0285

EXCESSIVE DAYTIME SLEEPINESS WITH LONG SLEEP DURATION INCREASES MYOCARDIAL INFARCTION RISK

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Introduction: Excessive daytime sleepiness (EDS) affects 10-20% of the population and is associated with cardiovascular diseases (CVD) and mortality. However, EDS is heterogeneous, associated with both short and long sleep duration. It is unclear whether each subtype is related to CVD.
Methods: To understand the association of EDS subtypes (stratified by sleep duration) with incident myocardial infarction (MI), we perform multivariable Cox proportional hazards regression on MI using longitudinal medical record data of 471,991 individuals free of CVD at baseline from the UK Biobank. Baseline EDS and sleep duration were assessed by self-reported questionnaires.
Results: After adjusting for multiple social-demographic and behavioral factors, EDS with long sleep (≥9 hours) was associated with a 91% increased incidence of MI (HR=1.91, 95% CI 1.34-2.71) compared to healthy sleep pattern (sleeping 6-9 hours without sleepiness), while EDS with normal (6-9 hours) or short sleep (≤6 hours) was not associated with incident MI. Long sleep without sleepiness was associated with a 39% increase in incident MI (HR=1.39, 95% CI 1.14-1.71). The association of EDS with long sleep was not explained by chronotype, insomnia, sleep apnea, depression, hypertension, or type 2 diabetes, but was confounded with self-reported overall health conditions (HR=1.46, 95% CI 1.02-2.08 after adjustment).
Conclusion: Our study suggests the previous association evidence of EDS increasing risk of MI may be primarily driven by its long sleep subtype (high "sleep propensity"), but the underlying mechanisms are unclear. Future work is needed to understand whether there are targetable interventions for this novel sleep phenotype that may improve cardiovascular health.
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0286

PRESENCE AND POTENTIAL IMPACT OF DEMOGRAPHICALLY BASED ATTRITION IN PEDIATRIC SLEEP MANIPULATION RESEARCH

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Introduction: Pediatric sleep manipulation studies have mostly enrolled youth from dominant-culture, higher-income families. Studies with more diverse samples have been underpowered to detect differential attrition and demographic effect moderators. Here we pool data from two adolescent sleep manipulation protocols in a Midwest US city to better examine demographic differences in attrition rates and effect sizes.
Methods: Samples were pooled from studies detailed in Beebe et al. (2017; SLEEP, zsw035) and Duraccio et al. (2021; JSR, e13054). Both involved a sleep stabilization week, followed by 5-night periods of sleep restriction (6.5 hours/night in bed) and healthy sleep (9.5-10 hours/night in bed) in randomized counterbalanced order. Primary caregivers and 14-17 year-old adolescents each completed attention and sleepiness questionnaires for both conditions. Here we compare adolescents who were caregiver-identified as Black vs. White, the two largest local racial groups. Caregivers also reported their own education, family income, and household structure (single- vs. two-parent). Non-parametric tests looked for differential attrition and MANCOVA tested for racial differences in effects.
Results: Of the 257 initially enrolled, Black adolescents and those from households with one parent, lower income, or lower caregiver education were differentially lost to attrition (all $p<.001$), even though the racial makeup of the final sample approximated the regional population (36% Black, 64% White). In the final sample, Black and White youth were equally able to change their sleep ($p>.90$). Manipulation effects were significantly smaller for Black than White adolescents for inattention (self-report $p=.026$; parent-report $p=.017$) and sleepiness (self-report $p=.002$, parent-report $p<.001$), but these differential effects were non-significant after controlling for family income, household structure, and caregiver education ($p>.05$).
Conclusion: Even when a final study sample seemingly approximates the diversity of the local population, differential attrition may affect results. In this case, it superficially appeared that being in a non-dominant group (self-identified Black) was protective against the impact of short sleep. However, this effect disappeared after controlling for demographic risk factors. Participation in sleep manipulation studies can be challenging, so families facing higher burdens may need more support; otherwise, only the most resilient of those families may succeed, which could distort findings.
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0287

EFFECTS OF SLEEP RESTRICTION AND RECOVERY ON THE CAPACITY OF GLUCOCORTICOIDS TO INHIBIT INFLAMMATORY MARKER EXPRESSION IN HUMAN MONOCYTES

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Introduction: Chronic low-grade systemic inflammation is involved in the pathogenesis of many human diseases. Common sleep patterns of restricting sleep during weekdays and catching up on sleep over the weekend induce inflammatory upregulation that may not resolve following weekend recovery sleep. We hypothesize that this sleep pattern leads to an inflammatory imbalance of markers regulating inflammatory homeostasis, including inflammatory markers (eg, interleukin-6 (IL-6) and cyclooxygenase 2 (COX-2)) and markers of counter-inflammation (eg, glucocorticoids (GCs)). The enzyme COX-2 is involved in prostaglandin synthesis and is the target of pain-relieving nonsteroidal anti-inflammatory drugs (NSAIDs). GCs are used in the treatment of many inflammatory diseases, including severe acute infection with SARS-CoV-2. We investigated if sleep restriction impairs the capacity of GCs to inhibit inflammatory COX-2 expression in a preliminary dataset.
Methods: The present preliminary dataset (N=6, 2F/4M) derives from an ongoing randomized controlled within-subjects trial consisting of three 11-day in-hospital protocols (2 restricted sleep arms, 1 control sleep arm). The ongoing study is blinded for administration of placebo or aspirin under sleep restriction. Under restricted sleep conditions, 2 nights of baseline sleep (8h/night) were followed by 5 nights of restricted sleep (4h/night), concluding with 3 nights of recovery sleep (8h/night).