development in humans remains poorly understood. The present study examined associations between maternal sleep quality in early, mid, and late pregnancy, and newborn hippocampal and amygdala volume, regions implicated in memory and emotion.
Methods: Pregnant individuals (N=94; Mage=30.5; SDage=5.3) reported on sleep quality three times during pregnancy. Newborn (Mageinweeks=5.1; SDageinweeks=2.7) hippocampi and amygdalae volumes were assessed during an unsedated sleep cycle using magnetic resonance imaging (MRI). Tissue segmentation was collected using a multiatlas iterative algorithm that individually segmented the regions of interest and subsequently combined T1- and T2-weighted high-resolution images (See neonate multiatlas at https://www.nitrc.org/projects/unc_brain_atlas/). Bivariate correlations examined the association between prenatal sleep quality and hippocampus and amygdala volume. Partial correlations examined these associations in the presence of significant cofounding variables including intracranial volume, body weight percentile, sex, and postconceptional age.
Results: Partial correlations revealed that poor maternal sleep quality early in pregnancy predicted larger newborn bilateral hippocampal volume (all rs<.25; ps<.038). Associations with sleep later in gestation persisted for the right hippocampus (all rs<.25; ps<.038). Prenatal maternal sleep quality did not significantly predict newborn amygdala volume (all rs<-.06; ps>.58).
Conclusion: This study provides novel evidence linking prenatal sleep quality and newborn hippocampal volume in humans, suggesting the presence of an intergenerational link between prenatal sleep health and offspring well-being.
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REGION-SPECIFIC CHANGES IN BRAIN PEPTIDOGLYCAN FOLLOWING SLEEP DEPRIVATION

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Introduction: Bacterial cell wall peptidoglycan (PG) and muramyl peptides (MPs), isolated from mammalian brains and urine following sleep deprivation (SD), promote non-rapid eye movement sleep. These PG/MPs likely originate from the host microbiome and have been quantified in neonatal murine brain. PG/MP amounts and dynamics in healthy, adult murine brain remain unknown.
Methods: Wildtype mice acclimated to standard lab conditions were sacrificed at Zeitgeber time (ZT) 3 or ZT15 with (treatment, N=8), or without (control, N=8) 3h of SD prior to time points. Hypothalamic (HT), somatosensory cortex (Sctx) and brain stem (BS) areas were dissected, homogenized in phosphate buffered saline and centrifuged. PG/MP contents in resultant supernatants were determined using an ELISA (MyBioSource), interpolating sample PG from the standard curve, and expressed as ng peptidoglycan per mg tissue wet weight (ng/mg).
Results: At ZT3 and ZT15, BS PG values were significantly higher than HT or Sctx values, while HT and Sctx values did not differ from each other. At ZT3, mean PG values from control mice were: 3.6 in HT, 3.7 in Sctx, and 8.6 ng/mg in BS. After SD, corresponding values were: 3.0, 4.8 (statistically significant increase, p<0.05), and 7.5 ng/mg. Further, within all 8 individual mice after SD prior to ZT3, PG levels in Sctx were higher than corresponding values in HT (p<0.001).At ZT15, PG control values were: 4.6 in HT, 4.6 in Sctx, and 8.9 ng/mg in BS. After SD, PG level at ZT15 was not significantly changed in any brain area assayed. However, PG values after SD at ZT15 compared to ZT3 SD values were significantly higher for HT and BS (p<0.0005 and p<0.005, respectively). In an independent experiment (see Dykstra-Aiello et. al. this volume) we confirmed PG values at ZT15 in BS were significantly higher than Sctx values.
Conclusion: Results indicate unique PG regulation by brain area, sleep loss, and time-of-day suggesting physiological roles for brain PG guiding host behaviors such as sleep. Thus, mammalian sleep-wake regulation and its various associated cognitive states, are the product of millions of years of co-evolutionary symbioses between microbes and their hosts.
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CENTRAL AND PERIPHERAL MARKERS OF OXIDATIVE STRESS AND SLEEP IN MOOD DISORDER: A PILOT MR SPECTROSCOPY STUDY

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Introduction: Sleep disturbance is both a symptom of and a risk factor for mood disorders. Oxidative stress may impact the relationship between sleep disturbance and mood. However, the relationships between sleep and in vivo measurements of oxidative stress in the brain have not been examined in humans. Therefore, we tested the relationship between patient-reported sleep disturbance with peripheral and central concentrations of glutathione (GSH), the primary antioxidant in the brain.
Methods: Participants (2 women and 7 men) were individuals with (n=2) and without mood disorder (n=7), ages 35-61 years. MR spectroscopy at 7 Tesla (TE=20ms, TM=10ms, TR=3000ms) was used to measure cortical GSH levels in the brain in the anterior cingulate cortex (ACC), ventromedial prefrontal cortex (VMPFC), and dorsolateral prefrontal cortex (DLPFC). Peripheral GSH was assayed from a fasted morning blood draw. Sleep disturbance and related impairment was assessed via the PROMIS Sleep Disturbance (PROMIS-SD) and Sleep-Related Impairment (PROMIS-SRI) questionnaires. Pearson correlations were assessed between peripheral and central GSH levels with PROMIS-SD and PROMIS-SRI T-scores.
Results: We observed significant negative correlations between DLPFC GSH with sleep disturbance (r=-0.744, p=0.022) and sleep-related impairment (r=-0.753, p=0.019). We did not detect correlations between sleep-related endpoints and peripheral GSH levels or GSH levels in the ACC or VMPFC.
Conclusion: These preliminary results suggest that more sleep disturbance and related impairment may be associated with lower levels of cortical GSH and implicate a role for sleep disturbance to impact oxidative stress in a region associated with rapid eye movement sleep and modulation of affective states.
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