SLOW WAVE ACTIVITY IN THE FIRST NREM EPISODE IS A TRAIT MARKER IN ADDITION TO A HOMEOSTATIC STATE MARKER

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INTRODUCTION

It has been well established that slow wave activity (SWA) in the NREM sleep EEG is increased during recovery sleep following total sleep deprivation (relative to baseline sleep).[1] However, recent experiments involving repeated exposure to sleep deprivation have indicated that SWA also varies systematically among individuals independently of prior wakefulness.[2] Thus, it appears that SWA is both a sleep-homeostatic state marker as well as a trait marker. Given that the effect or sleep deprivation on SWA is typically strongest in the beginning of the sleep period,[3] we re-examined the state and trait aspects of SWA using only data from the first NREM episode of recovery sleep.

METHODS

Twenty healthy good sleepers (ages 29.1 ± 5.4; 10 females) spent 11 consecutive days and nights in a sleep laboratory. During this time, they underwent three 36-hour periods of sleep deprivation. Each sleep deprivation period was preceded by a baseline sleep opportunity and followed by a recovery sleep opportunity. Every sleep period consisted of 12 hours time in bed (TIB), with subjects going to bed at 22:00 and getting up at 10:00. See Figure 1 for further details on the study protocol.

All sleep periods were recorded polysomnographically, with electrodes placed at EEG derivations Fz, C3, C4 and Oz referenced to A1/A2. The polysomnographic records were visually scored in 30-second epochs according to the criteria set by Rechtschaffen and Kales.[4] SWA, defined here as the average spectral power in the 0.75–4.5 Hz frequency band of the EEG per 30-second epoch of NREM sleep, was assessed for the first NREM episode of each sleep period by means of spectral analysis.

The three baseline and the three recovery nights were subjected to mixed-effects ANOVA, with prior sleep deprivation as the main effect, and a random effect over subjects on the intercept. The magnitude of the main effect, corresponding to the average difference in SWA between baseline and recovery sleep, was used as a marker of the state aspect of SWA in this experiment. This was compared quantitatively to the standard deviation over subjects for the random effect on the intercept, representing the systematic individual differences encountered across all six sleep periods, and constituting a marker of the trait aspect of SWA in the first NREM episode.
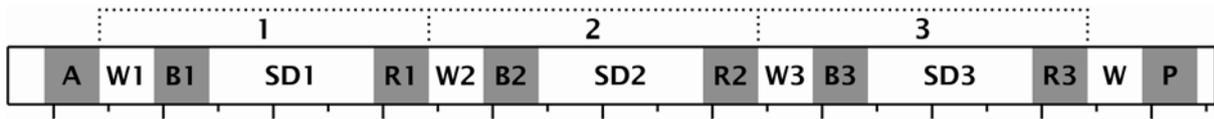
Figure 1. Schematic of the 11-day laboratory study protocol. Gray areas represent nighttime sleep opportunities; white areas represent periods of scheduled wakefulness. Bottom tic marks indicate midnight (long) and noon (short). The experiment began with a 12-hour adaptation night (A). Subsequently, there were three iterations (labeled 1 through 3) of the following pattern: 12 hours of scheduled wakefulness (W); 12 hours TIB for baseline sleep (B); 36 hours of total sleep deprivation (SD); and 12 hours TIB for recovery sleep (R). The experiment ended with an additional 12-hour wakefulness period (W) and a 12-hour pre-departure sleep opportunity (P). Each of the scheduled sleep periods began at 22:00 and ended at 10:00.

RESULTS

For every EEG derivation, there was a significant increase of SWA in the first NREM episode of recovery sleep compared to baseline sleep ($F_{1,56} > 33.1$, $P < 0.001$)—see Fig. 2. In addition, there was significant between-subjects variability, observed systematically across all six sleep periods considered ($Z > 2.7$, $P < 0.004$)—see Fig. 3.

For the Fz derivation, the standard deviation for systematic individual differences across all six sleep periods was 21.7% greater than the average difference in SWA between baseline and recovery sleep. Results for the other EEG derivations were similar, indicating that the magnitude of trait individual differences in SWA was systematically greater than the magnitude of the state effect of 36 hours of total sleep deprivation on SWA in the first NREM episode.

After removal of the subject with the most extreme SWA expression (farthest to the right in Fig. 3), there were no notable changes in the results, and trait individual differences remained the dominant source of variance.
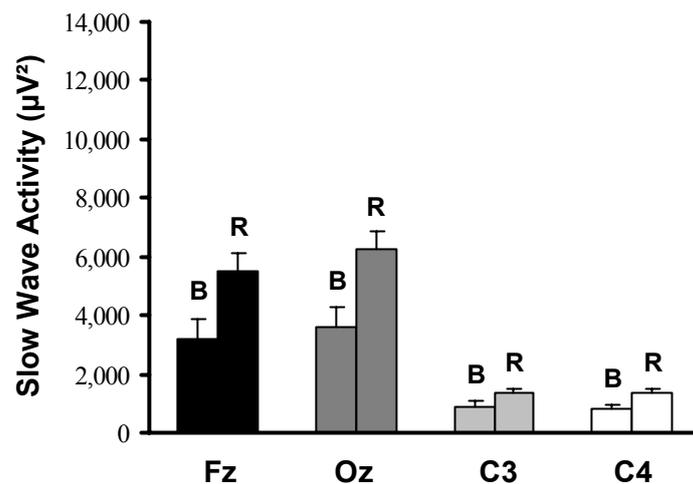
Figure 2. Group-average SWA in the first NREM episode during baseline (B) and recovery (R) nights for the Fz, Oz, C3 and C4 EEG derivations (whiskers represent standard error).
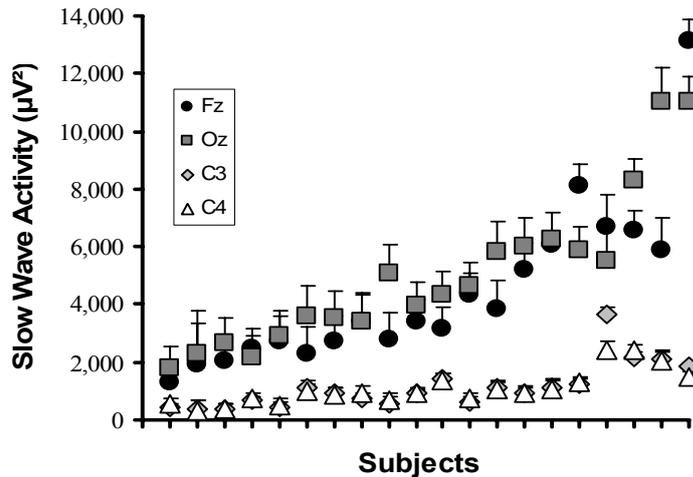
Figure 3. SWA in the first NREM episode for the four EEG derivations, averaged across nights for each individual subject and plotted in ascending order of overall SWA (whiskers indicate standard error). Note that the vertical scale is the same as in Fig. 2, facilitating visual comparison of state effects (Fig. 2) and trait effects (Fig. 3).

DISCUSSION

Our results indicate that SWA in the first NREM sleep episode was not only a robust marker of sleep homeostasis (i.e., it is a state marker)—it was also consistently different among individuals regardless of sleep homeostatic state (i.e., it is also a trait marker). The magnitude of systematic individual variability in our sample of healthy young adults (trait estimate) was greater than the magnitude of the effect of 36 hours of prior wakefulness (state estimate). By focusing on the first NREM sleep episode, any potential confounds from individual differences in sleep duration were avoided. Take together, our results suggest that SWA in the first NREM episode is *predominantly* a trait marker in addition to being a state marker.

The present results are in agreement with the qualitative observation by Finelli and colleagues that the magnitude of variability among individuals with regard to spectral power in the NREM sleep EEG exceeds the magnitude of the effect of prior sleep deprivation.[5] Furthermore, our finding parallels the earlier observation by our group that whole-night NREM sleep SWA is chiefly a trait marker in addition to being a state marker.[2]

Current models of sleep homeostasis do not account for trait individual differences in SWA.[1] Further research is needed to determine whether the trait and state aspects of NREM sleep SWA can *simultaneously* be understood in terms of sleep homeostatic mechanisms.

ACKNOWLEDGMENTS

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