COMMENTARY

DOI: 10.5665/SLEEP.1260

Sleep EEG, the Clearest Window through which to View Adolescent Brain Development

Commentary on Tarokh et al. Sleep EEG provides evidence that cortical changes persist into late adolescence. SLEEP 2011;34:1385-1393.

Ian M. Colrain, PhD[1,2]; Fiona C. Baker, PhD[1,3]

[1]Human Sleep Research Program, SRI International, Menlo Park, CA; [2]Department of Psychological Sciences, University of Melbourne, Parkville, Victoria, Australia; [3]Brain Function Research Group, School of Physiology, University of the Witwatersrand, Johannesburg, South Africa

It can be argued that the two periods of time in human development that show the most dramatic changes in the brain are from mid-gestation through age 6 years and during adolescence.[1] While the prenatal changes involve both proliferative neurogenesis and apoptosis, the postnatal changes are not so much in the number of neurons, but in the number and form of the interconnections between them.[1]

The human female pelvic girdle places limitations on the extent of brain development that can occur in utero. The rapid growth in brain and head size in the first two years of postnatal life are due to the rapid development of trillions of synaptic connections, providing young children with a rich interconnected network of neurons, as an ideal and highly plastic substrate to support learning. Thus children exposed to a bilingual environment from birth, have a greater facility with language, and a different organization of language areas in the brain compared to those of us drilled in a foreign language in high school.[2] Nonetheless, there is a cost to running a highly interconnected network. While it has great potential, it is inefficient, can be slow, and has a high metabolic demand.[3]

Starting in childhood, brain regions start losing plasticity and gain speed and efficiency, by pruning weaker synaptic connections and myelination of axons in those remaining. This use it or lose it pruning process starts in primary cortices (probably with the sensorimotor strip or occipital cortex), and then moves forward and outward eventually encompassing the whole brain.[4] The last area to lose plasticity and to take on its adult form is the prefrontal cortex.[5] Again, there are probably adaptive advantages to having this area remain plastic for as long as possible, but eventually it too must finish pruning, myelinate its axons, and take on its adult form.[6]

The paper by Tarokh and colleagues[7] in this issue of *SLEEP* highlights how longitudinal sleep EEG data is arguably the optimal functional measure of these structural brain changes. The use of longitudinal data removes much of the noise associated with individual differences and permits a more accurate determination of maturational effects on the brain. Together with data from other very recent papers,[8,9] data from Tarokh et al. present evidence of decreased EEG power across a wide spectrum in NREM and REM sleep. The comparison of the data showing differences between 15-16 and 17-18 years, with those collected in a similar design but from younger adolescents highlights the fact that later adolescents are still showing dramatic changes in sleep EEG power, reflecting ongoing changes in brain development.

Tarokh et al. show smaller changes in the occipital (particularly left occipital) derivations than in the central derivations, and inspection of their figures reveals that the power spectrum at the 15-16 year time point is already at the relatively lower level seen centrally at ages 16-18. This would imply that the occipital cortex develops earlier than regions sampled by central electrodes, consistent with the report by Baker et al.[8] that the occipital derivation showed greater decline than central and frontal derivations across the power spectrum in NREM and REM sleep, when looking at longitudinal changes earlier in adolescence. It is also consistent with the recent Feinberg paper[9] showing an earlier start to the decline in NREM delta for occipital than for central or frontal derivations.

Of particular interest in the report by Tarokh et al. is the lack of change in the high sigma band. While this may be, as they suggest, due to a change in the dominant frequency in the band, it may also reflect that neural mechanisms underlying sigma activity (and by extension, sleep spindles) develop early, an interesting implication given their sleep protective role. Baker et al.[8] found no change in the sigma band during NREM sleep, in a longitudinal assessment of younger adolescents. Menstrual phase effects on sigma activity, particularly fast sigma,[10,11] are a potential confound in these sorts of data sets however, particularly when as in the case of the Tarokh et al. paper, the majority of subjects are female. As pointed out by Tarokh and colleagues, the rate of decline in sleep EEG power over time was variable between individuals and unrelated to age at initial assessment. Further studies are needed to investigate what factors contribute to this variability in development in healthy adolescents and extend to investigate whether medical or psychiatric conditions impact changes in the sleep EEG across adolescence.

EEG power reflects the sum of inhibitory and excitatory postsynaptic potentials in thousands of neural columns sampled by an individual electrode,[12] and the curve describing changes in delta EEG over the lifespan is remarkably similar to those based on postmortem anatomic synaptic density measures, and cerebral metabolic rate.[13] While longitudinal MRI measures are informative of brain structural changes in adolescence,[12] even over short time periods,[14] structural MRI measures do not capture aspects of brain development such as neuronal connectivity and receptor density.[15] Longitudinal assessment of changes in EEG power, especially delta frequency power, provides an

Submitted for publication August, 2011
Accepted for publication August, 2011
Address correspondence to: Ian M. Colrain, PhD, SRI International, 333 Ravenswood Avenue, Menlo Park, CA 94043; Tel: (650) 859-3915; Fax: (650) 859-2743; E-mail: ian.colrain@sri.com

easily measured, noninvasive functional correlate of the developmental synaptic pruning process. Ideally, both sleep EEG and MRI measures would be taken in the same subjects.[16] The EEG data from Tarokh et al. show the developmental process extending, at least in regions anterior to occipital cortex, into ages at which adolescents give the outward physical appearance of having achieved adulthood.

DISCLOSURE STATEMENT

The authors have indicated no financial conflicts of interest.

REFERENCES

1. Stiles J, Jernigan TL. The basics of brain development. Neuropsychol Rev 2010;20:327-48.
2. Kim KH, Relkin NR, Lee KM, Hirsch J. Distinct cortical areas associated with native and second languages. Nature 1997;388:171-4.
3. Chugani HT, Phelps ME, Mazziotta JC. Positron emission tomography study of human brain functional development. Ann Neurol 1987;22:487-97.
4. Shaw P, Kabani NJ, Lerch JP, et al. Neurodevelopmental trajectories of the human cerebral cortex. J Neurosci 2008;28:3586-94.
5. Gogtay N, Giedd JN, Lusk L, et al. Dynamic mapping of human cortical development during childhood through early adulthood. Proc Natl Acad Sci USA 2004;101:8174-9.
6. Yakovlev P, Lecours, A. The myelogenetic cycles of regional maturation in the brain. Oxford: Blackwell; 1967.
7. Tarokh L, Van Reen E, LeBourgeois M, Seife R, Carskadon M. Sleep EEG provides evidence that cortical changes persist into late adolescence. Sleep 2011;34:1385-93.
8. Baker FC, Turlington SR, Colrain I. Developmental changes in the sleep electroencephalogram of adolescent boys and girls. J Sleep Res 2011.
9. Feinberg I, de Bie E, Davis NM, Campbell IG. Topographic differences in the adolescent maturation of the slow wave EEG during NREM sleep. Sleep 2011;34:325-33.
10. Driver HS, Dijk DJ, Werth E, Biedermann K, Borbely AA. Sleep and the sleep electroencephalogram across the menstrual cycle in young healthy women. J Clin Endocrinol Metab 1996;81:728-35.
11. Baker FC, Kahan TL, Trinder J, Colrain IM. Sleep quality and the sleep electroencephalogram in women with severe premenstrual syndrome. Sleep 2007;30:1283-91.
12. Lopes da Silva F, van Rotterdam A. Biophysical aspects of EEG and magnetoencephalogram generation. In: Lopes da Silva F, Niedermeyer E, eds. Electroencephalography: basic principles, clinical applications and related fields. 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2005:107-25.
13. Feinberg I, Campbell IG. Sleep EEG changes during adolescence: an index of a fundamental brain reorganization. Brain Cogn 2010;72:56-65.
14. Sullivan EV, Pfefferbaum A, Rohlfing T, Baker FC, Padilla ML, Colrain IM. Developmental change in regional brain structure over 7 months in early adolescence: comparison of approaches for longitudinal atlas-based parcellation. Neuroimage 2011;57:214-24.
15. Giedd JN, Stockman M, Weddle C, et al. Anatomic magnetic resonance imaging of the developing child and adolescent brain and effects of genetic variation. Neuropsychol Rev 2010;20:349-61.
16. Buchmann A, Ringli M, Kurth S, et al. EEG sleep slow-wave activity as a mirror of cortical maturation. Cereb Cortex 2011;21:607-15.