Evidence of objective sleep impairment in non-epileptic attack disorder: A naturalistic prospective controlled study using actigraphy and daily sleep diaries over six nights.

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Highlights

- This prospective daily actigraphy/sleep diary study observed objective impairments in sleep in those with NEAD compared to normative controls.

- Sleep in the NEAD group was characterised by disrupted and inefficient sleep.

- Where poor sleep is reported, clinicians should carefully assess for factors that may be impacting sleep

Poor sleep is reported by many with non-epileptic attack disorder (NEAD) with correlations evident between self-reported sleep quality and mood and functional impairment. However, it is contended that self-reported sleep impairment in NEAD is a subjective phenomenon, which represents a general tendency to over-report symptoms or mis-interpret bodily states in those with NEAD. The present study was therefore designed to investigate the extent of subjective and objective sleep impairments in those with NEAD. Over six nights we prospectively recorded comparable nightly objective (actigraphy) and subjective (consensus sleep diary) sleep parameters in a sample of 17 people with NEAD, and an age and gender matched normative control group ($N = 20$). Participants recorded daily measures of attacks, dissociation and mood. Alongside higher subjective sleep impairment, the NEAD group had significantly worse objective sleep on several metrics compared to the normative controls, characterised by disrupted sleep (frequent awakenings and wake after sleep onset, low efficiency.) Exploratory analyses using mixed effects models showed that attacks were more likely to occur on days preceded by longer, more restful sleep. This study, which had good ecological validity, evidences the presence of objective sleep impairment in NEAD, suggesting that in patient reports of problems with sleep should be given careful consideration in clinical practice.

People with Nonepileptic attack disorder (NEAD) are a large proportion of referrals to neurology and neuropsychology services [1]. This condition is associated with significant distress and low quality of life [2]. While there is growing consensus that NEAD emerges from a heterogeneous number of risk and maintaining factors [3], there are few treatments with empirical support. Available treatments usually focus on the mental health aspects of NEAD: psychotherapies to address aberrant illness beliefs, trauma or psychological conflicts and medications to regulate mood [4-7].

Poor sleep is commonly reported in NEAD and is associated with functional impairment and mood disturbance [8-12]. Ostensibly, it follows that sleep interventions are worth consideration in NEAD management. Indeed, trials in other mental and physical health conditions show that mood and quality of life improve following cognitive behaviour therapy for insomnia [13]. It is also possible that sleep could affect NEAD symptoms directly. In other neuropsychiatric and physical health conditions, sleep correlates with symptom severity, including dissociation [14] [15, 16], and many NEAD risk factors - such as antecedent trauma, PTSD or mood disturbance – implicate sleep as a factor in the development and severity of symptoms [17, 18].

However, there is much to understand about the nature of sleep in NEAD before we consider how we might respond to reports of poor sleep in our patients. Most saliently, it is contended that reports of sleep problems in NEAD need to be interpreted with caution, because they simply reflect a general bias towards a mis-interpretation of somatic symptoms in those with NEAD, leading to over-reporting [19]. The only study in this area examined the relationship between self-reported sleep quality and objective sleep data (EEG and actigraphy) in a group of people with NEAD and a group with epilepsy [10] and found that those with NEAD had

significantly poorer scores on self-reported sleep parameters than those with epilepsy, but little difference on objective measures. This mirrors findings in studies assessing emotion perception in those with NEAD, where subjective reports of emotional experience differ from autonomic correlates [20, 21]. This finding was taken as further indication that self-reported sleep impairments in NEAD reflect a general deficit in interpreting bodily states, with sleep impairment being "more somatic than physiologic in PNES subjects."[10] p127. The implication one might take from this is that specific sleep interventions have little to add to commonly applied interventions (e.g. CBT, psychodynamic therapy) targeting the general misinterpretation of bodily states. However, methodological issues in the Latreille study [10] suggest that conclusion may be premature: First the comparator condition used in this previous study, epilepsy, is itself characterised by objective sleep impairments [22], which renders the lack of difference between groups difficult to interpret. Second, subjective and objective data were not recorded using comparable parameters: subjective data was gathered with retrospective trait-level questionnaires, objective sleep data were recorded prospectively on a daily basis. Third, the study occurred in an inpatient hospital setting - a synthetic environment where sleep may be atypical.

The present study primarily aimed to assess whether reported sleep disturbance in NEAD reflects objective sleep impairment. To overcome the limitations of the previous study, we measured sleep parameters prospectively over several days using comparable objective (actigraphy) and subjective (daily sleep diary) methods in a group of people with NEAD and an age and gender-matched normative control group without known neurological or sleep conditions, and in naturalistic settings. In addition, to begin to explore the relationship between sleep and attacks, we collected daily data on occurrence of attacks, dissociation and

mood to investigate the possibility that NEAD symptoms were worsened by poor sleep the night before.

Method

Design

A prospective six-night study of subjectively and objectively measured daily sleep variables, next day functioning and attack experiences in a cohort of people with NEAD and a normative control group.

Participants

NEAD sample

People diagnosed with NEAD were recruited from UK National Health Service (a free at the point of access public health service) neurology, neuropsychology and psychotherapy clinics. The diagnoses of epilepsy or NEAD was based on the interpretation of all available clinical data (e.g. most recent clinical assessments, alongside knowledge of the patient history, information from seizure witnesses, home video or video-EEG recordings when available) by a fully trained neurologist at a specialist epilepsy service in a UK regional neuroscience centre. While video-EEG confirmation was not available in all cases, the neurologists were sufficiently certain of all diagnoses exclusively to recommend treatment for one disorder (i.e. psychotherapy for NEAD, antiepileptic drug treatment for epilepsy). Patients in whom there was any clinical suspicion of a mixed seizure disorder were excluded. Additional inclusion criteria were: aged 18-70 years; under the clinical care of the recruitment centres; able to wear an Actiwatch and complete daily questionnaires. Exclusion criteria were: comorbid epilepsy; presence of a previously diagnosed sleep disorder (eg. obstructive sleep apnoea, REM and NREM parasomnias); inability to speak and/or read English; reporting risk of self-

harm or suicide; any physical or intellectual disability that would hinder their ability to give consent or to participate.

Control sample

Control participants were recruited from the University of Leeds's School of Psychology pool of volunteers' mailing list. The inclusion criteria for the control sample were: aged 18-70 years; able to wear an Actiwatch and complete daily questionnaires. Exclusion criteria were: diagnosis of a seizure or sleep disorder; inability to speak and/or read English; reporting risk of self-harm or suicide; any physical or intellectual disability that would hinder their ability to give consent or to participate. We aimed to match the control sample, at least on gender and age, to the NEAD sample.

Materials

Participants gave baseline information on demographics and details of attacks frequency (where appropriate), and completed a one-off battery of trait sleep and mood questionnaires, collected via online questionnaires. Then, for six days, they completed daily sleep diaries wore wrist-mounted Actiwatches and completed daily measures of mood, dissociation and attack frequency.

Baseline Measures

Pittsburgh Sleep Quality Index (PSQI)

The PSQI [23] is a widely-used 19 item measure of self-reported sleep. It includes seven domains; sleep latency (SL), total sleep time (TST), wakefulness after onset of sleep (WASO), sleep efficacy (SE), sleep disturbances (awakenings) and day-time functioning.

Dissociative Experience Scale-II (DES-II)

The DES-II [24] is a trait measure of dissociation. It contains 28 items regarding dissociative experiences measured on a scale of 0 to 100% and contains three sub-scales: amnesia, depersonalisation/derealisation and absorption.

Generalised Anxiety Disorder 7 (GAD7)

The GAD7 [25] is a 7-item questionnaire measuring the severity of anxiety. Scores range from 0 – 21, where a total score within the range of 10-14 suggests moderate anxiety and a score of 15 and above indicates severe anxiety.

The Patient Health Questionnaire 9 (PHQ 9)

The PHQ-9 [26] is a 9-item measure of depressive symptoms. Scores range from 0 – 27, and 10 and 15 and above represent the cut-offs for moderate and severe depression, respectively.

Prospective daily measures

Actigraphy

Sleep polysomnography is the gold standard way to measure objective sleep parameters, but is difficult to use outside of hospital/laboratory settings. Actigraphy measures such as the Actiwatch are often used as an alternative to enable research in naturalistic settings, where ecological validity is important (Lee, 2016). The Actiwatch is a wrist-worn activity monitor that measures levels of activity, wakefulness and sleep by continuously capturing the level of movement and body position that indicate waking and sleep cycles. It is considered a valid tool in the measurement of objective total sleep time and wakefulness after the onset of sleep in clinical populations [27]. We used the ActiGraph wGT3X-BT Actiwatch. The data

collected from the Actiwatch is analysed via ActiLife (version 6) software.

Consensus Sleep Diary (CSD)

The CSD [28] is a standardised daily dairy where participants record the time gone to bed, onset of sleep, total sleep time, sleep disturbed, total time awake due to disturbances, wakefulness after sleep and perceived quality of sleep. It also records naps during the day, the number of caffeinated drinks and medication. We asked the participants to diarise their sleep over six nights.

Daily measure of Dissociation & Mood

We could find no brief daily dissociation scales. We therefore developed a short measure using relevant items from existing measures. We used four items from the 56-item State Scale of Dissociation (SSD) [29]. The items are scored from "*Not at all*" to "*Very much so*". Based on clinical experience, we selected two depersonalisation and two derealisation items with good face validity and altered the wording to measure the state of dissociation daily as opposed to momentarily.

- "Today, things around me seemed unreal or dreamlike."
- "Things around me looked different today, from the way they usually do."
- "Today, my body has felt vague, indefinite, strange."
- "Today, my body seemed disconnected from my thoughts, my feelings, myself"

Daily measure of Mood

The international short-form measure for positive and negative affect (I-PANAS-SF) [30] measures daily mood. It consists of 10 items, measuring across five negative affects (upset, hostile, ashamed, nervous and afraid) and five positive affects (alert, inspired, determined,

attentive and active). The I-PANAS-SF has been used as a daily measure of mood and modified to ask, “indicate to what extent you felt this way during the day today”, on a 10-point scale from “very little” to “very much”[31]. For continuity between the daily measures in our study, the wording of the first point-scale to was changed to “not at all” and the last point-scale to “very much so”.

Daily Attacks Record

Participants with NEAD were asked to note the number of attacks that they experienced on each day of the study.

Procedure

NEAD sample

Potential participants were approached by clinicians in routine appointments. Those interested were subsequently interviewed by the lead researcher in a pre-participation meeting lasting from 30 to 60 minutes at their hospital. Inclusion/exclusion criteria were checked, consent obtained, and information regarding use of the Actiwatch and questionnaires given. Participants were asked to wear the Actiwatch continuously for a week (six nights) and only to remove this temporarily when taking a shower. Participants were also asked to complete the CSD upon waking and record number of attacks experienced that day and complete the dissociation and the mood scales before going to bed. Participants were encouraged to put reminders on their phone to complete their daily measures, or to keep their folder next to their beds as a reminder. After this week, participants were asked to attend a half hour post-participation meeting in which they returned the Actiwatch and the completed diaries and questionnaires. Participants were thanked for their participation and reimbursed

for their travel costs with a £25 shopping voucher. Ethical approval for this aspect of the research was given by NHS Research Ethics Committee (**IRAS project number: 239312**).

Control sample

All control participants were recruited from the University of Leeds's School of Psychology pool of volunteers' mailing list. A recruitment email was sent to the list, outlining the study and how to volunteer. In an attempt to match the demographics of our prospective NEAD population, in the first instance we sought out female participants over the age of 25, in line with the findings that NEAD was more likely to be diagnosed from the 20s onwards (Ettinger et al., 1999). To ensure that our sample would include a wide range of ages, including older adults, we also recruited from the University of Leeds's older adults' panel for volunteer research participants over the age of 60. The procedure was then the same as for those with NEAD, with the exception that the control sample were not asked about attacks occurrence, and all participation meetings occurred at the University. Additional Ethical approval for this aspect of the study was given by The School of Psychology at the University of Leeds (PSC-431).

Data Analysis

The data collected from the Actiwatch was processed using the ActiLife software. Data from this was extracted to a database containing all other collected data. Independent sample t-tests with Bonferroni Corrections were run in SPSS to explore between group differences, the p-values and Cohen's *d* statistic are reported. A mixed effects logistic model was used to assess associations between sleep variables and next day attack occurrence, and a mixed linear effect model to assess whether sleep affected next day dissociation and negative mood.

The random effect for participant identity was used since we have repeated measures over multiple days. Models were fit using the lme4 package within R.

Results

Samples and baseline characteristics

Twenty-seven people with NEAD were approached by their neurologist, from which 17 consented and entered into the study. In total, 20 control participants were recruited, and all completed the study- albeit an error in Actiwatch administration meant that objective sleep parameter datasets could not be retrieved for 5 participants (Table 1). The groups were matched on age (NEAD group, M = 38.6 [SD, 16.2]; Control group, M = 38.3 [SD, 11.9]) and gender (NEAD group,76.5%; Control group, 75%), although those in the control group were more frequently in full (NEAD group, 35.3%; Control group, 70%) and part-time (NEAD group,5%; Control group, 11.8%) employment. On average, those in the NEAD group (M=12.8, SD= 4.4) reported significantly poorer overall sleep than the control group (M= 4.5, SD= 2.8); t (26.7) =6.8, $p \leq 0.001$ and with a large effect size, d = .80 (Table 1). The NEAD group also reported higher anxiety; t (21.5) = 5.4, $p \leq 0.001$ with a large effect size, d = .76; depression t (22.5) = 7.2, $p \leq 0.001$, d = .84 and higher levels of dissociation than those in the control group (M=23.9, SD=20.3); t (20.2) = 4.7, $p \leq 0.001$, with a large effect size, d = .72. (Table 1) The number of attacks experienced by the NEAD group ranged from 0 (less than 1 a month) to 150 attacks a month, with a median (IQR) of 8 (18.75) attacks per month (Table 1).

<Table 1>

Prospective examination of subjective and objective sleep parameters

Table 2 shows the means for subjective (sleep diary) and objective (actigraphy) sleep parameters across the week for the NEAD (N = 17) and control (N = 15 for objective and

N=20 for subjective data) populations. Overall the NEAD group reported subjectively worse sleep than the control group, with small to moderate differences. After Bonferroni correction, significant between group differences were only apparent in the subjective data for Sleep quality (*M*= 2, *SD*=.90) (*M*=.65, *SD*=.59) t (33.2) = - 4.2, $p \leq 0.001$, with a medium effect size of d = .57.

Similarly, in the objective sleep data, worse sleep was apparent in the NEAD group on all parameters, with small to medium differences. After Bonferroni correction, the NEAD group scored significantly worse on several parameters: The NEAD group (*M*= 121.6, *SD*=60.6) experienced significantly more Wakefulness After Sleep Onset (WASO) than the control group (*M*= 61.4, *SD*=28.6); t (23.5) = 3.7, $p \leq 0.001$ with a medium effect size, d = .60 and experienced significantly poorer SE (*M*= 77.8, *SD*= 8.4) vs (*M*= 86.4, *SD*= 4.4); t (24.9) = - 3.7, $p \leq 0.001$ with a medium effect size, d = 60. The NEAD group (*M*= 21.2, *SD*=7.2) also experienced significantly more awakenings during sleep when compared to the control group (*M*= 11.8, *SD*=7.6); t (34.6) = 3.8, $p \leq 0.001$, with a medium effect size, d = .55. As can be seen in Figure 1, both groups tended greatly to underestimate the number of awakenings that objectively occurred during sleep.

<Table 2.> *<Figure 1. >*

Relationships between daily measures of sleep, attack occurrence, dissociation and mood

Throughout the week, the control group (*M*= 28, *SD*=9.3) on average experienced significantly more Positive Affect (PA) than the NEAD group (*M*= 18, *SD*= 11); t (31.) = - 2.9, $p \leq 0.01$ and with a medium effect size of d = .46. Additionally, the NEAD group (*M*= 3.8, *SD*=1.5) reported significantly more dissociation than the control group (*M*=1.2, *SD*=

.48); t (18.8) = 6.9, $p \leq 0.001$ with a large effect size of d = .85. On average, the NEAD group experienced attacks daily (M = 1.1, SD = 1.7).

<Table 3.> *<Table 4.>*

A multivariate linear mixed model with the whole sample showed no significant impact of sleep metrics on next day mood or dissociation (Table 3). However, NEAD diagnosis generally led to a higher negative mood score (c=3.18, CI=[-1.1, 7.8], p=0.19). Here also, a strong negative mood the preceding day led to better mood the following day – likely observed because of regression to the mean. A high dissociation score predicted an increased dissociation score in the subsequent day. Focusing in the NEAD sample alone (Table 4), using a mixed effects logistic regression model (Table 4.), increased hours of sleep (OR=2.78 per hour of sleep, CI=[1.2, 10.6], p<0.05) and decreased number of awakenings (OR=0.8 per awakening, CI=[0.6, 0.9], p<0.05) correlated with an increased the likelihood of an attack the following day. Given this surprising result, we ran a post-hoc model, to test the opposite direction of relationship - whether attacks adversely affected sleep. The correlations between seizures and a later shortening of sleep time (approximately 14 minutes, CI=[-0.9, 0.42], p=0.48), and between seizures and a later number of awakenings (-2.0 awakenings, CI=[-7, 3], p=0.44) were non-significant. Sleep in the preceding night showed no clear associations with dissociation or mood.

Discussion

The results suggest that reported sleep impairment in NEAD is often objective in nature, as opposed to simply reflecting a general tendency to misinterpret bodily state or to over report somatic symptoms. Here, data from objective tests of sleep show those with NEAD experienced significantly poorer sleep efficiency, more awakenings and more time awake

after sleep onset, with only sleep latency showing evidence of subjective overestimation in those with NEAD. Indeed, as in our control group, it was notable that those with NEAD tended greatly to underestimate the actual number of awakenings they had during sleep. The observed pattern of sleep impairment is commensurate with findings from actigraphy studies in fibromyalgia, which have also found restless sleep in this sample compared to 'healthy controls' [32, 33].

Our pattern of results suggests that NEAD-specific subjective misinterpretations of bodily states that have been observed in several studies of emotion perception [20, 21], may not generalise to a misinterpretation of sleep. These findings differ from those from the previous study [10] that concluded that reported sleep impairments in NEAD are subjective in nature. This may be because of the differences in methodology used. The previous study compared hospital samples of those with NEAD to those with epilepsy, and retrospective trait measures of subjective sleep with direct state measures of objective sleep [22]. In contrast, in the present study we compared those with NEAD to an age and gender matched sample from the general population. Further, prospective daily monitoring of comparable objective and subjective sleep metrics was undertaken, and the study took place in participant's homes over several days, improving ecological validity.

The present study was designed to explore whether reported sleep disturbance in NEAD is better considered objective or subjective and not the possible reasons for any observed sleep impairment. The sleep impairment in our NEAD sample could have been caused by many factors. First, sleep disorders may be part of the NEAD symptom constellation. The pattern of sleep impairment in our sample was characterised by disrupted and inefficient sleep, which coheres with an earlier EEG sleep study that found 27% of participants with NEAD showed

probable periodic limb movement disorder NEAD [11]. Second, poor sleep in NEAD could represent the impact of mood disturbance. Our NEAD sample did report higher levels of depression and anxiety, and other sleep-affecting mental health conditions like PTSD are more prevalent in NEAD [34]. However, one should be cautious about concluding that mental health conditions have primacy in their relationship with sleep. It is well-documented that mental health and sleep have a bi-directional relationship, where sleep can propagate problematic mood [35] and sleep interventions improve mental health symptoms in physical and mental health conditions [13, 36]. In common with clinical cohort studies [37], our sample were less frequently in employment than our control sample, and an inconsistent sleep or activity pattern could also affect sleep. Also, physical health comorbidities such as chronic pain and asthma can affect sleep and may be more prevalent in NEAD [38]. Medications for physical or mental health co-morbidities may also help explain differences.

The clinical implications of the present study are that clinicians should look beyond the idea that reports of poor sleep in their patients represents another manifestation of a misinterpretation of bodily state. Where poor sleep is reported, clinicians may want to carefully assess for whether any of the aforementioned factors may be contributing to sleep impairments in any given case. While there have been no studies of sleep treatments for NEAD, sleep management may be helpful: we know that better sleep in this sample is associated with improved well-being, and there is trial evidence supporting sleep interventions as a means to improving functioning, distress and even symptoms in other neurological [39, 40] and neuropsychiatric [36] conditions.

In additional exploratory analysis we assessed prospective associations between objective sleep parameters and attack occurrence or dissociation on the following day. A study with a

similar design with comparable conditions (chronic fatigue syndrome) found no relationship between actigraphy parameters and symptom severity [41]. However, our results showed a surprising pattern: attacks were more likely to happen following nights characterised by longer, less disrupted sleep. One possible explanation for this involves 'sleep pressure'. Poor sleep for several nights increases a homeostatic drive for sleep ('sleep pressure'), which leads to a night of excessive sleep. Here, a night of excessive sleep following a period of poor sleep may help mood recover, but cognitive functioning remains poor on subsequent days [42-44]. It is perhaps at this stage that a person is most vulnerable to attacks. Our post-hoc model suggested that this was unlikely to be explained by the opposite direction of relationship – that attacks cause poor sleep. Given that the present study was underpowered for these analyses, and this pattern of results was unexpected, this explanation should appropriately be considered speculative. Further studies with larger sample sizes are recommended to formally test these associations. To this end, the present study serves as proof of principle that a proportion of people with NEAD can engage well with these types of daily diary sleep studies, which come with high participant burden.

Limitations

The primary analyses were powered for moderate-to-large differences between groups; however, the exploratory analyses were underpowered and results of these should be interpreted with caution. We acknowledge that EEG monitoring of sleep in experimental conditions such as hospital wards yields more accurate metrics of objective sleep than actigraphy [27]. However, this is obtained at a cost of ecological validity - it is likely that hospital settings make it difficult to capture 'normal sleep'. In terms of our measures, due to difficulties finding a validated and practicable (i.e. brief) measure of daily dissociation, we developed a daily measure of dissociation for the present study. The psychometric properties

of this measure are unknown. Our NEAD sample was matched in demographics to our control sample. Our study was not designed to assess for or to imply a cause for sleep impairment in NEAD, only to assess the extent to which they are objective. Thus we didn't specifically aim to include or exclude those with several co-morbidities that might affect sleep (e.g. mental health, physical health etc). In addition, presence of a sleep disorder was ascertained on the basis of general neurological assessment and the clinician's knowledge of the patient. A specialist sleep assessment was not undertaken in order to exclude participants. Finally, not all NEAD diagnoses were made based on video-EEG recording of typical events. This is a limitation, as we cannot entirely rule-out the possibility that some individuals without NEAD were included. However, this limitation may enhance the generalisability of our findings to clinical practice, at least in the UK: even in specialist services, such as the recruiting site for this study, 40% of diagnoses of NEAD are made without video-EEG [4].

Conclusion

Data collected from objective sleep monitoring (via actigraphy) in conditions with ecological validity (own homes, own bed) suggests that subjective reports of sleep impairment in NEAD reflect objective sleep impairments. In comparison to an age and gender matched control group, those with NEAD experience inefficient sleep characterised by wakefulness after sleep onset and frequent awakenings. There are many possible reasons for sleep impairments in NEAD, and future research should examine these for prevalence and impact on sleep. In the meantime, since the presence of poor sleep may not simply represent somatic misinterpretation, clinicians may want to carefully assess reports of poor sleep by their patients. Prospective longitudinal exploratory analyses of sleep as a predictor of next day attack occurrence showed that attacks occurred more often on days following longer, less disturbed sleep; this warrants further investigation in larger studies.

Conflicts of Interest: Authors declare that they hold no conflicts of interest that affect the content of this article

Ethics statement: The study was approved by the appropriate ethics committee, in line with the Declaration of Helsinki.

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Table 1. Baseline Measures of Sleep, Dissociation, Mood and Attack Frequency

	CONTROL (MEAN, SD)	**NES (MEAN, SD)**	***P*-VALUE AND COHEN'S *D***
PSQI	4.5 (2.8)	12.8 (4.4)	p = 0.000 (d = 0.80) **
DES-II	23.9 (20.3)	86.6 (51.5)	p = 0.000 (d = 0.72) **
GAD7	3.7 (2.9)	12.7 (6.4)	p = 0.000 (d = 0.76) **
PHQ-9	3.2 (3)	14.9 (6.1)	p = 0.000 (d = 0.84) **
ATTACK FREQUENCY (PER MONTH)	Not applicable-	*8 (18.75)	

**Median, (IQR)*

Table 2. Subjective & Objective Outcomes of Daily Sleep.

	SUB[B] CONTROL (*MEAN, SD*)	**SUB NEAD (*MEAN, SD*)**	**SUB DIFFERNCE P-VALUE AND COHEN'S D**	**OB CONTROL(*MEAN, SD*)[A]**	**OB NEAD (*MEAN, SD*)**	**OB DIFFERNCE P-VALUE AND COHEN'S D**
SL[A] (MINS)	15. 3 (11.3)	51.3 (58.5)	*p =0.023 (d=.52)*	13.4 (10.2)	14.7 (23.3)	*p = 0.836 (d=.04)*
SE (%)	81.6 (9.9)	71.8 (14.7)	*p = 0.027 (d=.41)*	86.4 (4.4)	77.8 (8.4)	*p = 0.001 (d=.60)* **
TIB (MINS)	533.1 (53.4)	564.6 (114)	*p = 0.308 (d= .22)*	537.3 (61.7)	565.3 (112.3)	*p = 0.384 (d= .17)*
TST (MINS)	433.2 (57)	399.5 (105.6)	*p = 0.250 (d= .24)*	462.5 (47.9)	429 (59.2)	*p = 0.087 (d=.31)*
WASO (MINS)	99.9 (56.8)	165.1 (104.3)	*p = 0.030 (d= .43)*	61.4 (28.6)	121.6 (60.6)	*p = 0.001 (d= .60)* **
AWAKENINGS (FREQUENCY)	1.5 (.86)	2.7 (2.1)	*p = 0.042 (d=.43)*	11.8 (7.6)	21.2 (7.2)	*p < 0.001 (d=.55)* **
SQ (0-4)	0.65 (.59)	2 (.90)	*p = 0.000 (d=.57)* **	-	-	-

* $p \leq 0.01$, ** $p \leq 0.001$

[A]Objective sleep data for 15 participants, due to loss of data

SL = Sleep Latency, SE = Sleep Efficiency, TIB = Total Time in Bed, TST = Total Sleep Time, WASO = Wakefulness after onset of Sleep, SQ = Sleep Quality, SUB = Subjective Sleep Outcome, OB = Objective Sleep Outcome;

Figure 2. Objective-Subjective Awakenings in the NES and control groups over the 6 days/nights of study

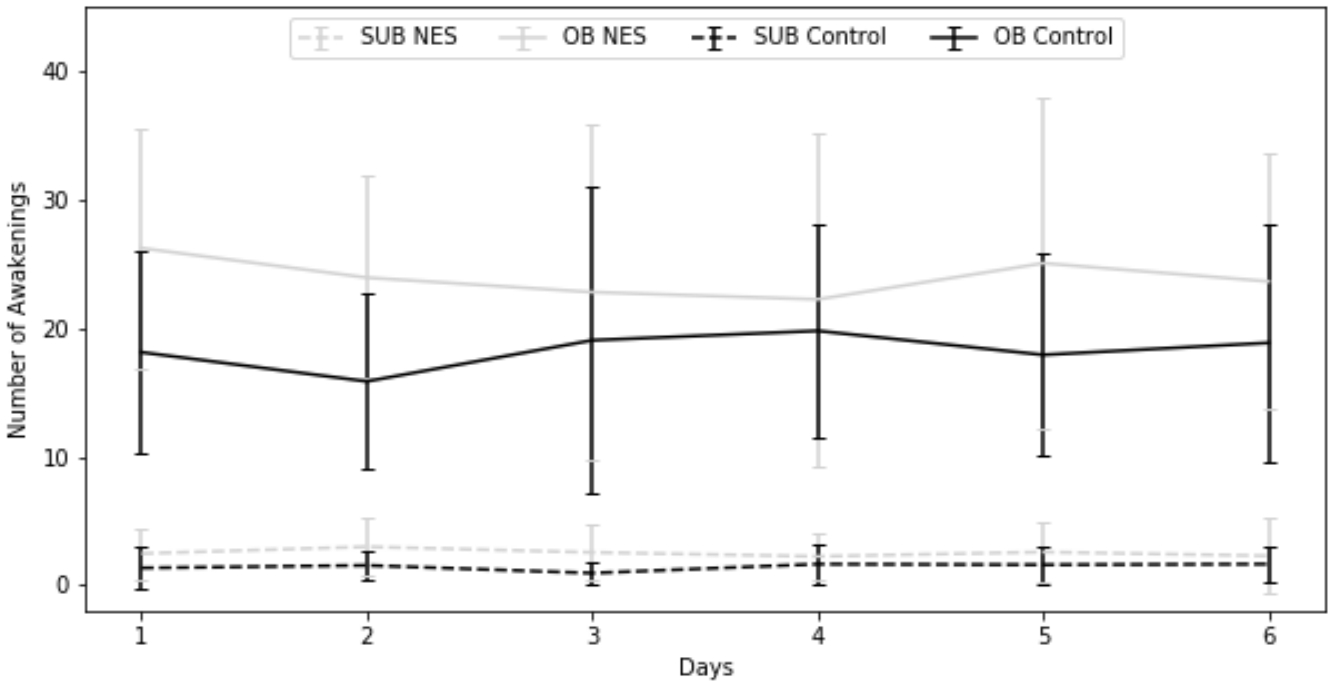

Table 3. Association of objective sleep metrics, mood and dissociation variables with next day mood and dissociation in the whole sample (NES and healthy controls combined, N = 32)

	Outcomes	
Independent Variables	Change in next day negative mood (Coef, 95% CI, p-value)	Next day disassociation (Coef, 95% CI, p-value)
Intercept	6.94, (-3.2, 14.6)	-0.24, (-2.1, 1.5)
Sleep time (hours)	-0.40, (-1.3, 0.5), 0.45	-0.01, (-0.2, 0.2), 0.91
Number awakenings	-0.06, (-0.2, 0.1), 0.47	-0.03, (-0.06, 0.0), 0.08
Disassociation	0.08, (-1.1, 0.8), 0.87	0.51, (0.3, 0.7), <0.001
Negative Mood	-0.67, (-0.9, -0.3), <0.001	0.02, (-0.0, 0.1), 0.29
NES	3.18, (-1.1, 7.8), 0.19	1.35, (0.6, 2.0), 0.008

Table 4. Association of objective sleep metrics, mood and dissociation variables with next day seizures, mood and dissociation in the NES sample alone (N = 17)

		Outcomes	
Independent Variables	Next day seizures (OR, 95% CI, p-value)	Change in next day negative mood (Coef, 95% CI, p-value)	Next day disassociation (Coef, 95% CI, p-value)
Intercept	0.02, (0.0, 4.6)	15.93, (4.4, 27.5)	3.35, (0.6, 6.1)
Sleep time (hours)	2.78, (1.2, 10.6), 0.05	-1.07, (-2.8, 0.6), 0.21	-0.10, (-0.5, 0.3), 0.63
Number awakenings	0.80, (0.6, 0.9), 0.02	-0.06, (-0.3, 0.2), 0.57	-0.03, (-0.1, 0.0), 0.20
Disassociation	1.13, (0.6, 2.1), 0.66	-0.47, (-1.6, 0.6), 0.41	0.44, (-0.1, 0.7), 0.002
Negative Mood	0.99, (0.8, 1.1), 0.84	-0.35, (-0.6, -0.1), 0.002	0.02, (-0.0, 0.1), 0.35