**Painful stimulation could elicit transient changes in ongoing electroencephalographic (EEG) activity, reflected as phase-locked event-related potentials (ERPs) and non-phase-locked modulations of ongoing EEG oscillations [1-2]. These modulations, usually confined to a specific frequency band, consist of a transient enhancement (event-related synchronization, ERS) or suppression (event-related desynchronization, ERD) of EEG power [3-4]. Among these modulations of EEG power, the effect of noxious stimuli on the suppression of beta band oscillations has been extensively investigated [1,3,5,6]. Several neurophysiologic studies demonstrated that pain induced alpha ERD over the contralateral somatosensory cortex [6, 9] may reflect the functional state of the primary somatosensory cortex (SI) [7] and its contributions to pain processing [6, 8]. In contrast, a widespread suppression of alpha oscillation after noxious stimuli was observed to be maximal at the posterior parietal and occipital regions [3-4, 10], thus reflecting the endogenous task related cortical processing [11]. In the present study, we hypothesized that pain induced alpha ERD captured multiple functions, each of which could be disentangled and represented as distinct features.**

**RESULTS AND SIGNIFICANCE**

1. **In the target condition**, the pain induced alpha ERD was maximally distributed at the contralateral-central regions at early latencies (250 ms to 350 ms) (Figs. 2 & 3), but maximally distributed at the posterior parietal and occipital regions at late latencies (400 ms to 750 ms). As revealed by source analysis, late latency alpha ERD was generated from bilateral occipital lobes (Fig. 1).

2. **In the non-target condition**, the pain induced alpha ERD was maximally distributed at the contralateral-central regions for a long-lasting period (250 ms to 750 ms) (Figs. 1-3), and source analysis revealed that the pain induced alpha ERD would reflect summation effects of stimulus-related and task-related cortical processing, and could be greatly influenced by the variation of subjects’ mental states.

3. **Alpha ERD magnitude** was significantly different across stimulus bins (different level of alpha ERD magnitude) in both target (F = 11.22, P < 0.001) and non-target (F = 12.44, P < 0.001) conditions, whereas post-stimulus alpha power was not significantly different across bins in both target (F = 1.07, P = 0.382) and non-target (F = 1.29, P = 0.269) conditions.

4. **Pre-stimulus alpha power** is significantly different across target and non-target conditions. In summary, pain induced alpha ERD would reflect summation effects of stimulus-related and task-related cortical processing, and could be greatly influenced by the variation of subjects’ mental states.

**Methodology**

Eighteen healthy right-handed volunteers (nine females) with a mean age of 22 years (range 19-29 years) participated in the study. All subjects gave written informed consent before participation, and the study was approved by the local ethics committee. The EEG data were recorded using a 64-channel Brain Products system (pass band: 0.01-100 Hz, sampling rate: 500 Hz) from an oddball task paradigm, in which the noxious sensation was triggered using the intra-epidermal stimulation [12]. Intra-epidermal electrical stimuli were constant-current square electric pulses of 0.5 ms duration delivered through a stainless steel concentric bipolar needle electrode consisting of a needle cathode (length: 0.1 mm, φ: 0.2 mm) surrounded by a cylindrical anode (φ: 1.4 mm). The stimuli were delivered to the medial and lateral side of the left hand dorsum, and all the noxious stimuli were reported as painful pinprick sensation for all subjects.

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**References**