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# Introduction

Non-restorative sleep (NRS) is a condition characterised by self-reports of unrefreshing sleep despite normal objectively measured sleep duration and architecture, leading to excessive daytime fatigue, sleepiness, and diminished quality of life (Roth et al., 2010). Despite the impact of this condition, there are no established guidelines for diagnosis or clinical management, and it is not included in the Diagnostic and Statistical Manual-5-TR (DSM-5-TR) (American Psychiatric Association, 2022). NRS has previously been clinically managed as a subtype of insomnia disorder (ID) despite patients not exhibiting the symptoms associated with ID such as difficulty falling asleep, difficulty maintaining sleep, and shortened sleep duration (Roth et al., 2010). ID has received significant attention and research due to being the most common sleep disorder in Australia and the associated health burden (Sweetman et al., 2021). It has been linked to an increased risk of physical and mental health disorders, and decreased quality of life (Kyle et al., 2010). In contrast, NRS is poorly understood and treated, largely due to limited available research on the aetiology of the condition. Currently, NRS symptoms are predominantly measured through subjective complaints, and it is unclear if the condition is associated with measurable neuropathology that may be different to ID. If there are measurable changes in brain activity as compared to healthy controls and to ID, this could aid in better aetiological classification and diagnosis of the condition.

Both NRS and ID have been consistently associated with increased daytime fatigue (Kim et al., 2019; Zhang et al., 2012), however findings on subjective and objective daytime sleepiness are inconsistent (Hein et al., 2017; Sarsour et al., 2010). While fatigue is a broad construct that refers to feelings of exhaustion or low motivation, sleepiness specifically refers to an individual’s sleep propensity, or their drive to sleep (Gradisar et al., 2007). Subjective sleepiness, being an individual’s self-assessed perception of their sleep drive at a given moment, and objective sleepiness, being the level of sleep-like brain activity recorded through electroencephalography (EEG), are correlated and increase in response to prolonged wakefulness and reduce with sleep in healthy populations (Åkerstedt & Gillberg, 1990). Although ID has been associated with decreased subjective and objective measures of sleepiness, it is not known how sleepiness manifests in an NRS population [@cite].

EEG recordings provide a spatiotemporally integrated recording of neuronal signals across the cortical surface, allowing for the non-invasive measurement of brain activity and mental states in humans (Buzsáki et al., 2012). This method enables identification of intrusions of sleep-like brain activity present during wake, that can occur in localised brain regions rather than entire lobes of the cortex (Siclari & Tononi, 2017). Quantitative analysis of brain activity using high-density electroencephalography (HD-EEG) data provides an accurate method of explore daytime sleepiness in these populations. HD-EEG enables exploration of cortical activity variations across brain regions with greater spatial resolution than traditional EEG, allowing for neural measures of sleepiness to occur within localised regions of the cortical surface. Therefore, this study will use self-rated sleepiness scores and HD-EEG data to answer the research question of whether there are differences in subjective or objective sleepiness during resting-state wakefulness directly after awakening from overnight sleep between NRS in comparison to ID and healthy controls.

# Sleep Disorders

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## Insomnia Disorder

ID is the most common sleep disorder in Australia with an estimated prevalence of 23.2% (Appleton et al., 2022). It is linked to detrimental outcomes for individuals, including increased risk of comorbid psychiatric disorders, reduced physical health, diminished quality of life, and significant daytime fatigue (Kim et al., 2019; Kyle et al., 2010; Morin et al., 2015; Perlis et al., 2022). ID is diagnosed through subjective reports of impaired overnight sleep, difficulty with sleep initiation, frequent overnight awakenings, and/or early morning awakenings without the ability to fall back asleep, leading to clinically significant distress or dysfunction in daily life (American Psychiatric Association, 2022).

**Non-Restorative Sleep**

NRS is distinct from ID as the primary complaint is unrefreshing sleep despite normal sleep duration of approximately 7-8 hours, and no subjective reports of difficulty initiating or maintaining sleep, or early morning awakenings (Roth et al., 2010). Daytime impairments associated with NRS include significant daytime fatigue, reduced cognitive performance, and reduced psychological well-being, leading to reduced quality of life and impaired daily function (Roth et al., 2010). The prevalence of the symptom of unrefreshing sleep upon awakening in Australia is estimated to be between 42-45%, however only approximately 10% of this population receive clinical treatment (Adams et al., 2017; Metse & Bowman, 2020). Despite the negative consequences of the condition, it was removed as a characteristic of ID with the release of the Diagnostic and Statistical Manual-5 (DSM-5) due to its inconsistency with other ID symptoms and lack of validated operational measures (American Psychiatric Association, 2013). Consequently, this population are diagnosed as “other specified insomnia disorder”, with no guidelines for diagnosis or treatment (American Psychiatric Association, 2022). As NRS may be its own unique disorder with an underlying neurobiological cause, it is essential to develop diagnostic criteria and understand the associated aetiology to improve outcomes for patients.

# Sleep

Sleep is a necessary behaviour for all humans that can be defined as a reversible reduction in responsiveness to external stimuli accompanied by a measurable change in brain activity patterns (Cirelli & Tononi, 2008). Sleep progresses through a series of stages throughout the night, including rapid eye movement (REM) sleep and non-rapid eye movement (NREM) sleep, which is further divided into stages N1, N2, and N3, each distinguishable through distinct patterns of brain activity and muscle tone. Sleep is regulated by two interacting systems, with the circadian system operating as a pacemaker that entrains the body to a 24-hour cycle in response to the external environment, while the homeostatic system is responsible for building up sleep pressure that accumulates with wakefulness and dissipates with sleep (Borbély, 1982).

Sleep pressure can only be reduced by sleep [@cite]. High sleep pressure at the start of the night is associated with increased prevalence and amplitude of slow wave activity (SWA) in the EEG, which reduces as sleep pressure dissipates (Vyazovskiy et al., 2011). Slow waves are synchronized oscillations of neuronal membrane potentials between hyperpolarised and depolarised states in the delta frequency range (0.5-4 Hz) that propagate throughout the brain in an antero-posterior cortical progression, with the greatest prevalence in N3 sleep (Achermann & Borbély, 2003; Riedner et al., 2007). SWA has topographic variations throughout the brain, occurring locally and asynchronously across brain regions and most prominently in areas associated with increased activity during wake, suggesting sleep homeostasis is a locally regulated phenomenon (Krueger et al., 2019; Siclari & Tononi, 2017). SWA dissipates with consecutive sleep cycles throughout the night, indicating that homeostatic sleep pressure also dissipates [@cite]. Emerging evidence suggests that IF is characterised by lower initial SWA at sleep onset (Grimaldi et al., 2021), indicating a deficiency in the homeostatic regulation of sleep, which may additionally explain daytime fatigue and sleepiness.

# Sleepiness and Fatigue

## Subjective Sleepiness and Fatigue

Increased sleep pressure is perceived subjectively as sleepiness, which is hypothesised to act as a primary motivational drive ensuring organisms sleep regularly despite the risks arising from reduced consciousness (Axelsson et al., 2020). It is a measure of an individual’s self-assessed level of sleep pressure, objective drowsiness, or sleep propensity, which fluctuates throughout the day in response to the influence of sleep homeostasis and circadian systems (Åkerstedt et al., 2014). Subjective sleepiness can be measured as either trait or state sleepiness, with trait sleepiness being an individual’s propensity to fall asleep in a given situation, and state sleepiness being a measure of sleepiness at a point in time (Åkerstedt et al., 2014; Johns, 1991). State sleepiness is most commonly measured using the Karolinska Sleepiness Scale (KSS), a one item nine-point Likert scale correlated with EEG measures of drowsiness in healthy populations (Cluydts et al., 2002; Kaida et al., 2006).

Excessive daytime sleepiness is one of the most common complaints associated with NRS but is inconsistently observed in ID (Hein et al., 2017; Sarsour et al., 2010). Although daytime fatigue is the most prevalent and detrimental complaint observed in ID, the inability to sleep that is characteristic of ID means that subjective sleepiness may not be a sensitive measure of daytime sleepiness in this population (Kyle et al., 2010; Raizen et al., 2023). Therefore, there is a critical need for improved measurement tools that can capture the experiences of ID and NRS.

## Objective Sleepiness

Objective sleepiness refers to the quantifiable level of sleep pressure within an individual measurable through behavioural or neurophysiological measures. The most commonly used measures in research and clinical practice sensitive to increased sleepiness are the Multiple Sleep Latency Test (MSLT), which evaluates sleep propensity by measuring how quickly an individual falls asleep; the Maintenance of Wakefulness Test (MWT), which measures the ability to stay awake; and the Psychomotor Vigilance Task (PVT), which assesses sustained attention through reaction time measurements (Basner & Dinges, 2011; Martin et al., 2023). However, these measures do not directly measure the level of neurobiological sleepiness that can be present in an individual without sleep onset, instead measuring the consequences of increased sleepiness. This limits their use within clinical populations, particularly ID which is characterised by an inability to fall asleep.

Neurobiological sleepiness can be directly measured through the Karolinska Drowsiness Test (KDT), which uses EEG data to quantify the level of arousal when the eyes are open and closed (Åkerstedt et al., 2014; Åkerstedt & Gillberg, 1990). Increased sleep pressure, such as that caused by experimentally manipulated sleep deprivation, can be observed through a shift of EEG spectral power from fast frequency activity in the alpha (8-12 Hz), beta (15-25 Hz), and gamma (13-35 Hz) frequencies to slower frequency activity in the delta (0.5-4 Hz) and theta (4.5-8 Hz) frequencies [@cite]. This shift is most prominent in the theta frequency range, with increased theta oscillations during eyes-open resting wake mirroring the predicted trajectory of sleep pressure (Snipes et al., 2023). Additionally, this increase occurs in a task-dependent manner within localised cortical regions, mirroring the process observed in sleep deprivation and subsequent recovery SWA during sleep [@cite]. Theta activity reflects fluctuations in circadian rhythms (Aeschbach et al., 1997; Cajochen et al., 2002), decreases following caffeine consumption (Landolt et al., 1995), and is increased in individuals suffering from excessive daytime sleepiness (EDS) (Melia et al., 2015), meaning it appears to be a marker of sleepiness, rather than just sleep pressure. However, as theta activity is also associated with fatigue, increasing with time-on-task independent of changes in other frequency bands (Li et al., 2020; Tran et al., 2020), it is insufficient as a measure of objective sleepiness alone.

The increase in theta oscillations and shift from fast-frequency to slow-frequency EEG activity can be quantified by the slowing ratio (SR), which measures the ratio of fast frequencies to slow frequencies. SR is associated with reduced sleep onset latency (Appleton et al., 2022), impairments to behavioural performance following sleep deprivation (Gibbings et al., 2022), and can be used to assess treatment efficacy in obstructive sleep apnoea (Tracey et al., 2024). Although this measure is commonly used to measure sleepiness, it does not consider that alpha power manifests differently with increasing sleepiness when the eyes are open **vs eyes closed.**

Under conditions of low sleepiness, alpha power is most prominent during eyes closed resting wake EEG recordings, with the greatest power in the occipital region. However, with increasing sleep pressure, alpha oscillations appear with eyes-open resting wake EEG and attenuate when the eyes are closed [@cite]. Increased eyes-open alpha power is correlated with increased sleep pressure accumulated through sleep deprivation and changes in body temperature occurring in response to circadian influences (Cajochen et al., 2002; Tian et al., 2018). Additionally, decreased eyes-closed alpha power represents the beginnings of sleep onset, **representing the transition between wake and N1 sleep [@cite].** This change in spectral power can be quantified as the alpha attenuation coefficient (AAC), which calculates the ratio of eyes-open alpha power to eyes-closed alpha power (Stampi et al., 1995). As alpha attenuation reflects declining alertness and the progression towards sleep, it can be used as an objective measure of sleepiness.

## The Association Between Objective and Subjective Sleepiness

Measures of subjective sleepiness were developed and validated in concordance with objective sleepiness outcomes. State subjective sleepiness as measured by the KSS is highly correlated with AAC, alpha, and theta power during the eyes-open KDT in healthy individuals, indicating validity for both measures in measuring the construct (Kaida et al., 2006). KSS additionally correlates with other behavioural measures of sleepiness, including reaction time, sleep latency, and behavioural lapses in a driving task (Baulk et al., 2001; Sandberg et al., 2011; Shin et al., 2024). However, this association is not linear, with associations being strongest at high levels of sleepiness (KSS ≥ 7) [@cite]. This suggests there may be a mechanism wherein the individual experiences increased awareness of the presence of sleep-like neural activity occurring despite maintaining consciousness (Åkerstedt et al., 2014).

This association may also vary significantly within different clinical groups. ID is characterised by cortical hyperarousal; the 24-hour increase of fast-frequency EEG activity occurring during wake and sleep (Colombo et al., 2016; Kao et al., 2021; Riemann et al., 2010). Increased fast-frequency activity in ID has been linked to sleep-state misperception, wherein individuals underreport their subjective sleep time in comparison to PSG measured sleep [@cite]. Furthermore, this association has not been researched in an NRS sample without a comorbid sleep disorder. Understanding if there are differences in the mechanisms of subjective and objective sleepiness in ID and NRS could provide greater insight into the aetiology and treatment for both disorders.

# Aim

This study aimed to explore if there are differences in how people with NRS, ID, and healthy controls experience subjective and objective sleepiness, and if differences are associated with topographic differences of spectral power during resting wake. First, we examined if there were group differences in subjective sleepiness levels upon awakening as measured by the KSS administered after habitual wake time, hypothesizing that subjective sleepiness would be highest in NRS, lowest in ID, with healthy controls in the middle. Secondly, we examined if there were group differences in HD-EEG measures of objective sleepiness as measured through AAC and SR in the morning KDT. We hypothesized that objective sleepiness would be highest in NRS, lowest in ID, with healthy controls in the middle. Third, we explored if the association between subjective sleepiness as measured by KSS and objective sleepiness as measured by AAC and SR differed across groups, hypothesizing there would be a significant main effect for group membership and subjective sleepiness, and a significant interaction.

# Method

## Participants

The study was approved by the Macquarie University Human Research Ethics Committee (FoRA ID 17112) and all participants provided written informed consent. Participation was voluntary and could be discontinued at any time.

The present study was derived from data captured during a larger neuroimaging research study on phenotyping of individuals with NRS. Due to the complexity of the study and large amount of outcome variables an a priori power analysis was not performed. A sample size of 12 participants from each population, with a total sample of 36 participants, was proposed due to funding constraints. Participants were sex and age matched (with a maximum difference ± 2.5 years) to control for to the influence of age and sex on sleep architecture (Mongrain et al., 2005).

Participants were excluded if they had comorbid sleep apnoea, as measured by WristOX pulse oximeter or the STOP-bang sleep apnoea questionnaire (STOP-Bang) which has a high sensitivity of detecting clinically relevant obstructive sleep apnoea syndrome (Chung et al., 2016; Nigro et al., 2009). Participants were additionally excluded if they had clinically significant depression (> 10) or anxiety (> 7) scores as measured through the Depression Anxiety and Stress Scale 21 (DASS-21) (Lovibond & Lovibond, 1995), heavy alcohol use, or pregnancy. Circadian rhythm disruption was controlled for with an exclusion criteria of recent (in the previous 30 days) shift work or international travel, or a natural sleep time outside the hours of 21:30 and 8:00 [@cite]. As certain medications can influence sleep architecture, participants taking regular medications affecting sleep were excluded.

The inclusion criteria for the ID group was clinical diagnosis of ID by a sleep physician following the DSM-5-TR criteria, with difficulty initiating or maintaining sleep persisting for over 1 month causing clinically significant distress or impairment in daily life (American Psychiatric Association, 2022). They were additionally required to have a Pittsburgh Sleep Quality Index (PSQI) score ≥ 5, and an Insomnia Severity Index (ISI) score ≥ 15.

Individuals in the NRS group could not have a weekly mean Total Sleep Time (TST) below six hours as measured by sleep diary or actigraphy, or a mean refreshed score ≥ 3 on a 5-point Likert scale as measured using the Karolinska Sleep Diary (Åkerstedt et al., 1994). Inclusion in this group required an overall PSQI score ≥ 5, with subcomponent scores ≥ 2 on the PSQI Component 1 and ≥ 10 on PSQI Component 5.

Healthy controls needed to have a PSQI score ≤ 4 or less and an ISI score of 6 or less.

## Procedure

Participants were recruited through referrals to the Woolcock Institute of Medical Research and the Royal Prince Alfred sleep clinics, and via social media advertising. The Woolcock Institute of Medical Research is a specialist clinic that conducts research in addition to clinical services for individuals experiencing sleep and respiratory disorders. Volunteers completed an online questionnaire to assess eligibility for inclusion in a clinical group (ID, NRS, healthy controls) which was then confirmed through telephone screening by a researcher and an in-person clinical screening by a sleep physician.

Prior to the study, participants attended the Woolcock Institute of Medical Research for initial screening by a sleep physician. Participant’s baseline sleep and activity patterns were measured via a Geneactiv Actigraphy watch for 7 days prior, which was validated against self-reported sleep diaries (Menczel Schrire et al., 2023). Participants additionally completed the Restorative Sleep Questionnaire Daily Version (RSQ-D) (Drake et al., 2014) and Karolinska Sleep Diary to assess baseline sleep quality (Åkerstedt et al., 1994).

On the day of the study, participants arrived at the laboratory at 17:00 and underwent a final medical screening and a series of cognitive assessments forming part of a larger study. They were then served dinner and fitted with a high-density electroencephalography (HD-EEG) cap. Participants went to bed at their habitual bedtime, as established by self-report and actigraphy data (Menczel Schrire et al., 2023).

Overnight PSG data were collected using standard American Academy of Sleep Medicine (AASM) clinical practice guidelines, measuring HD-EEG activity in addition to electrocardiogram (ECG), electrooculogram (EOG), and electromyogram (EMG) data (Berry et al., 2017). Any overnight disturbances were recorded by research staff.

Lights were turned on at the participant’s habitual wake time. The Karolinska Sleepiness Scale (KSS) and Karolinska Drowsiness Test (KDT) were administered five minutes post habitual wake time (Åkerstedt & Gillberg, 1990). Following the morning KDT, participants completed further cognitive testing and an MRI scan.

Participants were reimbursed for travel costs to and from the laboratory up to the value of $250 and remunerated $100 upon successful completion of the study.

## Measures

### Screening Questionnaire

An online screening questionnaire was administered to obtain participants’ age, sex, baseline alcohol consumption, absence of pregnancy, suitability for magnetic resonance imaging (MRI), and to screen for circadian disruption (Appendix A). Additional questionnaires included the STOP-Bang (Chung et al., 2016), Insomnia Severity Index (ISI; Bastien et al., 2001), Depression Anxiety Stress Scales-21 (DASS-21; Lovibond & Lovibond, 1995), and the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989).

### Insomnia Severity Index (ISI)

Insomnia symptoms were assessed using the Insomnia Severity Index (ISI), a seven item self-report measure of subjective insomnia symptoms (Bastien et al., 2001). Items (e.g. “Please rate the CURRENT (i.e. LAST 2 WEEKS) SEVERITY of your insomnia problem(s)”) are rated on a 5-point Likert scale ranging from 0 (“none”) to 4 (“very severe”). The scale ranges from 0 to 28, with scores of 10 or greater found to have 86.1% sensitivity and 87.7% specificity for detecting ID cases in a community sample and ISI scores ≥ 15 interpreted as moderate-severe insomnia (Morin et al., 2011). The ISI demonstrated good internal consistency within the current sample with a Cronbach’s alpha of .89.

### Pittsburgh Sleep Quality Index (PSQI)

Self-assessed sleep quality was measured using the Pittsburgh Sleep Quality Index (PSQI), a 19-item questionnaire assessing sleep quality and disturbance over the past month (Buysse et al., 1989). The PSQI measures a broader construct than insomnia severity as it measures sleep-related disturbances beyond sleep initiation and maintenance. The convergent validity between the PSQI and ISI within our sample was *r* = .79. The measure produces a global score (PSQI) comprised of seven component scores, relating to subjective sleep quality (PSQI-1; “During the past month, how would you rate your sleep quality overall”), sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances (PSQI-5; (“During the past month, how often have you had trouble sleeping because you…”), use of sleep medication, and daytime dysfunction. Items are rated on a 4-point Likert scale with greater scores indicating greater impairment. Global PSQI scores range from 0 to 21, with scores ≥ 5 recommended as the cut-off point for poor quality sleep in clinical populations (Buysse et al., 1989). The PSQI demonstrated acceptable internal consistency (α=.72) within the sample, which is consistent with previously reported values in clinical and non-clinical populations (Mollayeva et al., 2016).

### Flinders Fatigue Scale (FFS)

Daytime fatigue impairments was measured using the Flinders Fatigue Scale (FFS), a 7-item measure of fatigue characteristics (e.g. “was fatigue a problem for you”) over the previous two weeks (Gradisar et al., 2007). The scale produces a score ranging from 0 to 31, with greater scores indicating greater fatigue. The threshold for clinically significant daytime fatigue is a score of 13-15 for borderline fatigue, 16-20 for moderate fatigue, and ≥ 21 for severe fatigue (Cameron et al., 2017). The scale explicitly defines fatigue as being distinct from sleepiness, stating “We are interested in the extent that you have felt **fatigued** (tired, weary, exhausted) over the last **two weeks**. We **do not** mean feelings of **sleepiness** (the likelihood of falling asleep).” The FFS had good internal consistency within the sample(α = .86).

### Epworth Sleepiness Scale (ESS)

Trait sleepiness was measured using the Epworth Sleepiness Scale (ESS), an 8-item measure assessing the likelihood of dozing in specific situations (e.g. “sitting and reading”) (Johns, 1991). Items are rated on a 4-point Likert scale, ranging from 0 (“would **never** doze”) to 3 (“**high** chance of dozing”). Scores range from 0 to 24 with greater ESS scores indicating greater sleep propensity, with scores ≥ 10 indicating subjective excessive daytime sleepiness (Johns, 1991). The ESS had good internal consistency (α=.85) within the sample within the sample and was not correlated with FFS scores (*r* = .20).

### Karolinska Sleepiness Scale (KSS)

Subjective state sleepiness was assessed 5 minutes after natural wake time using the Karolinska Sleepiness Scale (KSS), a 1-item measure of an individual’s perceived sleepiness at a given point, with the instructions “Please measure your sleepiness over the past 5 minutes.” It uses a 9-point Likert scale with verbal anchors at every second step ranging from 1 (“Extremely alert”) to 9, “Extremely sleepy - fighting sleep” (Åkerstedt & Gillberg, 1990).

### Karolinska Drowsiness Test (KDT)

The Karolinska Drowsiness Test (KDT) was administered immediately following the KSS and was used to measure electrophysiological drowsiness as measured through HD-EEG recordings. Participants were instructed “Look at the dot in front of you and be as relaxed as possible while staying awake. Keep your head and body still and minimize blinking. After a few minutes, I’ll ask you to close your eyes and keep them closed for a few minutes. Finally, I’ll ask you to open your eyes again and keep them open for a few minutes.” The test is approximately 7 minutes long with 3 phases (eyes open/eyes closed/eyes open) each lasting 120 seconds. The eyes open conditions were concatenated during data analysis.

### PSG Sleep Scoring and Sleep Macroarchitecture

Overnight PSG sleep data were recorded and scored in 30 second epochs according to American Academy of Sleep Medicine Manual (AASM) criteria by an experienced rater (Berry et al., 2017). Sleep recordings were evaluated for the following parameters of sleep continuity: time in bed (TIB, measured as total time spent in bed independent from sleep state); total sleep time (TST, defined as time between first sleep onset and final awakening, excluding periods awake); sleep onset latency (SOL, measured as time from lights out until first epoch of sleep); snooze time (measured from time of final awakening to time out of bed); REM latency (minutes from sleep onset to first epoch of REM sleep); wake after sleep onset (WASO, time spent awake between sleep onset and final awakening); sleep efficiency (ratio of TST to time in bed × 100 %); and total minutes/percentage in N1, N2, N3 and REM sleep (as scored using the AASM criteria).

### HD-EEG

High-density EEG data were collected using 256-channel electrode caps and Net Amps 400 amplifiers (MagstimEGI, Eugene, OR, USA) with signals amplified and digitised at 500 Hz referenced to the vertex (CZ). Prior to starting any of the acquisitions, electrode impedance was below 50 kΩ. After acquisition, data were low-pass filtered at 70 Hz, high-pass filtered at 0.3 Hz, and notch filtered at 50 Hz.

## Data processing

### Visual Inspection and EEG Pre-Processing

All preprocessing was completed using the EEG Processor application for MATLAB (https://eeg-processor.readthedocs.io/). Data were visually inspected for artefacts and arousals which were removed across all channels. Poor quality channels were replaced with an interpolated EEG signal from all other good-quality channels using linear mixing, weighted by the squared distance to the poor-quality channel.

To enhance the local signal detection of each electrode and minimise the influence of the vertex (Cz) electrode, data were re-referenced to a common average signal (i.e., mean global signal across all EEG channels).

### Independent Components Analysis

Following visual inspection, independent components analysis (ICA) was used to identify and separate statistically independent components. This was done using a semi-automated process using the MATLAB program ICLabel, which automatically removed components classified as non-brain activity with a probability ≥ .8 (Pion-Tonachini et al., 2019). Further visual inspection was conducted to verify artefact removal and to remove components not reaching the weighting threshold that were visually identified as non-brain activity. Remaining components were back-projected to the EEG data signal, retaining only channels at cranial sites (i.e., channels on the cheek and neck were removed), resulting in a cleaned EEG time series dataset.

### Power Spectra

EEG data is characterized by oscillations of varying amplitude at different frequencies which can be quantified through power spectral analysis. The power spectra represent the distribution of power in a signal across frequencies, indicating which frequency components contribute the most to the EEG signal. Power spectra were obtained for each channel using a fast Fourier transform (FFT) to deconstruct the EEG signal from the time domain to the frequency domain. The power spectra were calculated using the Welch method with 6-second windows (50% overlap) and obtained for the eyes closed condition and the concatenated recording of the two eyes open conditions. EEG spectral power densities were integrated within the following frequency bands: low delta (0.5–1 Hz), delta (1–4.5 Hz), theta (4.5–8 Hz), alpha (8–12 Hz), sigma (12–15 Hz), beta (15–25 Hz), and gamma (25–40 Hz). Differences in absolute power can be caused by interindividual factors such as head size, scalp-tissue composition, and skull thickness. Therefore, absolute EEG spectral power densities were normalized to the grand-total power calculated as the average area under the curve between 0.5 and 40 Hz across all EEG channels.

### Slowing Ratio (SR)

The EEG slowing ratio (SR) is a biomarker of sleepiness reflecting the general slowing of brain activity that appears with increasing sleepiness, with a dominance of slow frequency activity being indicative of decreased arousal (D’Rozario et al., 2013). SR has been shown to be a valid measure of reduced alertness and increased drowsiness in clinical populations (Sweetman et al., 2021). The slowing ratio was calculated for each participant in the eyes open and eyes closed conditions using the formula log10(delta + theta)/(alpha + sigma + beta) (Vakulin et al., 2016). A higher SR score indicates increased electrophysiological sleepiness.

### Alpha Attenuation Coefficient (AAC)

The alpha attenuation coefficient (AAC) measures alpha frequency power differences between eyes open and eyes closed conditions (Stampi et al., 1995). Alpha activity increases with sleepiness when the eyes are open, but decreases with sleepiness when the eyes are closed (Putilov & Donskaya, 2014). The AAC is calculated as the log ratio of alpha power in the eyes closed condition to alpha power in the eyes open condition, log10(alpha eyes closed/ alpha eyes open]. A lower AAC score reflects decreased cortical activity and increased sleepiness.

## Statistical Analysis

Statistical analysis of descriptive measures was done using R version 4.3.2 (R Core Team, Vienna, Austria). Analyses involving HD-EEG data were run using the EEG processor application which makes use of the toolboxes Fieldtrip (Oostenveld et al., 2011) and EEGlab (Delorme & Makeig, 2004). An alpha level of *p* = .05 was used for all analyses.

A one-way analysis of variance (ANOVA) was calculated for group differences in categorical variables in demographic variables, survey response measures, and sleep macro-architecture between groups. Assumptions of normality of the distribution, residuals and outliers was conducted using Q-Q Plots, Shapiro-Wilk normality tests, and visual inspections of histograms. The assumption of homogeneity of variance was checked using Levene’s test, and if violated, Welch’s ANOVA was used. **Sphericity?**

To compare subjective sleepiness between groups, a one-way ANOVA was conducted with KSS scores as the dependent variable and group membership as the independent variable. Assumptions were checked as described above. Post-hoc pairwise comparisons using a Bonferroni correction were conducted in the case of a significant result controlling for multiple comparisons.

To compare objective sleepiness between groups, separate one-way ANOVAs were applied to test for group differences in the SR and the AAC at each of the 178 EEG channels. **Sentence about family-wise error rate for channels?** To control for the increase in type-I error rate, a cluster-mass permutation-based analysis of linear models (PALM) was applied (Winkler et al., 2014). Clusters were defined as neighbouring EEG channels. Electrodes showing a significant F-statistic for the factor group (*p* < .05), and their mass was derived as the integrated F-statistical value. This cluster mass was compared to an empirical null-distribution. This involved applying the same model to 10, 000 random shuffles of the data, and with each iteration the largest cluster mass was entered in the to build a reference null-distribution of cluster sizes that occur due to chance, which was then used to compare the found cluster mass size against. Clusters were deemed significant at an alpha threshold of *p* < .05.

To analyse if the association between subjective and objective sleepiness differed across groups, a general linear model was applied with SR and AAC in each EEG channel as the dependent variable, and subjective sleepiness and group membership as predictors. Interaction terms between KSS score and group membership were included to test whether the relationship between subjective and objective sleepiness differed across groups. As above, permutation-based analysis was used to control for type-I error rate. Clusters with a *p*-value < .05 were deemed significant.

# Results

## Participants

964 participants completed the online expression of interest questionnaire, with 352 (36.5%) meeting eligibility criteria. Of these, 169 participants (17.5%) were unable to be contacted or did not respond to a follow-up email. 180 participants proceeded to pre-screening, of whom 147 were excluded during the pre-screening and screening visits, with the most common exclusion reasons being medication use (n = 44) or the absence of an age- and sex-matched participant (n = 54). Due to the time constrains of this honours thesis, the final sample obtained was 33 participants (13 NRS; 11 ID; 9 Controls; 3% of participants who completed the expression of interest questionnaire). Two control participants were excluded from sleep macroarchitecture analysis due to missing data, as sleep studies could not be scored by an expert sleep technician in time. Participant demographic and survey response details are provided in Table 1, and sleep macroarchitecture Table 2.

Significant group differences were found for PSQI sleep quality scores, ISI insomnia severity scores, and FFS daytime fatigue scores, with the control group showing the lowest impairment and the ID group reporting the greatest impairment. Due to a violation of the assumption of homogeneity of variances (*p* = .009), a one-way Welch’s ANOVA was used to assess group differences in PSQI scores, finding a significant effect of group, *F*(2, 16.32) = 35.99, *p* = <.001. Post-hoc Games-Howell tests showed that the control group had significantly lower PSQI scores than both the ID (mean difference = −8.67, *p* < .001) and NRS groups (mean difference = −4.74, *p* < .001), and the ID group having significantly higher PSQI scores than the NRS group (mean difference = 3.93, *p* = .010). A Welch’s ANOVA showed a significant group effect for ISI scores, *F*(2, 18.96) = 112.60, *p* < .001. Post-hoc analyses revealed the control group had significantly lower ISI scores than the ID (mean difference = −15.2, *p* < .001) and NRS groups (mean difference = −12.02, *p* < .001). However, the ID group did not have significantly higher ISI scores than the NRS group (mean difference = 3.17, *p* = .125). Although all groups did not have clinically significant (≥ 13) daytime fatigue, a one-way ANOVA found a significant main effect for group, *F*(12, 30) = 10.56, *p* < .001. Post-hoc comparisons using a Bonferroni correction showed the control group had significantly lower scores compared to the ID (mean difference = −7.47, *p* < .001)

|  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Table 1 *Descriptive Measures by Group*   |  |  |  |  |  |  |  |  | | --- | --- | --- | --- | --- | --- | --- | --- | | Measure | ID | | NRS | | Control | | *p* | | *M* | *SD* | *M* | *SD* | *M* | *SD* | | Sex, male/female | 4/7 |  | 6/7 |  | 3/6 |  |  | | Age | 42.22 | 11.91 | 40.52 | 11.59 | 39.39 | 13.56 | .882 | | PSQI | 12.55 | 3.45 | 8.62 | 1.45 | 3.88 | 1.55 | <.001 † | | ISI | 17.64 | 3.20 | 14.46 | 4.37 | 2.44 | 1.59 | <.001 † | | FFS | 11.36 | 4.03 | 9.77 | 4.04 | 3.89 | 2.93 | <.001 | | ESS | 6.67 | 5.07 | 5.00 | 4.22 | 4.11 | 3.72 | .671 | | KSS AM | 5.09 | 2.17 | 5.77 | 1.92 | 4.22 | 1.09 | .168 |  |   † notes Welch’s ANOVA as homogeneity of variances was violated. PSQI = Pittsburgh Sleep Quality Index; ISI = Insomnia Severity Index; FFS = Flinders Fatigue Scale; ESS = Epworth Sleepiness Scale; KSS = Karolinska Sleepiness Scale |
| Table 2Sleep Macroarchitecture by Group  |  |  |  |  |  |  |  |  | | --- | --- | --- | --- | --- | --- | --- | --- | | Measure | ID | | NRS | | Control | | *p* | | *M* | SD | *M* | SD | *M* | SD | | Time in bed | 464.1 | 38.6 | 491.2 | 25.8 | 493.0 | 31.1 | .087 | | Total sleep time | 360.6 | 87.8 | 395.3 | 51.7 | 415.3 | 64.5 | .246 | | Sleep onset latency | 33.9 | 36.9 | 23.7 | 33.2 | 20.7 | 20.9 | .773 † | | REM latency | 95.6 | 31.9 | 88.1 | 29.1 | 136.4 | 62.6 | .041\*\* | | WASO | 51.2 | 33.0 | 66.4 | 44.9 | 56.9 | 44.1 | .637† | | Sleep efficiency | 77.1 | 15.5 | 80.6 | 10.4 | 84.0 | 10.2 | .460† | | N1 (minutes) | 29.5 | 11.4 | 34.7 | 20.7 | 27.1 | 9.8 | .556‡ | | N2 (minutes) | 181.5 | 54.7 | 196.6 | 37.0 | 224.8 | 58.2 | .202 | | N3 (minutes) | 66.8 | 38.6 | 74.0 | 24.5 | 92.6 | 34.6 | .876 | | REM (minutes) | 82.9 | 34.9 | 90.0 | 24.5 | 92.6 | 34.6 | .777 | | N1 % | 8.1 | 3.2 | 8.8 | 5.0 | 6.6 | 2.5 | .369‡ | | N2 % | 50.0 | 9.7 | 49.8 | 7.4 | 53.7 | 9.6 | .608 | | N3 % | 19.8 | 12.8 | 18.9 | 8.8 | 17.7 | 7.9 | .917 | | REM % | 22.1 | 5.1 | 22.5 | 4.1 | 22.0 | 6.7 | .974 |   † notes variable was log10 transformed as assumption of normality was violated. ‡ notes Welch’s ANOVA as homogeneity of variances was violated. WASO = wake after sleep onset. All times reported in minutes. Sleep macroarchitecture variables reported following AASM criteria. |

and NRS groups (mean difference = −5.88, *p* = .003). No significant difference was found between the ID and NRS groups (mean difference = 1.59, *p* = .933).

The only significant difference between groups in sleep macroarchitecture was in REM latency, *F*(2, 28) = 3.58, *p* = .042. Post-hoc comparisons using a Bonferroni correction showed the NRS group had significantly reduced REM latency in comparison to healthy controls (mean difference = –48.24 minutes, *p* = .044).

## Comparing Subjective Sleepiness Scores Between Groups

To assess if there were group differences in subjective sleepiness at habitual wake time between groups, a one-way ANOVA was conducted on KSS scores (Table 1). Assumption checks showed that the data met the assumption of homogeneity of variances, *F*(2, 30) = 1.77, *p* = .187, and the assumption of normality, *W* = 0.967, *p* = .395. The ANOVA revealed a medium non-significant effect of group, *F*(2, 30) = 1.90, *p* = .168, η² = 0.11 (Figure 1).

## Figure 1

*Non-significant Differences in Subjective Sleepiness Scores by Group*

A diagram of a group

Description automatically generated

*Note.* Horizontal lines represent median scores, while boxes show interquartile range (IQR). Whiskers extend to 1.5 times the IQR with outliers shown as dots.

## Comparing Objective Sleepiness Between Groups

To investigate if objective sleepiness upon awakening differed between groups, three one-way ANOVAs were conducted using a cluster mass permutation analysis (Figure 2). No significant cluster differences for AAC were detected between groups before or after correcting for multiple comparisons, smallest uncorrected *p*channel-value *F*(2) = 2.66 *p*uncorrected = .080, *p*fwe = .710. For SR in the eyes open condition, there were no significant cluster differences between groups before or after correcting for multiple comparisons, smallest uncorrected *p*channel-value *F*(2) = 1.45, *p*uncorrected = .252, *p*fwe = .826. For the SR in the eyes closed condition, there were also no significant cluster differences between groups before or after correcting for multiple comparisons, smallest uncorrected *p*channel-value *F*(2) = 1.15, *p*uncorrected = .334, *p*fwe = .939. These findings indicate there are no measurable differences in commonly used measures of EEG objective sleepiness between groups.

## Figure 2

*Non-Significant Differences in Objective Sleepiness EEG Measures by Group*

A diagram of different colored circles

Description automatically generated with medium confidence

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| ID | NRS | Control |  | F-Test |  | Global averages |

|  |  |  |
| --- | --- | --- |
| SR EC | SR EO | AAC |

*Note.* Topoplots represent group average AAC and SR values, and F-statistic values. No significant differences were observed between groups in any measures, as shown by F-test maps. All groups showed the greatest AAC values in the occipital regions, reflecting increased alpha activity when the eyes are closed, and therefore lower objective sleepiness. Blue areas indicate a prevalence of alpha activity when the eyes are open, or the reduction of alpha activity when the eyes are closed, and therefore higher objective sleepiness. Eyes-open SR topoplots indicate the greatest prevalence of slow frequency activity in the prefrontal regions across groups, while eyes-closed SR topoplots show the greatest prevalence of slow frequency activity in occipital regions. Box and whisker plots represent global AAC and SR values averaged across EEG channels per participant across groups.

**The Association Between Subjective and Objective Sleepiness Between Groups**

To investigate if there were differences in the association between subjective and objective sleepiness between groups, a general linear model with a cluster mass permutation analysis was conducted (Figure 3). For the AAC, no significant cluster differences were found for the main effect of KSS, smallest uncorrected *p*channel-value *T* = -1.94, *p*uncorrected = .064, *p*fwe = .595. Similarly, no significant cluster differences were observed for the main effect of group. One channel in the left temporal region was significant at the uncorrected level but was non-significant after correction for multiple comparisons, *F*(2)= 4.54, *p*uncorrected = .021, *p*fwe = .314. All other channels were non-significant at the uncorrected level. No significant interaction effects between groups were found for AAC. A cluster of 5 channels in the central cortical region was identified but did not reach significance, *F*(2) = 10.22, *p*cluster = .164.

For SR in the eyes-open condition, no significant cluster differences were observed for the main effect of KSS, smallest uncorrected *p*channel-value *T* = 2.01, *p*uncorrected = .056, *p*fwe = .402. No significant cluster differences were observed for the main effect of group, smallest uncorrected *p*channel-value *F*(2)= 1.63, *p*uncorrected = .211, *p*fwe = .830. There were no significant interaction clusters, smallest uncorrected *p*channel-value *F*(2) = 3.11, *p*uncorrected = .065, *p*fwe = .452.

Finally, for the SR in the eyes-closed condition, no significant cluster differences were found for the main effect of KSS. One channel in the left temporal region was significant at the uncorrected level but was non-significant after correction for multiple comparisons, *T* = -2.19, *p*uncorrected = .038, *p*fwe = .287. All other channels were non-significant at the uncorrected level. No significant differences were observed for the main effect of group, smallest uncorrected *p*channel-value *F*(2) = 1.16, *p*uncorrected = .327, *p*fwe = .938). No significant interaction clusters were identified, smallest uncorrected *p*channel-value *F*(2) = 2.09, *p*uncorrected = .145, *p*fwe = .685.

Overall, these results indicate that there were no significant associations between subjective sleepiness (KSS) and objective EEG measures across groups, and no significant group differences were found for the main or interaction effects of sleepiness.

**Figure 3**

*Non-Significant Differences in Objective Sleepiness by KSS Score and Group:KSS Interaction*

|  |  |  |
| --- | --- | --- |
| KSS t-test | KSS:Group | Global average regression |

A close-up of several colored lines

Description automatically generatedSeveral images of different types of data

Description automatically generated with medium confidence

|  |  |  |
| --- | --- | --- |
| SR EC | SR EO | AAC |

*Note.* Regression lines show global AAC and SR values averaged across all EEG channels per participant. **What would we expect from the graphs?** Orange dots show significant channels before correction for multiple comparisons.

# Discussion

This study examined if there were differences in subjective or objective measures of sleepiness, or the association between them both, in individuals with ID, NRS, and healthy controls. The hypothesis that subjective sleepiness upon awakening would be significantly higher in individuals with NRS and lower in ID in comparison to healthy controls was not supported. Furthermore, NRS was not associated with increased EEG measures of objective sleepiness, while ID was not associated with decreased measures of objective sleepiness in comparison to healthy controls. Finally, there were no significant differences in the association between subjective and objective sleepiness between groups, with subjective sleepiness and group membership not significantly predicting measures of objective sleepiness, and no significant interaction. These results suggest that although ID and NRS may have different underlying aetiology and causal mechanisms, **and be different to healthy controls,** we did not find group differences in subjective or objective measures of sleepiness upon awakening.

**Subjective Sleepiness**

Contrary to hypotheses, both ID and NRS groups exhibited non-significantly increased subjective sleepiness in comparison to healthy controls. **Medium effect?** This contrasts with prior research which consistently finds lower subjective sleepiness in ID populations [@cite]. Findings in NRS are mixed, with some studies finding increased subjective sleepiness in comparison to healthy controls [@cite] and some finding reduced subjective sleepiness [@cite]. As NRS is characterized by unrefreshing sleep, it was expected that they would show significantly increased sleepiness upon awakening in comparison to healthy controls. This non-significant finding is further supported by the non-significant group differences (ID-NRS) in trait sleepiness as measured by the ESS, indicating that there are no significant group differences in subjective sleepiness within the current sample. This finding suggests state subjective sleepiness upon awakening may not be a sensitive measure for distinguishing between NRS and ID.

Although we found a medium effect size (η² = 0.11) for subjective sleepiness, the KSS generally produces large effect sizes for clinical group differences. There are several potential explanations for this finding.

* Large degree of concordance in daytime impairments between ID and NRS: no significant difference in subjective fatigue measured by the FFS, or insomnia symptoms as measured by the ISI. However, there were group differences in sleep quality (PSQI) scores, with the ID group reporting lower subjective sleep quality than the NRS group. May suggest that daytime sleepiness and impairments are similar between groups.
* Could be due to low power, limited by small sample size and the timing of KSS administration immediately after awakening may not capture group differences.
* However, these findings suggest that there may be no difference in subjective sleepiness upon awakening between groups.

## Objective Sleepiness

The results of this study did not support the hypothesis that NRS would be associated with increased measures of objective sleepiness and ID would exhibit reduced objective sleepiness in comparison to healthy controls. Interestingly, we did not find increased fast-frequency activity in the ID group, contrary to previous studies which consistently report the finding of cognitive hyperarousal in ID in comparison to healthy controls. Furthermore, NRS was not increased with increased measures of objective sleepiness.

* Compare and contrast results to previous papers
* Implications – measuring by SR and AAC are not associated with group differences, indicating similar objective sleepiness.
* Reasons: no group differences in sleep architecture, which could then mean no group differences in objective sleepiness. Could be further explored by using KDT repeated throughout the day to see if there are differences in accumulation of sleep pressure, or comparing to previous night sleep and seeing if there are differences in SWA.
* Could be that SR is insufficient for capturing the hyperarousal present in ID, as it cannot capture differences in fast-frequency spectral bands. If ID has increased beta/gamma but reduced alpha, while others have increased alpha but reduced beta/gamma, this would not be captured.
* Overall implications: Limited by lack of specificity in spectral bands, however indicates there are no large group differences in objective sleepiness upon awakening (when there are no differences in PSG measured sleep)

A potential reason for this is there were no differences in PSG sleep architecture, indicating participants across groups may have had similar objective overnight sleep. If they had similar overnight sleep, and all groups experienced the same reduction in sleep pressure, EEG measures of sleepiness would be the same.

Another potential reason for this is that a single EEG recording taken upon awakening may not be powerful enough to detect group differences. A post-hoc sensitivity analysis conducted using G\*Power (Faul et al., 2007) found the study was sensitive to detecting a large effect size (Cohen’s f = 0.57) using an alpha level of .05 and a desired power of 80%. This suggests that while the study was underpowered to detect small to medium effect sizes, it was adequately powered to detect large effects. If there was a **clinically significant** difference, we would have found it (Discuss with reference to found effect sizes in literature). Other studies using the KDT finding a significant effect of group tend to use measurements taken multiple times, including before sleep and during the day.

Finally, participants may have been differentially affected by first-night effects. First night effects are where people sleep either better or worse on the first night in a clinical setting. For the ID group, they may have had better sleep than usual because of the loss of conditioning effects they experience at home in their normal bed. For the NRS and control groups, they may have slept worse than usual due to the unfamiliarity of the setting, noises, HD-EEG cap, etc etc.

## Association of Objective and Subjective Sleep Measures

This study did not find any significant associations between subjective sleepiness and objective sleepiness across groups. Furthermore, there were no group differences in objective sleepiness when including subjective sleepiness as a predictor. Finally, there were no significant clusters after controlling for multiple comparisons suggesting the association between subjective and objective measures of sleepiness do not vary between ID, NRS, and healthy controls.

Theoretical basis: KSS is a sensitive indicator of objective sleepiness in healthy controls, however these findings came from repeated measurements and is most accurate at high levels of sleepiness. ID is associated with sleep-state misperception, so may not be able to accurately assess their objective sleepiness. Unknown in NRS.

What does this mean: May mean there are no differences. More likely explanation: SR and AAC lose richness of data. If ID model of cortical hyperarousal is true, then would expect to see increased fast-frequency activity. However, two issues. 1. SR does not account for the different effects of alpha with increasing sleepiness depending on eyes open/eyes closed. 2. SR is too crude and loses richness of data.

What to do about limitation: analyse data in individual spectral bands to see what is associated with subjective sleepiness and if this differs between groups. Repeat rest over day.

Overall significance: The finding that subjective sleepiness and group did not act as predictors for objective sleepiness suggests there may not be group differences in how these clinical groups experience subjective/objective sleepiness, however needs better analysis.

## Strengths and Limitations

Strengths: age- and sex- matching, strict exclusion criteria.

Limitations: low power, could only detect for large effects. However, clinically significant differences would likely reveal large effects. Only tested for subjective sleepiness upon awakening, however this is when group differences reflecting overnight sleep are likely to be greatest.

## Practical implications and future directions

Why did we do this: to see if there are group differences in subjective/objective sleepiness upon awakening that could indicate different aetiology between ID/NRS in comparison to controls. Could help with understanding underlying neuropathology and assist in diagnosis, suggest an easier way to help with diagnosis than PSG.

What do our findings mean: preliminary findings suggest no group differences in sleepiness upon awakening. Could mean there are no differences, but could be obfuscated by no differences in PSG sleep.

Why do we care: NRS is associated with significant daytime impacts, similar impairment to ID, however not treated (only 1 out of 4 with NRS seek treatment) and no guidelines for objective diagnosis/treatment. Need to find if it is different to ID or if it should be treated the same. There are treatement guidelines for ID (CBT-I), however unknown if this will improve outcomes for NRS if they don’t meet ID DSM-5 criteria.

Future research: Future research analysing spectral power in each band, or across day, could provide greater understanding.

**Other notes, largely just a current dumping ground**

This contrasts to previous findings which support the hypothesis that subjective sleepiness is a sensitive and reliable predictor of objective sleepiness in healthy and clinical populations (Åkerstedt et al., 2014; Kaida et al., 2006; Shin et al., 2024) [@cite for clinical]. **However, these measurements were taken many times over the day and over several days.**

AAC and SR were not associated with subjective sleepiness within our sample. This may be because our sample was not excessively subjectively sleepy, and individuals cannot reliably predict sleepiness until they are already experiencing measures of sleep onset. Alternatively, this may be because although the measures were developed in order to aid with data analysis, they may lose the richness of EEG data and obfuscate differences in specific spectral bands.

* If there is cortical hyperarousal in ID, then it would not be captured by SR/AAC – because the differences are in faster frequency bands.
* Fatigue can be measured objectively by looking at theta. So there may be increased localised theta activity, that is being compensated for by increased fast frequency activity in beta/gamma bands. But if you lump all the fast frequencies together, you can’t see differences there.
* Additionally, SR does not account for the differential impact of alpha depending on eyes open vs eyes closed. As alpha activity increases with sleepiness in EO, it should probably be included with the slow frequency side of the equation. But, if this was the case, and SR was still a reliable measure, it would have been associated with KSS in EC and it wasn’t.
* If this finding is true, what does it mean for our understanding of ID and NRS?

## Strengths and Limitations

Power: A post-hoc power analysis using G\*power (Faul et al., 2007) determined that using a set alpha of .05 and the found effect size (η² = 0.11), the study achieved power of .39. However, the KSS generally produces large effect sizes for comparisons between clinical groups in conditions of **both acute and partial sleep deprivation** (Åkerstedt et al., 2014).

**Future thoughts**

* Why do we care about sleepiness? When fatigue is **RIGHT THERE**  and we can maybe measure that? Because we can mesasure that in the EEG and there amy be group differences in clinical disorders. If we look at theta only, that can also tell us about sleep homeostasis dysfunctions

## A post-hoc sensitivity analysis conducted using G\*Power (Faul et al., 2007) found the study was sensitive to detecting a large effect size (Cohen’s f = 0.57) using an alpha level of .05 and a desired power of 80%. This suggests that while the study was underpowered to detect small to medium effect sizes, it was adequately powered to detect large effe