

One Night Total Sleep Deprivation Alters Neural Correlates of Risk-Taking

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Introduction Sleep deprivation (SD) can induce significant deficits in multiple neurocognitive functions, particularly vigilant attention, working memory, and executive function (1). However, the effects of SD on risk-taking behavior are unclear, with some studies reporting greater risk-taking behavior (2) while others reporting no changes following sleep loss (3-4). The present study used functional MRI with a well-validated balloon analog risk task (BART) paradigm (5) and examined the effects of 24hr of total sleep deprivation (TSD) on the neural correlates of risk-taking.

Methods A total of 27 healthy adults (13 male, mean age 31.3 ± 6.7 years) participated in the study. The subjects were scanned on a Siemens 3 T Trio scanner while performing a modified BART task (5) at rested baseline (BL) and after 24hr of total SD, using a standard EPI sequence with the following parameters: TR=1.5s, TE=30ms, flip angle=90°, 25 interleaved axial slices with 5 mm thickness, in-plane resolution=3.44 x 3.44 mm. During the BART, subjects were required to sequentially inflate a virtual balloon that could either grow larger or explode. Larger balloons were associated with increasingly larger monetary rewards and risk of explosion. The average number of inflations participants made for the balloons provides an objective assessment of risk-taking propensity. Imaging data were analyzed by SPM5 using an event-related model. Risk-induced brain activation was isolated by parametric analysis of the levels of risk associated with balloon inflations. Both voxel-wise general linear modeling (GLM) and region of interest (ROI) analysis were conducted.

Results Behavioral data showed no changes from BL to SD. During the BART, subjects made 6.5 ± 1.3 inflations at BL and 6.6 ± 1.1 inflations after SD ($p > .7$). Neuroimaging data showed robust activation in the mesolimbic, frontal, and visual pathway areas during the BART risk taking both at BL and during SD (Fig.1a & b), which replicated the activation patterns in our previous studies (5-6). Direct comparisons between these brain activation patterns indicated no differences from BL to SD. However, direct comparisons between the neural responses to the loss events (balloon explosion) showed reduced activation in the left insula (Fig.2a). Furthermore, at BL, both SPM whole brain analysis and independent ROI analysis showing that insular and striatum activation level negatively correlated with risk-taking propensity (Fig.2b-c), which also replicated our previous finding (6). However, such negative correlations were not found after SD.

Discussions and Conclusions Consistent with previous studies (3-4), our data showed that 24hr of TSD did not change risk-taking behavior. However, significantly reduced activation in the insula was observed during loss events following TSD, which may reflect diminished negative emotional response to loss outcomes during risk-taking. Moreover, the loss of negative correlations between risk-taking behavior and activation level in insular and striatum following TSD, suggesting that one night sleep loss can alter neural mechanisms mediating the inter-individual differences in risk-taking propensity without actual behavioral changes. Overall, our data suggest that sleep loss alters neural responses associated with risk-taking and support the hypothesis that neuroimaging findings may be a precursor to the behavioral changes following long time sleep deprivation.

References

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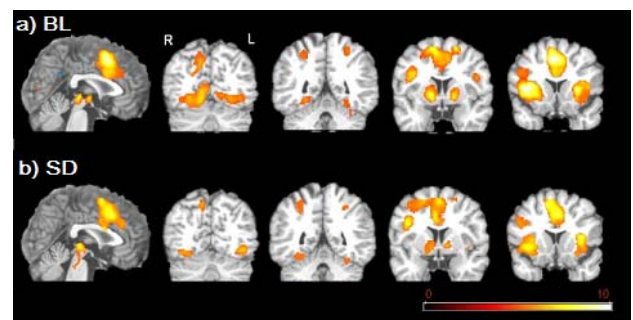


Fig.1 Similar risk-induced activation patterns at BL and after SD.

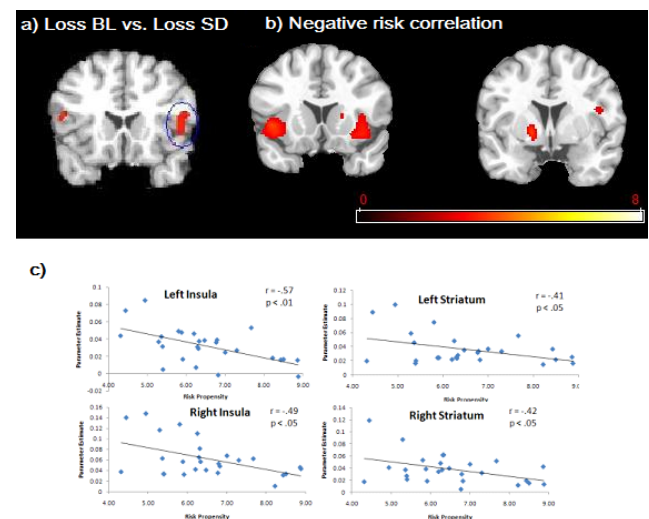


Fig.2 a) Loss events showing reduced activation in the insula after SD; b-c) Both whole brain and ROI analysis showing negative correlations between risk propensity and activation in insula and striatum at BL.