

Despite its significance, key predictors – particularly obstetric, sleep-related and social support factors – have rarely been studied in large population-based cohorts.

**Methods** We assessed associations between obstetric factors (e.g., gestational diabetes, multiple pregnancy, preterm birth, labour and postnatal complications), subjective sleep problems (insomnia, short sleep duration, difficulty getting up in the morning) and social support (loneliness, frequency of visits, and confiding in friends/family) with the probability of PND in up to 27,519 UK Biobank participants who had previously given birth.

Both a lifetime history approach and an analysis of linked International Classification of Diseases codes in the periods before and after first birth were used to identify PND cases. Individual predictors were first examined using separate logistic regression models, then included together in penalised regression (elastic net) models to identify the most informative predictors and evaluate their combined predictive performance. Models adjusted for age, neuroticism, Body Mass Index (BMI) and Townsend deprivation index.

**Results** Greater risk of PND was associated with younger age at first birth, higher number of births, history of stillbirth, miscarriage and preterm birth, insomnia, difficulty getting up, short sleep duration and loneliness. Elastic net models incorporating sociodemographic, obstetric, sleep and social support variables predicted lifetime history of PND with sensitivity of 65.4% and specificity of 75.2% and area under the curve (AUC) of 0.77. The most robust predictors included neuroticism, younger maternal age at first birth, higher BMI, greater number of births, loneliness, insomnia, difficulty getting up and previous miscarriage(s).

**Discussion** These findings suggest that new and expectant mothers with a tendency towards sleep difficulties, low social support, or prior pregnancy-related trauma/complications may benefit from targeted preventive resources and support.

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#### SEX DIFFERENCES IN NOCTURNAL AUTONOMIC RECOVERY FOLLOWING SLEEP RESTRICTION IN HEALTHY OLDER ADULTS

<sup>1</sup>Bindiya Shenoy\*, <sup>1,2</sup>Adriana Michalak, <sup>1</sup>Molly Scoble, <sup>1</sup>Ziad Shabana, <sup>1</sup>Michael Hornberger, <sup>1</sup>Alpar S Lazar. <sup>1</sup>Faculty of Medicine and Health Sciences, University of East Anglia, Norwich, United Kingdom; <sup>2</sup>Department of Biomedical, Metabolic and Neural Sciences, University of Modena and Reggio Emilia, Modena, Italy

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**Background** Sleep restriction and irregular sleep are common in older adults and can impair cardiac autonomic function. However, it remains unclear how recovery sleep and sex moderate these effects. We examined nocturnal heart rate variability (HRV) from baseline to recovery following sleep restriction or scheduled napping in healthy older adults.

#### Method

Thirty-eight individuals (mean±SD age, 64.9±9.0 years; 47% male) completed a 2.5-day laboratory protocol. After baseline polysomnography, participants were assigned to either a 40-hour sleep deprivation or a multimap protocol, followed by a recovery night.

HRV was assessed in continuous 5-minute segments during N2, N3, REM on both nights. Parameters included low frequency (LF), high frequency (HF), LF/HF ratio, standard deviation

of NN-intervals (SDNN), and root mean square of successive RR-interval differences (RMSSD).

Wilcoxon signed-rank tests compared HRV across sleep stages and nights in the full sample and by sex. HRV recovery-to-baseline ratios were entered into general linear models (GLMs) to assess effects of condition, sex, and their interaction, for each parameter and stage.

**Results** HRV varied significantly across sleep stages (except SDNN). RMSSD ( $23.2\pm 11.5$ ms) and HF ( $20.1\pm 11.2$ ms<sup>2</sup>) were significantly lower, and LF ( $62.2\pm 14.3$ ms<sup>2</sup>) and LF/HF ( $5.47\pm 3.96$ ) were higher, in REM compared with N2 and N3 ( $p<0.01$ ). Stage-related HRV differences were seen in both sexes, with stronger effects in women.

GLMs of HRV ratios revealed significant protocol and sex interactions across all parameters except SDNN, indicating sex-specific recovery responses. In N2, LF/HF showed a significant interaction:  $F(1)=6.50$ ,  $p=0.016$ , partial  $\eta^2=0.178$ . Women exhibited a more pronounced increase in LF/HF following sleep deprivation.

**Conclusion** REM was marked by reduced parasympathetic activity, compared to other stages, particularly in women. Recovery sleep modulated HRV in a sex and protocol-dependent manner, with the strongest effects in N2. These findings highlight the complex interplay between sleep stage and sex in shaping autonomic recovery following sleep restriction.

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#### REST ASSURED? HABITUAL SLEEP LIKELY DOES NOT FURTHER EXPLAIN AGE-RELATED CHANGES IN CEREBRAL BLOOD VELOCITY OUTCOMES (PRELIMINARY FINDINGS)

<sup>1,2</sup>Alice Lester\*, <sup>1</sup>Bert Bond, <sup>1</sup>Jon Fulford, <sup>2</sup>Dwayne Mann, <sup>2</sup>Simon Smith, <sup>2</sup>Jenna Taylor. <sup>1</sup>University of Exeter, Exeter, United Kingdom; <sup>2</sup>University of Queensland, Brisbane, Australia

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**Introduction** Habitual poor sleep is linked to dementia risk. Impairments in neurovascular coupling (NVC) may precede dementia symptoms, however, the impact of habitual sleep on cerebral perfusion and NVC remains poorly understood.

**Methods** Total sleep time (TST), time in deep sleep (N3) and sleep efficiency were quantified during three home sleep assessments using an electroencephalography-based device (Somfit, Compumedics) in 96 participants (60 female, age 10.5-76.8 years). Cerebral blood velocity (CBv) through the middle (MCAv) and posterior (PCAv) cerebral arteries was measured using transcranial Doppler ultrasound. Percent change in PCAv from eyes closed to peak PCAv during a visual search task was taken to represent NVC. Hierarchical multiple regressions were used to explore the associations between CBv, NVC, age, and sleep.

Preliminary results: TST ranged from 3.9-10.0 hours, time in N3 from 3-200 minutes, N3 sleep percentage from 0-39%, and sleep efficiency from 56-97%. Resting MCAv and PCAv ranged from 35-107 cm/s, and 24-75 cm/s respectively. NVC responses ranged from 6.8-53.8%.

Older age was associated with lower TST ( $R^2=0.223$ ), N3 sleep (time:  $R^2=0.262$ ; percentage:  $R^2=0.162$ ) (figure 1), and sleep efficiency ( $R^2=0.123$ ; all  $P<0.001$ ). Age was not asso-