant differences between young and older adults with regard to arousal regulation parameters. In addition to that, previously examined relations of these parameters to sleep quality, depression and daytime drowsiness were not replicated in this sample of participants. This could be attributed to the absence of major pathological conditions in the participants of this sample, which could be crucial for detecting effects. Alternatively, this may have also occurred due to technical limitations relating to the application of the VIGALL. Finally, the arguments laid out in this chapter hint at the need for precise approaches to accommodate age-related sensitivities when comparing age differences, a premise which will be given the spotlight in the next chapter.

Chapter 4: Narrowband Power Analysis, Negative Emotional States, Daytime Drowsiness & Sleep Quality

4.1. Introduction

4.1.1. Background Information

Thus far, the investigations within the present thesis centred around understanding the effects of psychological variables, such as negative emotional states, daytime drowsiness, and resting-state cognition, and dissecting the contributions of age-related factors to better understand these influences on sleep quality.

In the previous chapter, a focus was placed on the arousal regulation model, offering a testable framework that implicated both psychological and neurophysiological factors as contributors to sleep deficits. Notably, it connected these factors with neuroanatomical developments in ageing, including deteriorations to cortical areas that are crucial for arousal and sleep-wake regulation. Combining those factors provided an empirical perspective to evaluate whether age-related changes to the brain can be represented as an observable measure, and at the same time, provide a context in which it can be interpreted. For example, if deteriorations to the arousal regulation systems (i.e. ARAS) occur in older age, are they accompanied by measurable changes to indices of arousal stability, such as the mean vigilance, arousal stability score, and slope indices? And more importantly, do those measures explain sleep deficits?

However, despite the thorough exploration, the age-related comparisons, and correlations between arousal regulation parameters with the variables of interest in the previous chapter did not yield significant findings. This could be due to unaccounted age-related shifts to EEG measurements. As hinted at in the previous chapter, this suggests that investigations of the ageing brain, particularly those utilising electrophysiological approaches such as EEG require nuance. It is crucial to consider and properly account for age-related

changes to the brain, as these alterations may consequently affect the signals that are recorded from the scalp. Within the context of the arousal regulation model, the VIGALL, which is the primary tool to assess arousal regulation, may have reduced accuracy in older-aged adults, due to the shifting of the individual alpha frequency. This limitation of the VIGALL underscores a broader more foundational issue that has been gaining attention in recent years — that being the definitions and characterisation of EEG properties. This issue is a vital aspect of consideration relevant to the thesis, as it is related to the parameters investigated in the study, such as age, negative emotional states, and sleep quality. It is also relevant to the VIGALL as these EEG properties are vital to the staging and labelling of the arousal stages, which themselves are used to derive arousal stability measures.

As such, this chapter examines these foundational issues at the methodological level: (i) the individual alpha frequency (IAF) and individual alpha peak frequency (IAPF); (ii) the definition of alpha and theta band activity from a conventional, canonical approach, and the individualisation and parameterised approach.; (iii) and lastly, it explores the properties of electrophysiological signals, encompassing both periodic and aperiodic components. This is the central premise of the present chapter and is the final angle of exploration before a fuller interpretation of all variables explored in this thesis can be synthesised in the final chapter.

4.1.2. Individual Alpha Peak Frequency (IAPF) & Individual Alpha Frequency (IAF)

The human EEG in a state of relaxed wakefulness is characterised by the alpha rhythm, notably pronounced towards posterior regions of the brain, referred to as the posterior dominant rhythm (St. Louis & Frey, 2016). These alpha rhythms exhibit variability across individuals, occurring within a frequency band known as the individual alpha frequency (IAF). The IAF centres around the individual alpha peak frequency (IAPF), defined as the

frequency peak within a band region where the power of signals is highest, commonly within the 7.5-12.5 Hz range (Klimesch, 1999).

The IAF is a widely studied effect, beginning with its conception of alpha rhythms observed by Berger (1929), who observed brain rhythms occurring at frequencies of 8-11 Hz that seemed to be most prominent when people were relaxed with their eyes closed (Quigley, 2022). Contemporary investigations also link IAF variability to psychological well-being, cognitive processing, and the development of psychiatric and neurodegenerative disorders. For instance, decreased IAF has been linked to depressive symptoms and the onset of mild cognitive impairment (Tement et al., 2016). Likewise, reduced IAFs have been linked to lower general intelligence, slower processing speed, reduced performance on inhibition and memory tasks and poorer sleep-modulated memory consolidation (Cesnaite et al., 2023; Cross et al., 2020; Grandy, Werkle-Bergner, et al., 2013; Grandy, Werkle-Bergner, et al., 2013; Klimesch, 2012).

Despite high inter-individual variability, intra-individual IAF tends to remain stable across the lifespan in the absence of pathologies. As such, this makes the IAF a useful tool to track the development of pathologies, as slowing (i.e. decreasing from higher to lower frequencies) of the IAF may indicate early signs of a disorder, for example, dementia and schizophrenia (Grandy Werkle-Bergner, Chicherio, Lövden et al., 2013; Moretti et al., 2004; Ramsay et al., 2021). Beyond its utility in investigating psychological, cognitive, and pathological correlates, the IAF is also particularly useful in studying the ageing brain. The IAF has important consequences for other EEG-related measures, especially the individual alpha peak frequency (IAPF). Parallel to the IAF, the IAPF has been shown to also decrease in older age, falling between ranges of 8.5- 9.7 Hz in older adults 60 years and older, in contrast to averages of 9.8-10.5 Hz in younger age (Dustman et al., 1993; Scally et al., 2018).

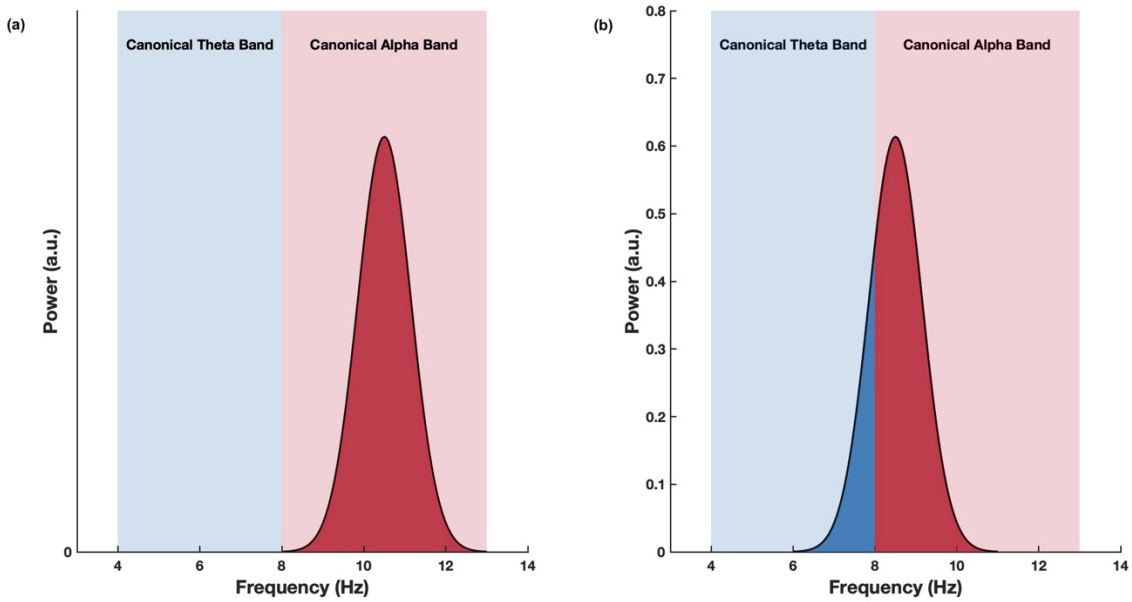
4.1.3. Calculating Alpha & Theta Power Using Canonical and Individualised Approaches

Alpha and theta power are pivotal and widely studied signals in EEG studies, as they can offer valuable insights into brain development and pathology and allow for inferences to be made about cortical activity. The conventional definition of alpha and theta power relies on canonically defined frequency bands, with alpha typically ranging from 8 to 12 Hz, and theta from 4 to 8 Hz (e.g. Marecek et al., 2016; Zink et al., 2016). There is however a lack of standardisation in the boundaries of these ranges, as alpha has also been cited as 8-13 Hz (e.g. Fumoto et al., 2004; van Netten et al., 2008) or 8-14 Hz (Palva & Palva, 2007), and theta bands have also spanned 4-7 Hz (Fumoto et al., 2004; Kay, 2005). Notably, the relationship between alpha and theta is strongly related, as the lower boundary of alpha often marks the upper boundary of the theta band (Klimesch, 1999).

Alpha and theta power demonstrate functional correlates with several aspects of cognition and show variability within individuals of different cognitive status and age (refer to Chapter 1.3). Particularly with theta power, it has been shown to both increase and decrease in older age (e.g. Babiloni et al., 2015; Barry & De Blasio, 2017; Vlahou et al., 2014). While these variabilities are modulated by other psychobiological factors, a portion of these findings may be attributed to misinterpretations of narrowband power findings resulting from the rigidity of pre-defined frequency bands and not accounting for shifts in individual alpha in older age. As an illustrated example of this, refer to the power spectral density (PSD) plots in Figure 4.1.3.

Figure 4.1.3

Simulated PSDs with ~10.5 Hz and ~9 Hz Alpha Peak Frequencies Within Canonical Alpha & Theta Bands



Note. PSDs (Power Spectral Densities) analyse a signal's frequency composition, describing the power of the signal at specific frequencies. In (a) the alpha peak frequency is ~10.5 Hz, with a 2.5 Hz half-width, illustrating an alpha band with a frequency range of 8-13 Hz. When the alpha frequency fits within the canonical boundaries, the entire signal of the alpha frequency is properly captured by the canonical shaded alpha window. However in (b) the alpha peak frequency is slower, ~9 Hz with a 2.5 Hz half-width, creating an alpha frequency range of 6-11 Hz. In these instances where the alpha frequency is shifted lower (i.e. to the left), the lower tail of the PSD crosses over into the canonical theta band. This can result in the conflation of lower alpha power as theta power due to the rigid pre-defined frequency bands. a.u. refer to arbitrary units; The PSDs plotted in the above figure are simulated Gaussian peaks centered around a peak alpha frequency.

In the context of ageing, the individual alpha peak frequency (IAPF) tends to slow, leading to a downward shift of the IAF. The failure to account for these shifts during the computation of alpha and theta power within the rigid canonical bands can result in a conflation of results. Specifically, the crossover of lower alpha power into the theta range

simultaneously leads to an increase in theta power and a reduction of alpha power (see Figure 4.1.3). This phenomenon may contribute to previous findings indicating an increase in theta power in older age, suggesting a potential misinterpretation due to the interference of lower-shifted alpha into the conventional theta frequency (Cesnaite et al., 2023). This is a foundational issue that highlights the limitation of the conventional canonical approach to frequency definition, which calls for more careful approaches that consider these shifts.

While recent hypotheses (e.g. Cesnaite et al., 2023) have addressed these issues that may explain inconsistencies in the ageing literature, the advocacy of individualised approaches to investigating spectral power dates to 1999 by Klimesch. Klimesch (1999) recognised the high inter-individual variability of alpha frequency and suggested adjusting the alpha frequency bands (i.e. IAF) based on the IAPF. Adopting such individualised approaches, where the IAF are uniquely fit to each individual, would help prevent the conflation of power between frequency bands. Supplementing this point, Cesnaite et al. (2023) specifically compared the canonical and individualisation approaches and revealed that group-level differences become nonsignificant once individual fits are accounted for. In addition to that, the variability of the IAPF is not unique across age groups but is also a concern among individuals with different psychological and cognitive statuses, such as dementia and schizophrenia (Moretti et al., 2004; Ramsey et al., 2021). Collectively, these considerations challenge the applications of the conventional approach, highlighting the need for the commonplace adoption of individualised approaches in EEG studies.

4.1.4. Parameterisation of EEG Signals into Periodic & Aperiodic Components

The interpretation of EEG-derived findings extends beyond understanding narrowband-limited power and the frequencies that define it, as it must also consider the nature and properties of electrophysiological signals themselves. When looking into EEG signals, there

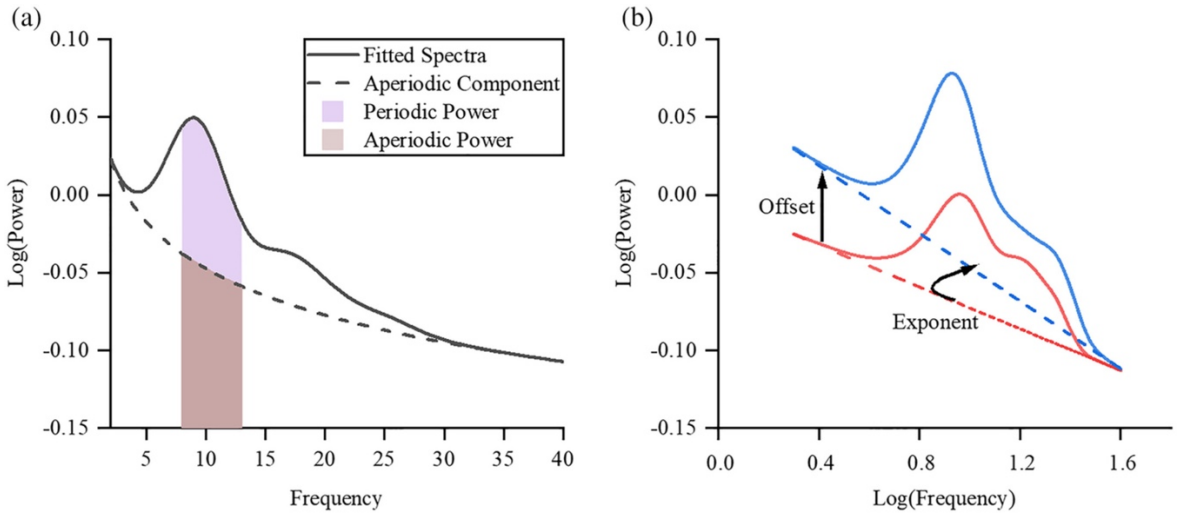
is a particular emphasis placed on establishing links between neural oscillatory behaviour and cortical activity, such as in cognitive or brain health measures. To be more specific, these oscillations of interest are rhythmic activity, which is often represented in the frequency domain, such as in theta and alpha power— conventionally calculated within canonical frequency bands (see section above). Inferences are then made based on the power within these frequency bands as calculated via spectral power analysis to form interpretations about a particular aspect of the brain. However, this simplistic approach overlooks an important feature of these electrophysiological signals, which is a critical signal that has direct effects on the measurements of rhythmic activity — the non-rhythmic activity.

Rhythmic and non-rhythmic activity refers to the periodic and aperiodic components that make up the EEG signal. While the periodic components (e.g. alpha and theta frequencies) may be the primary focus, they are embedded with aperiodic components characterised by a 1/f slope. This aperiodic component is believed to represent neural "noise", reflecting random background electrical fluctuations within the central nervous system (Donoghue et al., 2020). The slope of this aperiodic component is characterised by an offset (the y-intercept) and an exponent value, which reflects a balance of excitatory and inhibitory inputs in the brain, which are modulated by glutamatergic and GABAnergic connections respectively (Wang, 2020). Higher excitatory connections are associated with flatter 1/f slopes, in contrast to higher inhibitory connections which produce a steeper slope. Relatedly, an increase in excitatory connections produces higher "neural" noise, which resultantly creates higher interference in the brain, manifesting as more impulsive and faster reaction times, at the expense of accuracy (Cesnaite et al., 2023; Dave et al., 2018; Voytek et al., 2015a). Since these two components are heavily intertwined, changes affecting the aperiodic activity have a direct effect on the periodic activity, suggesting that it cannot be ignored when forming inferences about electrophysiological signals recorded from the brain.

As similarly argued in the previous section relating to the conceptual issues utilising canonical frequency bands, many EEG studies also do not make efforts to separate (i.e. parameterise) these different component sources of the EEG signal. This can be problematic when interpreting EEG findings, as periodic and aperiodic components reflect different aspects of cortical activity, where failure to account for the individual contributions of these components can also lead to misrepresentations of power. Due to the nature of the 1/f slope, narrowband power analysis will always estimate a non-zero power, even when no detectable oscillations are present, concerningly this would manifest as an effect, where there is none (Donoghue et al., 2020). Refer to Figure 4.1.4 for illustration and further explanation.

Figure 4.1.4

Illustration of Periodic and Aperiodic Power



Note. These illustrations describe the contributions of periodic and aperiodic power to an overall power spectrum. From "Separating the aperiodic and periodic components of neural activity in Parkinson's disease" by Wang, Z. et al., 2022, European Journal of Neuroscience, 56(6), pp. 4889–4900. In (a), the PSD is presented in semilog-power space, with log-spaced power values on the y-axis and linearly spaced frequencies on the x-axis. The solid line represents the overall power of the signal with the dashed line representing a fit of a 1/f slope. The shaded regions represent the overall power in the alpha frequency band. Note that the

shaded regions are divided into purple regions representing periodic power, and brown regions representing aperiodic power. The periodic component of the signal sits on top of the aperiodic component, and if directed approaches are not made to delineate between these components, there is a conflation of the periodic ("true" alpha power) and aperiodic power, which can lead to a misrepresentation of the alpha power. Also note that contributions of the aperiodic component are not linear across increasing frequencies, with aperiodic components showing higher interference at lower frequencies when compared to higher frequencies. (b) Illustrates how offset and exponent values affect the 1/f slope, represented in log-log space. The offset value represents the y-intercept of the slope and indicates the power of the signal; the exponent value represents the gradient, or how steep the slope is. The more negative the exponent, the steeper the curve.

Of immediate relevance to the topic of study in this thesis, the 1/f slope has been shown to become flatter in older age (Dave et al., 2018; Voytek et al., 2015), indicating higher excitatory connections (i.e. "noisier" background cortical activity). This is also a common feature observed in dementias and cognitive dysfunction, a connection that may have important implications in understanding the ageing brain, particularly the pathologies that accompany it (Voytek & Knight, 2015). In retrospect to the literature that has found increased theta in older age (e.g. Babiloni et al., 2016), together with sleep-related pathologies (i.e. increased theta in sleep apnea; e.g. Wu et al., 2020) and other neurodegenerative disorders, it may be possible that the effects observed may not be due exclusively to an increase of power in the theta band, but rather that the aperiodic "noise" may be higher in these populations, subsequently contributing to a higher overall theta, as both components are conflated with each other. These interpretations are supported by Cesnaite et al. (2023), who in their investigations found that the differences in theta power in

young and older adults became nonsignificant once the aperiodic components were subtracted from the overall signals.

Combining these interpretations with the canonical approach to narrowband frequency analyses might then explain the inconsistencies reported across the ageing literature. Increased theta power may be attributed to the interference of the lower alpha power within canonical theta bands, as well as due to the contribution of the higher aperiodic signals that disproportionately affect lower frequencies in comparison to higher frequencies (i.e. the aperiodic signal creates more "noise" at the theta band, compared to the alpha band). As such, precise methodological approaches must be incorporated into examinations of EEG activity, particularly when pertaining to older adults to disentangle different source effects to better understand the complex relationships between ageing, the brain, the development of poor psychological and health outcomes, and the focus of this study, sleep quality.

4.1.5. Goals of The Present Chapter

This chapter places a significant emphasis on methodological approaches related to EEG narrowband analyses. This emphasis is crucial as it examines the overall thesis research topic from a more fundamental perspective, which may provide reconciliatory evidence for existing findings. By having careful, directed efforts to contrast these approaches, it may also set the precedent for future investigations of a similar kind.

The following sections will provide detailed descriptions of these methodological approaches and how they were applied to the current dataset. Subsequently, analyses will be conducted using the conventional approach, adhering to canonical definitions of alpha and theta frequency bands and without parameterisation of the data. This approach will then be contrasted with the method that incorporates both the individualised fitting of alpha and theta frequency bands and the parameterisation of data into periodic and aperiodic components.

Age-group comparisons will then be run within these approaches to assess how well the findings agree with each other.

In addition to those comparative analyses, correlations will be explored to understand potential relationships between these narrowband EEG-derived parameters and factors of interest in this study, those being sleep quality, negative emotional states, and daytime drowsiness. Refer to Table 4.1 for an overview of the present research questions (RQ) and hypotheses.

Table 4.1

Table of Research Questions & Corresponding Hypotheses

RQ1	**How do conventional approaches to EEG narrowband analyses affect age-related differences in power?**
H1a	Alpha power will be lower in older adults compared to young adults.
H1b	Theta power will be higher in older adults compared to young adults.
RQ2	**How will the individualisation approach & parameterisation of EEG data affect age-related differences in power?**
H2a	Alpha power will not be significantly different between young and older adults.
H2b	Theta power will not be significantly different between young and older adults.
H2c	Older adults will have a reduced IAPF compared to young adults.
H2d	Older adults will show higher aperiodic power compared to young adults as indicated by more negative exponents and higher offsets.
RQ3	**How might EEG narrowband parameters correlate with self reported sleep quality, negative emotional states and daytime drowsiness?**

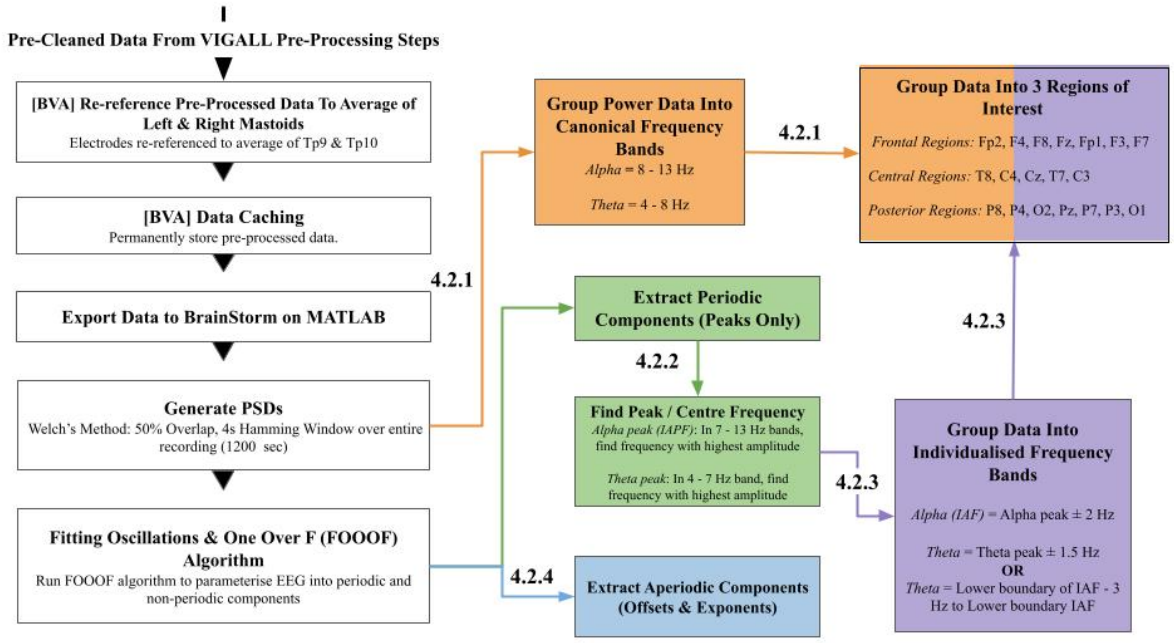
Note. RQ3 lacks specific hypotheses due to limited evidence linking parameterised and individualised parameters with the variables of interest. Therefore, the correlations conducted are exploratory.

4.2. Methods

The following subsections will describe the canonical approach to alpha and theta frequency power extraction (4.2.1). This will be followed by a description of the parameterisation of EEG data via the Fit-Oscillations-&-One-Over-F (FOOOF) algorithm via the MATLAB implement on BrainStorm (Tadel et al., 2011). This will include parameterising the data into periodic and aperiodic components, where the individual peak alpha frequency (IAPF) will be ascertained to define the individual alpha frequency (IAF) and theta frequency (4.2.2). Following that, narrowband frequency power extraction will be performed on the periodic components with the individualised alpha and theta frequency bands (4.2.3). Finally, the aperiodic components (i.e. offset and exponents) will be extracted (4.2.4). Refer to Figure 4.2.1 for the flowchart of operations.

Figure 4.2

Flowchart of Operations to Extract EEG-Derived Parameters



Note. Coloured boxes correspond to labelled subsections describing the procedures performed to extract EEG parameters. Orange = 4.2.1; Green = 4.2.2; Purple = 4.2.3; Blue =

4.2.4. [BVA] refers to the steps being run on BrainVision Analyzer (Version 2.2.2, Brain Products GmbH, Gilching, Germany).

4.2.1. Computation of Power According to Canonical Frequency Bands Without Parameterisation

As the data preprocessing pipeline of the VIGALL was extensive and included the typical steps used in data preparation for narrowband analysis (e.g. band-pass filtering, interpolation of bad electrodes, ICA removal), that pre-processed data was simply carried forward for computations used in this chapter (refer to Chapter 3 for data pre-processing pipeline). However, as electrodes were originally referenced to Cz, the data had to be re-referenced to the average of the mastoid electrodes (Tp9 & Tp10). This step was performed to "free up" the Cz electrode, as it would be included as an electrode of interest in subsequent analyses. In addition to that, this was also done to align this study's methods to the methodology of Barry and DeBlasio (2017), who compared resting-state EEG parameters between healthy young and older adults, a comparison relevant to the present study. The re-referencing was performed on BrainVision Analyzer (Version 2.2.2, Brain Products GmbH, Gilching, Germany), and was then exported to BrainStorm (Tadel et al., 2011), an open-source application operating within MATLAB that is dedicated to the analyses of neurophysiological data, such as MEG, EEG, fNIRS etc.

For each participant, power spectral densities (PSDs) were calculated for each channel's data across the entire rsEEG recording (~1200) with a 4-second Hamming window with a 50% overlap utilising Welch's method. Alpha and theta power were subsequently derived from these PSDs, calculated within the BrainStorm interface by computing the mean power within the frequency of 4-8 Hz for theta power, and 8-13 Hz for alpha power. The data was then exported to MATLAB (ver. R2022b) for further processing. For each participant,

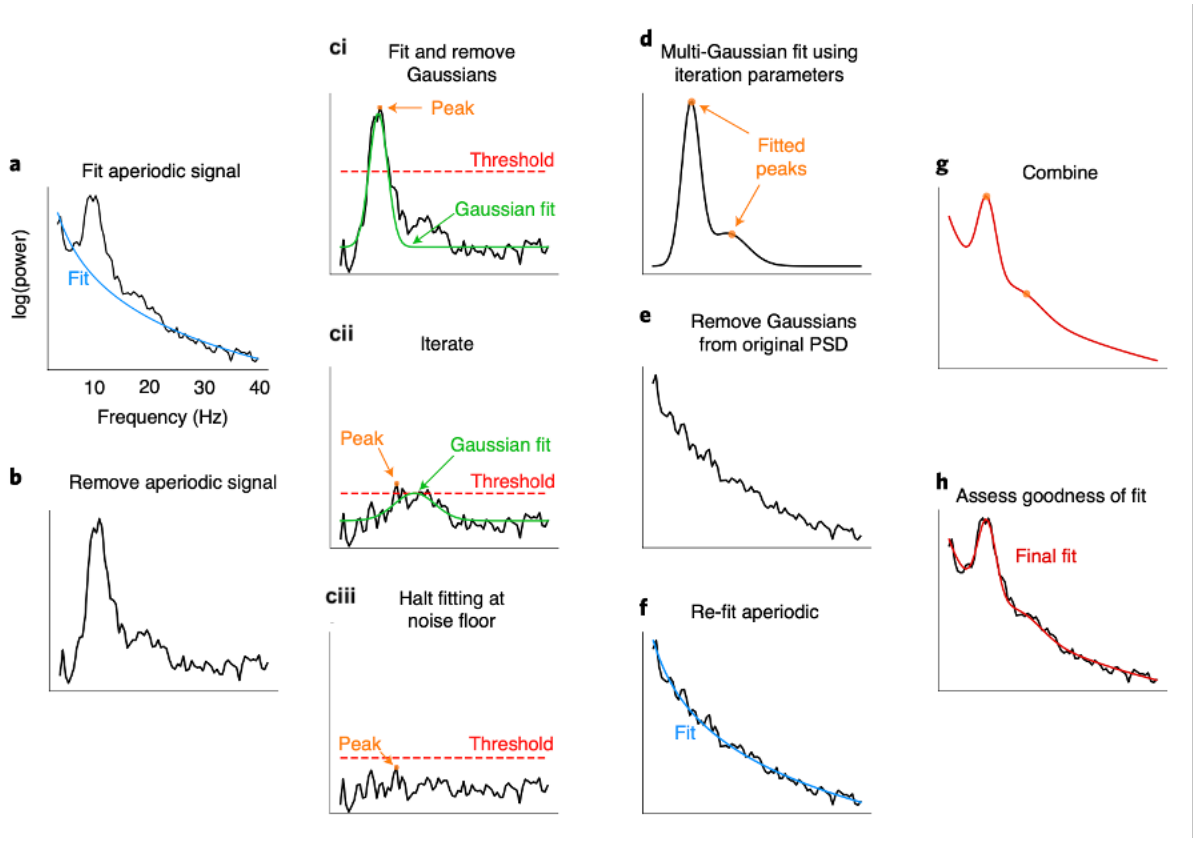
mean alpha and theta values were computed for each of the 28 channels. From these channels, 19 were utilised and categorised to form three regions of interest according to Barry and DeBlasio (2017): Frontal sites (Fp1, Fp2, Fz, F3, F4, F7, F8); centre sites (Cz, C3, C4, C7, C8); and posterior sites (Pz, P3, P4, P7, P8, O1, O2). The mean alpha and theta power within each of these sites were calculated by averaging the powers for all channels within the respective region. A global mean of alpha and theta power was also computed, representing the averaged power across all 28 channels. The PSD output values from BrainStorm were initially in V^2/Hz, which were then scaled into microvolts ($\mu V^2/Hz$) by dividing the signal values by $1E\text{-}6^2$.

4.2.2. Parameterisation of EEG Data, Extraction of Peaks & Individualised Frequency Bands

To parameterise the periodic and aperiodic components of the EEG signal, the Fitting-Oscillations-And-One-Over-F (FOOOF) algorithm by Donoghue et al. (2020) was utilised. The MATLAB implementation of the FOOOF was conducted within the BrainStorm interface (Tadel et al., 2011). For a conceptual explanation of the FOOOF algorithm, refer to Figure 4.2.2.

Figure 4.2.2

FOOOF Algorithm to Parameterise Signals into Periodic & Aperiodic Components



Note. Description of the FOOOF algorithm. From "Parameterizing neural power spectra into periodic and aperiodic components." By Donoghue, T., Haller, M., Peterson, E.J. et al, 2020, Nat Neurosci **23**, 1655–1665. The FOOOF algorithm of Donoghue et al. (2020) takes a computed PSD utilising the Welch method and parameterises the signals into periodic and aperiodic components. (a.) An estimated aperiodic component is fit onto the original PSD. (b.) The estimated component is then subtracted from the raw PSD, with the residual signals assumed to be a mix of periodic activity and additional noise. (ci.) A maximum peak is first identified from the residual signals, and if the peak is above the noise threshold (red dashed line), a Gaussian is fit around the peak based on the peak's frequency, power, and estimated bandwidth. This Gaussian is then subtracted from the residual signal. (cii.) This process is reiterated for each subsequently identified peak (ciii.) until the peaks no longer pass the noise

threshold. (d.) After identifying the number of putative peaks (and Gaussians), the multi-Gaussian is then fitted onto the original aperiodic-subtracted signal (i.e. the signal derived in [b.]) to account for the joint power of all the combined Gaussians. (e.) The multi-Gaussian model is then subtracted from the raw PSD (i.e. the original PSD). (f.) Using the residuals, a new aperiodic component is estimated. (g.) The re-fit aperiodic component is then combined with the multi-Gaussian model to give a final fit of the data. (h.) The fit of the final model is then assessed against the raw PSD. Donoghue et al. (2020) report that this method allows for >99% of variance in the raw PSD to be accounted for (Refer to the methods of Donoghue et al., 2020 for a detailed explanation of the algorithm, including formulas utilised by the models.).

Once the FOOOF algorithm was executed across all datasets, peak searches were performed to ascertain the individual peak alpha frequency (IAPF) and theta peaks for each participant. For the IAPF, the peak search was conducted between 7-13 Hz, with the IAPF defined as the frequency at which the amplitude was the highest. A similar search was performed to identify the theta peak within 4-7 Hz, though only 20% of participants exhibited a theta peak.

The alpha frequency band for each participant (IAF) was defined using the IAPF as the anchor point, extending a 2 Hz band to the left and right of the IAPF to define the alpha range (e.g. IAPF = 8.5 Hz, IAF = 6.5–10.5 Hz). In the case of the individual theta frequency band, if a theta peak was identified, a 1.5 Hz band was extended to the left and right of it to define the individual theta band. However, if no theta peak was present, the theta range was defined as a 3 Hz window extending from the lower boundary of the IAF and 3 Hz downward from it (e.g. if the lower boundary of IAF was 6 Hz, the individual theta range was defined as

3-6 Hz). This approach was also utilised by Cesnaite et al. (2023) when defining the individualised theta frequency bands.

4.2.3. Computation of Power According to Individualised Frequency Bands with Parameterisation

The extraction of alpha and theta power from the parameterised data (i.e. periodic components) follows similar steps used to extract alpha and theta power in the canonical bands (refer to 4.2.1).

Initially, the mean alpha and theta power for each channel were computed for each participant using their individualised alpha and theta frequency bands. This involved manually defining the corresponding frequency bands on each participant's dataset within BrainStorm. Subsequently, the data was exported to MATLAB (ver. R2022b) for further processing. The mean alpha and theta power were then calculated for the same three regions of interest (frontal, centre, and posterior), alongside a global alpha and theta power that included the average power across all 28 channels.

It is worth noting that the power derived from the periodic components of the FOOOF model is represented as $\log(\mu V^2/Hz)$. This representation is due to the FOOOF computing the model in semilog-power space, requiring that the interpretation of periodic signals be represented in log-space as well.

4.2.4. Extraction of Aperiodic Components

The aperiodic components (i.e. offset and exponents) were extracted from the FOOOF aperiodic model. Each participant had a total of 28 associated offset and exponent values, corresponding to the number of channels. To simplify subsequent statistical analyses, the

average of all 28 values was computed, resulting in a single value to represent the mean offset and exponent for each participant.

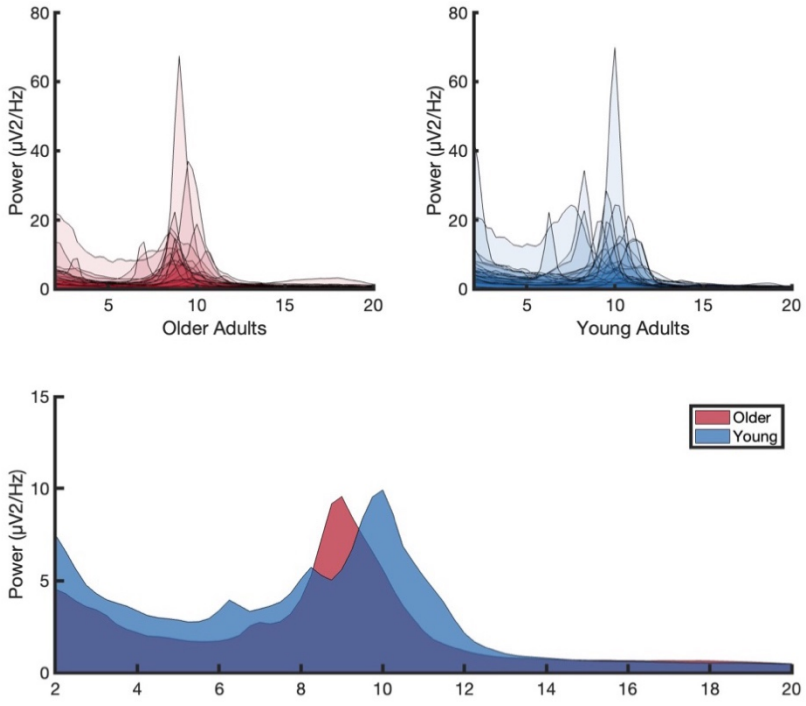
4.3. Results

No data was removed prior to analysis. In the instances where the data fit the assumptions of a parametric test, parametric approaches were favoured. However, if the normality or homogeneity of variances assumptions were not met, then non-parametric approaches were used instead. All comparisons were performed with an alpha criterion of 0.05 (α = .05).

4.3.1. Canonical Alpha & Theta Power

Figure 4.3.1a

Canonical PSDs Of Young & Older Adults



Note. Figures on the top indicate overlapped PSDs of all participants within the age group. The figure on the bottom indicates the overlap of the averaged PSDs of young and older adults.

Canonical alpha and theta power were not normally distributed across young and older adults. Additionally, they did not meet the homogeneity assumption for variances in certain regions (Shapiro-Wilk's test: p's < .01; Levene's test: $.002 \leq p \leq .86$). As such, Mann-Whitney U tests were conducted across all pair-wise comparisons across age groups for global power and power across regions. Refer to Table 4.3.1b and Figure 4.3.1b for the summary of test statistics for canonical alpha power; and Table 4.3.1c and Figure 4.3.1c for summary of test statistics for canonical theta power.

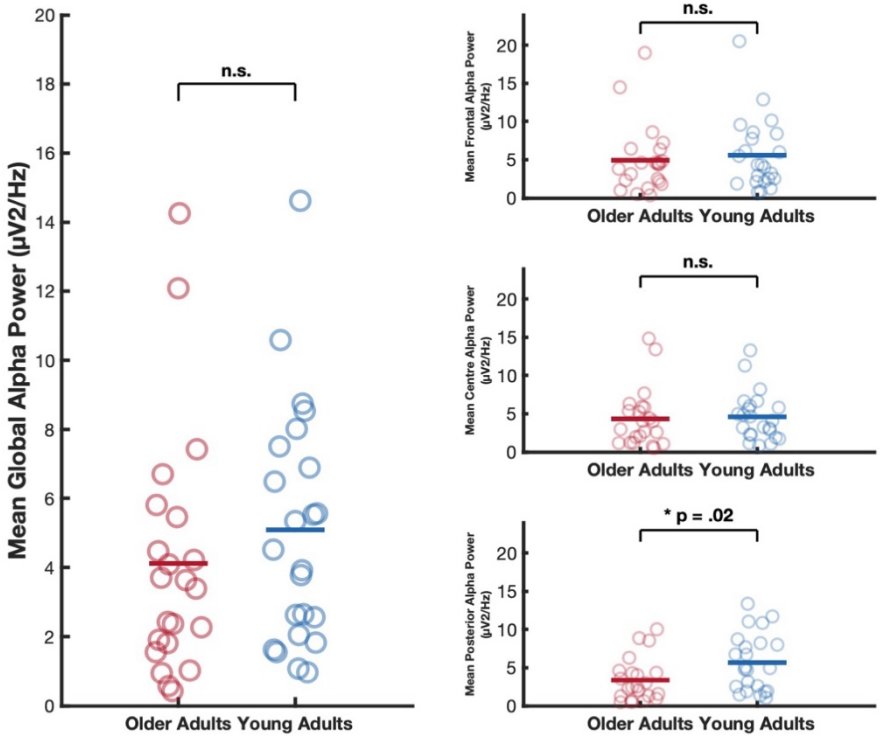
Table 4.3.1b

Summary Of Statistics Comparing Mean Canonical Alpha Power Across Age Groups & Regions

	Mean Canonical Alpha Power (μV² / Hz) / (*SD*)					
	Older Adults	**Young Adults**	**U**	**z**	***p***	**Effect Size (Cohen's *d*)**
Global Alpha	4.12 (3.53)	5.09 (3.47)	449	-1.28	.20	-.19
Frontal	4.93 (*4.46*)	5.58 (*4.64*)	487	-.42	.67	-.10
Centre	4.33 (*3.79*)	4.60 (*3.17*)	477	-.45	.52	.34
Posterior	3.37 (*2.84*)	5.68 (*5.68*)	404	-2.30	.02	-.20

Figure 4.3.1b

Comparisons of Canonical Alpha Power Between Young & Older Adults



Note. Horizontal lines going through points indicate mean values. Asterisk (*) indicates significance. * = $p < .05$

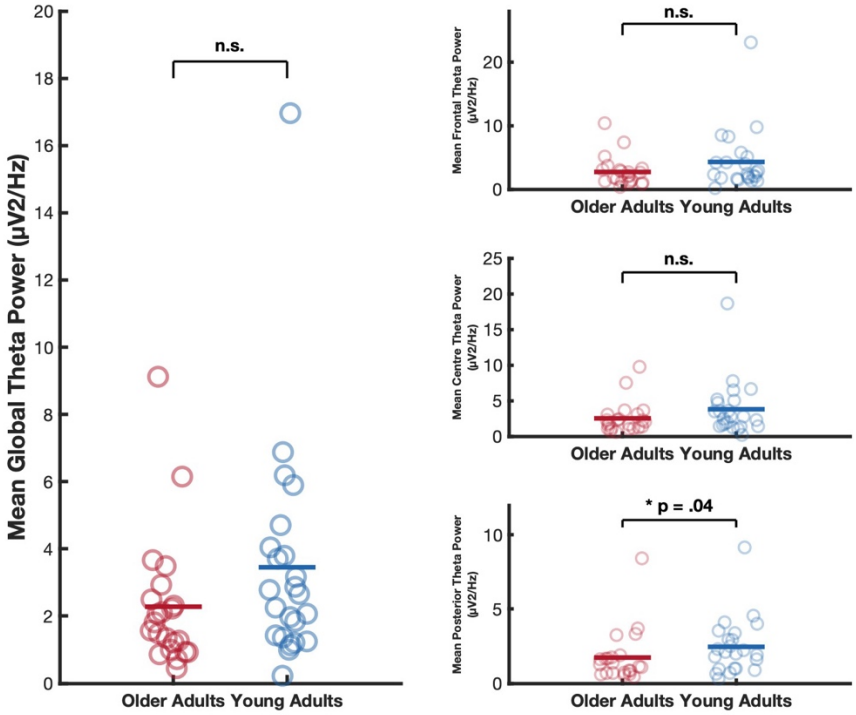
Table 4.3.1c

Summary Of Statistics Comparing Mean Canonical Theta Power Across Age Groups & Regions

	Mean Canonical Theta Power (µV² / Hz) / (*SD*)					
	Older Adults	**Young Adults**	**U**	**z**	***p***	**Effect Size (Cohen's *d*)**
Global Theta	2.28 (*1.99*)	3.45 (*3.44*)	432	-1.67	.10	-.25
Frontal	2.71 (*2.33*)	4.28 (*4.81*)	447	-1.33	.18	-.20
Centre	2.51 (*2.19*)	3.79 (*3.81*)	439	-1.51	.13	-.23
Posterior	1.74 (*1.76*)	2.46 (*1.89*)	414	-0.31	.04	-.31

Figure 4.3.1c

Comparisons of Canonical Theta Power Between Young & Older Adults



Note. Horizontal lines going through points indicate mean values. Asterisk (*) indicates significance. * = $p < .05$

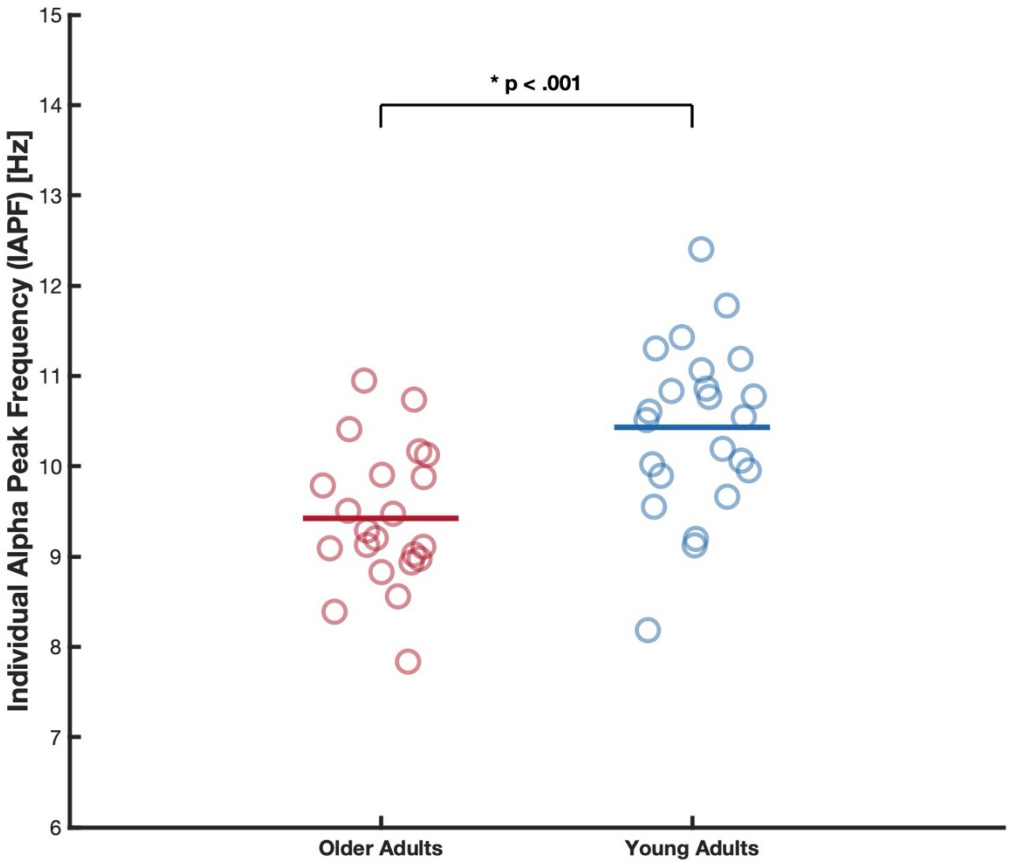
Posterior canonical alpha and theta power were significantly higher in young adults compared to older adults (p's ≤ .04). However, global, frontal, and centre canonical alpha and theta power were not significantly different between young and older adults (p's ≥ .10).

4.3.2. Individual Alpha Peak Frequency

The individual alpha peak frequencies (IAPF) were normally distributed and met the homogeneity assumption for variances (Shapiro-Wilk's test: young p = .88, older p = .92; Levene's test: p = .34). An independent samples t-test indicated that the IAPF was significantly higher in young adults (M = 10.43, SD = .94) compared to older adults (M = 9.42, SD = .77) ($t(43)$ = -3.92, p < .001, Cohen's d = -1.17; refer to Figure 4.3.2).

Figure 4.3.2

Comparison of Individual Alpha Peak Frequency (IAPF) Between Young & Older Adults

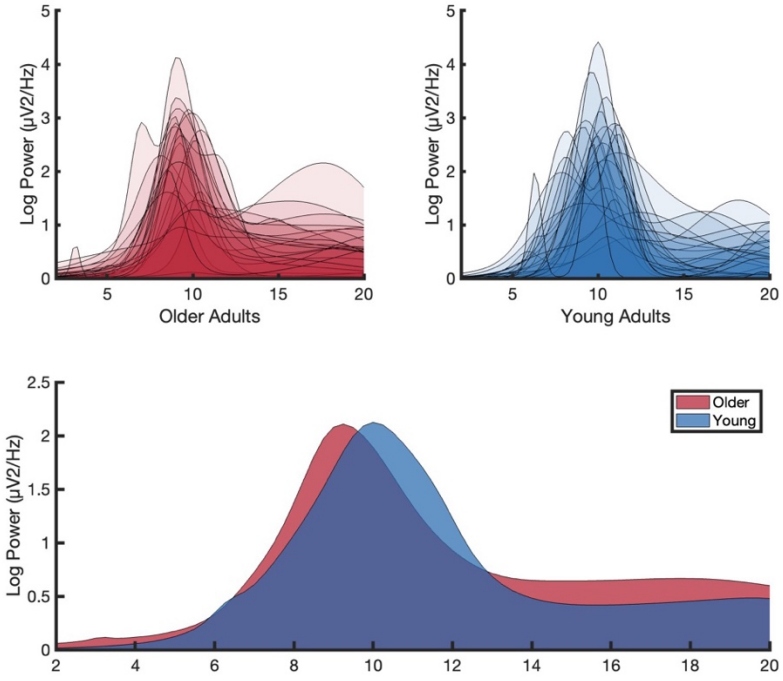


Note. Horizontal lines going through points indicate mean values.

4.3.3. Parameterised & Individualised Alpha & Theta Power

Figure 4.3.3a

Parameterised PSDs Of Young & Older Adults



Note. Figures on the top indicate overlapped PSDs of all participants within the age group. The figure on the bottom indicates the overlap of the averaged PSDs of young and older adults.

Parameterised and individualised alpha power was normally distributed across all regions (Shapiro-Wilk's test: p's ≥ .62), but theta power was not (Shapiro-Wilk's test: p's ≤ .005). Both alpha and theta power met the assumptions of variance homogeneity (Levene's test: p's ≥ .32). To maintain consistency across all narrowband power-wise comparisons, non-parametric tests were conducted across all pairwise comparisons, even though parameterised and individualised alpha power met the assumptions of a parametric test. Mann-Whitney U tests revealed no significant differences between young and older adults across globalised and

all regions of parameterised and individualised alpha and theta power (p's ≥ .06). Refer to Table 4.3.3b & Figure 4.3.3b for the summary of test statistics for parameterised and individualised alpha power; and Table 4.3.3c & Figure 4.3.3c for summary of test statistics for parameterised and individualised theta power.

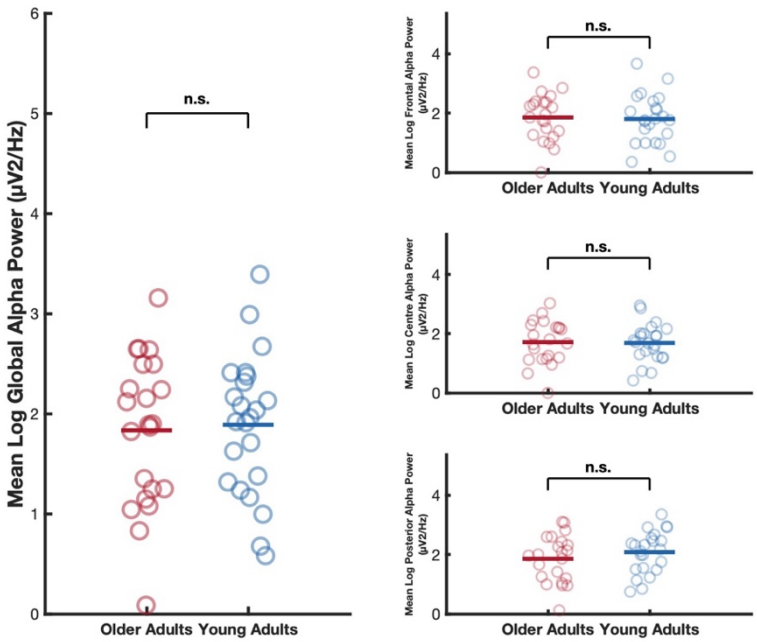
Table 4.3.3b

Summary Of Statistics Comparing Parameterised & Individualised Alpha Power Across Age Groups & Regions

	Mean Parameterised & Individualised Alpha Power log(μV² / Hz) / (*SD*)					
	Older Adults	**Young Adults**	**U**	**z**	***p***	**Effect Size (Cohen's *d*)**
Global Alpha	1.83 (0.75)	1.89 (0.70)	499	-.15	.88	-.19
Frontal	1.85 (*0.79*)	1.80 (*0.81*)	524	.40	.69	.06
Centre	1.72 (*0.73*)	1.69 (*0.64*)	515	.19	.84	.03
Posterior	1.86 (*0.79*)	2.08 (*0.70*)	464	-.94	.34	-.14

Figure 4.3.3b

Comparisons of Parameterised & Individualised Alpha Power Between Young & Older Adults



Note. Horizontal lines going through points indicate mean values.

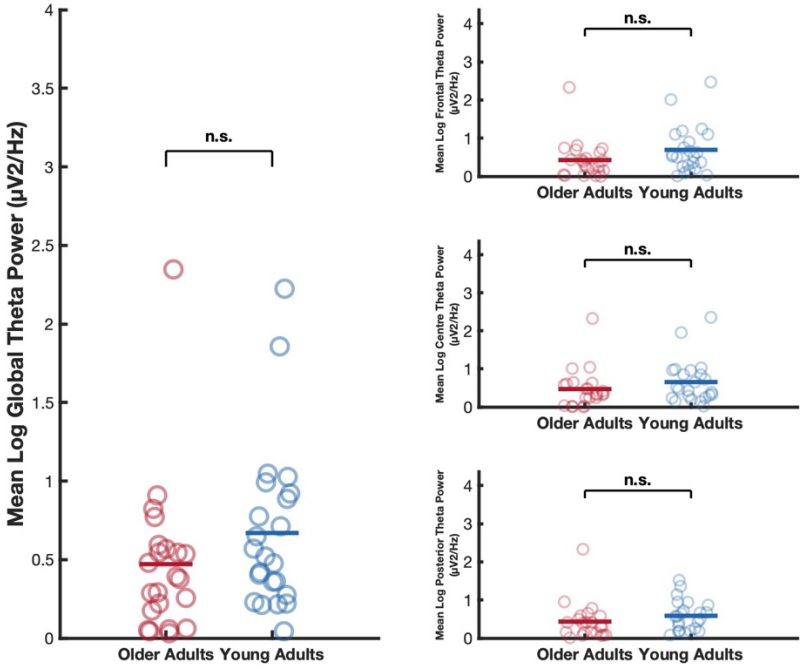
Table 4.3.3c

Summary Of Statistics Comparing Parameterised & Individualised Theta Power Across Age Groups & Regions

	Mean Parameterised & Individualised Theta Power log(μV² / Hz) / (*SD*)					
	Older Adults	**Young Adults**	**U**	**z**	***p***	**Effect Size (Cohen's *d*)**
Global Theta	0.47 (0.49)	0.67 (0.53)	437	-1.56	.12	-.23
Frontal	0.43 (*0.50*)	0.69 (*0.61*)	427	-1.78	.07	-.27
Centre	0.47 (*0.51*)	0.65 (*0.57*)	454	-1.17	.24	-.17
Posterior	0.43 (*0.49*)	0.59 (*0.40*)	421	-1.92	.06	-.29

Figure 4.3.3c

Comparisons of Parameterised & Individualised Theta Power Between Young & Older Adults]



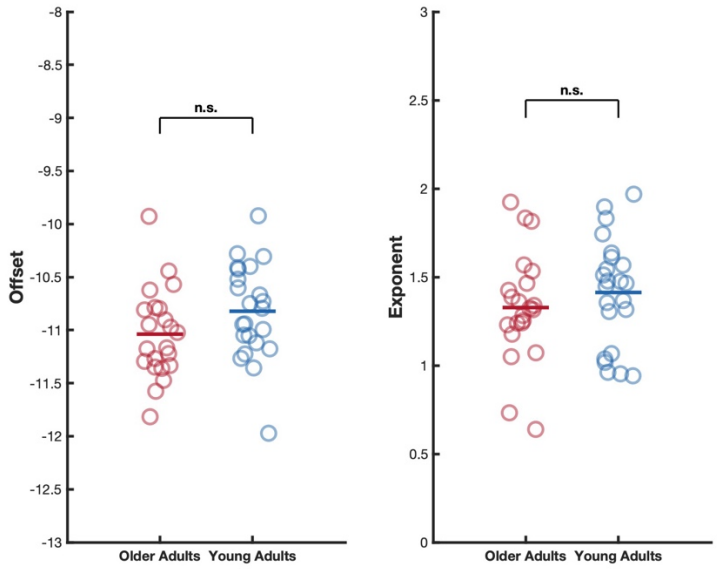
Note. Horizontal lines going through points indicate mean values.

4.3.4. Aperiodic Components

The aperiodic components across young and older adults were normally distributed and met the homogeneity assumption for variances (Shapiro-Wilk's test: young *p*'s ≥ .25, older *p*'s ≥ .20; Levene's test: *p*'s ≥ .80). The independent samples t-tests indicated that both offset (young: *M* = -10.82, *SD* = .47; older: *M* = -11.04, *SD* = .42) and exponent values (young: *M* = 1.41, *SD* = .31; older: *M* = 1.33; *SD* = .31) were not significantly different across young and older adults (*t*'s(43) ≥ -.93, *p*'s ≥ .1, Cohen's *d*'s = ≥ - .28) (Refer to Figure 4.3.4).

Figure 4.3.4

Comparisons of Offset & Exponent Values Between Young & Older Adults



Note. Horizontal lines going through points indicate mean values.

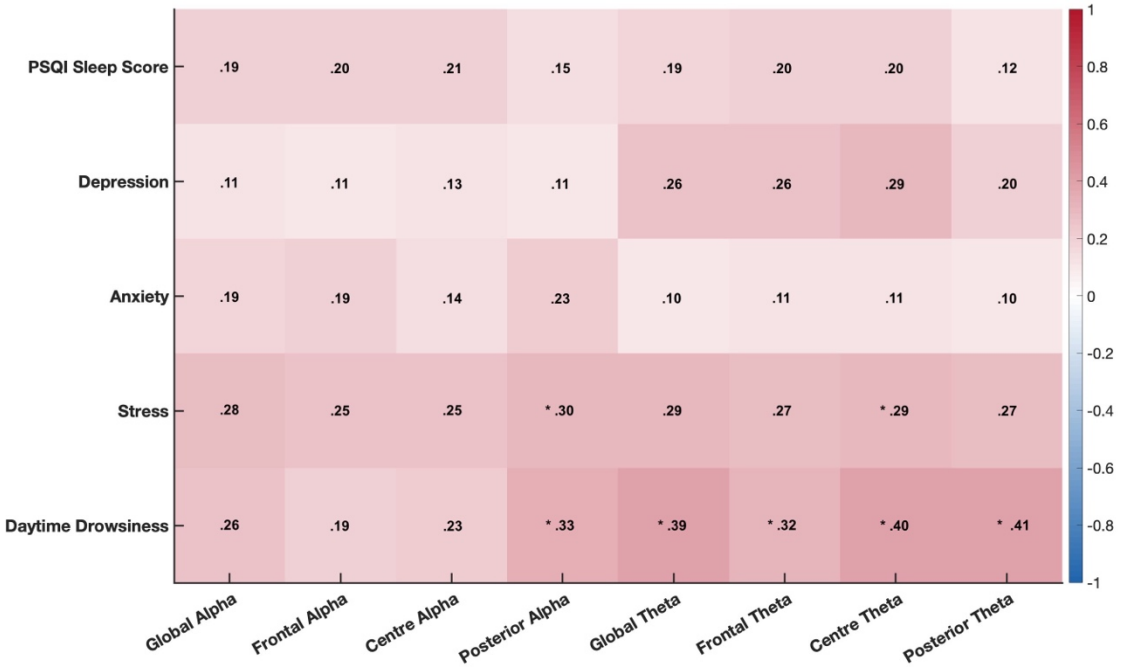
4.3.5. Correlations

To examine possible relationships between narrowband power measures with sleep quality, negative emotional states and daytime drowsiness, Spearman's correlations were conducted across all canonical measures and all parameterised and individualised measures. These are represented across two correlational matrices below (refer to Figure 4.3.5a for canonical

power; refer to Figure 4.3.5b for parameterised and individualised power). Correlations were also conducted across IAPF and aperiodic components along those dimensions (refer to Figure 4.3.5c).

Figure 4.3.5a

Correlational Matrix of Canonical Power Measures on Sleep Quality, Depression, Anxiety, Stress & Daytime Drowsiness

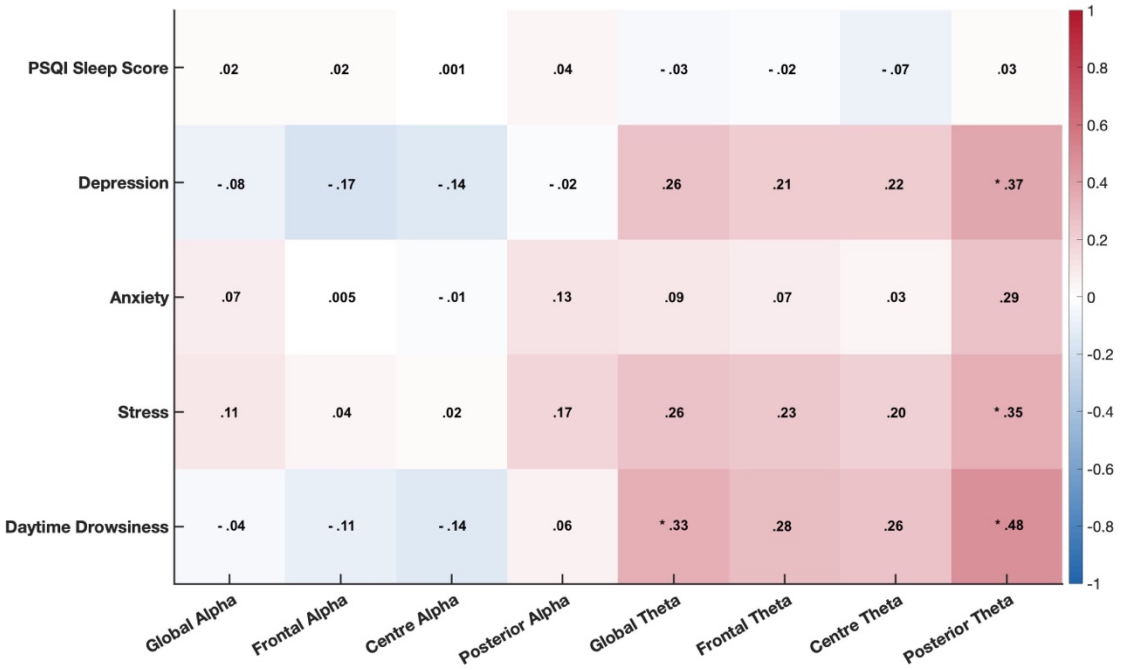


Note. Values within cells indicate Spearman's rho. Asterisk (*) indicates significance. * = $p < .05$

Stress correlated positively with canonical posterior alpha (Spearman's rho = .30, p = .049) and centre theta power (Spearman's rho = .29, p = .049), while daytime drowsiness correlated positively with posterior alpha power (Spearman's rho = .33, p = .03) and canonical theta power across all regions (Spearman's rho's ≥ .32, p's ≤ .02). All other correlations were not significant (p's ≥ .05).

Figure 4.3.5b

Correlational Matrix of Parameterised & Individualised Power Measures on Sleep Quality, Depression, Anxiety, Stress & Daytime Drowsiness

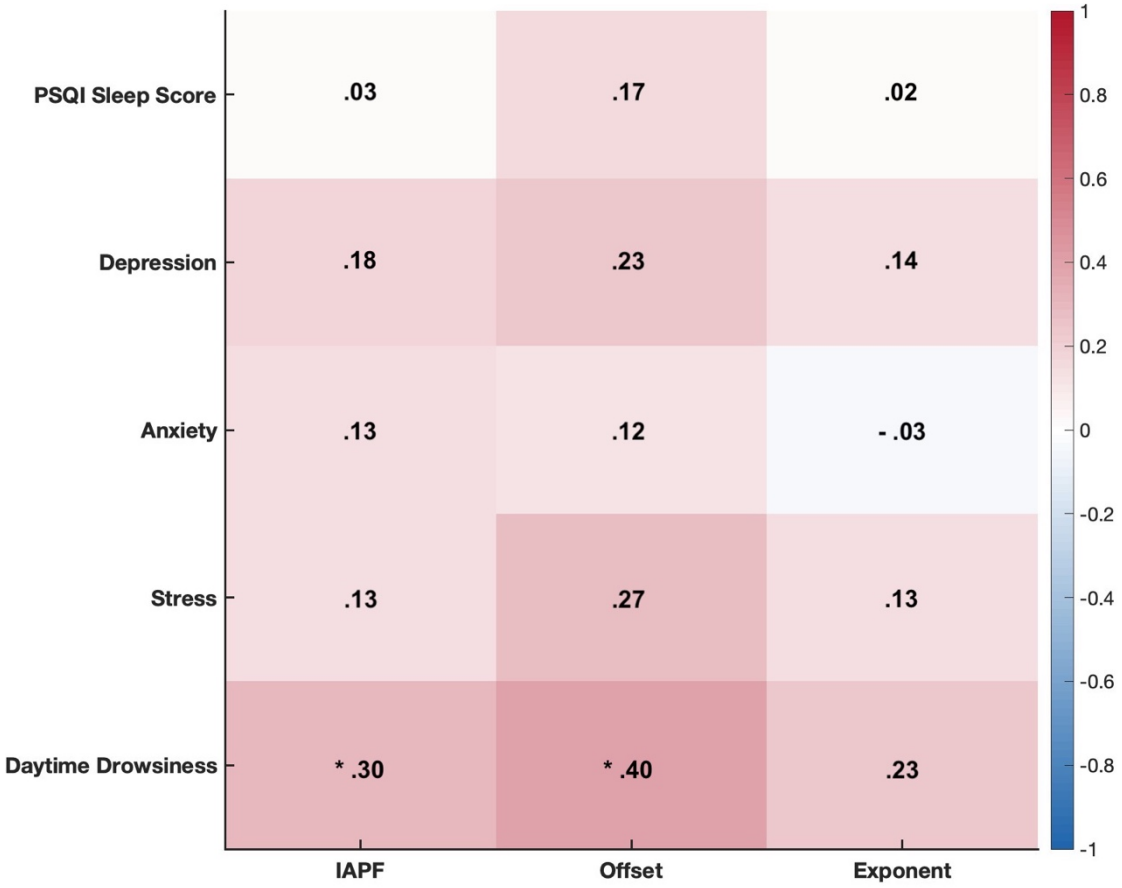


Note. Values within cells indicate Spearman's rho. Asterisk (*) indicates significance. * = $p < .05$

Parameterised and individualised alpha power did not correlate significantly across sleep quality, negative emotional states, or daytime drowsiness (Spearman's rho's ≥ - .14 to .11, p's ≥ .26). However, parameterised and individualised posterior theta power correlated positively with depression, stress, and daytime drowsiness (Spearman's rho's ≥ .35, p's ≤ .02). In addition to that, daytime drowsiness was also positively correlated with global theta (Spearman's rho = .33, p = .03). Besides that, all other correlations of theta power were not significant (p's ≥ .06).

Figure 4.3.5c

Correlational Matrix of IAPF & Aperiodic Components on Sleep Quality, Depression, Anxiety, Stress & Daytime Drowsiness



Note. Values within cells indicate Spearman's rho. Asterisk (*) indicates significance. * = $p < .05$

Daytime drowsiness correlated positively with individual alpha peak frequency (IAPF) (Spearman's rho = .30, $p = .04$) and offset values of the aperiodic component (Spearman's rho = .40, $p = .007$). No other correlations were significant (p's ≥ .07)

4.4. Discussion

The present chapter placed an emphasis on investigating the foundational bases of resting-state EEG studies. This approach served two main purposes: firstly, to establish a retrospective framework to examine existing literature relevant to the variables of interest in this thesis. Secondly, in contrast to the conventional approaches to narrowband measures, this chapter introduced the importance of considering age-related changes that may influence relationships between certain variables. By considering these factors, a more nuanced explanation can be offered alongside a more standardised approach to consolidate findings relevant to the present thesis.

Starting with the conventional approach, the present study found significantly lower posterior alpha and theta power in older adults compared to young adults. While this finding did not extend to the global power or centre and frontal theta, the appearance of a power difference is consistent with some previous literature, where age-related differences in narrowband power were reported (Rossini et al., 2007; Vlahou et al., 2014). However, upon utilising an individualised approach to account for age-related shifts in the individual alpha peak frequency (IAPF) and signal parameterisation, these power differences were no longer significant. This suggests that age-related differences in power observed through the conventional approaches might be attributed to the shift in the IAPF and aperiodic components, consistent with the propositions by Donoghue et al. (2020) and Cesnaite et al. (2023), who observed similar findings. In this sample of participants, only the IAPF showed significant differences between young and older adults, while the aperiodic components did not. The slowing of the IAPF is consistent with previous findings (Dustman et al., 1993, Scally et al., 2018), but this sample of older adults did not exhibit a flatter 1/f slope, which is inconsistent with previous suggestions that the 1/f slope tends to flatten in older age (e.g. Dave et al., 2018; Voytek et al., 2015). This finding could suggest that the slowing of the

IAPF and the flattening of the 1/f slope in older age may be driven by separate processes in the brain. Perhaps the slowing of the IAPF may be a common feature of ageing, while the increase in neural noise (i.e., flatter 1/f slope) may be indicative of pathology, rather than due to ageing.

Evaluating the present findings alongside concurrent evidence provides a limited scope of comparisons, as only a few studies have incorporated individualisation and parameterisation approaches. Comparisons with findings lacking these approaches would be challenging, as it would be difficult to disentangle the effects which may be attributable to unaccounted factors (e.g. shifts in IAF or contributions of aperiodic components). However, in relation to the few findings that have utilised these approaches, Pani et al. (2022) conducted a mini-review of 11 studies comparing nine clinical conditions that analysed aperiodic components as a useful tool for differential diagnoses. Of these reviews, only one is of tangential relevance to the present context, where Pani et al. (2022) found steeper 1/f slopes in patients with parasomnias, contrasted to those with sleep-related hypermotor epilepsy. However, that is the extent of coverage regarding sleep-related measures, which is also not immediately relevant to the present thesis.

In addition to that, Aggarwal & Ray (2023) replicated the findings of flatter 1/f slopes in older adults, though they found no significant differences between mild cognitive impaired patients after matching with age and gender-matched healthy older adults. This limited scope of comparison is further hindered by the lack of a standardised approach, even within the choice of extracting aperiodic components (Pani et al., 2022). Supplementing this point, (Kozma et al., 2023) also suggest that interpreting EEG signal measures must incorporate both periodic and aperiodic signals, as interpreting either component in isolation may be insufficient to draw significant conclusions. For instance, the FOOOF algorithm computes the periodic and aperiodic components in log-space, bounded by logarithm rules (e.g. log(a)

+ log(b) = log (ab) OR log(periodic) + log(aperiodic) = log(periodic x aperiodic)). As such, discretely parameterising the individual components may not provide the same interpretation of the data compared to if both components were not separated.

The solution to these computational approaches is beyond the scope of the present thesis, but it provides additional complexity that should be considered in future studies. Taken together, the change in findings after accounting for individual differences and parameterisation of EEG signals in previous studies (e.g. Cesnaite et al., 2023) as well as the present study highlight an important need to separate the contribution of different properties of cortical activity, as oversight of these parameters may lead to a misrepresentation of findings.

The implication of individualised and parameterised narrowband approaches extends to measures examined in the previous chapter as well. For example, the VIGALL acknowledges potential misclassifications when handling data with variant IAPF measures (i.e. IAPF < 8.5 Hz). Thus, if such tools are to be robustly applied across a wide population, especially in older adults, these considerations must be addressed, such as how to handle slowed IAFs. Furthermore, incorporating the parameterisation approach into the VIGALL may enhance its utility. As noted by Lendner et al. (2020) and Monchy et al. (2023), aperiodic parameters are modulated by fluctuating arousal states. Particularly during the transition from wakefulness to the onset of sleep to deeper sleep stages, a flattening of the 1/f slope occurs relative to wakefulness. Perhaps the inclusion of an additional 1/f slope parameter could improve the VIGALL classification, potentially bridging gaps where it currently struggles with variant alpha rhythms.

Concerning the variables of interest to this study, the correlations showed a significant shift in effects following individualisation and parameterisation. Notably, after individualisation and parameterisation of data, non-significant correlations emerged between

all alpha measures, despite initial positive correlations between canonical posterior alpha with daytime drowsiness and stress. In contrast, posterior theta exhibited correlations with more measures after individualisation and parameterisation, including depression, stress, and daytime drowsiness. As covered earlier, direct interpretations of these findings are challenging, as only a few recent studies have attempted similar approaches. However, it is noteworthy that theta power tends to increase during periods of heightened drowsiness and fatigue (e.g. Foong et al., 2015; Krishnan et al., 2020). While evidence regarding theta power's relationship with stress as a negative emotional state is scarce, increased theta power has been associated with higher mental stress, and theta power tends to increase in response to stressors (e.g. Awang et al., 2011; Okonogi & Sasaki, 2021). Those suggestions are consistent with the current findings. However, previous findings concerning the relationship between theta power and depression symptoms appear inconsistent, with studies reporting positive and negative correlations with depression (e.g. Fingelkurts et al., 2007; Grin-Yatsenko et al., 2009; Tas et al., 2015).

Regarding aperiodic components, daytime drowsiness showed positive correlations with exponent values and the IAPF. The correlation between larger exponent values with higher daytime drowsiness is consistent with previous findings (e.g. Cross et al., 2022; Zhang et al., 2021). However, the positive correlation between IAPF and daytime drowsiness is unexpected. There is no direct evidence linking IAPF with daytime drowsiness, although lowered IAPF is typically associated with pathological states. For instance, increased fatigue is linked to a decrease in IAPF, and lowered IAPF is observed in various psychiatric conditions (e.g. dementias and cognitive decline), where instances of daytime drowsiness are typically higher (Leibowitz et al., 2006). As such, it would have been expected for IAPF to exhibit a negative correlation instead of a positive one. Providing an explanation is challenging due to the lack of sufficient evidence or literature that can offer insight into this

finding. Lastly, of noteworthy importance, none of the measures showed a significant correlation with sleep quality.

In summary, this chapter stressed the importance of adopting careful and focused approaches when investigating the effects of ageing on the brain. This thesis characterised such an approach by integrating the individualisation of frequency band definition and the parameterisation of cortical components as measured through electrophysiological methods, such as EEG. The decision to dedicate this chapter to these methodological considerations was driven by the need to establish a reproducible approach that adheres to good practices that can provide a contextual framework to examine retrospective evidence, which has previously yielded inconsistent results. More importantly, examining these variables through these multiple facets can be used to support the primary objective of this thesis — to explore the key factors affecting sleep quality in older age. In these chapters, general associations were examined among the variables of interest, including arousal regulation parameters and other EEG narrowband-derived parameters, and their correlations with variables such as sleep quality, negative emotional states, and daytime drowsiness. Alongside that, age-group comparisons were also conducted across these variables. However, a dedicated exploration of the relationships between these variables at the age-group level has not yet been covered. As such, in the next final chapter, all these variables will be synthesised into age-group specific models to investigate their cumulative effects on sleep quality.

Chapter 5: Exploring Age-Group-Wise Predictors Of Sleep Quality

5.1. Introduction

In Chapter 2, the relationships between sleep quality, negative emotional states, daytime drowsiness, and resting-state cognition were investigated. Following that, Chapter 3 examined the arousal regulation model proposed by Hegerl and Hensch (2014) to explore potential connections between age-related changes to the areas of the brain involved in arousal and sleep-wake regulation, and whether such impacts could be observed via the VIGALL. Chapter 4 then expanded on the premise that age-related analyses should consider age-related sensitivities. It involved a critique of the conventional approach to EEG narrowband frequency analysis and contrasted it with an individualised and parameterised approach. Addressing such concerns enabled a retrospective review of existing literature relevant to the thesis, which provided context to previously inconsistent findings, and to provide a guiding point for future examinations of the same kind. While those previous chapters involved cursory investigations of these variables, a central question remains: how significant are these variables in predicting sleep quality, and more importantly, do they exert differential effects on older adults compared to young adults?

In this final chapter, all previously examined variables will be examined in relation to sleep quality to identify the potential factors that may be crucial in understanding sleep quality differences between young and older adults. Separate regression models were conducted for young and older adults to assess the extent to which these variables account for variations in sleep quality. This may help delineate the impact of the observed factors on sleep quality and whether they may produce different effects across age groups. Additionally, providing such a comprehensive model may facilitate future explorations by highlighting the important factors that may be causal in the development of poor sleep quality.

Across the previously examined features, only a few variables, namely depression, anxiety, stress, and daytime drowsiness, correlated significantly with sleep quality. Conversely, none of the resting-state cognition measures, VIGALL-derived arousal regulation parameters, or narrowband EEG spectral features showed significant correlations with sleep quality. As such, it is expected that these non-correlated variables may not be meaningful predictors of sleep quality, a hypothesis to be tested in the subsequent regression models.

In the next section, methods relating to the elastic net regressive model will be covered. This will be followed by an overview of the findings and an identification of meaningful, or 'significant' features that may explain sleep quality differences in young and older adults. Finally, this chapter will conclude the thesis with an overall discussion of the examined factors, including the implications and limitations of the present study.

5.2. Elastic Net Regression Methods

In the selection of predictor variables for the following regression models, conventional narrowband parameters were excluded. Instead, only individualised, and parameterised narrowband parameters were included. In addition to that, only global measures, excluding region-wise power (e.g., frontal alpha) were included to improve the parsimony of the models. Furthermore, the initial vigilance level was also excluded, as it was implicitly used in the calculation of parameters such as the slope index and would not be meaningful on its own. As such, a remaining total of 22 variables were retained as predictor variables with sleep quality as the outcome variable.

The present study encounters the challenge of "large-p, small-n", where the number of observations (n) is not significantly larger than the number of predictor variables (p). With separate regression models planned for each age group, 22 variables would be regressed against sleep quality with a small sample of 23 young adults and 22 older adults.

Additionally, due to the nature of variables, certain variables share high degrees of collinearity. For instance, the computation of arousal regulation parameters involved ratios of theta and alpha power, two variables which were treated as separate predictors in this model. Furthermore, a conceptual understanding of arousal regulation parameters may suggest non-linear relationships against sleep quality. For instance, both high and low arousal stability scores may be linked to non-stable arousal regulation patterns, which also impact sleep quality (refer to Chapter 3 for explanations of unstable and hyperstable arousal patterns).

Given these issues, the use of linear regression methods may be inappropriate for the current dataset. Additionally, the objective of this chapter is to identify 'significant' variables that may potentially contribute to poor sleep quality, which operate under the assumption that several variables may not hold predictive value and can be excluded. As such, the use of penalised regression methods may be more suitable, namely elastic net regression, even though it may not solve the requirements of all the variables (i.e., the non-linearity of arousal regulation parameters). Addressing the nature of such variables would require more tailored models, which will be elaborated on in the discussion section. Nonetheless, these variables were still included in the current model.

The elastic net regression combines two regularisation techniques, named lasso (L1) and ridge (L2) regression, which are used in regression analyses to address the problem of multicollinearity and overfitting (i.e., the model is "overfit" to the data, capturing noise in the data and failing to generalise to other datasets, which may occur in models trained on small-n, large-p datasets). In lasso (least absolute shrinkage and selection operator) regression, a standard linear regression model is fitted with a tuning parameter or penalty that minimises the residual sum of squares (i.e. creating a "better" fit of the model to the data) by multiplying the absolute values of coefficients by the penalty term (λ). Effectively, this process reduces coefficients of less important variables to zero, creating a subset of relevant

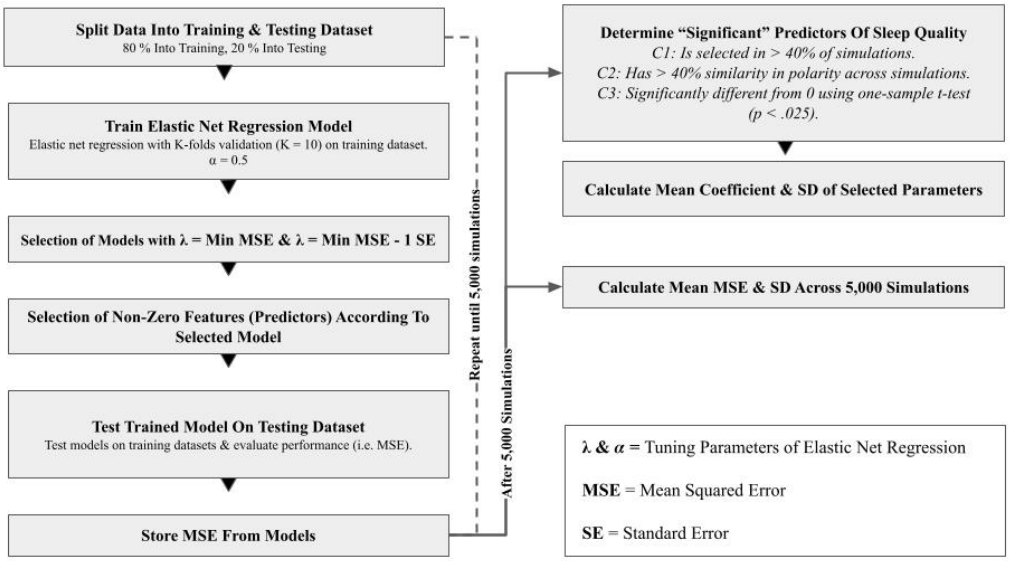
variables and discarding non-important ones from the model. Lasso regression is particularly useful for dimensionality reduction and feature selection. Similarly, ridge regression also utilises a tuning parameter that minimises the residual sum of squares by multiplying the sum of squared values of the coefficients with the penalty term (λ), rather than the absolute values. Functionally, this also minimises the coefficients of variables but does not fully eliminate them from the model, enabling the model to retain all variables to some extent. While the lasso method may simplify the model, it may produce overly sparse models with too few variables. In contrast to that, ridge regression enables the examination of all variables, but may not simplify the model (refer to Thevaraja et al. (2019) for more detailed explanations of the lasso and ridge regression).

The elastic net regression (Zou & Hastie, 2003) combines both methods by imposing two tuning parameters: λ and α. The λ dictates the penalty that minimises the sum of squared residuals, while α which determines the relative weightage of ridge to lasso (α = 0 corresponds to fully ridge, α = 1 corresponds to fully lasso). By controlling the relative weightage of lasso, the elastic net combines the advantages of both ridge and lasso methods, allowing the coefficients of non-relevant variables to be set to zero while avoiding overly sparse models by imposing the ridge to retain certain features. This approach effectively addresses both feature selection and multicollinearity concerns, making it suitable for the present study, particularly in addressing the “large-p, small-n” problem. The subsequent section elaborates on how the elastic net regression was employed in this study.

To explore potential age-related differences in variables affecting sleep quality and to assess the overall performance of the models, separate models were trained and tested for young and older adults. All data processing and analyses were performed within MATLAB (ver. R2022b), refer to Figure 5.2a for the pipeline of operations.

Figure 5.2a

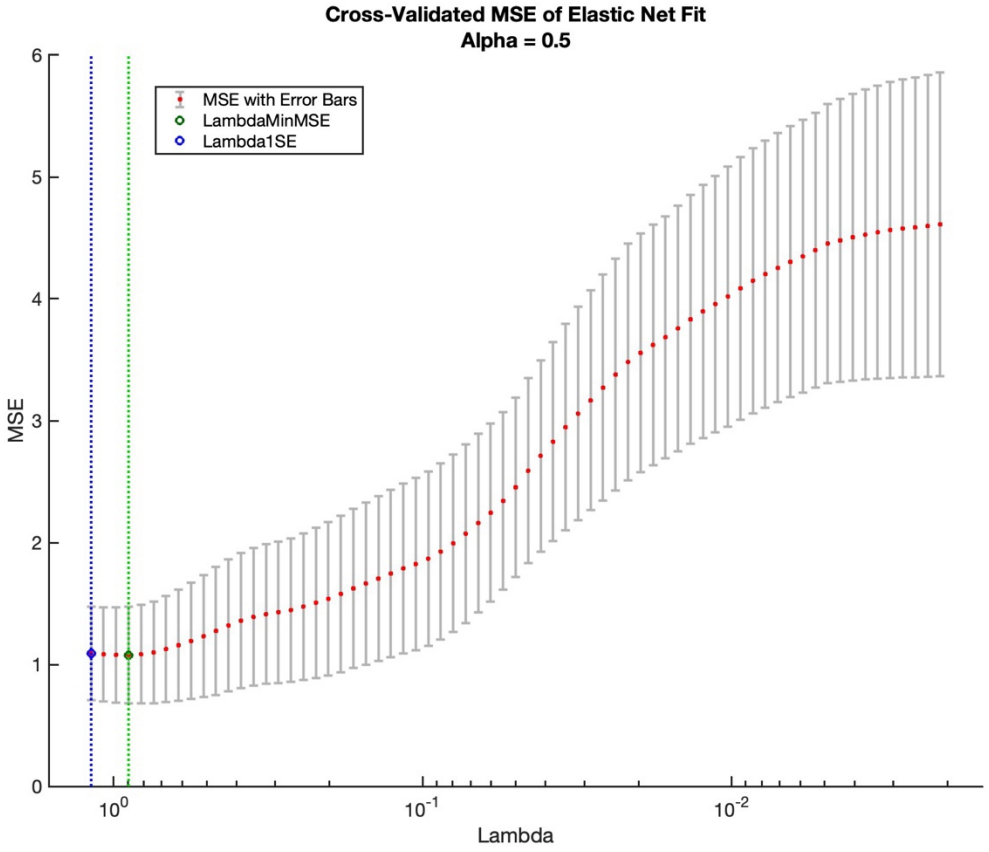
Elastic-Net Regression Pipeline to Determine Predictors of Sleep Quality



The initial dataset was divided into training and testing sets following an 80-20 split (80% training, 20% testing) Following that, the training data went through an elastic net regression with the α parameter set at 0.5, indicating an equal split of lasso and ridge methods. During model training, various λ (penalty) values were tested to determine the optimal fit model. This determination was based on evaluating the lowest mean squared errors (MSE), calculated by dividing the sum of squared (SSR) residuals by the number of observations. The MSE provides a measure of average prediction error, while the SSR represents the total prediction error. Across several lambda values, the value that produces the lowest MSE is taken as the optimal penalty value to produce the "best fit" model. This process was done via a 10-fold cross-validation process, which is a process that partitions the data into further subsets to enhance the estimates of the optimal λ value. For an illustrated example of cross-validation, refer to Figure 5.2b.

Figure 5.2b

Example of 10-Fold Cross-Validation Plot To Determine Lambda Values For MINMSE & MSE1SE Models



Note. Different lambda values are listed across the x-axis, with the MSE on the y-axis indicating the averaged error of the model. The green dotted line indicates the lambda value at which the MSE (MINMSE) is lowest; the Blue dotted line indicates the lambda value of minimum MSE minus 1 standard error (MSE1SE).

The models utilising the λ values corresponding to the lowest MSE (MINMSE) and the lowest MSE minus one standard error (MSE1SE) were used to estimate the number of features with a non-zero coefficient (i.e., 'significant' predictors on the outcome variable). While a MINMSE model tends to produce the model with "best fit", the MSE1SE model is often used instead, as it may prevent overfitting and produce the most parsimonious model

(Krystajic et al., 2014). To examine how the selection of predictors may change across models, this study examined both models. Subsequently, the trained models were run on the testing dataset to assess the model's fit to the data, with smaller MSE values indicating a better fit of the model.

Due to the inherent randomisation in the training process (i.e., random split of data into training and testing datasets), single iterations of the models may yield different results each time. To mitigate the potential arbitrariness of such findings, the entire process was reiterated 5,000 times for each model. This selection for the number of iterations was based on Han & Dawson (2021) for a balance of computational time and resource constraints. After 5,000 iterations, a selection of important, or 'significant' variables impacting sleep quality was determined. Note the use of 'significant' in this context. Penalised regression techniques, such as elastic net do not produce 'significant' values (i.e. p-values) in the traditional sense of hypothesis testing. For further details, refer to Goeman et al. (2012) and Lee et al. (2016).

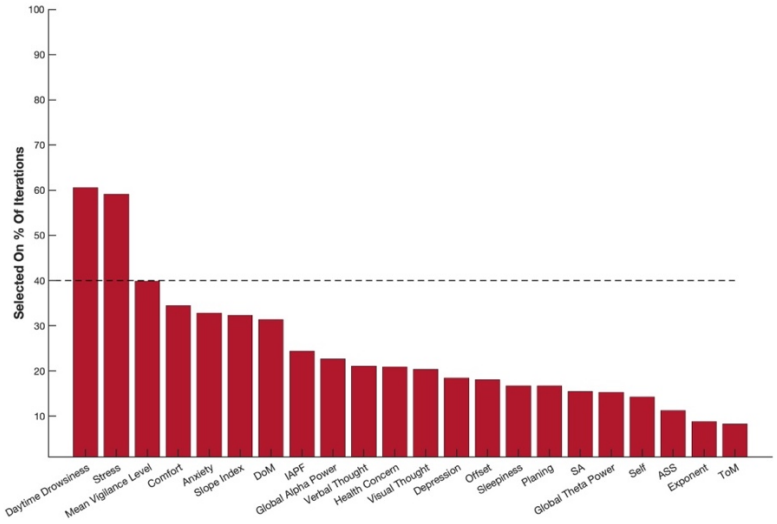
To ensure consistency in identifying significant predictor variables and to avoid arbitrariness, the present study adopted the hyperparameters of Jenul et al. (2021) combined with the thresholds used by Shah (2022) to define what a 'significant' variable means. Operationally, a variable was only selected as a significant predictor of sleep quality if it met the following three criteria: (i) the variable was selected as a non-zero variable in at least 40% of iterations; (ii) the variable showed coefficients of similar polarity in at least 40% of iterations; and (iii) the coefficients of the variables are significantly different from 0, as determined by a one-sample t-test with an α criterion of 0.025. Additionally, the overall fit of the models was also assessed using the mean and standard deviations of the models across 5,000 iterations.

5.3. Results of Elastic Net Regression

5.3.1 Older Adult Models

Figure 5.3.1a

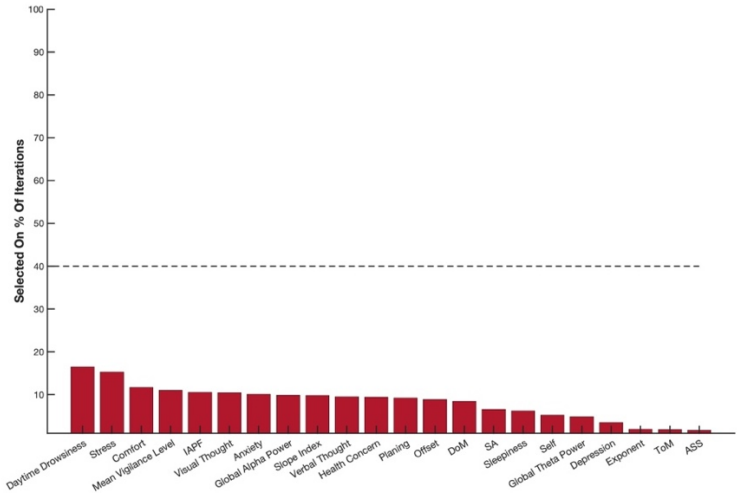
Variable Selection Frequencies Across 5,000 Iterations of MINMSE Model for Older Adults



Note. The dotted line indicates frequency threshold of 40%. Variables are organised in descending order of frequency.

Figure 5.3.1b

Variable Selection Frequencies Across 5,000 Iterations of MSE1SE Model for Older Adults



Note. The dotted line indicates frequency threshold of 40%. Variables are organised in descending order of frequency.

Table 5.3.1

Comparing Model Fits, Variable Selection Frequencies & Coefficients of MINMSE & MSE1SE Models for Older Adults

	MSE (MINMSE)		MSE - 1SE (MSE1SE)	
Mean Model MSE (SD)	1.70 (*1.30*)		1.48 (*0.94*)	
	Selected on % of Iterations	**Mean Coefficient (SD)**	**Selected on % of Iterations**	**Mean Coefficient (SD)**
Depression	18.42	0.20 (*0.27*)	3.46	0.27 (*0.31*)
Anxiety	32.80	0.12 (*0.16*)	10.12	0.17 (*0.16*)
Stress	* 59.14	0.19 (*0.12*)	15.30	0.17 (*0.11*)
Daytime Drowsiness	* 60.58	0.33 (*0.28*)	16.46	0.45 (*0.28*)
Arousal Stability Score (ASS)	11.24	0.20 (*0.26*)	1.66	0.20 (*0.23*)
Mean Vigilance Level	39.84	-0.13 (*0.13*)	11.00	-0.15 (*0.12*)
Slope Index	32.34	-0.13 (*0.11*)	9.80	-0.13 (*0.08*)
Global Alpha	22.66	0.33 (*0.19*)	9.86	0.31 (*0.15*)
Global Theta	15.26	-0.02 (*0.22*)	4.86	-0.02 (*0.15*)
Individual Alpha Peak Frequency (IAPF)	24.38	-0.21 (*0.16*)	10.50	-0.22 (*0.14*)
Offset	18.10	0.12 (*0.18*)	8.90	0.14 (*0.12*)
Exponent	8.82	-0.18 (*0.22*)	1.88	-0.17 (*0.17*)
Discontinuity of Mind (DoM)	31.34	0.09 (*0.09*)	8.44	0.10 (*0.07*)
Theory of Mind (ToM)	8.30	0.01 (*0.09*)	1.82	0.01 (*0.07*)
Self	14.26	0.18 (*0.15*)	5.22	0.15 (*0.11*)
Planning	16.70	-0.43 (*0.24*)	9.26	-0.39 (*0.17*)
Sleepiness	16.74	-0.16 (*0.2*)	6.18	-0.12 (*0.13*)
Comfort	34.46	-0.23 (*0.16*)	11.66	-0.31 (*0.13*)
Health Concern	20.90	0.14 (*0.13*)	9.48	0.16 (*0.11*)
Somatic Awareness	15.46	-0.16 (*0.15*)	6.56	-0.13 (*0.12*)
Visual Thought	20.36	0.29 (*0.19*)	10.48	0.27 (*0.13*)
Verbal Thought	21.06	-0.18 (*0.19*)	9.52	-0.20 (*0.13*)

Note. * Indicates that the variable had passed all three selection criteria for a 'significant' predictor of sleep quality.

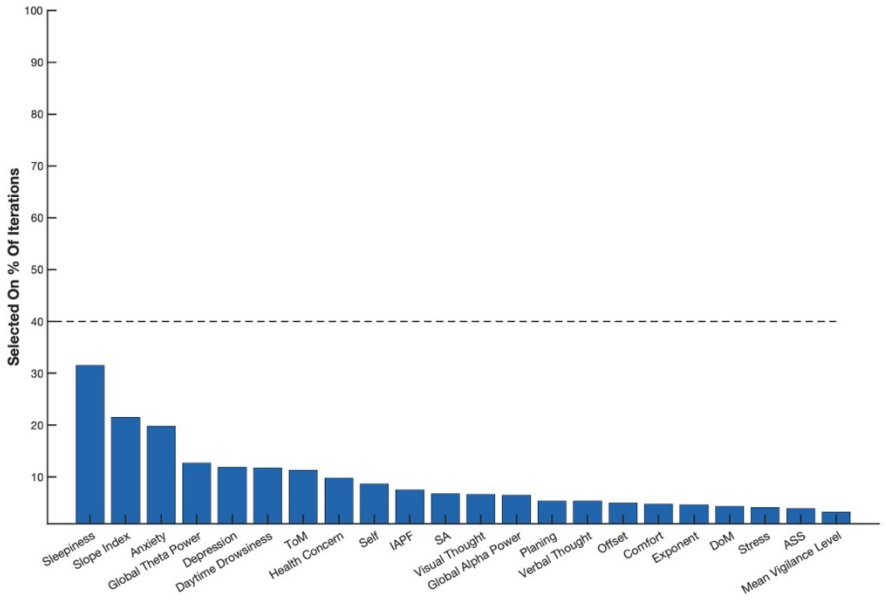
In the elastic net regression models conducted for the older adult data, daytime drowsiness and stress were the only two variables that met the 'significant' criteria in the MINMSE model, indicating that higher feelings of daytime drowsiness and stress predict poorer sleep quality (refer to Figure 5.3a). Daytime drowsiness was selected in 60.58% of the iterations,

with 100% of coefficients showing a positive polarity, and was significantly different from 0 ($t(4999) = 60.46$, $p < .001$, Cohen's $d = .86$). Stress was selected in 59.14% of the iterations, with 97.23% of coefficients showing a positive polarity, and was also significantly different from 0 ($t(4999)) = 52.09$, $p < .001$, Cohen's $d = .74$). None of the remaining variables in either the MINMSE or MSE1SE models met the 'significant criteria', suggesting that they may not be predictive of sleep quality according to the best-fit models (refer to Figure 5.3b & Table 5.3a).

5.3.2. Young Adult Models

Figure 5.3.2a

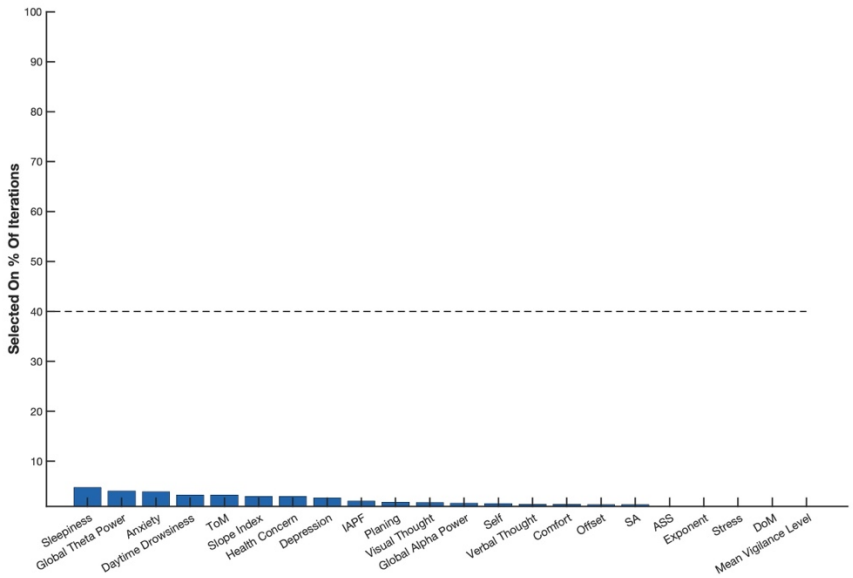
Variable Selection Frequencies Across 5,000 Iterations of MINMSE Model for Young Adults



Note. The dotted line indicates frequency threshold of 40%. Variables are organised in descending order of frequency.

Figure 5.3.3b

Variable Selection Frequencies Across 5,000 Iterations of MSE1SE Model for Young Adults



Note. The dotted line indicates frequency threshold of 40%. Variables are organised in descending order of frequency.

Table 5.3.2

Comparing Model Fits, Variable Selection Frequencies & Coefficients of MINMSE & MSE1SE Models for Young Adults

	MSE (MINMSE)		MSE - 1SE (MSE1SE)	
Mean Model MSE (SD)	0.80 (*0.56*)		0.68 (*0.42*)	
	Selected on % of Iterations	**Mean Coefficient (SD)**	**Selected on % of Iterations**	**Mean Coefficient (SD)**
Depression	11.88	0.12 (*0.11*)	2.72	0.14 (*0.12*)
Anxiety	19.80	0.17 (*0.16*)	3.90	0.18 (*0.15*)
Stress	4.14	0.07 (*0.13*)	0.86	0.06 (*0.14*)
Daytime Drowsiness	11.78	-0.19 (*0.12*)	3.30	-0.15 (*0.10*)
Arousal Stability Score (ASS)	3.94	-0.06 (*0.15*)	1.04	-0.07 (*0.11*)
Mean Vigilance Level	3.26	-0.04 (*0.14*)	0.48	-0.05 (*0.08*)
Slope Index	21.52	0.13 (*0.12*)	3.00	0.14 (*0.13*)
Global Alpha	6.48	-0.12 (*0.14*)	1.60	-0.12 (*0.12*)
Global Theta	12.66	-0.26 (*0.19*)	4.04	-0.23 (*0.17*)
Individual Alpha Peak Frequency (IAPF)	7.48	0.05 (*0.17*)	2.06	0.04 (*0.15*)
Offset	5.00	0.18 (*0.16*)	1.32	0.16 (*0.11*)
Exponent	4.66	0.09 (*0.16*)	1.02	0.1 (*0.13*)
Discontinuity of Mind (DoM)	4.32	0.06 (*0.18*)	0.72	0.01 (*0.16*)
Theory of Mind (ToM)	11.30	0.23 (*0.18*)	3.28	0.22 (*0.17*)
Self	8.64	-0.17 (*0.21*)	1.52	-0.13 (*0.17*)
Planning	5.34	-0.09 (*0.13*)	1.86	-0.11 (*0.10*)
Sleepiness	31.54	-0.16 (*0.13*)	4.74	-0.19 (*0.13*)
Comfort	4.74	-0.03 (*0.14*)	1.36	-0.04 (*0.13*)
Health Concern	9.84	-0.17 (*0.12*)	2.96	-0.14 (*0.09*)
Somatic Awareness	6.78	0.14 (*0.12*)	1.32	0.12 (*0.10*)
Visual Thought	6.66	-0.19 (*0.2*)	1.74	-0.2 (*0.17*)
Verbal Thought	5.32	0.04 (*0.13*)	1.40	-0.01 (*0.08*)

In the elastic net regression models conducted for the young adult data, none of the variables in either the MINMSE or MSE1SE model passed the ‘significant’ criteria, indicating that none of the measured variables were significant for predicting sleep quality, as indicated by the best-fit models (refer to Figure 5.3c, Figure 5.3d & Table 5.3b).

5.4. Overall Discussion

5.4.1. Model Discussion

Across the four elastic net regression models conducted for young and older adults, the MINMSE and MSE1SE models revealed that most of the tested variables in this thesis were not strong predictors of poor sleep quality. Specifically, only two variables in the older adult MINMSE model met the 'significant' criteria: daytime drowsiness and stress. These findings suggest that older adults who report higher levels of daytime drowsiness and stress may experience poorer sleep quality. However, these variables did not survive in the MSE1SE model, suggesting that even these variables may not be very significant predictors of sleep quality.

While a broad interpretation of these findings suggests that poor sleep quality may not be predicted by any specific dimension investigated in this study, such as age, negative emotional states, daytime drowsiness, resting-state cognition, arousal regulation parameters, or narrowband-EEG-derived measures like periodic and aperiodic components of neural activity, it is important to consider the participant characteristics of this study. This consideration may reveal interpretations of findings that could be specific to this sample of participants, comprising of "healthy" young and older adults.

5.4.2. Interpretations of Overall Thesis Findings

Across all observed dimensions of this study, the older adult participants did not conform to the typical descriptions reported in the literature. Previous studies often found that older adults experienced poorer sleep quality (e.g., Gulia & Kumar, 2018), higher incidences of negative emotional states (e.g., Sivertsen et al., 2015) with higher reports of daytime drowsiness (Zalai et al., 2017). Consequently, it was expected that similar patterns would manifest in this sample of older adults. Based on these assumptions, it was hypothesised that

the older adults would exhibit non-stable arousal regulation patterns or higher levels of "neural noise", (i.e. flatter aperiodic slopes). However, contrary to these expectations, the older adults in this sample did not demonstrate significantly poorer sleep quality compared to young adults. In addition to that, they reported fewer symptoms of depression, anxiety, and stress, as well as lower levels of daytime drowsiness. Moreover, older adults did not show significantly different arousal regulation patterns or aperiodic components when compared to young adults. Furthermore, after controlling for the individualised fit of narrowband power, age-related differences in alpha and theta power were also not significantly different between the two age groups. The only significant finding that was consistent with the literature was the lowered IAPF in older adults (Scally et al., 2018).

One possible interpretation of these findings suggests that the participants sampled in this study may not exhibit significant "pathologies", or if present, the severity may not be sufficient to have a negative impact on sleep quality. In relation to the effects of negative emotional states on sleep quality, as similarly discussed in Chapter 3 with regards to its effects on arousal regulation patterns, the symptoms of these states may only influence sleep quality when manifested as part of a disorder. Since all participants recruited in this study were specifically excluded if they had a diagnosed sleep or psychiatric disorder, it may be reasonable to infer that the participants in this sample may not actively suffer from such disorders or experience these negative emotional states to a pathological extent. Any variations in symptom severity observed in this sample may still fall within "normal" ranges, rather than indicating the presence of pathological disorders. For instance, the variances in negative emotional states, such as depression or stress in young adults could be attributed to typical environmental stressors in a university setting, such as academic or financial stressors, social pressure, or concerns regarding post-graduation life (Ibrahim et al., 2013, see Chapter

2 Discussion). As such, the absence of such pathological symptoms may have resulted in the relatively lacklustre findings in this study.

While conducting norm comparisons would be ideal to support the interpretation that the participants in this sample do not fall within "pathological" ranges, standardised norms that may be readily utilised, particularly across arousal regulation parameters, are presently unavailable (see Chapter 3 Discussion). Furthermore, the individualisation and parameterisation of narrowband EEG activity revealed that group-level differences in alpha and theta power no longer persisted. While these findings may align with contemporary evidence (e.g., Cesnaite et al., 2023), they place retrospective findings that do not utilise these methods under scrutiny. This further restricts the scope of comparisons that can be made, making it challenging to benchmark the overall profiles of the current sample. However, an overall evaluation of the evidence in this study would suggest that the participants in this sample, including the older adults may represent a "healthy" sample, and consequently do not suffer from sleep deficits in a pathological manner. Therefore, any differences in sleep quality in this study may simply be unaffected by any particular factor, and any variations observed could be due to individual differences or environmental effects not measured in this study.

Of particular interest and relevance to this interpretation is the age difference in individual alpha peak frequency (IAPF). Typically, a slowed IAPF is associated with a wide range of pathologies (e.g., Grandy Werkle-Bergner, Chicherio, Lövden et al., 2013, Ramsay et al., 2021). However, recent findings by Pathak et al. (2022) suggest that the slowing of IAPF in older age may not be pathological. Instead, Pathak et al. (2022) found evidence that the slowing of the IAPF may be indicative of compensatory processes that preserve cortical networks as a response to the deterioration of axonal tracts in ageing. Viewed in this context, the slowing of the IAPF, which was observed in this sample of older adults, may actually be

an indicator of “healthy” responses to the ageing brain. An interpretation that aligns with the overall observations in this sample.

5.4.3. Implications, Limitations & Future Directions

While these findings diverge from the original intent of the thesis, which was to identify older adults with poorer sleep quality and explore the factors underlying those developments, they offer an alternative perspective on the relationship between ageing and sleep quality. These results suggest that poor sleep quality may not be an inevitable outcome of ageing.

A crucial takeaway from the findings, as indicated by the older adults in this sample, is that poor sleep quality, commonly reported across the literature, may not be solely attributable to ageing itself, but may occur as a side effect of other pathologies that may be more prevalent in old age. These suggestions parallel the findings of Chaput et al. (2020), who also believed that onset of sleep disorders is due to age-related lifestyle, rather than solely due to ageing. For future investigations into the relationships between sleep and ageing, the focus should perhaps shift to linking age-related pathology and sleep pathology, rather than simply age and sleep pathology. Similar study designs to those utilised in the present study could be replicated in other samples where such pathologies may be present, for instance, older adults suffering from physiological or psychiatric conditions. This would allow for the research to examine hypotheses where age-related pathology may initiate the onset of other related factors that may subsequently modulate sleep quality.

Conversely, it is also possible that these variables could be influenced by sleep quality, rather than influencing sleep quality themselves. For instance, good sleep quality may produce healthy arousal regulation patterns (i.e. stable patterns), which may consequently regulate negative emotional states and affective disorders. It is possible that because no

differences in sleep quality were observed in this sample, significant differences were also not observed in most of the other measures. Notably, the psychological variables revealed significant correlations with sleep quality in Chapter 2, but this relationship was not reflected in the models in this chapter. It may be possible that significant effects may have been obtained if sleep quality was used as a predictor, rather than the outcome variable. However, such examinations are beyond the initially planned scope, and would benefit from longitudinal designs, rather than cross-sectional designs.

Expanding on the findings of this thesis, future studies should make efforts to account for individualised fits of narrowband measures and the parameterisation of EEG signals to examine the relationships of periodic and aperiodic activity. As demonstrated in this sample, the incorporation of such methods validates the claim that EEG narrowband differences observed in previous literature may be attributed to a failure of canonical frequency bands to adequately capture shifts in individual peak frequencies. Furthermore, the parameterisation approach would enhance the credibility of findings and support future investigations that include the examination of aperiodic components as an additional diagnostic tool, potentially enhancing the overall diagnostic capabilities of using EEG-derived measures (Monchy et al., 2023).

Furthermore, dedicated efforts should be made to outline and define characteristics of arousal regulation patterns. This could involve establishing normative scores based on the populations of interest to the arousal regulation model, such as those with affective disorders, or those with sleep quality deficits. Specifically, this could involve delineating profiles that may describe different arousal regulation patterns. For example, establishing criteria for what scores of arousal stability may qualify as “stable” in contrast to non-stable states, or how slope indices may aid the categorisation of such patterns. In addition to that, including additional measures to accommodate shifts in the IAPF across ageing, or incorporate

aperiodic parameters may improve its validity. This would facilitate future interpretations of VIGALL parameters and allow for more standardised cross-study comparisons.

Besides that, addressing the individual nature of each variable may enhance predictive abilities or reveal new insights into their effects on sleep quality. Particularly in this study, several VIGALL parameters may not produce linear effects on sleep quality. Given that high and low arousal stability scores may indicate non-stable regulation patterns, examining this non-linearity with sleep quality, which is measured on a linear dimension, may not fully capture their interactions in traditional linear models. As such, more explicit efforts could be made to model this relationship, either through non-linear regression models such as generalised additive models or through machine learning models such as random forests.

One of the possible limitations of this study may be that the recruited participants, particularly older adults, may represent a population that is “healthier” than the general population. As the present study only recruited “healthy” participants — defined as individuals without sleep or psychiatric disorders, it is plausible that the absence of findings in the examined variables is specific to this population. Given the absence of these underlying issues, the examined relationships may simply not be relevant to sleep disorders. Despite showing some group-level differences, these variances may not reflect true pathology, which may necessarily have created a bias in the sampled participants.

Furthermore, the older adult participants in this study may be an atypical sample compared to those in previous studies. Notably, these older adults did not show significantly poorer sleep quality compared to the young adults to begin with, which is a slight deviation from the literature. The older adults in this study were primarily recruited from community activity clubs for older-aged adults, where they may have engaged in highly active lifestyles. These include social and physical activities like dancing, hiking, and cycling, as well as choir and cooking. Since a majority of the older adult participants were drawn from such groups,

confounding relationships between lifestyle and social factors may have influenced the results found. For instance, participation in large social events may have reduced feelings of loneliness and depression, and many of the older adults were retirees, potentially experiencing minimal to no work-related stress. Similarly, the young adult sample in this study were all university students. As such, the findings observed in this sample of young adults may not extend to young adults who may be outside an academic setting, who may experience a variety of different environmental factors that could potentially affect the variables measured.

Besides that, another limitation of this study may include the choice of questionnaires used. As previously discussed in Chapter 3, alternative assessments of depression or other affective disorders could be explored in relation to the arousal regulation model. This could involve an evaluation of which specific dimensions of depression may be characteristic of hyperstability, and which validated questionnaires may adequately capture those relationships. Perhaps a delineation process could also be conducted by contrasting these negative emotional scores between samples of healthy versus psychiatric patients, which may help address whether variations in arousal regulation patterns may be due to the severity of symptoms, or whether they may be modulated by a disorder.

Furthermore, the findings related to the resting-state cognition dimensions of the ARSQ were not particularly robust in this study. This may be attributed to the construct of the statements comprising these dimensions, which may not accurately reflect the intended dimension. For instance, the ‘Theory of Mind’ dimension included statements like “I thought about others.” and “I thought about people I like.”, which may not align with conventional definitions of theory of mind, involving the attribution of mental states to oneself and others (Wimmer & Perner, 1983). Furthermore, the authors themselves addressed similar concerns about the ToM dimension, as their findings were counter-intuitive; they found that ToM did

not correlate with the identification and acceptance of others, two traits that theoretically should be linked. This may suggest that the conceptualisation of ToM in the ARSQ may not be valid (Diaz et al., 2014). Similarly, the 'Other' dimension of the ARSQ encompassed 19 statements that did not correspond to any of the ten defined dimensions. These statements covered a wide range of seemingly unrelated factors such as "I thought about the present.", "I felt nothing.", or "I had similar thoughts throughout the session.", making it challenging to form informative interpretations of this dimension. As such, future studies aiming to investigate similar dimensions of resting-state cognition could benefit from re-validating the construct validity or improving the specificity of the statements to improve dimension clarity and interpretability.

Lastly, it is important to note that the present study had a relatively small sample size for certain analyses (e.g., small-n, high-p problem). The sample size was determined with consideration for the EEG study design, which also included the time practicalities of conducting an experimental session. On average, each participant's experimental session lasted 2.5 to 3 hours, and sessions had to be scheduled between 9.00 am to 1.00 pm to prevent time-of-day confounding effects (e.g., post-lunch drowsiness; Reyner et al., 2015). This scheduling constraint limited the number of sessions that could be conducted per day to one or two sessions. While the current sample may have been sufficient compared to similar EEG studies (e.g., Awais et al., 2014), it may have been underpowered for some of the statistical comparisons performed in this study. As such, future studies could explore the possibility of conducting secondary analyses using existing datasets with larger sample sizes to re-evaluate the hypothesised relationships investigated in this study.

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Appendix A

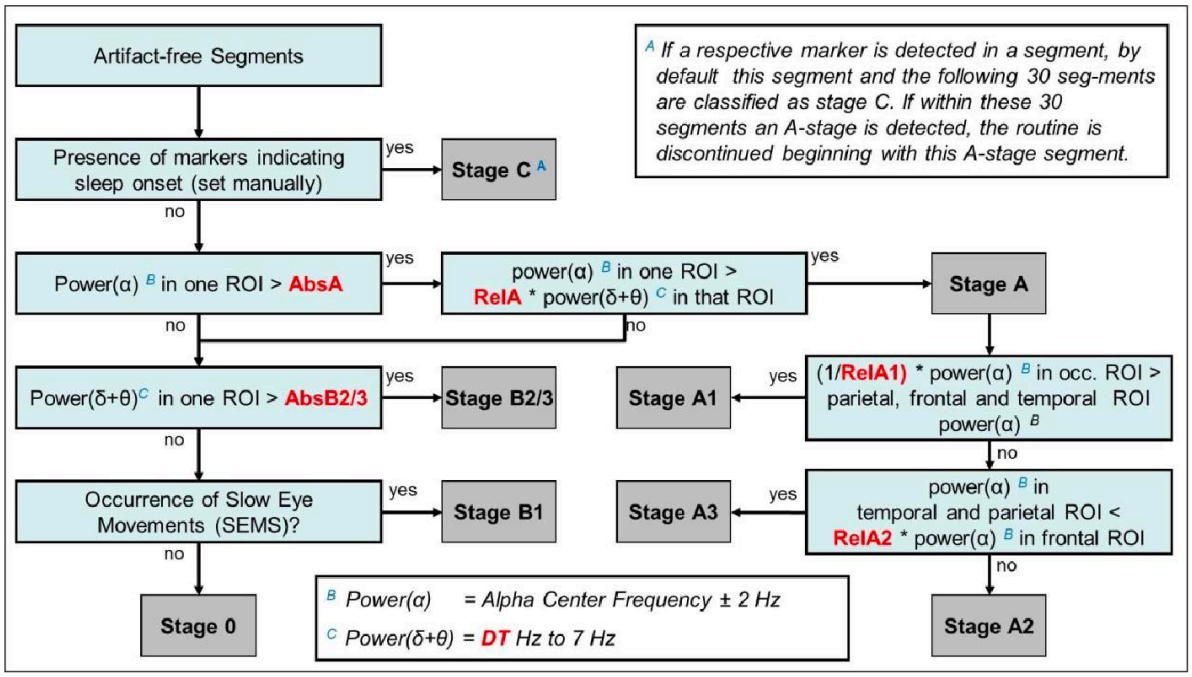
Berger Manoeuvre Instructions

The Berger manoeuver is conducted before EEG recordings to assess participant responsiveness, electrode reactivity, and to identify any abnormalities that could impact the recordings. The instructional script for this assessment was adapted from the VIGALL manual (Hegerl, Sander & Ulke et al., 2016), however specific instructions were adjusted to improve communication clarity, particularly for older adult participants. The sequence of instructions is detailed in the table below.

	Instruction	Assessment
1	"Please close your eyes and keep them closed for five seconds"	Observe EEG record for deflections on EOG electrodes, and appearance of posterior dominant rhythm
2	"Please open your eyes, and blink three times."	Observe EEG record for deflections on VEOG electrodes, take note if signal is propagated to other channels.
3	"Next, please move your eyes from left to right without moving your head."	Observe EEG record for deflections on HEOG electrodes, take note if signal is propagated to other channels.
4	"Please close your eyes again and keep them closed until I say so."	Observe EEG record for deflections on EOG electrodes, and appearance of posterior dominant rhythm
5	"Now, with your eyes closed, I want you to silently — in your head, continously subtract 5 from 100 until I tell you to stop. For example, start at 100, then 95, then 90..."	Allow participants to perform task for 15 seconds. Observe EEG record for change in posterior dominant rhythm, such as the appearance of beta waves.
6	"Alright, please open your eyes and tell me what number you stopped at."	Make note of participants' understanding of instructions by ensuring that the final number is a multiple of 5.

Appendix B

VIGALL Scoring Rules



Note. VIGALL Scoring Rules. From "Vigilance Algorithm Leipzig (VIGALL) Version 2.1 Manual" by Hegerl, U., Sander, C., Ulke, C. et al., 2016.