## 0036

### POPULATION WAKEFULNESS AND NOCTURNAL SUICIDE RISK

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**Introduction:** Nocturnal wakefulness may mediate the relationship between disrupted sleep and suicide risk since nighttime is associated with a peak in negative mood and altered reward processing and executive function. One example is a wakefulness-adjusted nocturnal peak in population suicide risk measured from 2003-2010 (Perlis et al, 2016), but these results have not been replicated with more recent data.
**Methods:** A total of 77,784 suicides with known time of fatal injury were extracted from the National Violent Death Reporting System (NVDRS) for 2003-2010 and 2011-2017. These data were then weighted by the estimated proportion of the population that was awake at each hour as derived from the American Time Use Survey (ATUS) for the same years. Suicides were tabulated by clock hours, age, sex, race, and ethnicity, and suicide counts were modeled using robust Poisson regression with hourly population wakefulness entered as an offset term, thus producing hourly incident risk ratios.
**Results:** A comparison of analyses between previously reported data (2003 to 2010) and new data (2011 to 2017) showed a consistently elevated risk of suicide at night (midnight to 6AM) that did not differ between time periods. After combining all fifteen years, the maximum number of suicides occurred at noon. Adjusting for population wakefulness, however, revealed a significant increased risk for suicide between 11PM and 5AM, with a 4.61-fold increase at 3AM (IRR: 4.61 [4.11-5.16]). Adjusting for age, sex, race, and ethnicity attenuated, but did not alter these results. In subgroup analyses, the nocturnal risk for suicide was elevated among those with bipolar disorder (5.2% of cases), those with a blood alcohol level greater than 80 mg/dl (14.9% of cases), and those who tested positive for a Z-drug (i.e., zolpidem, zaleplon, and eszopiclone) at autopsy (0.7% of cases).
**Conclusion:** Fifteen years of data from suicides across the United States show a significantly increased risk for suicide during the circadian night that peaks at 3AM. Nocturnal wakefulness remains a significant risk factor for suicide, and suicide prevention efforts may benefit from interventions to reduce nocturnal wakefulness and/or an increase in prevention resources at this time.
**Support (If Any):**

## 0037

### BLUE LIGHT EXPOSURE FACILITATES CORTICAL NEURAL EFFICIENCY EXCLUSIVE OF MELATONIN EFFECTS

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**Introduction:** Sleep and circadian rhythms are influenced by exposure to light at specific times of day. In particular, blue light exposure can suppress melatonin production, shift circadian timing of sleep and wake, and acutely enhance alertness. Despite the consistency of these effects, little is known about their underlying mechanisms. We propose that in addition to the melatonin and phase shifting effects of blue light, that it also produces acute changes in brain activation that lead to greater neural efficiency.
**Methods:** Twenty-six individuals (11 male; 15 female, Mean age=24.27, SD=6.27) completed a counterbalanced cross-over design study while undergoing two separate neuroimaging scans in a 3T MRI scanner separated by one week. During scanning, each participant was exposed to either BLUE light (470 nm; active condition) or AMBER (580 nm; placebo condition) light conditions on alternate weeks. All scans occurred between 11:00 a.m. and 1:30 p.m., a time that has been described as the "dead zone" when melatonin levels are generally unaffected by light exposure. Participants completed a well-established working memory task (i.e., N-back task) in the scanner while undergoing continuous exposure to the specific light wavelength for the duration of the task. We contrasted the simple 1-back memory condition versus the 0-back memory condition using SPM12. Contrast maps were then compared using a paired-samples t-test.
**Results:** Compared to AMBER light, the BLUE light was associated with significantly less deactivation within two large clusters comprising the default mode network (DMN). These included a large cluster (k=1343 voxels) in the medial prefrontal cortex and a large cluster (k=5075 voxels) encompassing the posterior cingulate, precuneus, and parietal cortex regions ($p<.05$ FDR cluster corrected). Melatonin levels did not differ from pre-to-post light exposure for either condition.
**Conclusion:** Despite no effect on salivary melatonin, BLUE light exposure was associated with significantly less deactivation of brain regions that are usually suppressed to engage in cognitively demanding tasks. This suggests that blue light appears to enhance cognitive efficiency, potentially leading to similar performance while taxing fewer brain resources. Such findings suggest a potential role for blue light in sustaining performance during periods of sleep loss.
**Support (If Any):**

## 0038

### ACUTE BLUE LIGHT EXPOSURE INCREASES ACTIVATION IN THE PULVINAR NUCLEUS

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**Introduction:** Blue-wavelength light can produce phase-shifts in the circadian rhythm. We have previously shown that morning blue light exposure was associated with advanced onset of sleep time and diminished daytime sleepiness. These changes were associated with increased gray matter volume in the left pulvinar nucleus of the thalamus, a key hub of the visual attention network. However, very little is known about the underlying mechanisms of these light activations. We hypothesized that the pulvinar may be affected more by the acute activating effects of light rather than its effects on melatonin. Therefore, we exposed individuals to blue or amber light while undergoing functional neuroimaging at a time of day when melatonin levels are almost non-existent.
**Methods:** Twenty-six healthy individuals (15 male; 11 female; age=24.27, SD=6.27) completed a counterbalanced cross-over study involving two 3T functional MRI sessions separated by one