Enhanced Resting-State Striatal Connectivity Correlates with Increases in Daily Caloric Intake after Sleep Deprivation

Fang Z, Spaeth AM, Zhu S, Taylor A, Goel N, Basner M, Detre JA, Dinges DF, Rao H

Center for Functional Neuroimaging, Department of Neurology, and Division of Sleep and Chronobiology, Department of Psychiatry, University of Pennsylvania Perelman School of Medicine, Philadelphia, PA, USA

Introduction: Sleep loss is a recognized risk factor for overeating and obesity, however the underlying neural mechanisms remain unclear. Previous imaging studies have indicated that sleep loss may enhance neural responses to food stimuli in the dopaminergic striatal pathways. In this study, we examined the effects of one night of total sleep deprivation (TSD) on resting-state striatal functional connectivity (FC) and its relationship to changes in caloric intake.

Methods: A total of 21 healthy adults (11 females, age 22-50 yrs, BMI 19.7-28.9) were scanned at rest on three occasions between 7-9 am using a standard EPI sequence on a Siemens 3T MR scanner: a baseline (BS) scan after 9h normal sleep, a SD scan after 24h TSD, and a third scan after two nights (20h) recovery sleep (RS). FC analyses using caudate as the seed region were conducted using SPM8 and the REST toolbox. Food and beverage intake was weighed before and after subjects ate a meal. Participants' food consumption was ad libitum and recorded. Changes in daily caloric intake were calculated and correlated with FC alteration following SD.

Results: Participants' caloric intake per hour was significantly higher following SD compared to BS (p<0.001). FC analyses showed no differences between BS and RS. However, SD significantly enhanced connectivity to caudate in the thalamus, dorsolateral prefrontal cortex, superior parietal cortex, precuneus, and putamen, and reduced connectivity in the sensorimotor regions (all p<0.001). Caudate-putamen connectivity changes correlated with increases in caloric intake from BS to SD (r=0.62, p=0.003).

Conclusion: Our results show that SD significantly altered resting-state striatal connectivity which predicted increases in daily caloric intake after sleep loss, supporting the role of the dopaminergic system in overeating. These findings suggest that disrupted dopamine pathway connectivity may be one mechanism by which SD increases food intake.

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