Frontal Electroencephalogram Alpha Asymmetry During Sleep: Stability and Its Relation to Affective Style

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Electroencephalogram (EEG) alpha (8–12 Hz) asymmetries were collected from the mid-frontal and central regions during presleep wakefulness and Stage 1, Stage 2, and rapid eye movement (REM) sleep in 11 healthy right-handed participants who were free of psychiatric, neurological, and sleep problems. The authors found significant correlations between presleep wakefulness and different stages of sleep in the frontal, but not central, EEG alpha asymmetry measure. The strongest correlation was between presleep waking and REM sleep, replicating and extending earlier work to a normal population. The high degree of association between presleep waking and REM sleep may be a result of high cortical activation common to these states and may reflect a predisposition to different styles of emotional reactivity.

A number of studies have shown that the pattern of resting electroencephalogram (EEG) alpha asymmetry recorded from the anterior portion of the scalp is related to a predisposition to experience positive and negative emotion and is predictive of individual differences in affective style (see Davidson, 1993, 2000, for reviews). For example, individuals who exhibit greater relative left frontal resting EEG activation are sociable and outgoing, whereas individuals who display greater relative right frontal resting EEG activation are shy and socially anxious (Schmidt, 1999). A similar relation between resting frontal EEG asymmetry and affective style has been noted in studies of EEG and temperament in infants and children (see, e.g., Fox, 1991; Fox, Henderson, Rubin, Calkins, & Schmidt, 2001, for reviews).

If the frontal EEG alpha asymmetry metric is a traitlike marker of dispositional affective style, then there should be stability across time and contexts in this metric. Davidson and his colleagues (Tomarken, Davidson, Wheeler, & Kinney, 1992) noted very good stability in waking frontal EEG alpha asymmetry and power measures across 3 weeks in adults. We have noted excellent short-term stability in frontal EEG asymmetry measures across several minutes in infants (Schmidt, 2001) and children (Schmidt, 1996). To date, although there have been relatively few studies that have examined the stability of the waking frontal EEG alpha asymmetry metric across time, there have been still fewer studies that have examined this issue across different levels of arousal such as varying depths of sleep stages. There appears to be only one study in the literature that has examined this very issue, and this was by Benca and Davidson and their colleagues (Benca et al., 1999).

Benca et al. (1999) examined stability and changes in regional EEG alpha asymmetries and power in waking and different sleep stages. The authors noted that there was stability across waking and sleep stages for frontal EEG alpha asymmetry, particularly during rapid eye movement (REM) sleep in adult participants. REM sleep, comprising about 20% of night, is
entered into periodically every 90 min following sleep onset, with each successive REM period being longer in duration. Cortical activation, binocularly synchronous eye movements, muscle paralysis, and vivid dreaming characterize REM sleep. These researchers also noted that, despite the stability in frontal EEG asymmetry across different sleep stages, there was a bias for greater EEG alpha power at right-sided sites compared with left-sided sites during sleep, particularly in the frontal region.

Another study by Davidson’s group (Donzella, Davidson, Stickgold, & Hobson, 1994) attempted to link the pattern of individual differences in waking frontal EEG alpha asymmetry to the emotional content of dreams. The authors found that individual differences in waking frontal EEG asymmetry predicted the emotional quality of dreaming, with individuals who exhibited greater relative right frontal EEG alpha activation reporting more negative affect during dreaming compared with left frontal activated individuals. However, it may be the waking dream report that differs in these individuals rather than a difference in the emotional content of the dream itself.

The primary goal of the present study was to replicate the overall findings from the Benca et al. (1999) study on the stability of frontal EEG alpha asymmetry during sleep and to extend these findings to a normal rather than patient population. Although the stability in waking frontal EEG alpha asymmetry has been replicated, there appears to be no studies that have attempted to replicate the findings reported by Benca and Davidson and their colleagues on the stability of this metric between waking and sleep (Benca et al., 1999; Donzella et al., 1994). We recorded EEG during presleep waking and different sleep stages from the left and right mid-frontal and central sites in healthy adults. We computed a separate EEG asymmetry measure for the mid-frontal and central sites using ln(alpha EEG power right) minus ln(alpha EEG power left). Because EEG power is thought to be inversely related to activation, negative scores on this metric are thought to reflect greater relative right activation (see Davidson & Tomarken, 1989). We predicted that there would be stability across presleep waking and different sleep stages for the frontal EEG alpha asymmetry measure, with a particularly strong relation between presleep waking and REM sleep, given that REM sleep is a state of high cortical activation similar to wakefulness and that it is associated with emotional reactivity of dreaming.

A second goal of the present study was to extend previous research by examining whether the pattern of frontal EEG alpha asymmetry during sleep was related to waking affective style. Participants completed a 20-item psychometric measure of Gray’s (1990) motivational model of the behavioral inhibition system (BIS) and the behavioral activation system (BAS) that are known to be reliable measures of affective style (i.e., the BIS/BAS scale developed by Carver & White, 1994). Previous studies have related this scale to individual differences in waking frontal EEG alpha asymmetry. Sutton and Davidson (1997), for example, found that individuals who exhibited greater relative left frontal EEG activation during waking were likely to score high on behavioral activation relative to behavioral inhibition. We predicted that greater relative right frontal EEG activation across waking and sleep stages would be related to high BIS scores.

Method

Participants

Participants were 11 adults (M = 33.27 years, SD = 6.71 years) who were part of a larger investigation of the effects of experimentally induced sleep fragmentation on daytime performance. Participants were recruited through advertisements at Brock University and through public service announcements in the community. The inclusion criteria for participation were good sleepers (e.g., keep regular sleep schedules between the approximate hours of 23:00–07:00, no history of difficulty initiating or maintaining sleep), 25–45 years of age, healthy (i.e., no current use of medications), right-handed, and nonsmokers. Through initial telephone interviews, participants were further excluded if they worked shifts, consumed excessive amounts of caffeine, or had any history of depression, head injury, neurological disease, or chronic pain. If suitable, candidates were invited to an orientation session at Brock University Sleep Research Laboratory to complete questionnaires regarding sleep/wake habits and history and personality measures (i.e., the BIS/BAS measure; Carver & White, 1994). The protocol was approved by the Research Ethics Board.

Procedures

The entire experiment involved 96 hr of continuous study of nighttime sleep and daytime performance. The data in the present study were taken from the initial baseline night (i.e., prior to any disruption from sleep deprivation procedures). Prior to the baseline night, participants were screened for sleep disorders in a single-night off-protocol using standard polysomnographic procedures. The data from their baseline night may thus be considered more typical of their
usual sleep quantity and quality because it was not their first night in the laboratory environment.

EEG was recorded with gold electrodes attached to the left and right mid-frontal (F3, F4) and central (C3, C4) regions of the scalp. All electrodes were referenced to the left mastoid site (A1). In addition, A2 was recorded for the purpose of offline rereferencing to a balanced reference (i.e., the average of A1 and A2). Analog filters for the Lamont digital amplifiers from the right mastoid site, Model HBX32-SLP (Stellate Systems, Montreal, Canada) were fixed at .05–70 Hz. In addition, eye movements were recorded from electrodes placed on the outer canthus of each eye, and electromyographic was recorded from electrodes under the chin. All physiological signals were sampled at 200 Hz. Data were sleep scored offline, according to standard methods (Rechtschaffen & Kales, 1968), by experienced raters. Sleep scorers had established their overall interater reliability to be above 92%.

Artifact was removed by visual inspection and 3-min epochs of sleep were selected for analysis. To examine the sleep onset period, 3-min epochs were taken from presleep wake, Stage 1, and Stage 2—the epochs were the first uninterrupted 3 min of the given stage during the sleep onset period. For the purpose of controlling for possible time-of-night differences, a 3-min epoch of Stage 2 was taken from the second half of the night. The selection of the late Stage 2 epoch was calculated for each participant separately (time in bed: sleep onset latency/2). The first 3 min of continuous Stage 2 following this halfway point marked the epoch for fast Fourier transformation (FFT) analysis. FFT values were calculated for every 5.12 s of data that were available after artifact rejection (spectra record length). Within each spectra record, 2.56-s FFT analyses were performed and averaged together. A hanning window was used, as well as a 75% overlap of spectra records. Slow-wavesleep (SWS) was investigated by taking the first 3 min of uninterrupted Stage 3 and Stage 4, respectively. Because of the nature of sleep/wake architecture across the night, the SWS data would have been mostly from the first half of the night. Each REM period was investigated (i.e., FFT on first uninterrupted 3-min epoch for all REM periods). Although most sleepers will experience 4–5 REM periods per night, the first REM period (expected within 60–90 min following sleep onset) is quite short in duration and sometimes absent. The first appearance of an REM period, containing at least three consecutive minutes, was categorized as “REM 1” for each individual participant, regardless of the time of night in which it occurred.

Power spectral analysis was conducted on presleep waking and Stage 1, Stage 2, and REM sleep. Prior to any analysis, data were rereferenced. The rereferencing montage was an average of A1 and A2. The EEG bandwidth preselected for analysis was 8–12 Hz (alpha). A separate asymmetry score (i.e., ln [alpha power right] minus ln [alpha power left]) was computed for mid-frontal and central sites for presleep waking and sleep data. Negative scores on this metric are thought to reflect greater relative right EEG activation (Davidson & Tomarken, 1989).

Results and Discussion

Is Frontal EEG Alpha Asymmetry Stable Across Waking and Different Sleep Stages?

Because frontal EEG alpha asymmetry has been found to be a traitlike marker of dispositional affective style, we first examined the stability of this metric across different sleep stages. We computed a series of Pearson product–moment correlations between EEG alpha asymmetry during presleep waking and each of the sleep stages separately for the mid-frontal and central regions (see Figure 1). As can be seen in Figure 1A, significant correlations emerged between mid-frontal EEG alpha asymmetry during presleep waking and Stage 1 REM sleep.1 As expected, individuals who exhibited greater relative right frontal EEG alpha activation during presleep waking were likely to exhibit a similar pattern of right frontal EEG alpha asymmetry during these sleep stages, replicating previous work (Benca et al., 1999). In addition, as predicted, we noted the strongest relation between frontal alpha asymmetry during presleep waking and REM sleep, replicating previous findings (Benca et al., 1999). It is also important to point out that none of the relations between central alpha asymmetry during presleep waking and the sleep stages was significant (see Figure 1B).

We should also note that our presleep wake period differed from that of the Benca et al. (1999) article (i.e., alert wakefulness). Specifically, presleep waking in good sleepers is a period of relaxed wakefulness that would be most similar to that of the eyes-closed data recorded by Benca et al. (1999; i.e., more alpha). There were no Stage 1 characteristics (i.e., EEG slowing, alpha suppression, or slow-rolling eye movements) in these epochs of presleep waking.

1 Note that when the one outlier is removed from the Stage 2/mid-frontal correlation (see Figure 1A, second panel), this correlation is also significant (r = .75, p = .01).
A. Mid-frontal (F4 – F3) Asymmetry

B. Central (C4 – C3) Asymmetry

Figure 1. Scatterplots of the relation between alpha asymmetry (right-left) during presleep waking and each sleep stage for the mid-frontal (A) and central (B) regions. REM = rapid eye movement.
Are There Changes in Regional EEG Alpha Power Across Different Sleep Stages?

Although we found stability in the frontal EEG alpha asymmetry measure between presleep waking and different sleep stages, there were changes in absolute EEG power across different sleep stages. Accordingly, we examined whether any of these changes were significant. We performed an analysis of variance (ANOVA), with region (mid-frontal, central), hemisphere (left, right), and condition (presleep waking, Stage 1, Stage 2, REM 1, REM 2) as within-subjects factors, on $\ln$ (8–12 Hz) EEG alpha power. Figure 2 presents the mean EEG power values for the mid-frontal (A) and central (B) regions by hemisphere and condition. The analysis revealed a significant main effect for hemisphere, $F(1, 10) = 38.34, p < .01$. As expected, participants exhibited significantly more EEG alpha power (i.e., less activation) in the right hemisphere across presleep waking and each sleep stage for both regions (see Figure 2). The analysis also revealed a trend for a significant main effect for condition, $F(4, 40) = 2.40, p = .066$. As expected, participants tended to exhibit more EEG alpha power

![Figure 2](image-url)
(i.e., less activation) during presleep waking and the least alpha power during the REM periods (see Figure 2), replicating previous work (Benca et al., 1999). A series of pairwise t tests supported these overall findings: waking > REM 2, t(10) = 2.35, p = .04; Stage 1 > REM 1, t(10) = 3.22, p = .01; Stage 2 > REM 2, t(10) = 4.13, p < .01; and REM 1 > REM 2, t(10) = 3.24, p = .01. Given that EEG alpha power is inversely related to activation (Davidson & Tomarken, 1989), these findings suggest that during sleep, participants were more left activated. This is consistent with the findings from Benca et al. (1999), and also as noted by a recent functional imaging study that described increased activation of left-sided thalamus during REM sleep (Maquet et al., 1996) and decreased activation of right-sided structures (e.g., precuneus and mediotemporal cortex) during slow wave sleep (Maquet et al., 1997), although it is also important to note that Maquet’s group also found decreased dorsolateral prefrontal activation during REM sleep, suggesting that there is some dissimilarity between the pattern of frontal activation observed on electrocortical and imaging indices during REM sleep.

Is the Pattern of Frontal Alpha Asymmetry During Sleep Related to Waking Affective Style?

If the frontal EEG alpha asymmetry measure is a traitlike marker of affective style, it should be related to affective style across different sleep stages. However, the magnitude of the expected correlation between the BIS/BAS measure and the frontal EEG alpha asymmetry metric during sleep was too small to be detected with the sample size reported in the present study, which is perhaps why none of the correlations between the BIS/BAS and the frontal EEG alpha asymmetry metric was significant, with the notable exception of Stage 2 sleep (r = -.67, p < .05). Individuals who exhibited greater relative right frontal EEG alpha activation during Stage 2 sleep were likely to score high on the BIS scale. Interestingly, Stage 2 sleep is characterized by 12–14 Hz spindle activity thought to reflect inhibition (Steriade & Amzica, 1998). It is also important to point out that the original Sutton and Davidson (1997) article on the relation between resting frontal EEG asymmetry and the BIS/BAS measure had considerably more participants in their study than the present study. As well, the participants in the Sutton and Davidson (1997) study were tested on two separate occasions in order to increase the reliability of the EEG asymmetry measure. The two points just noted highlight two possible limitations of the present study. First, we tested only 11 participants. Second, we tested participants on only one occasion. Although EEG/sleep studies are labor intensive and expensive to conduct, future studies need to test more participants and on separate occasions than we presented here to examine whether the present findings are reliable and generalizable.

Overall, the present findings suggest that the pattern of frontal EEG alpha asymmetry is stable across different sleep stages. Moreover, it is reasonable to speculate that given the stability in the pattern of frontal EEG alpha asymmetry between waking and REM sleep, frontal EEG alpha asymmetry during REM sleep thus may be predictive of emotional reactivity during dreaming, although we did not have any direct measures of the emotional content of the participants’ dreams to corroborate this notion. Future studies may want to consider waking the participant during or immediately after REM sleep and assessing dream content at that point. This may circumvent potential problems resulting from differences that may exist in participants’ waking dream subjective report versus actual differences in the emotional content of their dreams given that it would more closely link temporally to the actual dream state. Alternatively, the stability in the pattern of frontal EEG asymmetry between waking and REM sleep may be explained by the similar brain activation state between wakefulness and REM sleep (Kahn, Pace-Schott, & Hobson, 1997). Nevertheless, the present findings appear to be the first to replicate earlier work on the stability of frontal EEG alpha asymmetry during sleep in a normal, healthy group of good sleepers. Individual differences in frontal EEG alpha asymmetry during sleep, if shown reliable through subsequent study, may be useful to predict waking affective style.

References


**BRIEF REPORTS**

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