to their heterosexual peers. Minority stress (e.g., discrimination, victimization) may account for these differences, however little is known about these relationships and how generalized anxiety may play a role in sleep disturbance.
Objectives: Therefore, the aims of this study are to (a) understand the relationship between minority stress and sleep disturbance in a sample of sexual minority men, and (b) test whether these relationships are mediated by generalized anxiety.
Methods: In 2020, 241 sexual minority men were recruited across a south-eastern state in the USA. Participants were asked to respond to scales assessing perceived social stress, minority stress constructs (i.e., internalized homophobia, experiences of harassment, microaggressions), generalized anxiety, and sleep disturbance. Linear regressions were used to test the relationship between minority stress and sleep disturbance controlling for perceived social stress and to test mediation by generalized anxiety.
Results: Two minority stress constructs (experiences of harassment, and microaggressions) and perceived social stress were found to have a positive relationship with sleep disturbance. Generalized anxiety symptoms fully mediated the relationship between minority stress and sleep disturbance.
Conclusions: Because sleep quality has a profound impact on health, findings from this study suggest the need for psychological intervention to improve sleep for sexual minority men. Given that generalized anxiety fully mediates the relationship between minority stress and sleep, targeted anxiety-based interventions have the potential to reduce sleep disturbance disparities between heterosexual and sexual minority men.

Disclosure: No significant relationships.
Keywords: Anxiety; Sexual Minority; Sleep Disturbance; Minority Stress

EPV1476

Stress during the COVID-19 pandemic - impact on neuroplasticity

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doi: 10.1192/j.eurpsy.2022.2090

Introduction: The world's population has been exposed to traumatic events and high levels of stress due to the ongoing COVID-19 outbreak. Stress is known currently as a universal experience, but the concept was first defined in 1936 by Hans Selye. It has been shown that stress is associated with impairments in neuroplasticity (e.g. neuronal atrophy and synaptic loss in the hippocampus, prefrontal cortex) and has a crucial role in almost all mental disorders.
Objectives: In this paper we aim to highlight the recent theoretical and experimental advances in neuroscience regarding stress induced neuroplasticity.
Methods: We analyzed scientific literature written in English and published between 2019-2021. We used the electronic portal PubMed-NCBI.
Results: In the last few years, molecular and cellular studies on animal models of stress related and stress-induced psychopathologies revealed alterations in gene expression, micro ARNs expression, as well as in intracellular signaling pathways that mediate the stress induced adaptations. These findings have led to new theories regarding depression and anxiety in the molecular neurobiology field. It has been shown that stress reduces BDNF expression inducing neuronal atrophy in various brain areas. Contrastingly, other studies have demonstrated that chronic antidepressant treatment increases BDNF expression. Furthermore, a crucial role has been assigned to miRNAs in the development of chronic stress-induced depression-like behavior and neuroplasticity.
Conclusions: We hope that this paper will increase interest in the field of stress induced cellular and molecular changes. More research needs to be pursued in order to achieve a deeper understanding of the pathophysiology of stress-induced mental disorders.

Disclosure: No significant relationships.
Keywords: Depression; Stress; Neuroplasticity; Anxiety

EPV1479

Polysomnography Following Traumatic Brain Injury: A Systematic Review and Meta-Analysis

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doi: 10.1192/j.eurpsy.2022.2091

Introduction: Sleep disturbances are common following traumatic brain injury (TBI) worsening morbidity and other neuropsychiatric symptoms. Post-TBI alterations in sleep architecture require further study.
Objectives: (1) To evaluate polysomnographic measures of sleep architecture in participants with history of TBI compared to controls and as meta-analyses of pooled means. (2) To evaluate effects of timing and severity of TBI on polysomnographic outcomes.
Methods: PRISMA compliant systematic review was conducted of MEDLINE, PsycINFO, EMBASE and Scopus. Inclusion criteria: 1) reporting polysomnography in the context of TBI and 2) operationalizing TBI using clear/formalized criteria. Data were pooled in random-effects meta-analyses with outcomes expressed as mean differences (MD).
Results: In participants with TBI, sleep was comprised of 19.39% REM sleep, 8.13% N1, 51.18% N2, and 17.53% N3, as determined by meta-analyses of single means. Total sleep time was reduced in chronic (>6 months) TBI compared to acute-intermediate TBI (<6 months) (p=0.01). Compared to controls, participants with TBI differed with increased N1 sleep (MD=0.64%; 95% CI=0.02,1.25; p=0.04), reduced sleep efficiency (MD=-1.65%; 95%CI=-3.18,-0.12; p=0.03), and reduced sleep latency on the multiple sleep latency test (MD=-5.90mins; 95%CI=-10.09,-1.72; p<0.01). On sub-group analyses, participants with mild TBI differed from controls with reduced total sleep time (MD=-29.22mins, 95%CI=-54.16,-4.27; p=0.02). Similarly, participants with acute-intermediate TBI exhibited increased sleep latency compared to controls (MD=8.96mins; 95%CI=4.07,13.85; p<0.01) and differed significantly from participants with chronic TBI (X^2(1,N=608)=6.54; p=0.01).
Conclusions: Sleep architecture is altered following TBI with potential implications regarding functional outcomes and recovery.