Method Here we use data from the UK Biobank (N=5505, aged from 45 to 73) to elucidate the effect of poor sleep (insomnia, snoring, daytime sleepiness and short sleep duration) on WMH load. The sleep variables were obtained using a digital questionnaire, whereas the WMH load was derived from automated segmentation of T2 FLAIR magnetic resonance images using the BIANCA tool in FSL.

Results We show that age, snoring and daytime sleepiness significantly predict a higher WMH load (linear model, adjusted R²=0.13, p<0.0001). The WMH load of patients with potential sleep issues is significantly larger than those who reported no sleep issue (figure 1). Markers of poor sleep are associated with a higher body mass index (BMI) (linear model, adjusted R²=0.041, p<0.0001). A small but significant relationship exists between age, BMI and WMH (linear model, adjusted R²=0.14, p<0.0001).

Finally, a sleep burden score summing poor sleep markers significantly predicted the WMH load, when controlling for cardiovascular factors (table 1). Removing the sleep burden score leads to a significant decrease in the power of the model (ANOVA, p=0.027).

Discussion This exploratory analysis confirms the impact of measures of poor sleep on cerebrovascular health, proposing a complex relationship between sleep and WMH loads involving cardiovascular features in a large ageing population. Further work will examine the wider implications of measures of poor sleep on cognition and brain function.

REFERENCES
Results

Median (IQR) sleep duration was 8 hrs (7.5, 8.9) at baseline. The% TEI from each macronutrient (fat/carbohydrate/protein) at 0, 6 and 12 months was 35/42/18%, 34/43/18% and 33/44/19%. Substituting 1% TEI from protein or fat with carbohydrate was associated with 2.6 minutes shorter sleep cross-sectionally at 12-months but not at baseline or 6 months (figure 1). Higher carbohydrate intake (in place of protein or fat) was associated with a decrease in sleep duration by 1.5–2 minutes from 0–6 and 6–12 months. All longitudinal and cross-sectional macronutrient specific substitution models (but 12 months cross-sectional) suggested replacing protein with carbohydrate results in more sleep duration reduction (about one minute) than replacing fat with carbohydrate.

Discussion

Lower protein and higher carbohydrate intake are consistently associated with shorter sleep duration. Replacing 5% protein with 5% carbohydrate results in 10–15 minutes less sleep duration 6 months later.

REFERENCE


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Figure 1

Cross-Sectional and longitudinal association between carbohydrate intake (Explanatory variable) and sleep duration (Outcome variable)

Black lines represent the overall substitution of fat or protein with CHO
Red lines represent the substitution of protein with CHO
Yellow lines represent the substitution of fat with CHO

Introduction

Circadian rhythmicity is tightly regulated by clock genes. Polymorphic variations of the human PER3 gene have been shown to influence diurnal preference, sleep homeostasis and the development of mood disorders. However, it is unclear whether polymorphisms in this gene are associated with sleep duration and depression symptoms in real-life scenarios. We have investigated the relationship between sleep duration and mood traits among PER3 genotype groups in a Brazilian family-based cohort.

Methods

Baependi is a small rural town in Brazil that provides an opportunity to study the influence of sleep circadian patterns in a highly admixed rural population. We studied 1,100 subjects (mean age±SD 47.6±15.3, 42% male) and evaluated the effects of the coding-region variable number tandem repeat polymorphism in PER3 (rs57875989) on mood (Hospital Anxiety and Depression Scale) and self-reported sleep habits (Pittsburgh Sleep Quality Index), controlling for age and sex, using a general linear model.

Results

Our genotyping data showed that 33.5% of the subjects were homozygous for the shorter PER3 allele (4/4), 55.3% of participants were heterozygous (4/5) and 11.2% of subjects were homozygous for the longer allele (5/5). A main effect of PER3 genotype, sex and age was observed (p<0.01) on depression symptoms, as well as a significant interaction between sex and sleep duration ≤ 7 hours. The results revealed an increase in depression symptoms in the PER3(4/4) genotype compared to PER3(4/5). It was observed also an increase in depression symptoms according to the increase of age. The depression traits were also increased in women that have reported reduced sleep duration, despite controlling for age.

Discussion

We observed associations between PER3 genotype, sleep duration and depression symptoms, suggesting that PER3 gene may be behaviourally relevant for mental health, especially for women under chronic sleep loss conditions.

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PER3 POLYMORPHISM, SLEEP DURATION AND DEPRESSION SYMPTOMS IN A BRAZILIAN FAMILY-BASED COHORT, THE BAEPENDI HEART STUDY

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