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# Deficits in low beta desynchronization reflect impaired emotional processing in schizophrenia

Gábor Csukly [a,*], Kinga Farkas [a], Csilla Marosi [a], Ádám Szabó [b]

[a] Department of Psychiatry and Psychotherapy, Semmelweis University, Budapest, Hungary
[b] MR Research Center, Semmelweis University, Budapest, Hungary

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ABSTRACT

Empirical data from previous investigations showed that emotion processing is reflected in beta, and especially in low beta event related desynchronization (ERD) (i.e. a decrease in low beta power). While recognition of social information and emotion processing are impaired in schizophrenia, no previous study analyzed induced and evoked beta oscillations in patients with schizophrenia during emotion processing.

Twenty-eight subjects with schizophrenia and twenty-seven healthy controls subjects were enrolled in the study. The two study groups did not differ in age, gender and education. Participants viewed positive, neutral and negative scenes selected from the International Affective Picture System (IAPS) while 128-channel EEG was recorded.

A significantly weaker low beta ERD was detected in patients relative to controls for the negative stimulus condition in the right parieto-occipital and temporal regions. Patients with decreased beta ERD showed more prominent negative symptoms and more severe deficits in psychosocial functioning. Only in the control group stronger beta ERD was detected for the negative stimuli relative to positive and neutral stimuli in the same regions.

Our major finding is that impaired emotion processing in schizophrenia is reflected in decreased low beta ERD and in the diminished differences between low beta ERD to negative and non-negative emotional stimuli. Furthermore, it was found that patients with decreased beta ERD show more prominent negative symptoms and more severe deficits in psychosocial functioning.



## 1. Introduction

Recognition of social information and emotion processing are impaired in schizophrenia (Kohler and Martin, 2006; Pinkham, 2014; Taylor and MacDonald III, 2012).The majority of previous investigations studied the electrophysiological correlates of emotion processing in schizophrenia used facial expression recognition paradigms, which raise the possibility that the findings are specific to facial expression recognition to some degree. To our knowledge only two studies examined the electrophysiology of emotion processing in schizophrenia by the International Affective Picture Set (IAPS). Based on Event Related Potential (ERP) results they came to the conclusion that early stages of visual stimuli processing are abnormal in schizophrenia, however there the relatively small sample size ($n < 20$ in both groups), the significant difference between the study groups in level of education, and the inclusion of only male subjects limit the generalizability of the results (Pinheiro et al., 2013). Another study by De et al. (2013) examined emotion processing in schizophrenia by an inhibitory control task, and found a differentiation between negative and neutral stimuli in the early phase (<100 ms), which was diminished in patients with schizophrenia, however also in this study the groups differed significantly in education and again predominantly male subjects were enrolled.

While the ERP components linked to emotion processing are relatively well studied, only a few studies analyzed the oscillatory and the late (>800 ms) electrophysiology correlates of this process. Rhythms in the beta range (12–30 Hz) are found in many parts of the nervous system and are associated with attention, perception, and cognition. It has been suggested that beta oscillations are linked to the process of highlighting a stimulus as novel or salient that warrants further attention (Uhlhaas et al., 2008). Experimental and modeling efforts suggest that beta rhythms play an important role in higher level interactions involving more distant structures (Kopell et al., 2000) since their synchronization is less susceptible to long conduction delays compared to gamma band oscillations.

In a recent study Merkl et al. (2015) found a larger desynchronization in the beta band during depressed patients' reported emotional empathy for negative stimuli than when patients reported to have no empathy. Other empirical data from previous investigations also showed that emotion processing is reflected in beta (12–30 Hz), and especially in low (12–16 Hz) beta desynchronization respectively (Del Zotto et al., 2013; Cooper et al., 2013; Yoshino et al., 2012;

* Corresponding author at: 1083, Balassa 6, Budapest, Hungary.
*E-mail addresses:* csukly.gabor@semmelweis-univ.hu, csugab@yahoo.com (G. Csukly).

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Aftanas et al., 2006), while Miskovic and Schmidt (2010) found cross regional coherence in the beta band during affective image viewing. While based on these results we assumed that emotion processing would be associated primarily with low beta oscillations in general, little is known on the relationship between emotion processing and brain oscillations in schizophrenia. In a previous investigation (Csukly et al., 2014) we showed that oscillatory analysis (Event Related Spectral Perturbation = ERSP) brings important insights to emotion processing deficits in schizophrenia compared to conventional ERP analysis, since ERSP also captures induced activities, which is not phase-locked to the stimulus (Makeig, 1993) and therefore not represented in ERPs. Unlike evoked responses, induced activity is not phase-locked to the stimulus, and if a brain response to an event is not phase-locked across trials precisely enough, the potentially important induced activity will be averaged out from the ERPs. Accordingly, oscillatory changes 'induced' by experimental events can be poorly represented in, or completely absent from the time-domain features of the ERP 'evoked' by the same events. In order to gain a full insight into the electrophysiological activity linked to emotion processing, in this study evoked and induced activity were analyzed by calculating the ERSP during stimulus processing (Makeig, 1993). ERSP measures relative changes from the spectral power baseline, allowing the study of the time course of the EEG signal energy in specific frequency bands.

Only a few studies examined oscillatory brain activity linked to emotion processing in psychiatric disorders. Aftanas et al. (2003) found impaired event related synchronization over the left hemisphere in patients with alexithymia relative to healthy control subjects when viewing affective pictures. Furthermore, a recent theory put forward by Uhlhaas and Singer (2011, 2010) suggests that aberrant development of neural oscillations during adolescence in schizophrenia may lead to impaired neural activation and temporal coding and thus lead to neurocognitive dysfunctions, such as deficits in facial expression recognition and emotion processing. A study by Ramos-Loyo et al. (2009) found decreased theta activity over central and frontal regions in patients with schizophrenia in a facial emotion recognition task. However, in this study the changes from baseline in oscillatory activity (synchronization) were not measured. To our knowledge the present investigation is the first to analyze induced and evoked beta oscillations in patients with schizophrenia during emotion processing.

Due to impaired emotion processing in schizophrenia we expected decreased beta desynchronization in patients compared to healthy controls. Furthermore, we hypothesized that these deficits would be associated with negative symptom severity and psychosocial functioning in patients with schizophrenia.

## 2. Methods

### 2.1. Subjects and clinical measures

Twenty-eight subjects with schizophrenia (16 males, mean age 37.7 ± 8.4 years) and twenty-seven healthy controls subjects (15 males, mean age 38.2 ± 10.6 years) were enrolled in the study. The two study groups did not differ in age and education (Table 1). All participants were right-handed with the exception of one left-handed and one mixed handed patient and two left-handed healthy controls. All participants had normal or corrected-to-normal vision.

Selection criteria for all participants were no history of any CNS disease, mental retardation, epileptic seizure, substance dependence or substance abuse in the past 3 months, no history of head injury with loss of consciousness more than ten minutes. For healthy controls further exclusion criteria were any psychiatric disorder and a global severity index of >114 on the Symptom Checklist–90–R (Derogatis and Melisaratos, 1983), according to a Hungarian population sample (Unoka et al., 2004), in order to exclude subjects with high risk for psychiatric disorders.

Patients were recruited from the Department of Psychiatry and Psychotherapy, Semmelweis University, Budapest, Hungary. All patients met the criteria for schizophrenia based on the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV)(American Psychiatry Association, 1994). Psychiatric symptoms on the PANSS (Positive and Negative Syndrome Scale) (Kay et al., 1987) were evaluated by a trained psychiatrist. At the time of testing all patients took antipsychotic medication, the mean Chlorpromazine equivalent dose (Gardner et al., 2010) was 731 mg/day (SD = 322), while 11 patients took Benzodiazepines, and the mean Clonazepam equivalent dose was 0.7 mg/day (SD = 1.1 mg). Psychosocial functioning was measured by the Personal and Social Performance Scale (PSP) (Burns and Patrick, 2007). Demographic information for both study groups and the clinical characteristics of the patient group are presented in Table 1.

### 2.2. Stimuli and procedures

During EEG recordings, subjects were seated in a dimly lit, sound-attenuated room. Stimuli were presented on a computer screen at a viewing distance of approximately 50 cm using the Presentation 13.0 software (Neurobehavioral Systems, Inc.; Albany, CA). Each picture was displayed in color and occupied the entirety of a 19-in. (48.3 cm) monitor. At the viewing distance each picture occupied approximately 40° of visual angle horizontally and vertically. A total of 180 pictures were presented from the International Affective Picture System (IAPS) (Lang et al., 2008) of these, 60 depicted pleasant scenes (e.g., smiling faces, fun scenes depicting sports, family, and animals; valence dimension: Mean = 7.4 SD = 0.4; arousal dimension: Mean = 4.8, SD = 0.9), 60 depicted neutral scenes (e.g., neutral faces, household objects; valence dimension: Mean = 5.2 SD = 0.4; arousal dimension: Mean = 3.6, SD = 0.9), and 60 depicted unpleasant scenes (e.g., sad/angry faces, wreckages, aggressive/attack pictures; valence dimension: Mean = 2.5 SD = 0.4; arousal dimension: Mean = 5.6, SD = 0.8). Each picture was presented for 800 ms, and preceded by a fixation cross for 700 ms (ISI), which was similar to other EEG experiments applying paradigms with the IAPS pictures (Guntekin and Basar, 2010; Pinheiro et al., 2013; Foti et al., 2009; Hajcak and Dennis, 2009). This was a passive paradigm, and no response was needed by the subjects.

### 2.3. EEG recording and processing

EEG was recorded from DC with a low-pass filter at 100 Hz using a high-density 128-channel BioSemi ActiveTwo amplifier (Metting van Rijn et al., 1990). The electrode caps had an equidistant-layout and covered the whole head. EOG electrodes to monitor eye movements were placed below the left and above the right external canthi. Data were digitized with a sampling rate of 1024 Hz. Built-in and self-developed functions as well as the freeware EEGLAB toolbox (Delorme and Makeig, 2004) in the Matlab (MathWorks, Natick, MA) development environment were used for subsequent off-line data analyses. EEG was re-referenced to the common average potential and filtered off-line between 0.5 and 40 Hz using zero-phase shift forward and reverse IIR Butterworth filter.

Epochs from 400 ms pre-stimulus to 1500 ms post-stimulus were extracted from the continuous EEG for further analysis and corrected for the pre-stimulus baseline. The removal of muscle and eyes movement artifacts (detected by EOG) was performed by ADJUST (Mognon et al., 2010) an ICA (Independent Component Analysis) based automatic artifact detector. Furthermore epochs with a voltage exceeding ±100 μV on any EEG or EOG channel were rejected from the analysis. Total trial number per each picture type (neutral, positive, and negative) was 60. After artifact rejection, the average number of trials in the control group was 57.7 trials (SD = 4.1), 57.8 trials (SD = 3.3), and 57.5 trials (SD = 4.5) for the neutral, positive, and negative condition, respectively. For patients with schizophrenia the mean

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