NeuroImage 53 (2010) 629–637

Contents lists available at ScienceDirect

NeuroImage

journal homepage: www.elsevier.com/locate/ynimg

# EEG oscillations reflect visual short-term memory processes for the change detection in human faces

Hyoung-Dong Park [a,b], Byoung-Kyong Min [c], Kyoung-Min Lee [a,b,d,*]

[a] Interdisciplinary Program of Cognitive Sciences, Seoul National University, Seoul, Korea
[b] Institute for Cognitive Sciences, Seoul National University, Seoul, Korea
[c] Department of Radiology, Brigham and Women's Hospital, Harvard Medical School, Boston, USA
[d] Department of Neurology, Seoul National University, Seoul, Korea

ARTICLE INFO

*Article history:*
Received 9 November 2009
Revised 26 May 2010
Accepted 23 June 2010
Available online 30 June 2010

ABSTRACT

People often fail to notice a large change in the visual scene when the change occurs during a brief interruption of the viewing. Since the change is well above perceptual threshold in continuous viewing, the failure (termed change blindness) has been attributed to abnormal visual short-term memory (VSTM). However, it is still unclear where the abnormality lies among the phases in VSTM, namely, encoding, maintenance, and retrieval-comparison. EEG oscillations, especially the gamma activity, have been suggested as neural signatures of VSTM, but have not been examined in the context of change blindness. Thus, we asked in the present study whether change detection or failure is correlated with EEG oscillatory activities and, if so, whether the timing and the spatial distribution of the oscillations could pin-point the abnormal phase of VSTM in change blindness. While on EEG recording, subjects watched morphed pictures of human faces in trials which consisted of a 200-ms initial image display, a 500-ms blank period, and a 200-ms comparison image display. The two images were either the same or clearly different above threshold. Trials with different images were classified as hit or missed, based on subjects' responses, and EEG data were compared between the two types of trials. Enhanced gamma activity was observed in the right temporal-parietal region during all periods in the hit trials compared to the missed ones. Frontal theta activity was increased during initial image encoding, whereas beta activity was decreased during maintenance and retrieval-comparison in the hit trials. These results point to weak encoding of initial images as the culprit for a later failure in change detection, while abnormal processing in subsequent phases of VSTM may result from the weak encoding and also contribute to change blindness.



## Introduction

Change detection, the ability to detect changes in scenery, is one of the most fundamental cognitive skills for survival. In everyday life, our perception of the environment continuously changes, and failure to detect these changes could cause serious problems. However, we sometimes miss large changes in our environment. The term “change blindness” is used to describe the surprising difficulty observers have in noticing large changes to visual scenes because of saccades, blinks, blank screens, movie cuts, and other interruptions (Simons and Levin, 1998; Simons and Rensink, 2005). Change blindness can occur while viewing simple geometric shapes or colors, complex scenes, or familiar objects such as faces (Pourtois et al., 2006). Several factors, such as attention and short-term memory, could influence the change detection task. For instance, researchers studying one model of attention argued that observers can represent the details of only a few attended objects in a scene, suggesting the possibility that the representation of unattended objects is relatively sparse (Treisman, 1993). Although attention might be necessary for conscious change detection, it might not be sufficient (Simons and Rensink, 2005); observers often fail to detect changes even when attention is focused directly on the changing object (Levin and Simons, 1997; Simons and Levin, 1998). Other studies suggested that the key factor underlying change blindness is the limited processing capacity of the brain, resulting in the inability to retain an intact representation of sensory information over time and across brief interruptions in input (Pourtois et al., 2006; Simons and Rensink, 2005).

Theoretically, successful change detection in the change blindness paradigm depends on the initial encoding of a visual representation from the initial view, the retention of that representation in visual short-term memory (VSTM), and the comparison of the representation to relevant perceptual information in the changed image (Hollingworth, 2003). A deficit in any of these processes can induce change blindness, and each possibility has been suggested as the mechanism for it. First, change blindness may occur if insufficient

* Corresponding author. Department of Neurology, Seoul National University Hospital, 28 Yongon-Dong, Jongno-Gu, Seoul, 110-744, Republic of Korea. Fax: +82 2 3672 7553.
*E-mail address:* kminlee@snu.ac.kr (K.-M. Lee).

1053-8119/$ – see front matter © 2010 Elsevier Inc. All rights reserved.
doi:10.1016/j.neuroimage.2010.06.057

information is encoded from the first image (the encoding-failure hypothesis), as when focused attention is not directed to the changing object (Rensink, 2002). Second, even though sufficient information is encoded, change blindness may still occur if that information is not retained (the retention-failure hypothesis). The initial representation may simply be lost during the blank interval or overwritten by the second image (Shapiro et al., 1997). Finally, although the initial image is encoded and retained successfully, changes may not be detected if comparison with the subsequent image fails (the comparison-failure hypothesis). Subjects can sometimes remember and describe the visual characteristics of a deleted object even when they fail to detect the deletion (Simons et al., 2002). This suggests that implicit VSTM of the representation from an initial image is preserved and change blindness may be caused by comparison failure.

Previous functional magnetic resonance imaging (fMRI) studies have shown increased activity in the frontal and parietal lobes during change detection tasks (Beck et al., 2001; Pessoa and Ungerleider, 2004). In particular, Beck et al. (2006) suggested that the right parietal region might play a causal role in change detection of human faces in their study using repetitive transcranial magnetic stimulation (rTMS). However, since fMRI relies on the detection of relatively slow hemodynamic responses, fMRI techniques cannot evaluate what VSTM processes result in change blindness. On the other hand, several event-related potential (ERP) studies have been carried out to investigate the electrophysiological correlates of conscious change detection with faster temporal resolution (Eimer and Mazza, 2005; Koivisto and Revonsuo, 2003; Pourtois et al., 2006; Schankin and Wascher, 2007). These studies have shown that a negative amplitude shift around 200–300 ms detected at posterior sites after a change in the stimulus (i.e., N2) is associated with visual change awareness (Koivisto and Revonsuo, 2003). However, most of these ERP studies focused on neural correlates of change detection that occur after the second stimulus is presented (Eimer and Mazza, 2005; Koivisto and Revonsuo, 2003; Schankin and Wascher, 2007). Thus, specific processes of VSTM responsible for change blindness were not identified in these studies. Furthermore, ERP may not capture information about the dynamics of cell assembly formation, activation, and subsequent uncoupling, which may play a prominent role in different types of memory operations (Bastiaansen and Hagoort, 2003).

Complex cognitive processes could be implemented by the synchronization of neurons into transient oscillatory assemblies (Pesonen et al., 2007; Varela et al., 2001). Gamma band oscillations are particularly reported to be correlated with encoding, maintenance, matching, and retrieval of VSTM (Herrmann et al., 2004; Jensen et al., 2007; Jokisch and Jensen, 2007; Osipova et al., 2006; Tallon-Baudry et al., 1998). These hypotheses are in accordance with the proposal that neuronal representations can be sustained by the persistent firing of recurrently connected neurons (Hebb, 1949; Jensen et al., 2007). In addition, the gamma band and other frequency bands (e.g., theta and beta) have been reported to be related to memory processing tasks. For example, increased theta oscillation at the frontal region is closely correlated with encoding of VSTM (Jensen and Tesche 2002; Sederberg et al., 2003). Recent evidence also suggests that decreased beta activity reflects increased short-term memory load (Pesonen et al., 2006, 2007).

Although the limited capacity of VSTM is assumed to play an essential role in change blindness, and theta/beta/gamma synchronization is critically related to VSTM, to the best of our knowledge, no EEG oscillation studies that are directly related to change detection or change blindness tasks have been performed. Therefore, in the present study, we investigated whether the change detection phenomenon is reflected in EEG oscillatory activities by using morphed pictures of human faces as stimuli. Note that each of the three hypotheses (the encoding-failure hypothesis, the retention-failure hypothesis, and the comparison-failure hypothesis) makes a different prediction on the timing of neural activity differences when the change detection and the change blindness trials are compared, and in the current study we asked which prediction was actually supported by the data. More specifically, the encoding-failure hypothesis suggests that differences in neural activities between the change detection and change blindness trials will be observed during the memory-encoding period (i.e., when the first image is shown). In contrast, the retention-failure hypothesis predicts that neural activities will not differ during the encoding period but they will during the retention period (i.e., inter-stimulus interval between two images). Finally, according to the comparison-failure hypothesis, differences between two conditions will be detected after the changed image is presented (i.e., when the second image is shown).

Furthermore, it should be noted that the hypotheses may be tested using features of neural oscillations. For instance, the spatial distribution and frequency band of an observed difference in brain oscillation between the change detection and the change blindness trials will indicate which cognitive process differs between the trial types. On the basis of the current view on brain oscillations, one may predict that in successful change detection trials, gamma and theta synchronizations will increase before the second stimulus is presented, as they are closely associated with encoding and maintenance in VSTM (Herrmann et al., 2004; Jensen et al., 2007; Sederberg et al., 2003). One may also predict that beta activities will decrease in the change detection trials compared to the missed ones because they tend to decrease when memory load increases (Pesonen et al., 2006, 2007). In addition, enhanced gamma activity after the appearance of the second image is expected for successful change detection, since gamma oscillations have been implicated in the matching and utilization of VSTM (Herrmann et al., 2004).

## Materials and methods

### *Participants*

Ten healthy subjects with no history of neurological disorders participated in the experiment (mean age, $26.5 \pm 4.4$ years; 1 female; 1 left-handed) after providing written informed consent in accordance with the Declaration of Helsinki. All participants had normal or corrected-to-normal vision. Two subjects were excluded from data analysis because of their low accuracy rates (<25%).

### *Stimuli*

Six non-morphed and 147 morphed pictures of human faces were used as visual stimuli. These stimuli were frontal view images that were converted to grayscale and cropped by a uniform oval and subtended roughly 10° vertical (Webster et al., 2004). We chose 6 non-morphed pictures (3 pairs of male and female human faces) and then morphed them into 49 intermediate pictures that correspond to 2–98% morph levels in each pair, using Fantamorph software (Abrosoft, USA). The left and right columns of Fig. 1 show non-morphed endpoint pictures, and the middle column shows morphed pictures according to their specific morph levels in each set, which will be explained later.

We used these morphed faces as our visual stimuli for the following 2 reasons. First, in previous brain imaging studies on change blindness, researchers used completely different pictures of human faces as stimuli during their change detection tasks (Beck et al., 2001; Eimer and Mazza, 2005; Pourtois et al., 2006); this led to difficulty in quantifying differences across pictures. This implied that the difference between the initial and subsequent face could not be controlled among multiple trials. In addition, to control the task difficulty to produce balanced rates of “hits” and “misses”, they urged subjects to perform a dual task, which could be a compounding factor (Beck et al., 2001; Pourtois et al., 2006). For

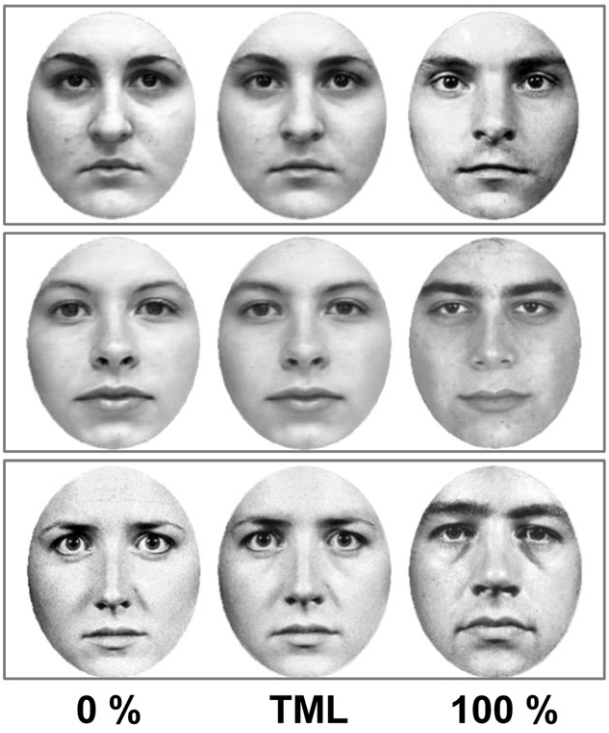


**Fig. 1.** Samples of original and morphed human faces. Faces in the left and right columns show non-morphed endpoint pictures. Faces in the middle column show morphed pictures according to the degree of the threshold-morph level (TML), in which subjects made a correct response about 50% of the time when the second stimulus had been changed. The corresponding TMLs for each set are 28 ± 2%, 26 ± 6%, and 24 ± 4% on the way from the left non-morphed face to the right non-morphed face.

example, to reduce the hit rate and to gather enough data on change blindness trials, subjects were urged to perform a change detection task and letter detection task at the same time (Beck et al., 2001). In contrast, using morphed pictures as shown in Fig. 1, we could better control differences across pictures and thus produce a nearly equal proportion of hit and miss trials without using the dual task paradigm.

*Experimental procedures*

In this experiment, we used a modified one-shot paradigm that presents an original and modified image in single rapid alternation with a blank screen between them (Beck et al., 2006; Beck et al., 2001; Eimer and Mazza, 2005; Pourtois et al., 2006; Schankin and Wascher, 2007). The EEG recording session consisted of 8 blocks, with 50 trials per block. In each trial, one of 153 pictures was randomly presented for 200 ms during the first stimulus-presented interval (SPI-1), and no image was presented for 500 ms during the subsequent inter-stimulus interval (ISI-1). Then, a changed (morphed) or unchanged picture followed for 200 ms (SPI-2). After another subsequent inter-stimulus interval (ISI-2), subjects were instructed to judge whether there was any change in the presented stimuli between SPI-1 and SPI-2 (cf. Fig. 2). Then, 1500 ms of an inter-trial interval followed. During the EEG recording session, the second pictures were changed according to the degree of threshold-morph level (TML), in which subjects made a correct response approximately 50% of the time when the second stimulus was changed.

TML was measured by the staircase method (Cornsweet, 1962) as a pre-experiment for every participant. During the pre-experiment, the first stimulus was chosen randomly among 51 pictures in each set. Then, in the change condition, the amount of morph of the second stimulus varied from 2% to 100%, depending on the response of the preceding trial, and the level at which the subjects made six continuous reversals was designated as TML. Because subjects were nearly 50% accurate when the second stimulus was changed by the degree of TML, regardless of what picture was presented during SPI-1, we assumed that perceptual distances were nearly the same along the whole set of pictures. Faces in the middle column of Fig. 1 show morphed pictures according to the degree of the mean TML in each set.

A fixation mark was always placed at the center of the monitor screen, except during the response period. To minimize guessing, subjects were instructed to respond to a change only when they were confident about seeing the change. Visual display and response recordings were carried out using MATLAB (Math Works, Natick, MA) and the Psychophysics Toolbox (Brainard, 1997).

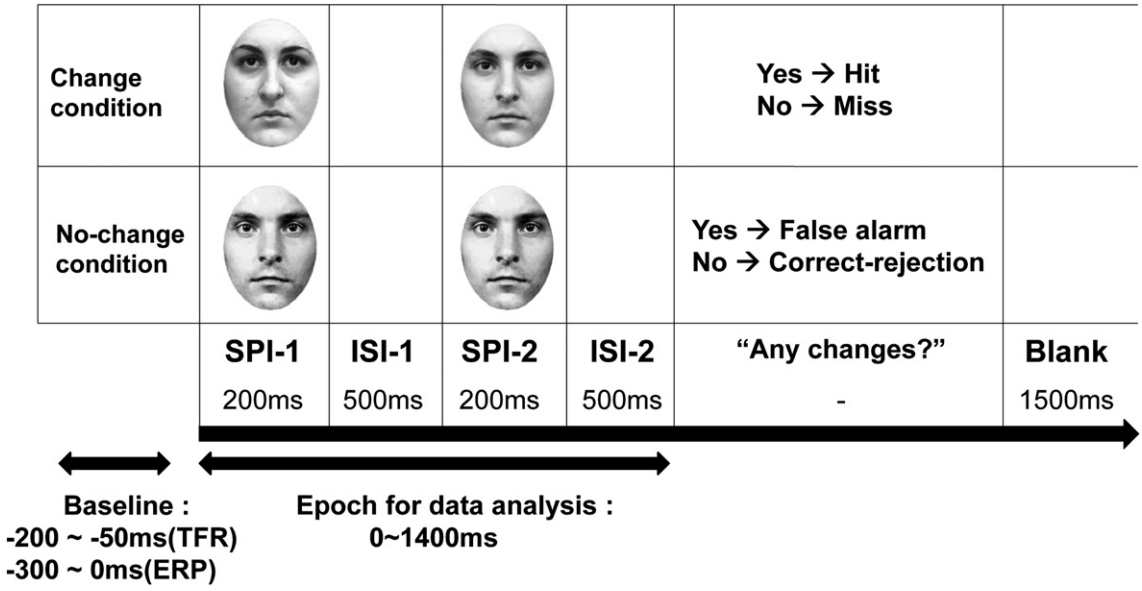


**Fig. 2.** A schematic diagram of the experimental design. In each trial, one of the morphed faces was presented for 200 ms (SPI-1) and no image was presented for 500 ms (ISI-1). Then, a changed or unchanged picture followed for 200 ms (SPI-2). After another ISI (ISI-2), subjects were instructed to judge whether there was a change or not. A blank was subsequently followed for 1500 ms for the inter-trial interval. The baseline for TFR and ERP were set differently (−200 to −50 ms and −300 to 0 ms, respectively).

*EEG recording*

As shown in Fig. 3, EEG was performed using 32 Ag-AgCl electrodes according to the extended international 10-20 system (BRAINTRONICS, Netherlands). All signals from each electrode were referenced to linked earlobes and were later re-referenced against a common average reference. Vertical and horizontal EOGs were also recorded to monitor eye movements. The impedance of each electrode was kept below 10 kΩ, and the sampling rate was 256 Hz.

*Data analysis*

Preprocessing was conducted using the EEGLAB toolbox (Delorme and Makeig, 2004). For ERP and time-frequency analysis, we used the Fieldtrip toolbox (http://www.ru.nl/fcdonders/fieldtrip). EEG epochs were obtained from 300 ms before to 1400 ms after the first stimulus onset. All trials were divided into four categories according to the signal detection theory (Green and Swets, 1966): correctly identified as changed (hit or change detection); not recognized (miss or change blindness); incorrectly identified as changed (false-alarm); and correctly rejected as not changed (correct rejection). We analyzed hit, miss, and correct-rejection trials, while false-alarm trials were not analyzed as the number of trials was too low for analysis (Fig. 2).

For ERP analysis, the raw EEG signals were band-pass filtered at 0.5–20 Hz with a finite impulse response (FIR) filter (Melloni et al., 2007). Trials that contained voltage fluctuations exceeding ±100 μV were rejected. Eye movement artifacts were corrected using the Independent Component Analysis (Delorme and Makeig, 2004). The baseline for ERP analysis was designated from −300 to 0 ms.

For time-frequency analysis, an FIR band-pass filter (1–55 Hz) was applied. We limited the upper cutoff of our band-pass filter to 55 Hz since a lower band of gamma activity (30–60 Hz) was reported to be correlated with VSTM (Jensen et al., 2007; Tallon-Baudry and Bertrand, 1999; Tallon-Baudry et al., 1998) and 60 Hz is associated with power noise. For the theta and beta bands, time-frequency representations (TFRs) of the spectral power were computed using a multi-tapered approach (Jokisch and Jensen, 2007; Osipova et al., 2006; Percival and Walden, 1993). For these lower frequency bands (4–30 Hz), we applied an adaptive time window of 3 cycles for each frequency ($\Delta T = 3/f$) and an adaptive smoothing of $\Delta f = 1/\Delta T$ (Osipova et al., 2006). For the lower band of gamma activity (30–55 Hz), wavelet transformation was used to calculate the TFRs of the power (Tallon-Baudry et al., 1998). Data were convolved using complex Morlet's wavelet: $w(t, f) = A\exp(-t^2/2\sigma_t^2)\exp(2i\pi ft)$, using a constant ratio of $f/\sigma_f = 7$ where $\sigma_f = 1/(2\pi\sigma_t)$ and the normalization factor $A = (\sigma, \sqrt{\pi})^{-1/2}$ (Anaki et al., 2007; Jensen et al., 2002; Tallon-Baudry et al., 1998). The mean power value of the prestimulus (−200 to −50 ms) was considered to be the baseline and was divided from the poststimulus power value. This correction was done for the individual frequencies (Jokisch and Jensen, 2007). This time window was chosen to avoid temporal smearing of the poststimulus activity into the baseline (Min and Herrmann, 2007).

The significance of the difference in ERP and frequency power between conditions was tested using the cluster-level nonparametric randomization test implemented in the Fieldtrip toolbox (Jokisch and Jensen, 2007; Mazaheri et al., 2009; Nieuwenhuis et al., 2008; Osipova et al., 2006; see Maris and Oostenveld, 2007, for a detailed review of the method). This method effectively controls type-I error rate in a situation involving multiple comparisons by clustering the selected (sensor, frequency, time)—samples on the basis of spatial, spectral, and temporal adjacency (Maris and Oostenveld, 2007). For the statistical analysis, all the electrodes were clustered into ten groups as regions of interest (cf. Fig. 3). For the ERP data, the mean potentials in a 250- to 320-ms (the N2 component) post-second stimulus time window were examined. For the TFR data, each data samples were averaged over time and frequency range of interest. As shown in Fig. 2, we examined the differences across four time windows (stimulus-presented interval 1 [SPI-1; 0–200 ms], inter-stimulus interval 1 [ISI-1; 200–700 ms], stimulus-presented interval 2 [SPI-2; 700–900 ms], and inter-stimulus interval-2 [ISI-2; 900–1400 ms]) and three frequency bands (theta [5–10 Hz], beta [12–30 Hz], and gamma [30–50 Hz]). The frequency boundary of the theta band was based on visual inspection of the grand average of TFR data. For these data points, ordinary $t$ values were computed. Then, contiguous ROI-time points exceeding the threshold ($p<0.05$) were clustered by cluster-finding algorithm and cluster-level test statistic was defined from the sum of $t$ values of data points in a given cluster. Subsequently, the Monte Carlo estimate of the permutation $p$ value of the cluster was computed by comparing the cluster-level test statistic to a randomization null distribution obtained by 1000 times randomly permuting the conditions in subjects (Nieuwenhuis et al., 2008; Mazaheri et al., 2009).

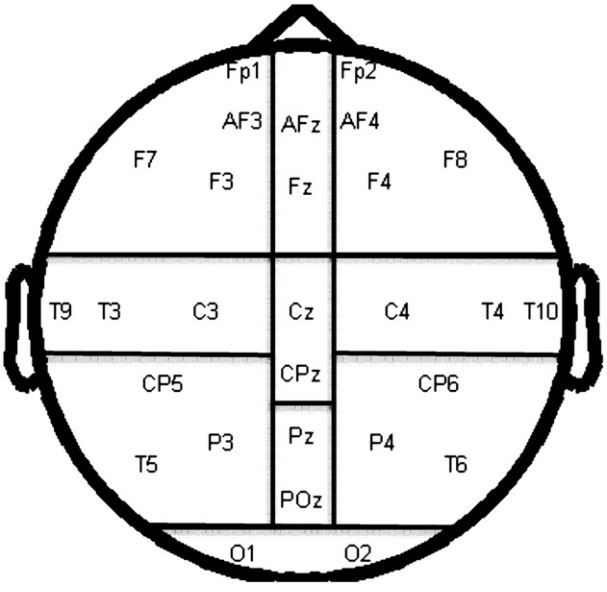


**Fig. 3.** Ten regions of interest with the locations of electrodes for statistical analysis.

## Results

*Behavioral data*

During the pre-experiment, we measured the threshold-morph level (TML) in each subject. The mean value of TML for the 3 sets was 28 ± 2%, 26 ± 6%, and 24 ± 4%, respectively. The middle column of Fig. 1 shows one typical picture morphed by the degree of TML in each set.

During the recording sessions, the stimulus was changed according to the degree of TML, and subjects produced a balanced rate of change detection (hit) and change blindness (miss) trials (mean 51% and 49%, respectively). In addition, the false-alarm rate remained very low (mean 3%) compared to the correct-rejection rate (mean 97%) as subjects were instructed to report change detection only when they confidently detected changes (cf. Fig. 2).

*ERP analysis*

As shown in Fig. 4, we found increases in the N2 component in change detection trials compared to change blindness trials. Enhanced negativity was observed about 200 ms after the second stimulus presentation in the posterior-occipital region for detected-change trials compared to undetected-change trials (Fig. 4B). During the time window for the N2 analysis (250–

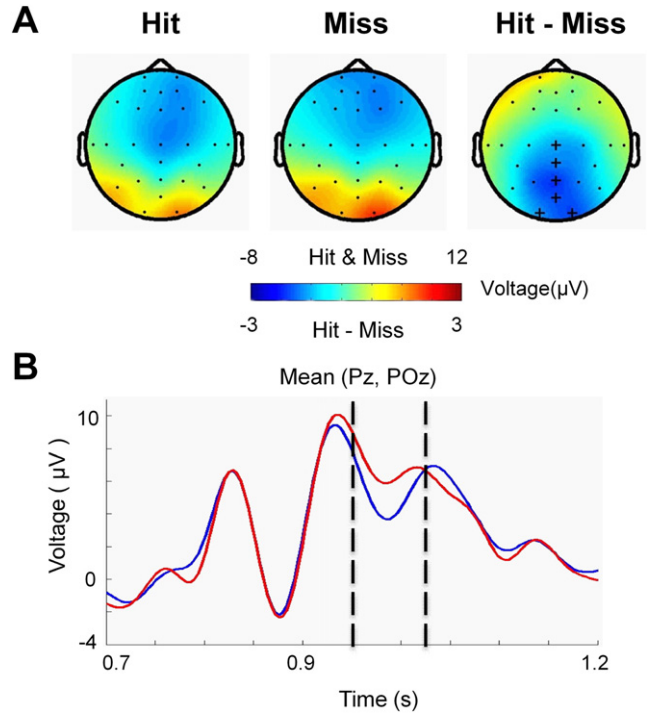


**Fig. 4.** The N2 component at the parieto-occipital region. (A) The grand average of the topography of relative potentials for hit and miss trials and their difference ("+" marks indicate statistically significant electrodes, $p<0.001$; cluster randomization routine). (B) The blue line indicates the ERP from the hit trials and the red line represents the miss trials. "0.7 s" in the time axis indicates the second stimulus onset and the dashed lines indicate the time window for N2 (250–320 ms after the second stimulus onset).

320 ms after the second stimulus onset), three regions of interest showed statistically significant differences (electrodes Cz and CPz: $p<0.001$; Pz and POz: $p<0.001$; O1 and O2: $p<0.001$; Fig. 4A).

*Modulations in the low gamma band (30–50 Hz)*

Time-frequency representations of power (TFRs) were calculated for the change detection (hit) and change blindness (miss) trials (Figs. 5–7). In the low gamma band (30–50 Hz), we found increased activity in the right temporal and right parietal regions (electrodes C4, T4, T10, P4, T6, and CP6) for the change detection condition compared to the change blindness condition during the first stimulus-presented interval (electrodes C4, T4, and T10: $p<0.01$; P4, T6, and CP6: $p<0.01$; cf. SPI-1 in Fig. 5A). Furthermore, enhanced gamma activity in the right temporal region (electrode C4, T4, and T10) was continuously found during ISI-1, SPI-2, and ISI-2 ($p<0.01$; Fig. 5A). Fig. 5B presents the TFRs of the gamma power changes of hit and miss trials and their differences over the electrodes included in the right temporal region (averaged across electrodes C4, T4, and T10).

*Modulations in the theta band (5–10 Hz)*

The frequency boundary of the theta band (5–10 Hz) was based on visual inspection of grand average of TFR data (see Fig. 6B). We found increased theta activity in the frontal region (electrodes Fp1, Fp2, AF3, AF4, F3, F4, F7, F8, AFz, and Fz) for the change detection (hit) condition compared to the change blindness (miss) condition during SPI-1 (Fig. 6, SPI-1) (electrodes Fp1, AF3, F3, and F7: $p<0.05$; Fz, AFz: $p<0.05$; Fp2, AF4, F4, and F8: $p<0.05$; cf. SPI-1 in Fig. 6A). After SPI-1, no significant effects were observed in the theta band. Fig. 6B presents the TFRs of the theta power changes of hit and miss trials and their differences over the electrodes included in the frontal region (averaged across electrodes Fz and AFz).

*Modulations in the beta band (13–30 Hz)*

We observed decreased beta activity in the parietal-occipital region (electrodes O1, O2, Pz, and POz) for the change detection (hit) condition compared to the change blindness (miss) condition during ISI-1, SPI-2, and ISI-2 (electrodes Pz and POz: $p<0.05$; O1 and O2: $p<0.05$; cf. Fig. 7A). Furthermore during ISI-2, decreased

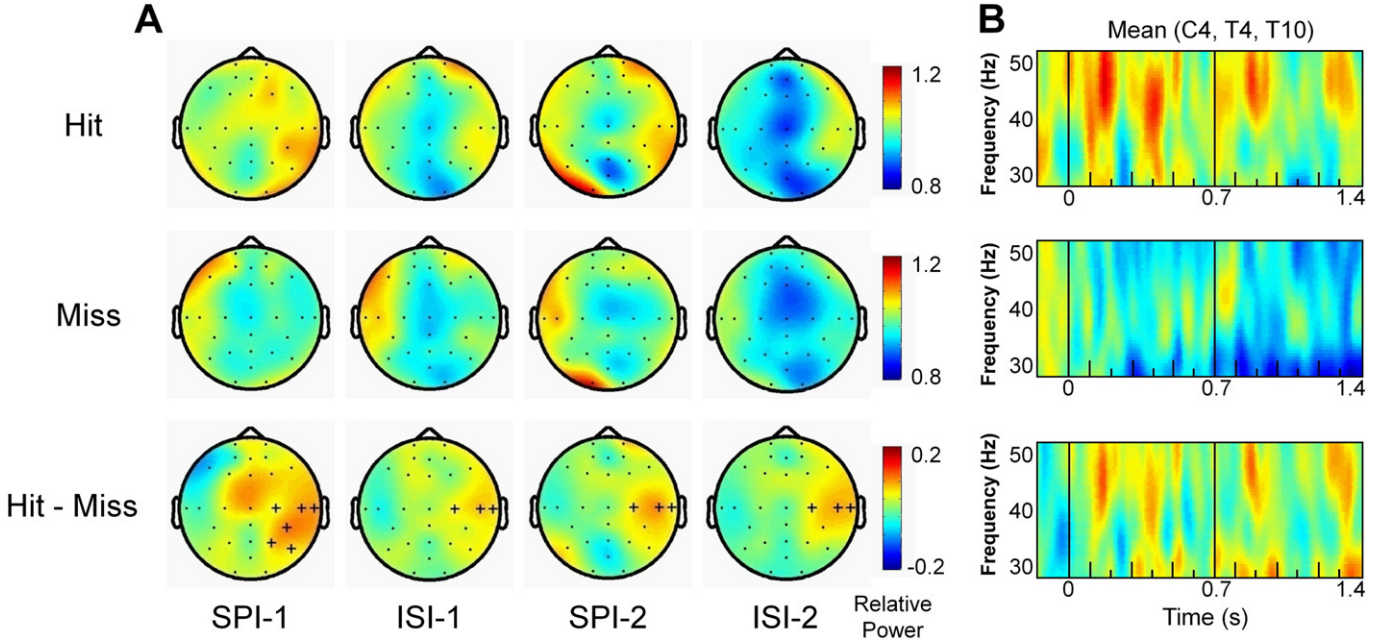


**Fig. 5.** (A) Topographies of the grand averaged gamma power of the hit and miss trials with their differences (hit − miss) during SPI-1, ISI-1, SPI-2, and ISI-2 periods ("+" marks indicate statistically significant electrodes, $p<0.01$; cluster randomization routine). (B) Time-frequency representations of gamma power averages of electrodes C4, T4, and T10. "0 s" and "0.7 s" in the time axis indicate the first and second stimulus onset, respectively.

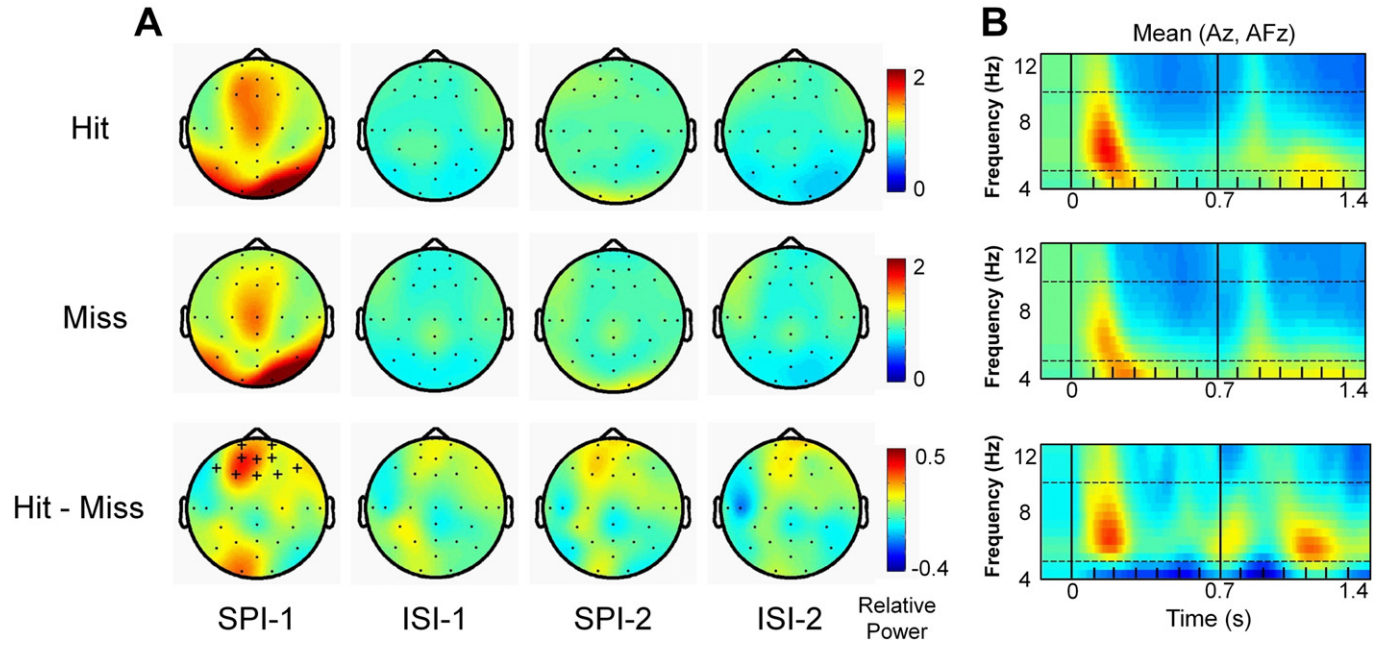


**Fig. 6.** (A) Topographies of the grand averaged theta power of the hit and miss trials with their differences (hit − miss) during SPI-1, ISI-1, SPI-2, and ISI-2 periods ("+" marks indicate statistically significant electrodes, $p<0.05$; cluster randomization routine). (B) Time-frequency representations of theta power averages of electrodes Fz and AFz. "0 s" and "0.7 s" in the time axis indicate the first and second stimulus onset, respectively. The dashed lines indicate the frequency boundary of the theta band (5–10 Hz).

beta oscillations were found around the central-frontal regions (electrode Cz and CPz: $p<0.05$; Fz and AFz: $p<0.05$). Fig. 7B presents the TFRs of the beta power changes of hit and miss trials and their differences over the electrodes included in the parietal-occipital region (averaged across electrodes Pz and POz).

We also compared the baseline period (−200 to −50 ms) between the 2 conditions, and no significant differences were found in the gamma, theta, and beta bands. Thus, the statistically significant differences in each frequency we reported are not due to the prestimulus effect.

*Comparing the correct-rejection trial*

We analyzed the correct-rejection trials to investigate whether there is an enhancement in either theta or gamma band activity during SPI-1. Although we analyzed the gamma band (30–50 Hz) activity in correct-rejection trials in the regions of interest that showed significant enhancement in hit trials, we could not detect any significant increases during SPI-1 in correct-rejection trials compared to miss trials (electrodes C4, T4, T10, P4, T6, and CP6: n.s). The theta band activity also showed no significant enhancement in correct-

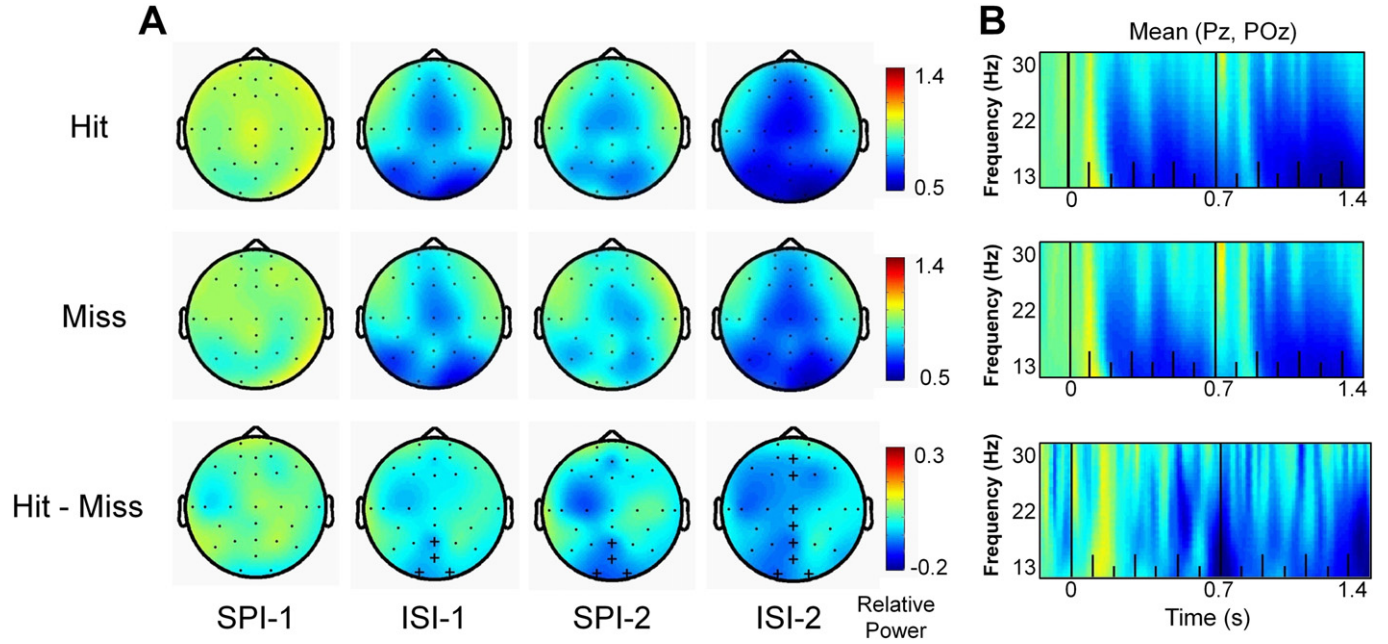


**Fig. 7.** (A) Topographies of the grand averaged beta power of the hit and miss trials with their differences (hit − miss) during SPI-1, ISI-1, SPI-2, and ISI-2 periods ("+" marks indicate statistically significant electrodes, $p<0.05$; cluster randomization routine). (B) Time-frequency representations of beta power averages of electrodes Pz and POz. "0 s" and "0.7 s" in the time axis indicate the first and second stimulus onset, respectively.

rejection trials compared to miss trials during SPI-1 (electrodes Fp1, AF3, F3, F7, Fz, AFz, Fp2, AF4, F4, and F8: n.s).

## Discussion

In the present study, we investigated the EEG oscillatory activity produced while subjects detected changes in morphed human face pictures. Successful change detections were accompanied by increased gamma power in the right temporo-parietal region. Moreover, enhancement of gamma activity was already observed when the first stimulus was presented, and this activity was sustained until the subjects responded. We also observed increased theta activity in the frontal region when the first stimulus was presented. In contrast, beta oscillation decreased in the parieto-occipital region during delayed periods.

### *Change blindness may occur with a single object in the scene*

Almost all previous brain imaging studies on the change blindness paradigm used visual stimuli that have multiple potentially changing objects (Beck et al., 2001; Eimer and Mazza, 2005; Koivisto and Revonsuo, 2003; Pessoa and Ungerleider, 2004; Pourtois et al., 2006), whereas we used a single object for our stimuli. Our experimental design is more similar to what Simons and Levin designed in their change blindness studies (Levin and Simons, 1997; Simons and Levin, 1998). They sequentially presented a single person as a potentially changing object, with a visual interruption between displays. What really makes this kind of "change blindness" situation interesting is that when there is a visual interruption between displays, one sometimes becomes blind to the relatively large changes being presented, although there is only a single attended object. Selective attention to a specific object is possibly necessary for change detection. However, attention may not be sufficient for conscious change detection, and observers often fail to detect changes even when attention is focused directly on the changing object (Levin and Simons, 1997; Simons and Levin, 1998). Using morphed and non-morphed pictures that have a single object, we could control the influence of attentional selection and investigate the functional role of VSTM and focused attention in change blindness.

Furthermore, the changes in our task were relatively large. The failure to detect small changes thus does not seem to be surprising. However, the change according to the degree of TML is large in the sense that if there is no blank between two stimuli, subjects cannot miss the change because of the ability to detect retinal differences (Simons and Levin, 1998).

It can be argued that what the subjects essentially did in our task was make a gender discrimination, as the pairs of non-morphed pictures differed in gender. To avoid such confounding interpretation, we instructed subjects to judge whether there were "any changes." As gender discrimination would require more perceptual information than just finding "any changes," we believe that the subjects had actually performed change detection and not gender discrimination.

### *N2 and conscious change detection*

Previous ERP studies on change blindness mostly focused on neural correlates of change detection that occur after the second stimulus is presented. Previous studies reported that successful change detection was linked to a negative amplitude modulation at posterior electrodes, which started about 200 ms after change onset (Eimer and Mazza, 2005; Koivisto and Revonsuo, 2003; Pourtois et al., 2006; Schankin and Wascher, 2007). Koivisto and Revonsuo (2003) suggested that posterior negativity is an electrophysiological correlate of phenomenal visual change awareness. We consistently observed decreased N2 component after the second stimulus onset during change detection trials compared with change missed trials. One study reported that positive amplitude enhancement, which started around 100 ms after the first stimulus onset (i.e., P1), correlates with the detection of a changed visual stimulus, and interpreted that enhanced P1 might reflect a greater engagement of attentional resources on the initial display (Pourtois et al., 2006). Although we did not find increased P1 component in the occipital-parietal region as in the Pourtois et al. (2006) study, we instead observed enhanced gamma and theta oscillations during a similar period.

### *Gamma activity and VSTM*

Since increased gamma oscillation has been implicated in both attention and stimulus encoding (Fries et al., 2001; Jensen et al., 2007; Steinmetz et al., 2000), our observation of enhanced gamma oscillation during the first stimulus presentation interval might reflect both focused attention and encoding by VSTM. Previous studies have consistently reported that attention might rely on gamma band synchronization to integrate neural activities related to a specific sensory object into a stable, salient, and coherent representation (Jensen et al., 2007). The intact encoding of VSTM might play a critical role in subsequent processing of VSTM, and our results demonstrate that change blindness correlated with failure in encoding the first visual stimulus.

On the other hand, increased gamma oscillation during the first delay period (ISI-1) might reflect successful maintenance of VSTM. In accordance with this interpretation, Tallon-Baudry et al. (1998) reported that gamma oscillation increased during the retention period of a delayed match-to-sample task and concluded that the enhancement might reflect the rehearsal of an object representation in short-term memory; our results differ from theirs in some respects. Whereas they observed increased gamma oscillation in occipital and frontal regions, we found enhanced gamma oscillation in the right temporal and parietal regions. This difference might be due to the difference of visual stimuli. Whereas they used a simple geometric display as a stimulus, we used human faces. Moreover, they contrasted gamma activities between memory and non-memory conditions, whereas we contrasted between the hit and miss trials which were both memory conditions. These differences in stimuli and comparing conditions may account for the difference of spatial distribution of enhanced gamma activity. Furthermore, Sederberg et al. (2003) observed, using intracranial EEG, an increase in gamma activity at widespread cortical sites during successful memory encoding, and interpreted that the functional role of gamma oscillation is not confined to the specific brain region.

Using fMRI, Beck et al. (2001) reported dominant BOLD (blood oxygen level dependent) responses of the right intra-parietal sulcus in successful change detection tasks (Beck et al., 2001). Recent evidence shows that gamma synchronizations and BOLD responses have positive correlation (Lachaux et al., 2007; Niessing et al., 2005). In addition, using rTMS, they showed that the right parietal cortex plays a critical role in conscious change detection (Beck et al., 2006). In our data on the successful performance of the change detection task, the dominant gamma activation in the right hemisphere was already observed during SPI-1. Presumably, increased gamma activity in the right temporal region during SPI-1 and ISI-1 seems to be critical for subsequent change detection.

If enhanced gamma activity during SPI-1 is due to intact encoding of VSTM, then these would also be present in some correct-rejection trials, as the participant could not predict the second stimulus and intact VSTM would be necessary for correct-rejection. However, in our experimental design, the correct-rejection condition also contains some trials that do not properly encode VSTM. As we instructed subjects to answer "yes" only when they confidently detected changes, when intact VSTM was not formed by gamma oscillation, their responses would be biased to "no." This means that in the no-change condition, although VSTM was not properly formed, subjects would choose "no," and the trials would be categorized as correct rejection. Whereas the hit condition consists of trials that have intact VSTM and the miss condition consists of trials that have sparse VSTM,

the correct-rejection condition contains trials that have intact and sparse VSTM at the same time. Indeed, 97% trials in the no-change condition were categorized as correct rejection. Consequently, we could not observe significantly increased gamma band activity during correct-rejection trials compared to miss trials in SPI-1. This explanation would be also applicable to enhanced theta activity during SPI-1 in the same manner.

Enhanced gamma oscillation was also observed after the second stimulus was presented. Our results are in accordance with the previously proposed explanation of the functional role of gamma oscillation (Herrmann et al., 2004). According to Herrmann et al. (2004) study to perform target detection, subjects have to activate a template of the target in short-term memory, and every stimulus needs to be compared with this template, and consequently, the target stimuli which matched with short-term memory contents lead to significantly more gamma activity than the novel or un-matched stimuli (Herrmann et al., 2004). Subsequently, after this matching process, the same information can be used for other processes, such as coordinating behavioral performance. According to this match-and-utilization model (MUM), "early" evoked activity is correlated with matching process, whereas "late" induced activity is associated with the utilization process. Gamma oscillation which occurs around 150 ms after stimulus presentation typically reflects "early" evoked activity, whereas "late" induced gamma oscillation is usually observed around 300 ms after stimulus presentation and later (Herrmann et al., 2004). Our observation suggests that enhanced gamma activity during SPI-2 (0–200 ms from the second stimulus onset) reflects this memory-matching process, whereas pronounced gamma activity during ISI-2 (200–700 ms from the second stimulus onset) might reflect utilization processes such as judgment of change detection.

The nature of the stimuli raises the question of what extent the observed enhanced gamma oscillations are specific to face stimulus or generalizable across other stimulus material. For example, Zion-Golumbic et al. (2008) reported that induced gamma (20–80 Hz) activity elicited by human faces was higher and peaked earlier than that elicited by other categories, such as ape faces, human hands, buildings, and watches. However, because we only used human faces as visual stimuli, we cannot answer this question. This question must be addressed in future studies in which several kinds of visual stimuli could be used.

### *Theta activity and memory encoding*

As shown in Fig. 6B, we observed enhanced frontal theta activity particularly in successful change detection trials. Our findings are consistent with the hypothesized role of the theta activity in memory encoding. It has been reported that theta synchronization commonly occurs during the cognitive process induced by task conditions requiring (i) the maintenance of information in short-term memory, (ii) sustained attention, and (iii) memory encoding and retrieval (for a review, see Klimesch et al., 2008). That is, since theta oscillations have been found to be enhanced during encoding of successfully recalled words (Klimesch et al., 1997; Sederberg et al., 2003) and pictorial stimuli (Osipova et al., 2006), our observations could also be interpreted in terms of reflecting successful memory encoding. Consistently, Mölle et al. (2002) reported that effective encoding of pairs of words was associated with a theta oscillation over the frontal regions of the scalp. Taken together, stimulus encoding processes during SPI-1 seem to be required for the subsequent successful change detection.

The frequency boundaries of the theta band have been defined differently in various studies (Jensen and Tesche, 2002). The hippocampal theta activity in animals is usually identified from 3 to 12 Hz (Klimesch, 1999), whereas the theta band in humans is typically identified from 4 to 8 Hz (Kahana et al., 2001). However, with intracranial recordings, the theta frequency band was identified in the interval from 4 to 9 Hz in humans (Jensen and Tesche, 2002; Raghavachari et al., 2001). Scalp MEG recording studies also reported increases in the theta activity peaking at 8 Hz during a Sternberg task (Jensen and Tesche, 2002). Also, the topography of the 7-Hz centered oscillations are in accordance with recent findings of frontal-midline theta activity in memory and attention paradigms (Jensen and Tesche, 2002; Mazaheri et al., 2009; Mazaheri et al., 2010; Sederberg et al., 2003). Therefore, the theta activity peaking at 7 Hz observed in our results belong to the upper margin of the theta band.

### *Beta activity and memory load*

Finally, we also observed decreased beta activity in posterior and occipital regions during ISI-1, SPI-2, and ISI-2. Beta oscillation has been generally suggested to be associated with motor preparation or execution. However, since preparatory motor activity would have been required equally for both types of trials, our finding of decreased beta oscillation in the posterior region, on the hit trials compared to the miss trials, may not be related to motor preparation. Rather, it is noteworthy that decreased beta oscillation already existed even before the second stimulus was displayed.

Decreased beta oscillation has recently been proposed to be related to complex cognitive processes (Pesonen et al., 2007; Sheth et al., 2009). For instance, during the performance of a visual n-back task, more decreased beta activity in the posterior region was observed with increasing memory loads (Pesonen et al., 2007). Sheth et al. (2009) also suggested that decreased beta oscillation is related to complex cognitive processes such as transformative reasoning which is believed to necessitate high memory load. In our data, decreased beta activity in the posterior region was observed after the disappearance of the first visual stimulus. Presumably, successful memory encoding of the first stimulus subsequently requires high memory load, and decreased beta oscillation during ISI-1, SPI-2, and ISI-2 might reflect that kind of highly memory-loaded mental states.

### *Encoding-failure hypothesis*

In the current investigation, we found evidence supporting the encoding-failure hypothesis: a significant hit-versus-missed difference was observed starting from the memory-encoding period, suggesting that weak encoding of initial images is at least partially responsible for the change detection failure. However, caution should be exercised not to over-interpret this finding: Since the methods employed in this investigation were correlative in nature, we cannot be sure whether the observed neural differences have caused the behavioral failure. In fact, significant differences were observed in oscillatory activities that have been implicated in each of all three component processes of VSTM (encoding, retention, and comparison). Any one, or perhaps all, of these differences might or might not be causative. Furthermore, an encoding failure is not necessarily the culprit in all change blindness situations. Simons has emphasized that some of the proposed explanations in the literature may be appropriate only for certain specific situations of change blindness (Simons, 2000). For example, when observers cannot anticipate a change, subjects who failed to detect a change sometimes recalled features of the initial object rather than the second one (Levin and Simons, 1997), indicating that the subject's VSTM of the representation from the initial view was intact. In our experiments, observers anticipated on which object a change may occur, namely, the face shown alone in the initial image. Our data show that at least in such a situation where attention is already directed to the potentially changing object, an encoding failure is the primal cause of change blindness.

In conclusion, by examining EEGs while subjects detected changes of morphed human faces, we showed that gamma, theta, and beta oscillations reflect successful detection or failure of changed stimulus.

Enhanced gamma and theta oscillations during the first stimulus-presented interval in advance of change onset might reflect successful encoding of VSTM and focused attention, respectively. Furthermore, successive increased gamma activity and decreased beta activity correlates with subsequent maintenance, matching, and retrieval of VSTM. Indeed, simultaneous theta, alpha, beta, and gamma oscillations have been proposed to be required for unified cognitive processes (Palva and Palva, 2007). Our results suggest that successful change detection of morphed human faces involves highly complex patterns of brain oscillations at different frequencies that reflect intact processes of VSTM.

## Acknowledgments

The work was supported by a grant from the National Research Foundation of Korea (No. 800-2009-0003). The authors would like to thank the Swartz Center for Computational Neuroscience (SCCN) and the Donders Institute for Brain, Cognition and Behavior (DCCN) for their generous gift of analysis toolboxes, Professor Ione Fine for kindly providing the pictures used as stimuli, and Dr. Hyon Lee for editorial help.

## References

Anaki, D., Zion-Golumbic, E., Bentin, S., 2007. Electrophysiological neural mechanisms for detection, configural analysis and recognition of faces. Neuroimage 37, 1407–1416.
Bastiaansen, M., Hagoort, P., 2003. Event-induced theta responses as a window on the dynamics of memory. Cortex 39, 967–992.
Beck, D.M., Rees, G., Frith, C.D., Lavie, N., 2001. Neural correlates of change detection and change blindness. Nat. Neurosci. 4, 645–650.
Beck, D.M., Muggleton, N., Walsh, V., Lavie, N., 2006. Right parietal cortex plays a critical role in change blindness. Cereb. Cortex 16, 712–717.
Brainard, D.H., 1997. The Psychophysics Toolbox. Spat. Vis. 10, 433–436.
Cornsweet, T.N., 1962. The staircrase-method in psychophysics. Am. J. Psychol. 75, 485–491.
Delorme, A., Makeig, S., 2004. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. J. Neurosci. Meth. 134, 9–21.
Eimer, M., Mazza, V., 2005. Electrophysiological correlates of change detection. Psychophysiology 42, 328–342.
Fries, P., Reynolds, J.H., Rorie, A.E., Desimone, R., 2001. Modulation of oscillatory neuronal synchronization by selective visual attention. Science 291, 1560–1563.
Green, D.M., Swets, J.A., 1966. Signal Detection Theory and Psychophysics. Wiley, New York.
Hebb, D.O., 1949. The Organization of Behavior: A Neuropsychological Theory. Wiley, New York.
Herrmann, C.S., Munk, M.H., Engel, A.K., 2004. Cognitive functions of gamma-band activity: memory match and utilization. Trends Cogn. Sci. 8, 347–355.
Hollingworth, A., 2003. Failures of retrieval and comparison constrain change detection in natural scene. J. Exp. Psychol. Human. 29, 388–403.
Jensen, O., Tesche, C.D., 2002. Frontal theta activity in humans increases with memory load in a working memory task. Eur. J. Neurosci. 15, 1395–1399.
Jensen, O., Gelfand, J., Kounios, J., Lisman, J.E., 2002. Oscillations in the alpha band (9–12 Hz) increase with memory load during retention in a short-term memory task. Cereb. Cortex 12, 877–882.
Jensen, O., Kaiser, J., Lachaux, J.P., 2007. Human gamma-frequency oscillations associated with attention and memory. Trends Neurosci. 30, 317–324.
Jokisch, D., Jensen, O., 2007. Modulation of gamma and alpha activity during a working memory task engaging the dorsal or ventral stream. J. Neurosci. 27, 3244–3251.
Kahana, M.J., Seeling, D., Madsen, J.R., 2001. Theta returns. Curr. Opin. Neurobiol. 11, 739–744.
Klimesch, W., 1999. EEG alpha and theta oscillations reflect cognitive and memory performance: a review and analysis. Brain Res. Rev. 29, 169–195.
Klimesch, W., Dopplemayr, M., Schimke, H., Ripper, B., 1997. Theta synchronization and alpha desynchronization in a memory task. Psychophysiology 34, 169–176.
Klimesch, W., Freunberger, R., Sauseng, P., Gruber, W., 2008. A short review of slow phase synchronization and memory: evidence for control processes in different memory system? Brain Res. Rev. 1235, 31–44.
Koivisto, M., Revonsuo, A., 2003. An ERP study of change detection, change blindness, and visual awareness. Psychophysiology 40, 423–429.
Lachaux, J.P., Fonlupt, P., Kahane, P., Minotti, L., Hoffmann, D., Bertrand, O., Baciu, M., 2007. Relationship between task-related gamma oscillations and BOLD signal: New digits from combined fMRI and intracranial EEG. Hum. Brain Mapp. 28, 1368–1375.
Levin, D.T., Simons, D.J., 1997. Failure to detect changes to attended objects in motion pictures. Psychon. Bull. Rev. 4, 501–506.
Maris, E., Oostenveld, R., 2007. Nonparametric statistical testing of EEG- and MEG-data. J. Neurosci. Meth. 164, 177–190.
Mazaheri, A., Nieuwenhuis, I.L., Van Dijk, H., Jensen, O., 2009. Prestimulus alpha and mu activity predicts failure to inhibit motor response. Hum. Brain Mapp. 30, 1791–1800.
Mazaheri, A., Coffey-Corina, S., Mangun, G.R., Bekker, E.M., Berry, A.S., Corbett, B.A., 2010. Functional disconnection of frontal cortex and visual cortex in attention-deficit/hyperactivity disorder. Biol. Psychiatry 67, 617–623.
Melloni, L., Molina, C., Pena, M., Torres, D., Singer, W., Rodriguez, E., 2007. Synchronization of neural activity across cortical areas correlates with conscious perception. J. Neurosci. 27, 2858–2865.
Min, B.K., Herrmann, C.S., 2007. Prestimulus EEG alpha activity reflects prestimulus top-down processing. Neurosci. Lett. 422, 131–135.
Mölle, M., Marshall, L., Fehm, H.L., Born, J., 2002. EEG theta synchronization conjoined with alpha desynchronization indicate intentional encoding. Eur. J. Neurosci. 15, 923–928.
Niessing, J., Ebisch, B., Schmidt, K.E., Niessing, M., Singer, W., Galuske, R.A., 2005. Hemodynamic signals correlate tightly with synchronized gamma oscillations. Science 309, 948–951.
Nieuwenhuis, I., Takashima, A., Oostenveld, R., Frenández, G., Jensen, O., 2008. Visual areas become less engaged in associative recall following memory stabilization. Neuroimage 40, 1319–1327.
Osipova, D., Takashima, A., Oostenveld, R., Fernandez, G., Maris, E., Jensen, O., 2006. Theta and gamma oscillations predict encoding and retrieval of declarative memory. J. Neurosci. 26, 7523–7531.
Palva, S., Palva, J.M., 2007. New vistas for alpha-frequency band oscillations. Trends Neurosci. 30, 150–158.
Percival, D.B., Walden, A.T., 1993. Spectral Analysis for Physical Applications: Multitaper and Conventional Univariate Techniques. Cambridge University Press, Cambridge, New York, NY, USA.
Pesonen, M., Björnberg, C.H., Hämäläinen, H., Krause, C.M., 2006. Brain oscillatory 1–30 Hz EEG ERG/ERS responses during the different stages of an auditory memory search task. Neurosci. Lett. 399, 45–50.
Pesonen, M., Hämäläinen, H., Krause, C.M., 2007. Brain oscillatory 4–30 Hz responses during a visual n-back memory task with varying memory load. Brain Res. 1138, 171–177.
Pessoa, L., Ungerleider, L.G., 2004. Neural correlates of change detection and change blindness in a working memory task. Cereb. Cortex 14, 511–520.
Pourtois, G., De Pretto, M., Hauert, C.A., Vuilleumier, P., 2006. Time course of brain activity during change blindness and change awareness: performance is predicted by neural events before change onset. J. Cogn. Neurosci. 18, 2108–2129.
Raghavachari, S., Kahana, M.J., Rizzuto, D.S., Caplan, J.B., Kirschen, M.P., Bourgeois, B., Madsen, J.R., Lisman, J.E., 2001. Gating of human theta oscillations by a working memory task. J. Neurosci. 21, 3161–3174.
Rensink, R., 2002. Change detection. Annu. Rev. Psychol. 53, 245–277.
Schankin, A., Wascher, E., 2007. Electrophysiological correlates of stimulus processing in change blindness. Exp. Brain Res. 183, 95–105.
Sederberg, P.B., Kahana, M.J., Howard, M.W., Donner, E.J., Madsen, J.R., 2003. Theta and gamma oscillations during encoding predict subsequent recall. J. Neurosci. 23, 10809–10814.
Shapiro, K.L., Arnell, K.A., Raymond, J.E., 1997. The attentional blink: a view on attention and a glimpse on consciousness. Trends Cogn. Sci. 1, 291–296.
Sheth, B.R., Sandkuhler, S., Bhattacharya, J., 2009. Posterior Beta and anterior gamma oscillations predict cognitive insight. J. Cogn. Neurosci. 21, 1269–1279.
Simons, D.J., 2000. Current approach to change blindness. Vis. Cogn. 7, 1–15.
Simons, D.J., Levin, D.T., 1998. Failure to detect changes to people during a real-world interaction. Psychon. Bull. Rev. 5, 644–649.
Simons, D.J., Rensink, R.A., 2005. Change blindness: past, present, and future. Trends Cogn. Sci. 9, 16–20.
Simons, D.J., Chabris, C.F., Schnur, T.T., Levin, D.T., 2002. Evidence for preserved representations in change blindness. Conscious. Cogn. 11, 78–97.
Steinmetz, P.N., Roy, A., Fitzgerald, P.J., Hsiao, S.S., Johnson, K.O., Niebur, E., 2000. Attention modulates synchronized neuronal firing in primate somatosensory cortex. Nature 404, 187–190.
Tallon-Baudry, C., Bertrand, O., 1999. Oscillatory gamma activity in humans and its role in object representation. Trends Cogn. Sci. 3, 151–162.
Tallon-Baudry, C., Bertrand, O., Peronnet, F., Pernier, J., 1998. Induced gamma-band activity during the delay of a visual short-term memory task in humans. J. Neurosci. 18, 4244–4254.
Treisman, A., 1993. The perception of features and objects. In: Baddeley, A., Weiskrantz, L. (Eds.), Attention: Selection, awareness and control. A tribute to Donald Broadbent. Clarendon Press University, Oxford, pp. 5–35.
Varela, F., Lachaux, J.P., Rodriguez, E., Martinerie, J., 2001. The brainweb: phase synchronization and large-scale integration. Nat. Rev. Neurosci. 2, 229–239.
Webster, M.A., Kaping, D., Mizokami, Y., Duhamel, P., 2004. Adaptation to natural facial categories. Nature 428, 557–561.
Zion-Golumbic, E., Golan, T., Anaki, D., Bentin, S., 2008. Human face preference in gamma-frequency EEG activity. Neuroimage 29, 1980–1987.