items of the Sexual Addiction Screening Test – Revised (SAST-R): a score of at least 11 in those seeking treatment, or a score of at least 6 in the volunteers. The DAT1 3' VNTR has been genotyped for 535 total (235 cases by the SAST-R definition, 500 controls) using the method as described by Vandenberg et.al (1992).
Results: In the 235 cases, the frequency of the DAT1 3' VNTR 8, 9, 10, and 11 repeat alleles was 0.004, 0.238, 0.743, and 0.015 respectively. The 129 controls defined as above, the frequency of DAT1 3' VNTR was 0.004, 0.229, 0.764, and 0.004. There was a nominally significant difference in the genotypic distribution of the cases versus controls ($p=0.048$), with a similar difference being seen between those defined as positive for a reward deficiency phenotype versus controls ($p=0.049$).
Conclusions: In this interim analysis, consistent with prior data, the DAT1 3' VNTR appears to be associated with a reward deficiency phenotype.
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Keywords: Addiction, Sex, Internet Addiction, ADHD, Psychiatric Genetics

S252. Fronto-Temporo-Occipital Cortical Thickness Measures Predict Poor Sleep Quality in At-Risk Youth

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Background: Poor subjective sleep quality (SQ) is a prominent risk factor for most forms of psychiatric illness, yet objective biomarkers of SQ have remained elusive. Our goal was to identify neural markers of SQ in at-risk youth using a combination of structural and functional neuroimaging assessments.
Methods: A transdiagnostic sample of 40 youth (8-17yr) completed an MRI assessment and rated past-week SQ with a modified Pittsburgh Sleep Quality Index (N=22 good SQ [PSQI$\leq$5]; N=18 poor SQ [PSQI$>$5]). Group-lasso logistic regression identified non-zero predictors of SQ from cortical thickness measures; BOLD response to reward and emotion fMRI tasks; and demographic/clinical features.
Results: Poor SQ was associated with higher depression severity and cortical thickness in sensory regions (thinner right superior temporal sulcus and left temporal pole, thicker right lateral occipital cortex). Age interacted with right superior frontal[SFC] cortical thickness to predict SQ, such that SFC thickness and age were positively associated in youth with good SQ and negatively related in those with poor SQ. Anxiety severity interacted with right rostral anterior cingulate[rACC] cortical thickness to predict SQ, such that rACC thickness and anxiety were negatively related in youth with good SQ and positively related among youth with poor SQ. Predictors explained 51.2% of the variance in SQ and correctly classified 85% of cases.
Conclusions: Age, internalizing symptoms, and cortical thickness in sensory and frontal midline regions, were useful classifiers of SQ. A combination of measures may be necessary to understand the neural basis of poor SQ and its role in psychiatric illness.
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Keywords: Sleep, Cortical Thickness, Task fMRI, Transdiagnostic, Youth

S253. Sleep Disturbances, Disruption of Circadian Rhythm and Loss of Daily Melatonin Secretion in CAF Military Personnel Suffering From Post-Traumatic Stress Disorder

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Background: According to DSM-5, sleep disturbance is a core feature of PTSD. About 70% of individuals with PTSD have co-occurring sleep problems, reporting greater trouble initiating and maintaining sleep. Melatonin is secreted by the pineal gland during physiologic night, synchronizing circadian timing and regulating sleep–wake cycle. This prospective investigation examined sleep characteristics and nocturnal melatonin production in military PTSD sufferers with endorsed sleep difficulties.
Methods: Volunteers included seven treatment-seeking Canadian Armed Forces (CAF) members, aged 31–45 years, with a diagnosis of PTSD under DSM-5 criteria, including CAPS$\geq$50. Healthy CAF members with no history of PTSD or sleep disorders served as controls. Participants wore wrist actigraphy during sleep for 7-days to derive estimates of sleep quality/quantity (total sleep time [TST], sleep latency [SLAT], wake after sleep onset [WASO], sleep efficiency [SE]). On day-8, participants remained in dim-light ($<$5 lux) for 24 h, during which saliva was sampled bi-hourly to measure endogenous melatonin levels (ELISA, pg/ml) and assess dim-light melatonin onset (DLMO).
Results: PTSD patients exhibited significant sleep disturbances, with lower (mean$\pm$SD, min) TST (385.6$\pm$64.1), greater SLA (12.8$\pm$12.1) and WASO (51.4$\pm$25.1), with an increased number of awakenings (17.8$\pm$9.1) and poor SE compared to healthy controls. Peak salivary melatonin levels in PTSD patients averaged 2.6$\pm$1.1 (range=1.4–4.7) over 24-h, relative to controls (range=20–100). Melatonin production was insufficient to calculate a DLMO in PTSD patients.
Conclusions: Our findings support research linking variable sleep patterns and circadian disruption to PTSD pathogenesis, suggesting chronodisruption may play a causal role in development of PTSD symptoms. Further preclinical and clinical studies are needed.