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The first-night effect suppresses the strength of slow-wave activity originating in the visual areas during sleep

Masako Tamaki, Ji Won Bang, Takeo Watanabe, Yuka Sasaki *

Department of Cognitive, Linguistic, and Psychological Sciences, Brown University, Box 1821, 190 Thayer Street, Providence, RI 02912, USA

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ABSTRACT

Our visual system is plastic and adaptive in response to the stimuli and environments we experience. Although visual adaptation and plasticity have been extensively studied while participants are awake, little is known about what happens while they are asleep. It has been documented that sleep structure as measured by sleep stages using polysomnography is altered specifically in the first sleep session due to exposure to a new sleep environment, known as the first-night effect (FNE). However, the impact of the FNE on spontaneous oscillations in the visual system is poorly understood. How does the FNE affect the visual system during sleep? To address this question, the present study examined whether the FNE modifies the strength of slow-wave activity (SWA, 1–4 Hz)—the dominant spontaneous brain oscillation in slow-wave sleep—in the visual areas. We measured the strength of SWA originating in the visual areas during the first and the second sleep sessions. Magnetoencephalography, polysomnography, and magnetic resonance imaging were used to localize the source of SWA to the visual areas. The visual areas were objectively defined using retinotopic mapping and an automated anatomical parcellation technique. The results showed that the strength of SWA was reduced in the first sleep session in comparison to the second sleep session, especially during slow-wave sleep, in the ventral part of the visual areas. These results suggest that environmental novelty may affect the visual system through suppression of SWA. The impact of the FNE may not be negligible in vision research.

1. Introduction

A growing body of evidence suggests that our visual system continues to be plastic during sleep. In the early postnatal brain, ocular dominance plasticity takes place not only during wakefulness after visual experiences, but also during subsequent sleep in young cats and mice (Aton et al., 2009, 2013; Frank, Issa, & Stryker, 2001; Hensch, 2005). Performance in visual tasks significantly improves after sleep in adult humans (Gais et al., 2002; Karni et al., 1994; Stickgold, James, & Hobson, 2000; Yotsumoto et al., 2009). Moreover, sleep deprivation has been shown to nullify sleep facilitation effect in visual tasks (Stickgold, James, & Hobson, 2000). These studies suggest that sleep plays a critical role in visual plasticity. However, the detailed neural mechanism of this sleep-dependent visual plasticity is not completely understood.

Importantly, to investigate the neural mechanism of sleep-dependent visual plasticity, sleep quality would be a crucial factor that needs to be controlled, because it may affect brain activities and plasticity during sleep (Dresler et al., 2010; Manoach & Stickgold, 2009). The first-night effect (FNE) is a sleep disturbance that is observed particularly in the first session of sleep experiments (Agnew, Webb, & Williams, 1966; Rechtschaffen & Verdone, 1964; Tamaki et al., 2005a), which could be a confounding factor in the field of sleep research. In the first sleep session, latencies to sleep onset and to rapid-eye movement (REM) sleep are longer, the proportion of wakefulness during bed time is increased, and total sleep time is decreased, compared to the second sleep session (Agnew, Webb, & Williams, 1966; Curcio et al., 2004; Tamaki et al., 2005a, 2005b; Tamaki, Nittono, & Hori, 2005). Sleep quality improves significantly in the second sleep session in younger and healthy participants (Agnew, Webb, & Williams, 1966; Lorenzo & Barbanoj, 2002). To control for the FNE, it has been recommended to incorporate adaptation sleep sessions before experimental sleep sessions, which would let participants adapt to a new sleep environment (Tamaki, Nittono, & Hori, 2005).

Although the FNE impact on sleep structures has been well documented, the FNE impact on spontaneous oscillations and plasticity during sleep is yet to be investigated. In particular, it is imperative to examine how the FNE modifies slow-wave activity (SWA, 1–4 Hz), one of the spontaneous oscillatory activities during non-rapid-eye movement (NREM) sleep, in the visual area, because SWA is suggested to be involved in various types of learning including visual perceptual learning (Aeschbach, Cutler, & Ronda,

* Corresponding author.
E-mail addresses: tamaki@brown.edu (M. Tamaki), ji_won_bang@brown.edu (J.W. Bang), takeo_watanabe@brown.edu (T. Watanabe), yuka_sasaki@brown.edu (Y. Sasaki).

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2008; Born & Wilhelm, 2012; Tamaki et al., 2013; Tononi & Cirelli, 2003). If SWA in the visual area is affected by the FNE, this will pose a critical confound for studies investigating the mechanisms of visual plasticity during sleep. In this case, using the data of the first sleep session should be discouraged. On the other hand, if the impact of the FNE on SWA originating in the visual area is small, we may not need to consider the FNE in future studies of sleep-dependent visual plasticity, which would save time and money for vision sleep research. Thus, it is necessary to ask whether the FNE modulates SWA in the visual cortex.

To our knowledge, however, there is no clear consensus on whether the FNE modifies SWA in the visual area, as argued below. First, we have found 58 papers on the FNE, among which there were only 14 papers that employed a young (age range: 16–39 yrs) and healthy population to investigate the FNE using polysomnography (PSG) measurement of at least two nights (Agnew, Webb, & Williams, 1966; Browman & Cartwright, 1980; Coble et al., 1974; Curcio et al., 2004; Kajimura et al., 1998; Lorenzo & Barbanoj, 2002; Rechtschaffen & Verdone, 1964; Rosadini et al., 1983; Sharpley, Solomon, & Cowen, 1988; Suetsugi et al., 2007; Tamaki et al., 2005a, 2005b; Tamaki, Nittono, & Hori, 2005; Toussaint et al., 1997). Among those, there were only a handful of studies that examined the impact of the FNE on the strength of SWA, using a power spectral analysis (Curcio et al., 2004; Tamaki et al., 2005b; Toussaint et al., 1997). The impacts reported were contradictory: increase in central brain regions (Curcio et al., 2004), decrease in frontal brain regions (Tamaki et al., 2005b), and no changes (Toussaint et al., 1997) in the first compared to the second sleep session. Unfortunately, there is no study that has investigated SWA in the occipital area.

Second, which brain regions, including in the visual areas, are susceptible to the FNE is also unclear, as modification of SWA strength could take place not only evenly in the brain but also in a particular region. Recent studies have suggested that SWA can be modulated and generated locally (Huber et al., 2004; Tononi & Cirelli, 2003; Vyazovskiy et al., 2011), not uniformly across brain regions, and may propagate across other brain regions from the frontal regions (Massimini et al., 2004; Nir et al., 2011). Moreover, the susceptibility of SWA to the FNE may not be consistent across the brain (Cajochen et al., 2006). The study by Cajochen et al. (2006) reported that the age-related reduction of SWA was prominent only in the frontal region, but not in the occipital region. This line of studies suggests that SWA in the visual area could be either suppressed by or resilient to the FNE locally, independent of other brain regions such as the frontal (where SWA decreased, as reported by Tamaki et al., 2005b) or central (where SWA increased, as reported by Curcio et al., 2004) brain regions. On the other hand, global decreases of SWA, including occipital areas, is also possible, due to sleep deterioration (Aeschbach, Cutler, & Ronda, 2008; Carrier et al., 2011; Walsh, 2009; Westerberg et al., 2012). Thus, it is also possible that SWA could be decreased globally including the visual area by the FNE.

In other words, whether the FNE impacts SWA in the visual area may depend on how globally the FNE impacts the brain. That is, if the FNE impacts the brain globally, it is possible that the strength of SWA is decreased in the visual areas, as well as in the frontal areas, due to deterioration in sleep quality. On the other hand, if the FNE impacts locally in a limited region (i.e., frontal areas), the strength of SWA may not be decreased in the visual area in the first compared to the second session. Moreover, the impact of the FNE on SWA may differ among the visual areas, because functional distinctions including attention and perception among the visual areas, such as the ventral and dorsal early visual areas, and the higher visual areas, have been suggested (Goodale, 1993; He, Cavanagh, & Intriligator, 1996; Mishkin & Ungerleider, 1982; Previc & Mullen, 1990; Rubin, Nakayama, & Shapley, 1996). Furthermore, the strength of SWA could even be increased in the visual area in the first sleep session: in the first sleep session, subjects are exposed to a new sleep environment with new objects and equipment, which could increase visual usage during wakefulness, thereby potentiating visual neurons to facilitate visual plasticity (Torasdotter et al., 1996, 1998). According to the synaptic homeostasis hypothesis (Tononi & Cirelli, 2003), SWA should be increased in the brain regions that have been extensively used and in synapses potentiated during prior wakefulness. Thus, the extensive usage of the visual area during wakefulness may result in SWA increase in the visual area during the subsequent sleep in the first sleep session.

In the present study, we tested whether the SWA strength is changed in the visual area in the first compared to the second session. If the FNE impacts the brain globally, the strength of SWA may be *decreased* in the visual area in the first compared to the second sleep session. On the other hand, if the impact of the FNE is local, then the strength of SWA may not be decreased in the visual area due to the FNE; in this case there would be two scenarios. In one, the strength of SWA may be *increased* in a visual area if the synaptic strength were greatly increased in the visual area (Tononi & Cirelli, 2003). In the other, there would be no change in SWA of a visual area in the first sleep session compared to the second, if neither the FNE nor visual plasticity affect the visual area, or if both affect the visual area and cancel each other out. To test these possibilities, we compared the strength of SWA between the first and second sleep sessions in three visual areas: the ventral and dorsal early visual areas, and the object area of the higher visual area. Furthermore, to compute SWA originating in these visual areas, we employed a source-localization technique (Ahveninen et al., 2007; Lin et al., 2004) with a combination of magnetoencephalography (MEG), PSG and magnetic resonance imaging (MRI). We used this method because the advanced source-localization technique would allow us to measure the SWA strength in the sub-regions within the visual area with high spatial resolution (Ahveninen et al., 2007; Dale & Sereno, 1993; Dale et al., 2000; Lin et al., 2004).

2. Material and methods

The present paper is based on a new analysis of our past projects that utilized the MEG and MRI source localization technique on sleeping brain. The past projects had different purposes, thus, the original experimental designs were slightly different. This section describes both the original designs and how we conducted the analysis of the present paper.

2.1. Subjects

After experimenters thoroughly described the purpose and procedure of experiments to the subjects, potential subjects completed questionnaires regarding their sleep–wake habits; usual sleep and wake time, regularity of their sleep–wake habits and lifestyle, habits of nap-taking, and information regarding their physical and psychiatric health including sleep complaints. Anyone with physical or psychiatric disease, currently receiving medical treatment, or suspected of having a sleep disorder was excluded. People who had the habit of taking a nap, consuming alcoholic beverages before sleep, or smoking were also excluded. Only people who had regular sleep–wake cycles were included, i.e., differences between average bedtimes, sleep durations and wake-up times on weekdays and weekends were less than 2 h. The average sleep duration for each potential subject ranged from 6 to 9 h regularly. The studies were approved by the institutional review board of the Massachusetts General Hospital where the data were collected and of

Brown University where the data were analyzed. All subjects gave written informed consent for their participation in experiments.

A total of 10 subjects data sets (5 females and 5 males, mean age 26.5 ± 0.99 years) were analyzed in the present paper. These subjects were chosen because they had MEG recordings for both the first and second night sleep sessions.

2.2. Experimental procedures

In the original experiments, three to four sleep sessions were conducted in total, depending on the project, followed by one MRI session. In all sessions, MEG and PSG were measured during sleep (for detail, see Section 2.3). In the original experiments, taking into account individual circadian rhythm variations, each subject's sleep time was set to their habitual sleep time for both sessions instead of enforcing a uniform time. On average, the lights were turned off around midnight, and the termination of recording was determined depending on the purpose of each project. In one project, the recording was terminated either when the subject showed REM sleep or when 90 min had elapsed. In another project, the recording was terminated when 180 min had elapsed. After the recording, the subjects were allowed to sleep for the rest of the night. The first and second nights were not consecutive in any of the projects. Starting three days before the onset of the experiment, subjects were instructed to maintain their sleep–wake habits, (i.e., their daily wake/sleep time and sleep duration). One day before each experiment, they were instructed to refrain from alcohol consumption, unusual physical exercise, and nap taking. Their sleep–wake habits were monitored by a sleep log to ensure the success of this procedure.

2.3. Data acquisition

MEG and PSG were simultaneously recorded in a magnetically shielded room. PSG consisted of EEG, electromyogram (EMG), electrooculogram (EOG), and electrocardiogram (ECG). EEG was recorded at 4–7 scalp sites (including C3, C4, O1, O2, Fz, Cz, Pz) according to the 10–20-electrode system referenced to the nasion. EOG was recorded from two electrodes placed at the outer canthi of both eyes (horizontal EOG). EMG was recorded bipolarly from the mentum. ECG was recorded from two electrodes placed at the right clavicle and the left rib bone. Electrode impedance was kept below 5 kΩ. MEG data were collected using a 306-channel whole-head Vectorview system (Elekta Neuromag, Helsinki, Finland) with 204 planar gradiometers and 102 magnetometers. Both MEG and EEG data were recorded at a sampling rate of 600 Hz. The data was filtered between 0.1 and 99 Hz, and was re-sampled at 198 Hz. All epochs with changes that exceeded 3000 fT/cm at any MEG channel or that were contaminated by artificial noises were discarded. The positions of all scalp electrodes, anatomical landmarks including the nasion and two auricular landmarks, and four head-position indicator coils were measured using a FastTrack 3D digitizer (Polhemus, Colchester, VT). Head position within the MEG sensor array was measured at the beginning of the session. Five-minute empty room MEG recordings were also made immediately prior to each experiment for the purpose of estimating the noise covariance matrix (Ahveninen et al., 2007).

MRI anatomical data was used for determining the conductor geometry for the boundary element model (BEM) of the head (Hamalainen & Ilmoniemi, 1984; Hamalainen & Sarvas, 1989), and for registering the MEG sensors' locations with the individual subject's anatomy (Dale, Fischl, & Sereno, 1999; Fischl, Sereno, & Dale, 1999). Subjects were scanned in a 3 T MR scanner (Trio, Siemens); a head coil was used in all experiments. Three T1-weighted MR images (MPRAGE; TR = 2.531 s, TE = 3.28 ms, flip angle = 7°, TI = 1100 ms, 256 slices, voxel size = 1.3 × 1.3 × 1.0 mm) were acquired. Data was inflated for each participant for brain parcellation to localize individual gyri and sulci (Destrieux et al., 2010; Fischl et al., 2004). This information was used for the region-of-interest (ROI) analysis described later.

2.4. Sleep-stage scoring procedure and the definition of the FNE

EEGs were scored and classified into sleep stages for every 30-s epoch according to standard criteria (Rechtschaffen & Kales, 1968). EEG recordings from the C3 electrode were used for this scoring; if the C3 recordings were contaminated by artifacts, C4 recordings were used instead. The same scalp area was used for scoring across individuals and across nights. The following terminology was used for the stages of sleep: stage W (wakefulness), stage 1 (NREM sleep stage 1), stage 2 (NREM sleep stage 2), slow-wave sleep (NREM sleep stages 3 + 4), stage REM (sleep stage REM). Sleep onset was defined as the time taken to reach the first epoch of stage 2 after lights off. The following variables were calculated for each subject for assessment of basic sleep structure (sleep variables): latency to each sleep stage (min), the time spent in each sleep stage (min), and percent sleep efficiency [(Total sleep time/Total recording time) × 100].

While there could be various measures to indicate the presence of the FNE, we decided to use the latency to sleep onset since this has been shown to be indicative as the measurement of the FNE (Tamaki, Nittono, & Hori, 2005). Although the latency to stage REM may also be useful to quantify the FNE, this could not be used because data of stage REM was not necessarily measured due to the experimental procedure adopted in a past project (see Section 2.2).

2.5. Data entered into the present analysis

In the present study, we decided to analyze the data that were collected from the first two sleep sessions for the following two reasons. First, the experimental intervention to sleep took place between the end of the second sleep session and the start of the third sleep session in the majority of subjects. The task performed before the sleep session may have affected the subsequent sleep (Gais et al., 2000; Karni et al., 1994; Mascetti et al., 2013; Stickgold, James, & Hobson, 2000; Tamaki et al., 2013; Yotsumoto et al., 2009). Second, we were particularly interested in the difference between the first and second sleep sessions, because the FNE is largely reduced in the second session at least with regard to NREM sleep variables (Agnew, Webb, & Williams, 1966).

To compare the strength of SWA between the first sleep session and the second, we used the first sleep cycle of NREM sleep that occurred within 90 min from lights out, because all of the subjects had this segment.

2.6. MEG wavelet and source localization analysis

A Morlet wavelet analysis (Ahveninen et al., 2007; Lin et al., 2004) was applied to MEG raw data every 30-sec epoch to obtain spectral power from 1 to 4 Hz that corresponds to sleep SWA frequency range (Huber et al., 2004). To localize the current sources underlying the MEG signals, we employed the cortically constrained minimum-norm estimate (MNE) using individual anatomical MRI and constrained the current locations to the cortical mantle (Ahveninen et al., 2007). Information of the MEG sensors' locations and the structural MRI segmentation were used to compute the forward solutions for all source locations using a single-compartment boundary element method (BEM; Hamalainen & Sarvas, 1989). The individual forward solutions constituted the rows of the gain (lead-field) matrix. For inverse computations, the cortical surface was decimated to ~20,000

vertices per hemisphere. The noise covariance matrix was computed from the empty-room MEG data. These two matrices were used to calculate the inverse operator to yield the estimated source activity, as a function of time, on a cortical surface (Ahveninen et al., 2007). We did not use EEG data for the source localization, because the number of EEG channels was very small (4–7 channels) compared to MEG (306 channels).

We computed the average strength of SWA (1–4 Hz) on the first (Night 1) and the second (Night 2) sleep sessions in each of the ROIs (see Section 2.7). As the strength of SWA changes with elapsed time from sleep onset as well as with sleep stage, we examined the time-dependent and sleep-stage dependent components separately. First, to examine the time-dependent component of SWA, the mean of the MEG currents were calculated every 1 min for each ROI (see Section 2.7). Then, the time-course data was further divided into three epochs to test whether the FNE had an impact on a specific time window. Each epoch corresponded to a specific phase of the development of SWA, that is, initiation (epoch 1), development (epoch 2), and maintenance/decline (epoch 3) of SWA. Second, to examine the differential impact of the FNE among the sleep stages, a sleep-stage dependent component was computed. To compute the sleep-stage dependent component, the strength of SWA for every 30-s epoch that corresponded to each sleep-stage epoch, was averaged separately for each sleep stage. Since we defined sleep onset as the onset of stage 2, SWA during stage 2 and slow-wave sleep were computed as SWA during sleep. In addition, SWA during stage W was calculated as SWA during wakefulness.

2.7. ROI

We defined three ROIs: the ventral and dorsal early visual areas and the higher visual area, to test whether there are differences in the impact of the FNE among the regions. The procedure to localize these ROIs was as follows. We first identified the ventral and dorsal part of V1, V2, and V3 for each subject by a standard fMRI retinotopic mapping technique (Engel et al., 1994; Fize et al., 2003; Sereno, Mcdonald, & Allman, 1994; Yotsumoto, Watanabe, & Sasaki, 2008; Yotsumoto et al., 2009). The higher visual area was defined anatomically using an automated parcellation system (Fischl et al., 2004), as the posterior part of the inferior temporal sulcus that roughly corresponds to higher visual areas specializing in object processing (Kourtzi & Huberle, 2005).

For the retinotopic mapping, blood oxygen level dependent (BOLD) signals were acquired using a gradient echo EPI sequence (TR = 2 s, TE = 30 ms, Flip Angle = 90°). Twenty-five contiguous slices (3 × 3 × 3.5 mm) orientated orthogonal to the calcarine sulcus were acquired covering the occipital to parieto-temporal cortices. Data were analyzed with FSFAST and FreeSurfer (http://surfer.nmr.mgh.harvard.edu) software. All functional images were motion corrected (Cox & Jesmanowicz, 1999), spatially smoothed with a Gaussian kernel of 5.0 mm (FWHM), and normalized individually across scans. Functional data were registered to the individual reconstructed brain (Dale, Fischl, & Sereno, 1999; Fischl, Sereno, & Dale, 1999). The individually reconstructed brain was also used for an automated parcellation (Destrieux et al., 2010; Fischl et al., 2004) for objective anatomical segregation to localize higher visual area in this study.

3. Results

3.1. Sleep variables

First, we compared sleep structures on Night 1 and Night 2 to confirm that the FNE had occurred (Table 1 and Fig. 1). Sleep stages kept changing back and forth between different sleep stages on Night 1. In contrast, the sleep stage transition was smoother on Night 2 (Fig. 1). The sleep onset latency was significantly longer on Night 1 than on Night 2 ($t(9) = 2.438$, $p = .038$; two-tailed paired t test), which confirmed the presence of the FNE. In addition, latency to stage 1 was significantly different ($t(9) = 2.428$, $p = .038$). The time spent in stage W (Table 1, $t(9) = 2.190$, $p = .056$) tended to increase, the time spent in slow-wave sleep decreased ($t(9) = 2.389$, $p = .041$), and percentage sleep efficiency was significantly lower ($t(9) = 2.247$, $p = .049$;) on Night 1 compared to Night 2.

3.2. Time-course changes of SWA in the visual areas

We next investigated the time course of SWA on Nights 1 and 2. In both sessions, SWA increased over time; however, there was a notable difference between sessions (Fig. 2). The result showed that SWA was profoundly decreased on Night 1 throughout the epochs. A 3-way repeated measures ANOVA was conducted to see whether the following factors impacted the strength of SWA; the factors were ROI (ventral and dorsal part of early visual areas, and higher visual area), epoch (1–3), and session (Night 1 and 2). There was a significant main effect for all three factors (ROI, $F(2,18) = 53.642$, $p < .001$, $\varepsilon = 1$; epoch, $F(2,18) = 14.307$, $p < .001$, $\varepsilon = 1$; session, $F(1,9) = 5.156$, $p = .049$, $\varepsilon = 1$). In addition, the ROI × epoch interaction was significant ($F(4,81) = 9.707$, $p < .001$, $\varepsilon = .57$). However, no significant interaction was found for ROI × session, epoch × session, or ROI × epoch × session. This analysis indicates that the impact of FNE was present in all ROIs.

3.3. Decreased SWA during slow-wave sleep in the visual areas

Next, SWA was calculated for each sleep stage (stage W, stage 2, and slow-wave sleep) to examine the influence of the FNE on different sleep stages (Fig. 3). First, a 3-way repeated measures ANOVA was conducted to test whether the following factors impact on the strength of SWA; the factors were ROI (ventral and dorsal part of early visual area, and higher visual area), sleep stage (stage W, stage 2, and slow-wave sleep), and session (Night 1 and 2). There were significant main effects for ROI ($F(2,18) = 64.866$, $p < .001$, $\varepsilon = 1$) and sleep stage ($F(2,18) = 106.667$, $p < .001$, $\varepsilon = 1$), but not for session. In addition, significant interactions were found between ROI × sleep stage ($F(4,81) = 44.873$, $p < .001$, $\varepsilon = .37$), sleep stage × session ($F(2,54) = 6.328$, $p < .01$, $\varepsilon = .53$), and ROI × sleep stage × session ($F(4,162) = 5.496$, $p < .05$, $\varepsilon = 40$). No significant interaction was found in ROI × session. These results suggest that the impact of FNE differed depending on the sleep stage and on the ROI.

Since a significant interaction associated with the ROI factor was found, a 2-way repeated measures ANOVA was conducted subsequently for each ROI to test whether the following factors impacted the strength of SWA; the factors were sleep stage (stage W, stage 2, and slow-wave sleep) and session (Night 1 and 2). First, in the ventral part of the early visual area, there was a significant main effect for sleep stage ($F(1,9) = 149.478$, $p < .001$, $\varepsilon = .60$). Moreover, a sleep stage × session interaction was significant ($F(2,18) = 6.554$, $p < .001$, $\varepsilon = .97$). Subsequently, post hoc paired t-tests were conducted for each sleep stage to examine the difference in the impact of the FNE among sleep stages. There was a significant difference between sessions in slow-wave sleep ($t(9) = -2.225$, $p = .027$; two-tailed paired t test), but not in stage W or stage 2 ($ps > .05$). This analysis suggests that the strength of SWA in the ventral part of the early visual area was significantly reduced in the first-night sleep, especially in slow-wave sleep. In the dorsal part of the early visual area, a 2-way repeated measures ANOVA (factors = sleep stage and session) showed that

Table 1
Basic sleep structure on Night 1 and Night 2.

Sleep variables	Night 1 ($m \pm$ s.e.m.)		Night 2 ($m \pm$ s.e.m.)		p-Value[a]
Latency to sleep stage (min)					
Stage 1	7.7	2.36	3.0	0.65	0.038
Stage 2	15.2	3.98	7.4	1.91	0.038
SWS	31.1	5.60	21.1	3.09	0.112
Time in sleep stage (min)					
Stage W	15.2	5.32	5.0	1.55	0.056
Stage 1	7.5	1.67	5.2	1.04	0.128
Stage 2	29.4	2.96	33.6	3.37	0.390
SWS	25.9	5.39	37.9	3.64	0.041
Percentage sleep efficiency (%)	82.1	5.94	93.9	2.04	0.049

Since the total recording time varied depending on study protocol, all the sleep variables were calculated from the first sleep cycle of NREM sleep. Percentage sleep efficiency was calculated by [(Total sleep time/Total recording time) × 100]. SWS, slow-wave sleep.
[a] Two-tailed paired t test (alpha = 0.05).

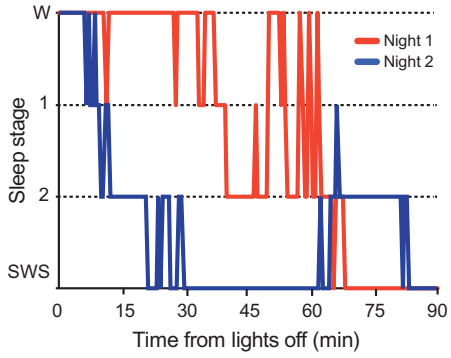

Fig. 1. Sleep hypnogram of a representative participant. Data were recorded for 90 min from lights out. *X*-axis corresponds to the elapsed time from lights out (min). *Y*-axis corresponds to each sleep stage (stage W, stage 1, stage 2, and slow-wave sleep). The red line indicates the stage transition during Night 1, and the blue line indicates the stage transition during Night 2. SWS, slow-wave sleep.

SWA was significantly different among sleep stages ($F(2,18) = 80.059$, $p < .001$, $\varepsilon = .56$). No significant main effect for session or sleep stage × session interaction was found. The analysis suggests that the FNE on the strength of SWA in the dorsal part of the early visual area is limited. For the higher visual area, a 2-way repeated measures ANOVA (factors = sleep stage and session) showed a significant main effect for sleep stage ($F(1,9) = 112.429$, $p < .001$, $\varepsilon = .40$). A sleep stage × session interaction was also significant ($F(2,18) = 6.950$, $p = .006$, $\varepsilon = .79$). Post hoc paired t tests conducted for each sleep stage revealed that there was a significant difference between sessions in slow-wave sleep ($t(9) = -1.997$, $p = .039$; two-tailed paired t test), but not in stage W or stage 2. This suggests that the strength of SWA in the higher visual area was significantly reduced in the first-night sleep, especially in slow-wave sleep.

The analyses so far showed that the impact of the FNE on SWA was significant in slow-wave sleep, especially in the ventral part of the early visual areas and the higher visual area, but not in the dorsal part of the early visual area.

3.4. Individual differences and the gender effect

We also examined individual differences and the gender effect on SWA. We subtracted the average SWA strength of Night 2 from that of Night 1 during slow-wave sleep for each visual area individually, and the FNE was determined to be present if the difference was smaller than zero. The results showed that nine out of ten subjects showed the FNE in at least one visual area (Table 2). When we divide the result for each visual area, seven (dorsal part of early visual area) or eight (ventral part of early visual area and higher visual area) out of ten subjects displayed the FNE in slow-wave sleep in the visual areas (Table 2). Next, we sorted the frequency of the FNE by the gender. There seems to be no significant gender differences in the frequency of the FNE in any of the visual areas (all $p > .05$, chi-square test for each visual area, Table 2).

4. Discussion

The present study examined whether the FNE has any impacts on the strength of SWA originating in the visual area. Because of limitations in experimental procedures, we could not compare all the sleep variables obtained here with those in previous studies that were computed from full-night recording. There is evidence, however, that the FNE that we documented in the present study may be comparable to previous studies. For instance, the change in sleep onset latency from Nights 1 to 2 (Table 1) were similar to those in previous studies (e.g., sleep onset latency was 14 min on Night 1, 7 min on Night 2, in Tamaki et al., 2005a). Duration of stage W tended to be larger on Night 1 compared to Night 2. Percent sleep efficiency was significantly lower on Night 1. Although the changes in some of the parameters did not reach significance, the alteration of the sleep structure recognized in previous research (Agnew, Webb, & Williams, 1966; Curcio et al., 2004; Toussaint et al., 1997) has also been indicated in our results.

Our results indicated that the impacts of the FNE were slightly different among the visual areas. First, we conducted the time-course analysis of SWA (Section 3.2, Fig. 2), which showed a reduction of SWA in Night 1 in comparison to Night 2 in all of three visual areas. This seems to support the view that the FNE has a global impact across brain regions when the analysis does not consider sleep stages. However, the second analysis by sleep stages illustrated the possibility that the impact of the FNE may not be ubiquitous in the visual system. The analyses on sleep stages (Section 3.3, Fig. 3) showed a significant main effect of ROI and a significant ROI × sleep stage × session interaction, among others. This suggests that the impact of the FNE is varied in different ROIs. Furthermore, the post hoc tests demonstrated a significant suppression of SWA, especially in the ventral part of the early visual area and the object area of the higher visual area, whereas we did not find a significant suppression in SWA in the dorsal part of the early visual area. These results may be a piece of counterevidence to the claim that the impact of the FNE is homogeneous across brain regions.

Interestingly, there was little suppression in SWA in the dorsal part of the early visual area. This rejects the assumption that the FNE should reduce the strength of SWA uniformly in the brain. The result may suggest that the impact of the FNE was negligible in this region, or that the influence of the FNE may have been

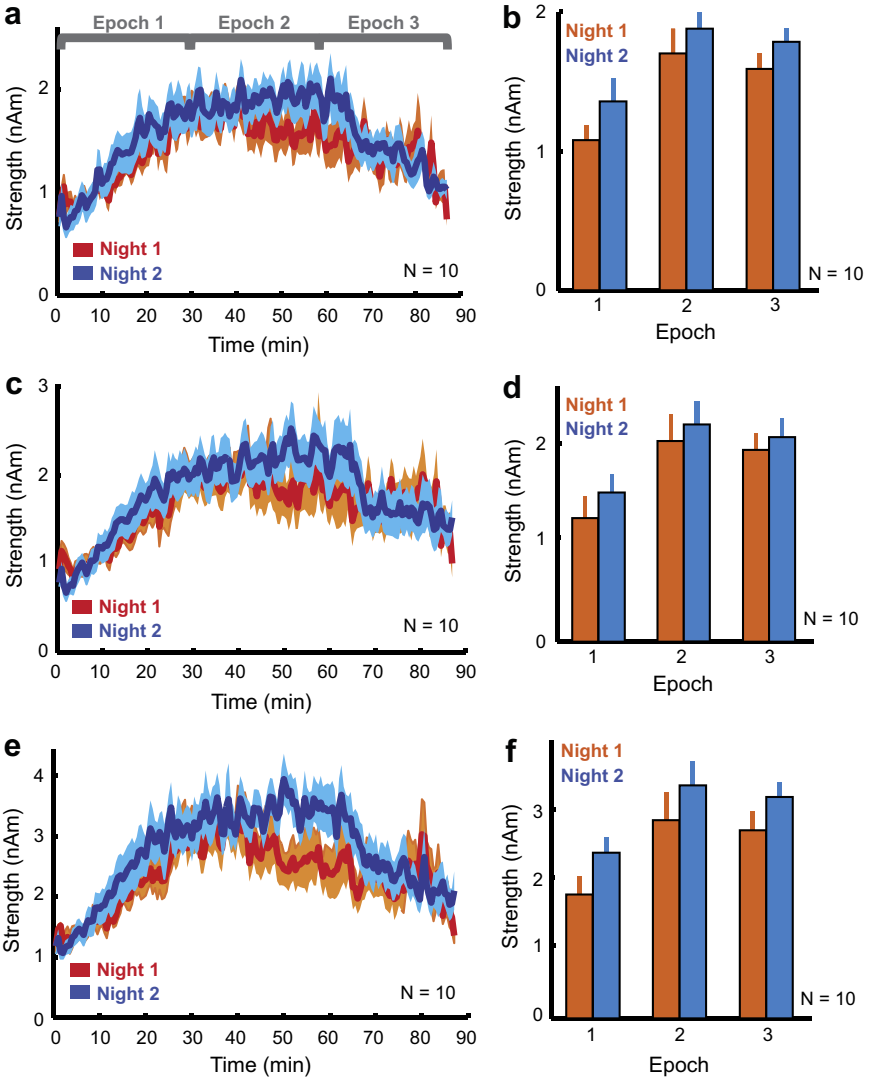

Fig. 2. The time-dependent modification of SWA in the early and higher visual areas on Night 1 and Night 2. (a, c and e) The time course of SWA in the *ventral* part of the early visual area (a), the *dorsal* part of the early visual area (c), and the higher visual area (e). Red line shows the mean SWA strength and shaded light orange area shows standard error of Night 1. Blue line shows the mean SWA strength and shaded light blue area shows standard error of Night 2. *X*-axis shows time from lights out (min). *Y*-axis shows the strength of SWA (nA m). (b, d and f) The time course activity (shown in (a), (c), and (e)) was further divided into three epochs in the *ventral* part of the early visual area (b), the *dorsal* part of the early visual area (d), and the higher visual area (f). Orange bars are the mean SWA on Night 1, and blue bars are the mean SWA on Night 2. *X*-axis shows epoch. *Y*-axis shows the strength of SWA (nA m). Error bar, s.e.m.

cancelled out by a local increase of SWA due to the synaptic homeostatic regulation (Tononi & Cirelli, 2003) in the dorsal stream (Goodale, 1993; Mishkin & Ungerleider, 1982). It is known that the lower visual field, which is projected to the dorsal part of the visual area, has an advantage in perception of particular stimuli (Previc & Mullen, 1990; Rubin, Nakayama, & Shapley, 1996). Furthermore, asymmetry in attentional resolution between the upper and lower visual field has also been suggested (He, Cavanagh, & Intriligator, 1996). The dorsal part of the visual area might have been stimulated in the first sleep session where novel objects and equipment are exposed to the subject. This may result in a local need of synaptic homeostatic regulation during sleep and nullify the reduction of SWA caused by the FNE.

We included only younger and healthy participants in the present study. Nine out of ten participants showed suppression of SWA during slow-wave sleep in at least one of the three visual areas. Thus, the FNE was present in a majority of the participants. Furthermore, there was no significant difference between genders in the frequency of the FNE. However, the impact of the FNE on SWA or individual and gender differences may vary with populations. Previous research suggests that the impact of the FNE differs by age (Wauquier et al., 1991; Webb & Campbell, 1979) or physical conditions such as respiratory disorders (Le Bon et al., 2000; Levendowski et al., 2009), chronic fatigue syndrome (Le Bon et al., 2003), depression (Rotenberg et al., 1997; Toussaint et al., 2000), or post-traumatic stress disorder (Herbst et al., 2010). Some groups are suggested to require more days for adaptation (Herbst et al., 2010; Rotenberg et al., 1997). Thus, greater SWA suppression may be shown in particular groups.

There are several limitations in the present study. Because we used the data collected from our past projects that had different purposes and employed different procedures, the data analyzed here was restricted to the first sleep cycle that occurred in the initial 90 min of nightly sleep. Moreover, it is still unclear which brain regions are affected by the FNE and which are not, because we restricted the regions of interest in the visual area. Whereas the present study shows that the visual areas are affected by the FNE through reduction of SWA in slow-wave sleep, the FNE in other

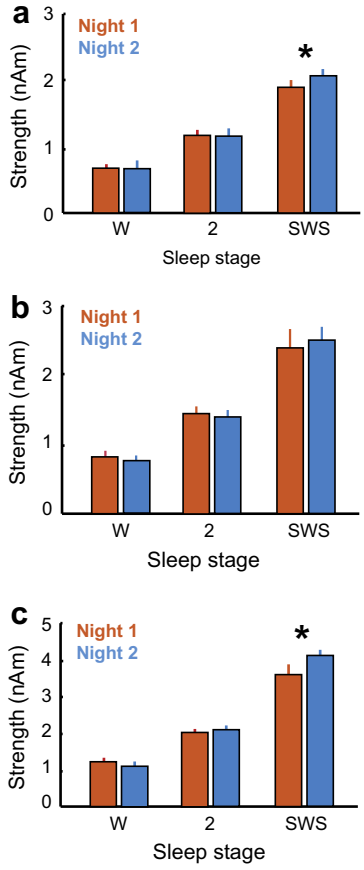

Fig. 3. The sleep-stage dependent modification of SWA in stage W, stage 2, and slow-wave sleep in the *ventral* part of the early visual area (a), the *dorsal* part of the early visual area (b), and the *higher* visual area (c). Orange bars show SWA on Night 1, blue bars show SWA on Night 2. *X*-axis is sleep stage, and *Y*-axis is SWA strength (nA m). SWS, slow-wave sleep. Error bar, s.e.m. $^{*}p < .05$ (two-tailed pared *t* test).

Table 2
Frequency of the FNE for each visual area and the gender effect.

	FNE presence	Visual areas		
		Ventral	Dorsal	Higher
Female ($N = 5$)	5	4	3	4
Male ($N = 5$)	4	4	4	4
Total # of FNE ($N = 10$)	9	8	7	8

Each numerical value shows the number of subjects that showed the FNE in each visual area in slow-wave sleep. The FNE presence shows the total number of subjects who showed the FNE in at least one of the visual areas. Ventral, ventral part of early visual area; Dorsal, dorsal part of early visual area, Higher, higher visual area.

cortical regions has yet to be studied in detail. Also, which spontaneous oscillations other than SWA, such as alpha or theta oscillations, are affected by the FNE in the visual area is unclear. Stage REM parameters should be also tested, because these may be sensitive to adaptation as well (Lorenzo & Barbanoj, 2002). Moreover, the relationship between neural plasticity and the FNE needs to be clarified.

In summary, our study is the first to show that the strength of SWA originating in the visual areas is largely suppressed by the FNE, although the impact of the FNE may be slightly different among the visual areas. As our results showed that the strength of SWA in the visual areas was reduced by the FNE, we suggest that it is crucial to reduce the FNE in order to study the roles of SWA in visual learning. Incorporating at least one adaptation session would be necessary.

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