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Journal search and commentary

Article reviewed: Evidence from the waking electroencephalogram that short sleepers live under higher homeostatic sleep pressure than long sleepers[☆]

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Objective

Determine if differences in spectral power analyses of the waking EEG indicate that short compared to long sleepers have a greater daytime physiological sleep pressure from an increase in the homeostatic sleep drive independent of the circadian effects.

Study design

Between Group comparison of spectral analyses of waking EEG using both absolute spectral power and parameters providing the best fit for a theoretical model combining circadian and homeostatic factors to predict the spectral power.

Study population

Generally healthy subjects recruited from a newspaper advert meeting study criteria. The following two groups were selected: Nine short sleepers (female:male 5:4; ages 22–31 with average = 25.9) who habitually slept less than 6 h and no more than 7 h on weekends. Eight long sleepers (female:male 4:4, ages 20–30 with average 24.1) who habitually slept more than 9 h and not less than 9 h for an average weeknight. The sleep pattern must have been reported to be stable for the past year. Subjects were excluded for any sleep disorder (other than abnormal length of sleep), shift work, psychiatric disorder (Axis I), or use of any medications, drugs or tobacco.

Methods

Each subject had two consecutive nights of EEG sleep recording scheduled to occur at the subject's normal sleep times, immediately followed by a 37–42 h constant routine (CR) starting at the end of the second night of sleep recording and continuing until 23:00 h the next night. A protracted 36-h rest period in total darkness, except for times eating or using the bathroom, permitted recovery sleep following the CR. During this rest period subjects were instructed to sleep as much as possible. The CR followed standard procedures: light <10 lux, subject lying awake in 'propped-up position' in bed, fluids every hour and isocaloric meals every 2 h both at room temperature, and no external time cues available. Rectal temperature was recorded during the CR and the temperature minimum and maximum were estimated by a cosine curve fitting to data for each minute.

Every 30 min during the CR the standard minimal sleep polysomnogram (left and right EOG, C3/A2 and C4/Al EEC and submental EMG) was recorded

[☆] Aeschbach D, Posolache TT, Sher L, Matthews JR, Jackson MA, Wehr TA. Neuroscience 2001;102:493–502.

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for 3-min. During this recording the subject was told to relax, keep eyes opened and focused on a spot on the wall and avoid frequent blinking or eye movements. The EEC recordings excluding sleep or movement artifact epochs were analyzed in 4-s epochs using a fast-Fourier transform with power spectra calculated in 1 Hz bins for frequencies of 0.25–20 Hz.

Changes in EEG power density were used to determine parameters of an equation with two primary additive components: a saturating exponential function of time awake representing the presumed homeostatic sleep drive and a circadian function with sinusoidal fluctuation between the temperature extremes. The homeostatic parameters estimated the amplitude and rate of saturation for the homeostatic sleep drive.

Results

The EEG spectral analyses during sustained wakefulness showed clear changes consistent with expected effects from increased duration of wakefulness and circadian cycle. Short vs. long sleepers showed overall power density significantly greater in the high frequency theta to low frequency alpha range (TLFA) (5.25–9.0 Hz) with some less significant increases in the higher frequency ranges of 12.5–20 Hz A two-way analysis of variance for group and time-awake by frequency ranges showed that time awake was a significant factor for all frequencies ($P < 0.003$). The group effect was significant for the 5.25–9 Hz frequencies ($P < 0.003$) and the interaction of group and time awake was significant for 0.75–5.0 Hz frequencies ($P < 0.03$). The higher amplitude of the power densities were consistent with increased homeostatic sleep pressure. The time constants for the exponential saturation of the homeostatic sleep drive with increased wake calculated from the two-process model did not differ between the long and short sleepers. The changes in slow wave activity during sleep showed similar group effects for the recovery sleep period.

Conclusion

The increases in the homeostatic sleep drive with increased wakefulness was associated with frequency-specific increases in the waking EEC, particularly in the TLFA range (5.25–9.0 Hz). The circadian and homeostatic effects on spectral power in the waking EEG were also determined separately. These analyses permit assessing the kinetics as well as amplitude of the homeostatic sleep drive. Natural short sleepers compared to natural long sleepers show increased amplitude but similar kinetics for the homeostatic sleep drive.

The increased amplitude of the sleep drive was seen as relating to either adaptation to chronic sleep loss or genetic differences, or possibly both.

Comment

While it is always a pleasure to read a carefully thought-out and detailed research article such as this one, it is easy to lose the potential significance of the article in the necessary presentation of the details. It may not seem surprising that the EEG expresses increased sleep need in the short sleepers. but it is somewhat surprising that this is expressed in the waking as well as the sleep EEC, occurs mainly in one frequency band and occurs without indication for any change in the time-constant or kinetics of the homeostatic drive. Changes in sleep EEG for short sleepers would be expected and have been previously shown [1]. What is particularly unique in this study is the documentation of significant difference in the wake EEG. The wake as well as the sleep EEG differs in the short sleeper indicating increased sleep pressure. Finding these results for the wake time is particularly significant since the sleep measures may be affected by differences in thermal load with longer times awake. This potential source of artifact should presumably not be present for equal amounts of wake time after sleep, but the relevant temperature data were not provided in this article.

So does increased wake-time sleep pressure mean the wake time functioning of the short sleeper is impaired? The waking EEG frequency range reflecting increased sleep drive is about the same frequency range associated in other studies with cognitive and vigilance performance decrements when the EEC activity is increased [2]. The wake EEC changes

with the increased sleep drive for short sleepers could, however, either reflect adaptation without any behavioral effects or a state with some subtle cognitive and vigilance decrements. Short sleepers appear to live with increased sleep drive in the daytime. Whether or not this disrupts daily function is not known?

References

[1] Aeschbach D, Cajochen C, Landolt HP, Borbély AA. Homeostatic sleep regulation in habitual short sleepers and long sleepers. Am J Physiol 1996;270:R41–R53.

[2] Lorenzo I, Ramos J, Arce C, Guevara MA, Corsi-Cabrera M. Effect of total sleep deprivation on reaction time and waking EEC activity in man. Sleep 1995;18:346–354.