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Linking Adolescent Sleep, Brain Maturation, and Behavior

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Despite the ubiquity of sleep across species, surprisingly little is known about its functions. It is known that during human adolescence, sleep patterns typically undergo marked changes. Electroencephalogram (EEG) studies indicate a 50% reduction of deep (stage 4) sleep and a 75% reduction in the peak amplitude of delta waves during nonrapid-eye movement sleep in adolescence [1]. Changes in circadian rhythms impart a tendency for later sleep onset. School schedules are often incompatible with a corresponding delay in sleep offset, leading to a less than optimal amount of sleep for the majority of adolescents.

Adolescence is also a time of dramatic changes in body, brain, and behavior. Converging evidence from structural and functional magnetic resonance imaging, electrophysiological, and postmortem studies indicate dynamic changes in the adolescent brain. Three robustly replicated themes emerging from these studies are: (a) greater “connectivity” exemplified by increases in white matter volumes, functional magnetic resonance imaging (fMRI) correlations among disparate regions during tasks, and EEG coherence; (b) a changing balance between earlier maturing/puberty-related limbic systems and later-maturing frontal executive function systems; and (c) a pattern of childhood peaks followed by adolescent decline for gray matter volumes [2]. If these gray matter volume reductions are partially accounted for by decreases in synaptic density, it may be related to the EEG changes noted above, as EEG signals emanate from spatially coherent activity of synapses [3]. A recent study examined the possible relationship between anatomical brain changes and EEG changes by acquiring both MRI and quantified EEG in 138 healthy subjects from ages 10 to 30 years [4]. Curvilinear reductions in gray matter volume of the frontal and parietal cortex were matched by similar curvilinear reductions in EEG power of the corresponding regions, supporting the connection between gray matter volume reductions, EEG changes, and synaptic pruning.

The behavioral manifestations associated with these changes vary considerably, depending on social context, but adolescents in all social mammals tend to demonstrate greater propensities for affiliation with same age peers and increases in sensation seeking and risk taking.

The link between these adolescent changes in sleep, brain, and behavior remain poorly understood. In “Reward-Related Brain Function and Sleep in Pre/Early Pubertal and Mid/Late Pubertal Adolescents,” published in the current issue of the *Journal*, the authors [5] provide an early step to address this void of knowledge. Specifically, they examine the relationship between various measures of sleep and fMRI measures during a task designed to assess reward anticipation and reward outcome.

The study of reward systems during adolescence is particularly relevant because of their centrality to decision making. Many of the sources of adolescent morbidity and mortality are directly related to decision making. Whether to use drugs, have unprotected sex, drive recklessly, or take other risks are all influenced by how the brain’s reward systems assess cost and benefit. One of the challenges addressed by the study is to untangle the pubertal/

hormonal effects of adolescence from other maturational effects. To partially isolate puberty-specific effects the authors examined subjects in a relatively narrow age range who differed on Tanner stage. Perhaps because of the relative ease of manipulating hormone levels in animal models a preponderance of the literature focuses on pre/post puberty comparisons, although future studies should also address the many brain and sleep changes occurring well after the completion of puberty. The results of the study indicate that poorer sleep is related to less activation of the caudate nucleus during reward anticipation and reward outcome. It is hypothesized that this lower activation may result in compensatory increases in the reward-driven behavior characteristic of teens. As with all studies relating imaging findings to behavior, caution is merited in interpretation, particularly with regard to causality. There is rarely a one-to-one correspondence between a particular brain region and a discrete function. Most functions involve many regions, and most regions are involved in many functions. Longitudinal studies following changes in an individual's sleep, fMRI activations, and behavior will help to elucidate the brain/sleep/behavior causal relationships. Also of interest would be to assess whether genetic differences relate to individual variance in sleep parameters.

In addition to the possible association of sleep changes with depression, substance abuse, and accidents noted in the article, there may also be connections with other disorders typically emerging during adolescence, such as schizophrenia. Based on his interpretation of the dramatic decreases in delta sleep of healthy adolescents as reflecting robust synaptic pruning, Irvin Feinberg, in 1982, postulated that schizophrenia may be a consequence of an exaggeration of typical synaptic elimination [6]. Subsequently, studies of membrane phospholipids, prefrontal metabolism, and frontal cortical gray matter changes have lent support to this "exaggeration of typical adolescent changes" hypothesis for schizophrenia [7–10].

An important point made by Holm and his colleagues [5] is that biological tendencies for sleep changes during adolescence do not mean that interventions are futile. The tendencies can be modulated by efforts to promote more sleep, and the effects of such interventions can be assessed to further elucidate the relationship between sleep quality and behavioral outcomes.

All in all, the current article represents an important but early step in our exploration of the relationship between sleep, brain development, and behavior in adolescence. Given that one-third of our lives are spent asleep and that the amount and quality of sleep has profound impact on our health, cognition, and behavior, it seems prudent to devote more resources to research this understudied domain. The dramatic changes in sleep, brain, and behavior during the second decade of life make this an even more compelling mandate for adolescent health research.

References

1. Dustman RE, Shearer DE, Emmerson RY. Life-span changes in EEG spectral amplitude, amplitude variability and mean frequency. *Clin Neurophysiol* 1999;110:1399–409. [PubMed: 10454276]
2. Giedd JN. The teen brain: insights from neuroimaging. *J Adolesc Health* 2008;42:335–43. [PubMed: 18346658]
3. Matsuura M, Yamamoto K, Fukuzawa H, et al. Age development and sex differences of various EEG elements in healthy children and adults—quantification by a computerized wave form recognition method. *Electroencephalogr Clin Neurophysiol* 1985;60:394–406. [PubMed: 2580690]
4. Whitford TJ, Rennie CJ, Grieve SM, Clark CR, Gordon E, Williams LM. Brain maturation in adolescence: concurrent changes in neuroanatomy and neurophysiology. *Hum Brain Mapp* 2007;28:228–37. [PubMed: 16767769]

5. Holm SM, Forbes EE, Ryan ND, et al. Reward-related brain function and sleep in pre/early pubertal and mid/late pubertal adolescents. *J Adolesc Health* 2009;45:326–34. [PubMed: 19766936]
6. Feinberg I. Schizophrenia: caused by a fault in programmed synaptic elimination during adolescence? *J Psychiatr Res* 1982;17:319–34. [PubMed: 7187776]
7. Pettegrew JW, Keshavan MS, Panchalingam K, et al. Alterations in brain high-energy phosphate and membrane phospholipid metabolism in first-episode, drug-naive schizophrenics. A pilot study of the dorsal prefrontal cortex by in vivo phosphorus 31 nuclear magnetic resonance spectroscopy. *Arch Gen Psychiatry* 1991;48:563–8. [PubMed: 1898445]
8. Andreasen NC, Reza K, Alliger R, et al. Hypofrontality in neuroleptic-naive patients and in patients with chronic schizophrenia. Assessment with xenon 133 single-photon emission computed tomography and the Tower of London. *Arch Gen Psychiatry* 1992;49:943–58. [PubMed: 1360199]
9. Sporn AL, Greenstein DK, Gogtay N, et al. Progressive brain volume loss during adolescence in childhood-onset schizophrenia. *Am J Psychiatry* 2003;160:2181–9. [PubMed: 14638588]
10. Keshavan MS, Anderson S, Pettegrew JW. Is schizophrenia due to excessive synaptic pruning in the prefrontal cortex? The Feinberg hypothesis revisited. *J Psychiatr Res* 1994;28:239–65. [PubMed: 7932285]