Subjective sleep-related variables in those who have and have not experienced sleep paralysis

Short title: Sleep-related variables predicting sleep paralysis

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Summary

Research suggests that poor sleep quality is related to the occurrence of sleep paralysis, although the precise relationship between these two variables is unknown. This association has generated interest due to the related possibility that improving sleep quality could help to combat episodes of sleep paralysis. To date, studies examining the association between sleep quality and sleep paralysis have typically measured sleep quality using general measures such as the global score of the Pittsburgh sleep quality index (PSQI). The aim of this study was to increase the precision of our understanding of the relationship between sleep paralysis and other aspects of sleep by investigating associations between different sleep-related variables and sleep paralysis. Using data from the G1219 twin/sibling study, analyses were performed on 862 individuals aged 22-32 (66% female). Results showed two components of the PSQI, sleep latency and daytime dysfunction, were predictors of sleep paralysis. In addition, a number of other sleep-related variables were significantly related to sleep paralysis. These were: insomnia symptoms, sleep problems commonly related to traumatic experiences, pre-sleep arousal, cognitions about sleep, and excessive daytime sleepiness. There was no relationship with sleep-disordered breathing, diurnal preference, or sleeping arrangements. Potential mechanisms underlying these results, and suggestions for future research are discussed.

Keywords

anomalous sleep experiences, disruptive nocturnal behaviours, parasomnia

Introduction

Sleep paralysis is a period of inability to perform voluntary movements at either sleep onset or upon awakening from sleep (American Academy of Sleep Medicine, 2014). Both laboratory studies and subjective self-reports have revealed a link between sleep quality and episodes of sleep paralysis (e.g. Denis et al., 2015; Takeuchi et al., 2002; for a review see Denis et al., 2017). Laboratory studies have shown that systematic disruption of rapid eye movement (REM) sleep is associated with increased frequency of sleep paralysis episodes. This work has characterised sleep paralysis as a continuation of REM muscle atonia (i.e. paralysis) into waking consciousness (Takeuchi et al., 2002; Takeuchi et al., 1992). Sleep paralysis commonly occurs in the context of narcolepsy (Andlauer et al., 2012; Dodet et al., 2015), though also occurs independently. When sleep paralysis occurs in individuals without narcolepsy, the term isolated sleep paralysis is preferred. As we were unable to definitively rule out a narcolepsy diagnosis, we refrain from using the term isolated sleep paralysis here. Studies using self-report measures have shown a link between poor sleep quality and both the occurrence and frequency of sleep paralysis (Denis et al., 2015; Denis and Poerio, 2017).

Utilising measures related to different aspects of sleep in a large sample, the aim of this work was to explore associations between sleep paralysis and specific aspects of sleep quality, with the aim of further refining our understanding of the link between sleep paralysis and sleep quality. To date, studies investigating these relationships have typically used global scores from measures such as the Pittsburgh sleep quality index (PSQI) (Buysse et al., 1989) and have used this score to show associations with sleep paralysis (e.g. Denis et al., 2015). The PSQI however also contains seven sub-scales tapping into different aspects of sleep. Therefore, the first aim of this study was to investigate which components of the PSQI are associated with the presence of sleep paralysis. As there is no existing work taking this approach, no specific hypotheses were formulated.

It was expected that insomnia symptoms would be linked to the presence of sleep paralysis, based on previous research findings (Ohayon et al., 1999; Szklo-Coxe et al., 2007). Disruptive nocturnal behaviours (such as nightmares, trouble sleeping due to general nervousness, and episodes of terror or screaming during sleep) are a subset of sleep disturbances that often show a heightened prevalence in post-traumatic stress disorder (PTSD) (Germain et al., 2005; Insana et al., 2013). As stress and trauma have been consistently linked to sleep paralysis (McNally and Clancy, 2005; Mellman et al., 2008), it was expected that stress-related sleep disturbances would be related to sleep paralysis (Denis et al., 2017). Sleep-disordered breathing was investigated based on a small body of evidence that patients with obstructive sleep apnea show a heightened prevalence rate of sleep paralysis compared to healthy controls (Vernet et al., 2011).

Higher levels of arousal in periods preceding sleep paralysis episodes compared to normal sleep have been found in experimental work on sleep paralysis (Takeuchi et al., 2002). Here we investigate for the first time in a large sample if self-reported pre-sleep arousal is linked to sleep paralysis. No previous studies have investigated the possible role of cognitions about sleep and this might provide important insights into treatment options for sleep paralysis. A cognitive behavioural therapy for sleep paralysis manual now exists, though systematic evidence for its effectiveness is lacking (Sharpless and Doghramji, 2015). Studies of CBT for insomnia have shown that dysfunctional beliefs about sleep can be modified and this is related to a reduction in insomnia symptom severity (Eidelman et al., 2016). If sleep paralysis is associated with higher dysfunctional beliefs about sleep, this may be an area that could be targeted in potential intervention strategies.

Excessive daytime sleepiness was evaluated based on previous studies reporting an association (Hsieh et al., 2010; Munezawa et al., 2011). The role of diurnal preference was investigated based on studies showing that certain genes involved in the variance of diurnal preference (Carpen et al., 2006; Lee et al., 2007; Parsons et al., 2014) may also be associated with sleep paralysis (Denis et al., 2015). Finally, possible effects of sleeping arrangements (room and bed sharing) were examined, as these have not been investigated before.

Method

Participants

The present analyses used data from wave 5 of the G1219 longitudinal twin study (McAdams et al., 2012), the only wave at which sleep paralysis has been assessed. At wave 5, research assistants attempted to trace those who participated at wave 4 and their siblings (N = 1817). Following tracing, all participants were sent a 12-page booklet to complete. A total of 883 were returned. The total sample included in these analyses was 860 individuals who had responded to the sleep paralysis item. The mean age was 25.30 years (SD = 1.81, range = 22-32 years) and 66% of the sample were female. The study received ethical approval from the Research Ethics Committee at Goldsmiths, University of London.

Measures

Sleep paralysis

Sleep paralysis was measured by a single item: ‘Sometimes, when falling asleep or waking up from sleep, I experience a brief period during which I feel I am unable to move, even though I think I am awake and conscious of my surroundings’. The label ‘sleep paralysis’ was not used, as labelling the experience can affect the reported prevalence rate (Fukuda, 1993). This item has been used in other work to provide a reliable estimate of sleep paralysis frequency (Cheyne et al., 1999).

Sleep quality

Sleep quality was assessed using the Pittsburgh sleep quality index (PSQI) (Buysse et al., 1989). This 18-item questionnaire assesses seven components of sleep quality and disturbances (sleep quality, sleep latency, sleep duration, sleep efficiency, sleep disruption, use of sleep medication, and daytime dysfunction). The global score has a theoretical range of 0-21, with a higher score indicating higher levels of sleep problems. The scale showed a reliability of α = 0.71 in the current sample. Each of the subscales have a theoretical range of 0-3 and have been shown to be valid and reliable measures in non-clinical populations (Mollayeva et al., 2016).

Insomnia symptoms

Insomnia symptoms were measured using 6 items from the insomnia symptoms questionnaire (Okun et al., 2009) (e.g. difficulty falling asleep; difficulty staying asleep). Items measure the frequency of insomnia symptoms occurring per week during the past month on a 5-point scale, ranging from never/don’t know (0) to always (4). The total scale score is the sum of these responses, ranging from 0-24. A higher score indicates more severe insomnia. The total scale demonstrated good internal consistency (α = .87).

Disruptive nocturnal behaviours

The PSQI addendum (PSQI-A) was used to measure disruptive nocturnal behaviours in the last month (Germain et al., 2005). The items assess sleep disturbances that are common in PTSD (disruptive nocturnal behaviours). Frequency of 7 disruptive nocturnal behaviours (e.g. memories or nightmares of a traumatic experience, trouble sleeping due to general nervousness and episodes of terror or screaming during sleep) was assessed on a four-point scale, ranging from not during the past month, to three or more times a week. The theoretical range was 0-21. A higher score indicates higher frequency of disruptive nocturnal behaviours. The scale showed a reliability of α = .73.

Sleep-disordered breathing

Three items were used to assess sleep-disordered breathing (Okun et al., unpublished). The items were 1) snorting or gasping during sleep, 2) loud snoring, and 3) your breathing stops or you choke or struggle for breath during sleep. Each item was assessed on a 5-point scale, ranging from never/don’t know (0) to always (4). This measure was analysed as a continuous scale, with a theoretical range between 0-12. Higher scores indicate more problems with sleep-disordered breathing. The internal reliability of the scale was acceptable (α = .69).

Cognitions about sleep

The dysfunctional beliefs and attitudes about sleep scale (DBAS) is a 10-item scale assessing sleep-disruptive cognitions such as faulty beliefs, worry, and attentional bias (Espie et al., 2000). For each item, participants are asked how strongly they agree/disagree with the statement on a scale of 1 (strongly disagree) to 10 (strongly agree). The theoretical range was between 10-100, with higher scores indicating higher levels of dysfunctional beliefs about sleep. The scale showed good internal reliability (α = .78).

Pre-sleep arousal

The pre-sleep arousal scale (PSAS) (Nicassio et al., 1985) is a 16-item questionnaire measuring symptoms of arousal experienced around bedtime. Items focused on both cognitive (8 items, e.g. intrusive thoughts) and somatic (8 items, e.g. sweating) arousal. Each item is answered on a five-point scale ranging from 1 (not at all) to 5 (extremely). A total score from 8 to 40 was computed for both the cognitive and somatic arousal sub-scales, with higher scores indicating greater arousal. Both the cognitive (α = .91) and somatic (α = .78) sub-scales showed good internal reliability.

Excessive daytime sleepiness

The Epworth sleepiness scale (ESS) is an 8-item questionnaire assessing the severity of daytime sleepiness (Johns, 1991). Participants are asked how likely they would be to fall asleep in a number of situations (e.g. whilst sitting and reading, and whilst sitting and talking to someone) on a four-point scale ranging from 0 (would never doze, i.e. fall asleep) to 3 (high chance of dozing). The scale has a theoretical range of 0 - 24, with higher scales indicating greater daytime sleepiness. The scale showed good internal reliability (α = .74).

Diurnal preference

The morning evening questionnaire (MEQ) was developed to assess degree of preference regarding morning and evening (Horne and Ostberg, 1976). The scale consists of 19 items. Fourteen items were answered on a four-point scale ranging from 1 to 4. The other 5 items were responded to with any hour of the day. Total score ranges from 16 – 86, with higher scores indicating a greater preference for mornings. The scale showed good internal reliability (α = .80).

Sleeping arrangements

Two questions tapped sleeping arrangements. Participants were asked *“Who sleeps in the same room as you?”*. Possible responses were: Nobody, partner, sibling, baby/child, and other. Due to low frequency of the “sibling”, “baby/child”, and “other” responses (n < 30), only the “nobody” and “partner” responses were analysed. The second question was *“Who sleeps in the same bed as you?”*, and possible responses were: Nobody, partner, baby/child, and other. Again, due to low frequency of “baby/child” and “other” responses (n < 20), only “nobody” and “partner” responses were analysed.

Statistical analysis

The sleep paralysis measure was positively skewed (skew = 1.93) and transforming the variable did not sufficiently improve the distribution. Due to this, and in order to keep our analysis consistent with previous work using this dataset (Denis et al., 2015), the variable was dichotomised into sleep paralysis absent = 0 (n = 605) and sleep paralysis present = 1 (n = 255). Participants who had experienced sleep paralysis at least once in their lives were categorised as sleep paralysis present. As such, all results reflect predictors of lifetime sleep paralysis occurrence, an approach that is commonly used in research on this topic (Denis et al., 2017). All analyses were performed on this dichotomised variable.

Independent samples t-tests were first used to explore associations between sleep paralysis and other sleep-related variables. Note that due to sleeping arrangements being frequency data, it was not possible to perform t-tests on this variable. Therefore, chi-square analysis was performed instead.

Next, multiple logistic regression was applied to ascertain significant predictors of sleep paralysis following adjustments. For each sleep-related variable, three regression models were used. In model 1, sleep paralysis presence was predicted from the sleep variable(s) of interest. In model 2, age and sex were added to the model. Finally, in model 3, global PSQI scores were added to control for the well documented effect of general sleep quality on sleep paralysis. Note that global PSQI was not added to the model using PSQI sub-components, as this would effectively lead to the same information being entered twice in the model.

All analyses were performed using Stata 9 (StataCorp, USA). As twin/sibling data were used in the analysis, the Stata command ‘cluster’ was used for the tests to be robust against the non-independence of observations found in a twin sample. The sleep-related variables assessed were: 1 – Sleep quality, as assessed by PSQI sub-components; 2 – Insomnia symptoms, as assessed by items from the ISQ; 3 – Disruptive nocturnal behaviours, as assessed by the PSQI-A; 4 – Sleep disordered breathing, as assessed by items from Okun et al., unpublished; 5 – Pre-sleep arousal, as assessed by the PSAS; 6 – Cognitions about sleep, as assessed by the DBAS; 7 – Excessive daytime sleepiness, as assessed by the ESS; 8 – Diurnal preference, as assessed by the MEQ; and 9 – Sleeping arrangements. To correct for multiple comparisons, a Bonferroni corrected *p* value of .006 (calculated as .05/9) was used when assessing for significant predictors. As Bonferroni corrections are sometimes considered to be overly conservative (Perneger, 1998), uncorrected values are also reported.

It is possible that only ever experiencing one episode of sleep paralysis would differ in terms of its predictors compared to experiencing multiple episodes of sleep paralysis. In order to examine this, analyses were also run on a reduced dataset that excluded individuals who reported only experiencing sleep paralysis once (therefore only examining those who reported multiple sleep paralysis episodes – at least several lifetime episodes). The results of these analyses are reported in the **supplementary materials**.

Results

Descriptive statistics

Of the sample, 29.7% (n = 255) reported experiencing sleep paralysis at least once in their lives. A smaller percentage, 7.9%, reported sleep paralysis several times a year. The distribution of sleep paralysis scores is displayed in **Figure 1**. Of note, this study uses a dataset that has been used in previous studies of sleep paralysis (Denis et al., 2015). Therefore, prevalence rates are the same as those reported previously.

Descriptive statistics for all variables are provided in **Table 1**. Independent samples t-tests showed all variables apart from use of sleeping medications to be significantly related to sleep paralysis in the expected direction. When participants who only experienced sleep paralysis once were excluded, all variables apart from use of sleeping medication and age were significantly related to sleep paralysis (see **Table S1**). Chi-square analysis of the sleeping arrangements variables suggested no association with sleep paralysis. This was also true when participants who experienced sleep paralysis once were excluded (see **Table S1**).

Sleep paralysis and other sleep-related variables

Sleep quality

A regression model predicting sleep paralysis presence from the seven PSQI sub-components simultaneously was performed. After controlling for age and sex (see model 2, **Table 2**), two components of sleep quality were found to independently predict sleep paralysis. These were sleep latency, odds ratio (OR) = 1.36, 95% confidence intervals (CI) = 1.12 – 1.66, *p* = .002; and daytime dysfunction, OR = 1.47, CI = 1.24 – 1.74, *p* < .001. Sleep disruption was significantly associated with sleep paralysis, although not after adjusting for multiple comparison corrections, OR = 1.28, CI = 1.06 = 1.54, *p* < .009. Sleep quality, sleep duration, sleep efficiency, and use of sleep medications were not significant predictors of sleep paralysis (all *p* > .10).

Other sleep-related variables

After age, sex, and global PSQI score had been controlled (see model 3 in **Table 2**), insomnia symptoms significantly predicted sleep paralysis, OR = 1.64, CI = 1.30 – 2.07, *p* < .001. Of note, in this model, global PSQI score *did not* significantly predict sleep paralysis, OR = 1.18, CI = 0.93 – 1.50. Disruptive nocturnal behaviours also significantly predicted sleep paralysis, OR = 1.60, CI = 1.32 – 1.91, *p* < .001 after adjusting for age, sex, and global PSQI score (model 3 in **Table 2**).Whilst sleep disordered breathing significantly predicted sleep paralysis, this association did not remain significant after adjusting for age, sex, and global PSQI score (model 3 in **Table 2**), OR = 1.16, CI = 0.98 – 1.38, *p* = .09.

After adjusting for age, sex, and global PSQI score (model 3 in **Table 2**), both cognitive, OR = 1.37, CI = 1.11 – 1.69, *p* = .004, and somatic, OR = 1.52, CI = 1.24 – 1.85, *p* < .001, pre-sleep arousal predicted sleep paralysis. Of note, in this model, global PSQI score was *not* a significant predictor of sleep paralysis, OR = 1.22, CI = 0.99 – 1.49, *p* = .051. This is of interest because in every other model bar insomnia, global PSQI score was a significant predictor (see **Table 2**). Dysfunctional beliefs about sleep were a significant predictor of sleep paralysis, OR = 1.35, CI = 1.15 – 1.58, *p* < 001 after age, sex, and global PSQI score had been controlled for (model 3 in **Table 2**).

Excessive daytime sleepiness was a significant predictor of sleep paralysis, OR = 1.37, CI = 1.17 – 1.61, *p* < .001, after adjusting for age, sex, and global PSQI score (model 3 in **Table 2**). Diurnal preference was *not* found to significantly predict sleep paralysis, OR = 0.86, CI = 0.73 – 1.01, *p* = .07 after adjusting for age, sex, and global PSQI score (model 3 in **Table 2**). Finally, sleeping arrangements did *not* predict sleep paralysis (room sharing: OR = 1.00, CI = 0.34 – 2.96, *p* = .99; bed sharing: OR = 1.03, CI = 0.36 – 2.99, *p* = .96) after adjusting for age, sex, and global PSQI (model 3 in **Table 2**).

When regression analyses were run excluding participants who only experienced sleep paralysis once, the same pattern of results was found. These results can be found in **Table S2**.

Discussion

The aim of this paper was to examine associations between different sleep-related variables and sleep paralysis. The results provide a number of unique and interesting insights into the relationship between sleep paralysis and other aspects of sleep assessed cross-sectionally.

After adjustments, two components of the PSQI were associated with sleep paralysis. Sleep latency here refers to the subjectively rated length of time it takes to fall asleep. As long sleep latency is a hallmark symptom insomnia, it is unsurprising that insomnia symptoms were also found to predict sleep paralysis in this study. Interestingly, in the model predicting sleep paralysis from insomnia symptoms, global PSQI score *did not* predict sleep paralysis independently of insomnia symptoms. This may suggest that symptoms of insomnia are a more specific predictor of sleep paralysis occurrence than are global PSQI scores.

The daytime dysfunction PSQI component was the other significant predictor of sleep paralysis. This item appears to tap into daytime sleepiness (Buysse et al., 2008). In our study, ESS score was a significant predictor of sleep paralysis. Taken together, these findings suggest excessive daytime sleepiness is associated with sleep paralysis, replicating previous findings (Hsieh et al., 2010; Munezawa et al., 2011).

The fact that disruptive nocturnal behaviours (which includes vivid nightmares and episodes of screaming and terror during sleep) were also associated with sleep paralysis experiences suggests that sleep paralysis does not occur in isolation, but can also co-occur with other ‘anomalous’ sleep experiences. This finding is consistent with other research showing that sleep paralysis is associated with a variety of other sleep experiences including exploding head syndrome and lucid dreaming (Denis and Poerio, 2017; Sharpless, 2015). It is possible that underlying sleep disruption could lead to a multitude of unusual experiences, as well as possibly being related to underlying shared genetic factors (Denis et al., 2017).

Pre-sleep arousal was shown to be related to sleep paralysis. Previous research has shown that both cognitive and somatic levels of pre-sleep arousal are linked to difficulties initiating and maintaining sleep (Harvey, 2000; Lichstein and Rosenthal, 1980; Tang and Harvey, 2004). This may in turn lead to an increased risk of sleep paralysis (Takeuchi et al., 2002). It has also been suggested that pre-sleep arousal acts as a mediator in the relationship between anxiety and sleep quality (Yeh et al., 2015). As anxiety levels are also known to be related to sleep paralysis (Denis et al., 2017), an interesting future study would be to investigate whether pre-sleep arousal mediates the relationship between anxiety and sleep paralysis.

Sleep paralysis was also related to beliefs individuals have about sleep. Negative cognitions about sleep may be associated with sleep paralysis in a circular relationship. A bout of sleep paralysis episodes may lead to increased worry and negative beliefs about sleep, which could lead to increased general anxiety, worry, and possibly an attempt to avoid going to sleep. As all of these individual factors are known to be related to sleep paralysis, it is possible that when an individual eventually sleeps their risk of sleep paralysis is increased further. This perspective is similar to that taken by models of insomnia which suggest a period of acute insomnia leads to dysfunctional beliefs about sleep, causing anxiety about sleep leading to sleep avoidance and a failure to exert control over sleep (Broomfield and Espie, 2005; Espie et al., 2006).

This finding suggests potential avenues for treating episodes of sleep paralysis. Cognitive behavioural therapy (CBT) is highly effective in treating insomnia (van Straten et al., 2017). A CBT for sleep paralysis manual now exists (CBT-ISP), though systematic testing of its effectiveness has yet to be performed (Sharpless and Doghramji, 2015). The current CBT-ISP manual already includes screening for insomnia at initial assessment, an approach supported by this research. In addition, the findings of this study suggest that dysfunctional beliefs about sleep, and levels of pre-sleep arousal, should also be assessed in the first CBT-ISP. If present, techniques to reduce these should be implemented in subsequent sessions. These factors have been shown to be effectively combated through a CBT approach (Trauer et al., 2015). Finally, the co-occurrence of other sleep disturbances, such as nightmares, should also be considered. When considering these proposals, the cross-sectional nature of the study should be kept in mind.

There are a number of limitations that must be considered. First, the study was largely exploratory in nature, capitalising on a large number of variables available. As such, independent replication of these findings is paramount. Second, the cross-sectional nature of the design means that it is not possible to assess the direction of the relationships reported. For example, whilst factors such as pre-sleep arousal and faulty cognitions about sleep predicted sleep paralysis, it was impossible to ascertain whether they represent a causal factor in sleep paralysis, arise as a consequence of experiencing sleep paralysis, or were associated for other reasons. Future longitudinal designs are needed to elucidate the nature of these relationships. Only subjective sleep-related variables were assessed here. Whilst this allowed for a large sample size, it is known that subjective and objective measures of sleep do not always align (Zhang and Zhao, 2007). Polysomnography (PSG) is considered the gold standard in assessing sleep, however its increased precision comes at the cost of smaller sample sizes. Whilst laboratory investigations of sleep paralysis have been conducted, they are limited by the relative rarity of sleep paralysis episodes, making it hard to directly study in a laboratory environment (Takeuchi et al., 2002; Takeuchi et al., 1992; Walther and Schulz, 2004). Finally, a dichotomous variable to measure sleep paralysis was used for statistical reasons. However, this approach loses detail. It is questionable whether a person who experiences sleep paralysis only a few times in their life would have the same risk factors for experiencing episodes as someone who experiences sleep paralysis almost every night. The accuracy of recalling episodes that may have happened a long time ago should also be considered, and the use of prospective measures of sleep paralysis should be more readily used in future studies.

Despite these concerns, this study makes an important contribution to the limited literature on sleep paralysis. We have shown that a complex relationship exists between sleep paralysis and sleep more generally. For example, results suggesting that other disruptive sleep behaviours co-occur with sleep paralysis suggest that this needs to be considered when assessing the clinical impact of sleep paralysis on an individual’s life. Furthermore, we showed significant associations between sleep paralysis and insomnia symptoms, pre-sleep arousal, and dysfunctional beliefs about sleep. These factors have all been shown to improve with CBT, suggesting possible future intervention strategies for reducing episode frequency in severe cases.

Author contribution

DD and AMG designed the study. DD analysed the data. All authors were involved in the preparation of the manuscript.

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*Table 1 – Descriptive statistics for variables included in the study*

|  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | Theoretical range | Overall | | Sleep paralysis present | | Sleep paralysis absent | | Sig |
| Variable |  | Mean | SD | Mean | SD | Mean | SD | *p* |
| Sleep paralysis | 1 – 7 | 1.65 | 1.16 | - | - | - | - | - |
| Age | 22 – 32 ± | 25.30 | 1.81 | 25.03 | 1.62 | 25.41 | 1.87 | \*\* |
|  |  |  |  |  |  |  |  |  |
| PSQI global | 0 – 21 | 5.38 | 2.87 | 6.47 | 2.98 | 4.92 | 2.70 | \*\*\* |
|  |  |  |  |  |  |  |  |  |
| Sleep quality |  |  |  |  |  |  |  |  |
| PSQI sleep quality | 0 – 3 | 0.98 | 0.65 | 1.13 | 0.66 | 0.92 | 0.64 | \*\*\* |
| PSQI sleep latency | 0 – 3 | 1.15 | 0.83 | 1.40 | 0.89 | 1.05 | 0.79 | \*\*\* |
| PSQI sleep duration | 0 – 3 | 0.56 | 0.64 | 0.66 | 0.66 | 0.52 | 0.64 | \*\* |
| PSQI sleep efficiency | 0 – 3 | 0.48 | 0.80 | 0.62 | 0.88 | 0.41 | 0.75 | \*\*\* |
| PSQI sleep disruption | 0 – 3 | 1.16 | 0.51 | 1.31 | 0.52 | 1.10 | 0.49 | \*\*\* |
| PSQI use of sleeping medications | 0 – 3 | 0.15 | 0.54 | 0.20 | 0.61 | 0.13 | 0.51 | ns |
| PSQI daytime dysfunction | 0 – 3 | 0.89 | 0.68 | 1.12 | 0.66 | 0.79 | 0.67 | \*\*\* |
|  |  |  |  |  |  |  |  |  |
| Insomnia symptoms |  |  |  |  |  |  |  |  |
| ISQ | 0 – 24 | 6.48 | 5.22 | 8.71 | 3.23 | 5.54 | 4.83 | \*\*\* |
|  |  |  |  |  |  |  |  |  |
| Disturbed nocturnal behaviours |  |  |  |  |  |  |  |  |
| PSQI-A | 0 – 21 | 2.08 | 2.65 | 3.23 | 3.36 | 1.59 | 2.11 | \*\*\* |
|  |  |  |  |  |  |  |  |  |
| Sleep-disordered breathing |  |  |  |  |  |  |  |  |
| Sleep-disordered breathing items | 0 – 12 | 0.54 | 1.36 | 0.43 | 1.24 | 0.77 | 1.58 | \*\*\* |
|  |  |  |  |  |  |  |  |  |
| Pre-sleep arousal |  |  |  |  |  |  |  |  |
| PSAS cognitive | 8 – 40 | 17.09 | 6.76 | 20.24 | 7.42 | 15.76 | 6.00 | \*\*\* |
| PSAS somatic | 8 – 40 | 11.30 | 3.84 | 13.17 | 4.64 | 10.51 | 3.13 | \*\*\* |
|  |  |  |  |  |  |  |  |  |
| Cognitions about sleep |  |  |  |  |  |  |  |  |
| DBAS | 10 – 100 | 50.26 | 15.25 | 54.67 | 14.47 | 48.39 | 15.19 | \*\*\* |
|  |  |  |  |  |  |  |  |  |
| Excessive daytime sleepiness |  |  |  |  |  |  |  |  |
| ESS | 0 – 24 | 5.52 | 3.66 | 6.44 | 3.77 | 5.14 | 3.55 | \*\*\* |
|  |  |  |  |  |  |  |  |  |
| Diurnal preference |  |  |  |  |  |  |  |  |
| MEQ | 16 – 86 | 52.22 | 8.44 | 50.73 | 8.75 | 52.86 | 8.24 | \*\*\* |
|  |  |  |  |  |  |  |  |  |
| Sleeping arrangements |  |  |  |  |  |  |  |  |
| Same room: Nobody | N/A | 3471 |  | 1111 |  | 2361 |  | ns2 |
| Same room: Partner | N/A | 4451 |  | 1281 |  | 3171 |  | ns2 |
| Same bed: Nobody | N/A | 3511 |  | 1071 |  | 2441 |  | ns2 |
| Same bed: Partner | N/A | 4771 |  | 1371 |  | 3401 |  | ns2 |

*Note*. SD = standard deviation, sig = independent samples t-tests (chi-square where appropriate) between sleep paralysis present and sleep paralysis absent significance level, \*\* = *p* < .01, \*\*\* = *p* < .001. ± = denotes actual range, not theoretical.

1 Count data representing response frequency to each sleeping arrangement option. Counts don’t add up to full sample size due to either missing data, or response option that was not included due to low n. See methods for full details.

2 Significance testing performed using chi-2 due to count data.

PSQI = Pittsburgh sleep quality index. A higher score indicates greater sleep problems.

ISQ = insomnia symptoms questionnaire. A higher score indicates greater problems with insomnia.

PSQI-A = Pittsburgh sleep quality index – addendum. A higher score indicates greater frequency of disruptive nocturnal behaviours.

Sleep-disordered breathing items. A higher score indicates greater problems with sleep-disordered breathing.

DBAS = dysfunctional beliefs about sleep scale. A higher score indicates greater level of dysfunctional beliefs about sleep.

PSAS = pre-sleep arousal scale. A higher score indicates greater pre-sleep arousal.

ESS = Epworth sleepiness scale. A higher score indicates higher levels of daytime sleepiness.

MEQ = morning evening questionnaire. A higher score indicates a greater preference for mornings.

*Table 2. Logistic regression models testing the association between sleep-related variables and the presence of sleep paralysis*

|  |  |  |  |
| --- | --- | --- | --- |
| Independent variable  [predicting the dependent variable sleep paralysis] | Model Information | | |
|  | Model 1 | Model 2 | Model 3 |
| Sleep quality [all PSQI components run together in the same models] | χ2 (df = 7) = 62.41, *p* < .001 | χ2 (df = 9) = 68.30, *p* < .001 | - |
|  |  |  |  |
|  | OR (95% CI) | OR (95% CI) | - |
| Age |  | 0.87 (0.80 – 0.96) \*\* | - |
| Sex |  | 1.02 (0.71 – 1.47) | - |
| PSQI sleep quality | 0.96 (0.78 – 1.17) | 0.96 (0.78 – 1.18) | - |
| PSQI sleep latency | 1.39 (1.15 – 1.70) \*\*\* | 1.36 (1.12 – 1.66) \*\* | - |
| PSQI sleep duration | 1.09 (0.89 – 1.34) | 1.11 (0.91 – 1.36) | - |
| PSQI sleep efficiency | 0.94 (0.76 – 1.17) | 0.93 (0.75 – 1.15) | - |
| PSQI sleep disruption | 1.27 (1.06 – 1.53) | 1.28 (1.06 – 1.54) ± | - |
| PSQI use of sleeping medications | 1.07 (0.91 – 1.26) | 1.06 (0.90 – 1.26) | - |
| PSQI daytime dysfunction | 1.44 (1.22 – 1.71) \*\*\* | 1.47 (1.24 – 1.74) \*\*\* | - |
|  |  |  |  |
| Insomnia | χ2 (df = 1) = 57.80, *p* < .001 | χ2 (df = 3) = 67.45, *p* < .001 | χ2 (df = 3) = 67.45, *p* < .001 |
|  |  |  |  |
|  | OR (95% CI) | OR (95% CI) | OR (95% CI) |
| Age |  | 0.88 (0.80 – 0.96) \*\* | 0.88 (0.80 – 0.96) \*\* |
| Sex |  | 0.90 (0.63 – 1.27) | 0.90 (0.63 – 1.27) |
| PSQI global |  |  | 1.18 (0.93 – 1.50) |
| ISQ | 1.83 (1.57 – 2.14) \*\*\* | 1.84 (1.58 – 2.15) \*\*\* | 1.64 (1.30 – 2.07) \*\*\* |
|  |  |  |  |
| Disruptive nocturnal behaviours | χ2 (df = 1) = 52.67, *p* < .001 | χ2 (df = 3) = 59.11, *p* < .001 | χ2 (df = 4) = 62.30, *p* < .001 |
|  |  |  |  |
|  | OR (95% CI) | OR (95% CI) | OR (95% CI) |
| Age |  | 0.90 (0.83 – 0.98) ± | 0.89 (0.82 – 0.97) ± |
| Sex |  | 0.89 (0.64 – 1.24) | 0.92 (0.65 – 1.29) |
| PSQI global |  |  | 1.39 (1.17 – 1.66) \*\*\* |
| PSQI-A | 1.83 (1.56 – 2.16) \*\*\* | 1.83 (1.55 – 2.16) \*\*\* | 1.60 (1.34 – 1.91) \*\*\* |
|  |  |  |  |
| Sleep-disordered breathing | χ2 (df = 1) = 15.28, *p* < .01 | χ2 (df = 3) = 22.08, *p* < .01 | χ2 (df = 4) = 51.50, *p* < .001 |
|  |  |  |  |
|  | OR (95% CI) | OR (95% CI) | OR (95% CI) |
| Age |  | 0.89 (0.82 – 0.97) \*\* | 0.88 (0.80 – 0.96) \*\* |
| Sex |  | 1.098 (0.77 – 1.51) | 1.06 (0.75 – 1.50) |
| PSQI global |  |  | 1.64 (1.39 – 1.94) \*\*\* |
| Sleep-disordered breathing items | 1.25 (1.06 – 1.49) \*\* | 1.25 (1.06 – 1.49) ± | 1.16 (0.98 – 1.38) |
|  |  |  |  |
| Pre-sleep arousal | χ2 (df = 2) = 84.49, *p* < .001 | χ2 (df = 4) = 88.03, *p* < .001 | χ2 (df = 5) = 86.07, *p* < .001 |
|  |  |  |  |
|  | OR (95% CI) | OR (95% CI) | OR (95% CI) |
| Age |  | 0.91 (0.83 – 0.99) ± | 0.90 (0.82 – 0.98) ± |
| Sex |  | 0.89 (0.64 – 1.25) | 0.93 (0.66 – 1.31) |
| PSQI global |  |  | 1.22 (0.99 – 1.49) |
| PSAS cognitive | 1.48 (1.22 – 1.79) \*\*\* | 1.47 (1.21 – 1.78) \*\*\* | 1.37 (1.11 – 1.69) \*\* |
| PSAS somatic | 1.56 (1.28 – 1.90) \*\*\* | 1.56 (1.28 – 1.91) \*\*\* | 1.52 (1.24 – 1.85) \*\*\* |
|  |  |  |  |
| Cognitions about sleep | χ2 (df = 1) = 31.96, *p* < .001 | χ2 (df = 3) = 39.86, *p* < .001 | χ2 (df = 4) = 59.40, *p* < .001 |
|  |  |  |  |
|  | OR (95% CI) | OR (95% CI) | OR (95% CI) |
| Age |  | 0.88 (0.81 – 0.96) \*\* | 0.88 (0.80 – 0.96) \*\* |
| Sex |  | 0.89 (0.64 – 1.25) | 0.92 (0.65 – 1.30) |
| PSQI global |  |  | 1.56 (1.31 – 1.85) \*\*\* |
| DBAS | 1.53 (1.32 – 1.78) \*\*\* | 1.54 (1.33 – 1.79) \*\*\* | 1.35 (1.15 – 1.58) \*\*\* |
|  |  |  |  |
| Excessive daytime sleepiness | χ2 (df = 1) = 21.52, *p* < .001 | χ2 (df = 3) = 30.70, *p* < .001 | χ2 (df = 4) = 58.10, *p* < .001 |
|  |  |  |  |
|  | OR (95% CI) | OR (95% CI) | OR (95% CI) |
| Age |  | 0.87 (0.80 – 0.95) \*\* | 0.87 (0.80 – 0.95) \*\* |
| Sex |  | 0.92 (0.66 – 1.28) | 0.93 (0.66 – 1.31) |
| PSQI global |  |  | 1.62 (1.37 – 1.92) \*\*\* |
| ESS | 1.41 (1.22 – 1.64) \*\*\* | 1.44 (1.24 – 1.67) \*\*\* | 1.37 (1.17 – 1.62) \*\*\* |
|  |  |  |  |
| Diurnal preference | χ2 (df = 1) = 11.10, *p* < .01 | χ2 (df = 3) = 19.15, *p* < .01 | χ2 (df = 4) = 53.35, *p* < .001 |
|  |  |  |  |
|  | OR (95% CI) | OR (95% CI) | OR (95% CI) |
| Age |  | 0.88 (0.82 – 0.97) ± | 0.88 (0.81 – 0.96) \*\* |
| Sex |  | 1.12 (0.81 – 1.57) | 1.08 (0.76 – 1.53) |
| PSQI global |  |  | 1.65 (1.40 – 1.96) \*\* |
| MEQ | 0.78 (0.67 – 0.90) \*\* | 0.77 (0.66 – 0.90) \*\* | 0.86 (0.73 – 1.01) |
|  |  |  |  |
| Sleeping arrangements | χ2 (df = 2) = 0.48, *p* = .78 | χ2 (df = 4) = 6.91, *p* = .14 | χ2 (df = 5) = 42.40, *p* < .001 |
|  |  |  |  |
|  | OR (95% CI) | OR (95% CI) | OR (95% CI) |
| Age |  | 0.89 (0.81 – 0.97) ± | 0.89 (0.81 – 0.97) ± |
| Sex |  | 1.04 (0.74 – 1.46) | 1.03 (0.72 – 1.46) |
| PSQI global |  |  | 1.20 (1.13 – 1.28) \*\*\* |
| Same room | 0.81 (0.20 – 3.30) | 0.89 (0.22 – 3.61) | 1.14 (0.24 – 3.80) |
| Same bed | 1.12 (0.35 – 0.57) | 1.07 (0.27 – 4.32) | 0.90 (0.27 – 2.95) |

*Note. \*\*\* = p <* .001, *\*\** = *p* < .01, ± = *p* < .05, not significant after Bonferroni correction. OR = Odds ratio, CI = confidence interval. Sleep paralysis presence/absence is the dependent variable in all models. Model 1 shows results before age, sex, and PSQI global score have been controlled for. In model 2, age and sex are controlled for and in Model 3, PSQI global score was also controlled for where appropriate (all models except sleep quality).

PSQI = Pittsburgh sleep quality index

ISQ = insomnia symptoms questionnaire

PSQI-A = Pittsburgh sleep quality index – addendum

PSAS = pre-sleep arousal scale

DBAS = dysfunctional beliefs about sleep scale

ESS = Epworth sleepiness scale

MEQ = morning evening questionnaire