Brain’s change detection elicited by emotional facial expressions in depressed and non-depressed individuals

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ABSTRACT

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Previous research has shown that people suffering from depression typically have deficits in emotional information processing. These deficits can be seen in the level of behavior and also in the brain processes. The aim of our study was to investigate with event-related potentials, whether depressed individuals have differences in their brain processes compared to non-depressed individuals when emotional facial expressions are used as stimuli. We applied an oddball paradigm in order to elicit visual mismatch negativity and thus investigate brain's change detection. The brain reacts to infrequently presented stimuli and recognizes the change in the pattern. We also investigated whether short psychotherapy (Acceptance and Commitment Therapy, ACT) alters the brain processes of depressed individuals. Individuals were shown pictures of human faces. In 80% of the faces, the expression was neutral. In the rest of the faces, the expression was emotional. Half of the pictures depicted fearful expression. The other half depicted happy expression. Pictures were presented in quasi-random order on a computer screen. Twenty depressed and ten non-depressed individuals participated in the study. During the experiment, a participant's EEG was recorded with electrodes attached to their scalp. Depressed individuals were randomized in to two groups. The first group received therapy immediately. The second group received therapy after six weeks from the first EEG recording. Those in the immediate treatment group had their EEG recorded twice. Those in the delayed treatment group had EEGs recorded three times. The non-depressed group had an EEG recorded only once. Stimuli were the same in each recording session. The results suggested that depressed and non-depressed individuals processed emotional facial expressions differently. Non-depressed individuals were able to differentiate emotional and neutral expression from one another. In the depressed group, this differentiation did not occur. The P240 component's discriminative power seemed to be lateralized to the posterior parts of right hemisphere, whereas N240's discriminative power was localized in fronto-parietal regions. Component N170 seemed to be the best to reflect a visual mismatch negativity. N170 was able to differentiate between the two emotional expressions which components P240 and N240 were not able to do. These results were in line with previous research which has shown that depression is often characterized by the dysfunctional processing of emotional information.

Keywords: event-related potentials (ERPs), visual mismatch negativity (vMMN), depression, N170, brain's change detection, emotional stimuli, facial expressions, oddball

TIIVISTELMÄ

Aivojen muutosdetektio emotionaalisiin kasvonilmeisiin masentuneilla ja ei-masentuneilla ihmisillä

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Aikaisemmissa tutkimuksissa masentuneilla ihmisillä on löydetty sekä käytöksen että aivojen tasolla näkyvää poikkeavuutta emotionaalisen informaation käsittelyssä. Tutkimuksemme tarkoituksena oli herätevasteita mittaamalla selvittää, eroavatko masentuneiden ja ei-masentuneiden henkilöiden aivoprosessit toisistaan, kun ärsykkeinä käytetään emotionaalisia kasvokuvia. Käytimme oddball -asetelmaa. Oddball -asetelmassa aivot reagoivat harvoin esitettyyn ärsykkeeseen tunnistaen ärsykevirrassa tapahtuneen muutoksen. Tarkoituksenamme oli aikaansaada visuaalisten ärsykkeiden aiheuttama poikkeavuusnegatiivisuus ilmiö ja näin tarkastella aivojen muutoksen havaitsemisen kykyä. Lisäksi tutkimme, vaikuttaako lyhyt psykoterapia (hyväksymis- ja omistautumisterapia) masentuneiden henkilöiden aivoprosesseihin. Oddball -asetelmassa tutkittaville näytettiin ihmiskasvoja, joista 80%:lla oli neutraali ilme. Loppujen kuvien ihmiskasvoilla oli joko iloinen tai pelästynyt ilme. Kummankin emotionaalisen ilmeen esiintymistodennäköisyys oli 0.10. Kuvat esitettiin satunnaisessa järjestyksessä tutkittaville tietokoneen ruudulta. Kokeeseen osallistui 20 masentunutta ja 10 ei-masentunutta henkilöä. Kokeen aikana tutkittavilta mitattiin aivosähkökäyrää päänahan pinnalle asetetuilla elektrodeilla. Masentuneet henkilöt satunnaistettiin kahteen ryhmään, välittömän hoidon ja viivästetyn hoidon ryhmään. Välittömän hoidon ryhmään kuuluvilta EEG mitattiin kaksi kertaa, viivästetyn hoidon ryhmäläisiltä kolme kertaa ja ei-masentuneiden ryhmältä kerran. Ärsykkeet olivat samat joka mittauskerralla. Masentuneet henkilöt saivat terapiaa, joka toteutettiin kuuden viikon sisällä ennen viimeistä EEG-mittausta. Tuloksista kävi ilmi, että masentuneiden ja ei-masentuneiden henkilöiden aivot käsittelivät emotionaalisia kasvonilmeitä eri tavoin. Ei-masentuneiden henkilöiden aivot kykenivät erottelemaan neutraalin ja emotionaalisen ilmeen toisistaan, mutta masentuneiden ryhmässä tällaista erottelua ei tapahtunut. Komponentin P240 erotteluvoima näyttäisi olevan lateralisoitunut oikealle aivopuoliskolle takaraivolohkolle, ja N240:n otsalohkon ja päälakilohkon keskivaiheille. Näyttäisi siltä, että komponentti N170 on paras kolmesta tutkitusta komponentista ilmentämään visuaalista poikkeavuusnegatiivisuutta. N170 pystyi erottelemaan myös pelokkaan ja iloisen ilmeen toisistaan, jota komponentit P240 ja N240 eivät tehneet. Saadut tulokset tukevat aiempia tutkimustuloksia, joiden mukaan masennukseen liittyy häiriöitä emotionaalisen informaation prosessoinnissa.

Avainsanat: herätevasteet, visuaalinen poikkeavuusnegatiivisuus, masennus, N170, aivojen muutosdetektio, emotionaaliset ärsykkeet, kasvonilmeet, oddball

CONTENTS

1. Introduction

The human face is a unique object for human beings as the nature of face recognition and processing has a special significance for humans (Nelson, 2001; Kanwisher & Moscovitch, 2000). Facial expressions of human emotion have strong attention-directing power and communicative value. They are powerful indicators of emotional states and intentions of others. Therefore, the human face is an essential form of social nonverbal communication.

The ability to correctly distinguish and to recognize facial expressions is a central aspect in effective social interaction (Erickson & Schulkin, 2003). Six basic emotions (fear, happiness, disgust, anger, surprise, and sadness) have been distinguished and shown to be universal across cultures (Ekman & Friesen, 1971; Ekman et al., 1987). Evolutionary views of emotions perceive them as adaptations. It seems plausible to suggest that the rapid production and recognition of emotional cues, would enhance individual's survival. For example, fearful faces have been shown to elicit a rapidly-processed involuntary orienting of spatial attention towards their location (Pourtois, Grandjean, Sander & Vuilleumier, 2004). The nature of face perception is complex and no definite agreement exists between researchers. Kanwisher and Moscovitch (2000) state, that neuropsychological evidence suggests that dissociable neural systems exist for the recognition of individual faces, the discrimination of emotional expressions, and the discrimination of the direction of overt attention (i.e., gaze). Our study aims to expand the knowledge of face processing by investigating how people with and without depression process emotional expressions. This information will help to build a better understanding of depression and the information processing deficits which often characterize depression.

1.1. Objectives of the present study

We investigated the following three issues: (1) whether one or more of the ERP components, N170, P240 or N240, would reflect visual mismatch negativity as suggested by the study of Zhao and Li (2006); (2) whether there are differences in the processing of emotional facial expressions between depressed and non-depressed subjects; (3) whether brief psychotherapy modulates the depressed participant's processing of facial expressions. We applied an oddball paradigm and used emotional facial expressions as stimuli.

1.2. Mismatch negativity in the visual modality

An oddball paradigm was applied to the present study to investigate the possible appearance of visual mismatch negativity.

Event-related potentials (ERPs) are voltage fluctuations in the EEG. ERPs are time locked to sensory, motor or cognitive events and they directly measure the electrical response of the cortex to those events. The ERP waveform can be characterized across three main dimensions: amplitude, latency, and scalp distribution (Sanei & Chambers, 2007). Mismatch negativity was first found in the auditory modality (Näätänen, Gaillard & Mäntysalo, 1978). Mismatch negativity (MMN) is an electromagnetic response to a discriminable change in any regular aspect of auditory stimulation, which usually peaks 150-200 ms from change onset and is elicited even in the absence of attention (Näätänen et al., 1978). Therefore, MNN represents brain's change detection. More particularly, visual MMN is considered to be a negative wave usually appearing in a 100-400 ms post-deviance latency range. It is, like auditory MNN, an ERP response to infrequent stimuli deviating from a repeated standard stimulus (Czigler, Weisz & Winkler, 2006). However, it is still debated among researchers whether visual MMN exists and what would be the best ERP candidate for it.

Wei, Chan and Luo (2002) used an oddball paradigm to investigate mismatch negativity in auditory and visual modalities. They concluded, that visual early deviant-related negativity might be a possible analogue to auditory MMN because of their common features. Pazo-Alvarez, Cadaveira and Amenedo (2003) state in their review that, a deviant-related MMN-like component, possible analogue to auditory MMN, seems to exist in the visual modality. The Pazo-Alvarez review was focused on studies concerned with changes in simple visual features such as shape, colour and motion direction. It appeared that a greater difference between standards and deviants was necessary to elicit visual MMN compared with the auditory MMN (Pazo-Alvarez et al., 2003). Furthermore, a study by Maekawa et al. (2005) obtained a valid visual MMN using a windmill-pattern stimulus. A deviant-related component was significantly modulated by deviancy and upon which Maekawa et al. concluded that it was possible to detect and to assess preattentive processing in the visual modality.

Zhao and Li (2006) investigated visual MMN elicited by facial expressions under non-attentional condition. The N170 was significantly enhanced and the P250 significantly reduced when elicited by deviant stimuli (sad and happy faces) rather than by standard stimuli (neutral faces). Also, the visual MMN elicited by sad faces was larger (more negative) than that elicited by happy faces. Zhao et al. concluded that the modulations of N170 and P250 reflected visual MMN. Lastly, Susac, Ilmoniemi, Pihko and Supek (2004) concluded that a MMN-like negativity was obtained in their study when faces were used as stimuli in an oddball paradigm.

Research where complex visual stimuli (e.g. faces) are used to elicit visual mismatch negativity has not been yet carried out extensively. Our research therefore aims to shed light on this rather new branch of research.

1.3. The face-specific N170 component

The existence of the N170 component is well documented and its central role in face processing is acknowledged.

Faces, compared with other visual objects, typically elicit an ERP component known as N170. It peaks approximately 170 ms after stimulus onset at occipital-temporal recording sites (Bentin, Allison, Puce, Perez & McCarthy, 1996; Itier & Taylor, 2004). The N170 component is face-specific; it is elicited by human faces, but not for example by animal faces, cars and items of furniture (Bentin et al. 1996). It has been suggested that different facial expressions of emotion elicit different responses in the brain and that N170 is affected. However, researchers have obtained differing results.

In the study of Eimer, Holmes and McGlone (2003) faces expressing six basic emotions were compared with neutral faces by recording ERPs. Two sets of designs were used: one where attention was directed toward facial expressions (emotion task), and another where it was directed elsewhere. The results showed that an enhanced positivity for emotional, relative to neutral faces, was elicited in the emotion task. This effect of emotional expression was visible at fronto-central sites starting about 160 ms post-stimulus. The N170 component was unaffected by different facial expressions. Emotional effects obtained in the emotion task were remarkably

similar for all six basic facial expressions. When attention was directed away from the faces toward a demanding perceptual discrimination task, the emotional expression effects were completely eliminated. However, Batty and Taylor (2003) received differing results. Their data suggested an early automatic encoding of emotional facial expressions reflected by N170. Latency and amplitude differences among emotional expressions (the six basic emotions compared to neutral) were seen from 140 ms post-stimulus. Positive emotions evoked N170 significantly earlier than negative emotions while the amplitude of N170 evoked by fearful faces was larger than for neutral or surprised faces. The study's participants' task was to respond to target stimuli (pictures of cars, etc.), which were not concerned with the facial expressions. Similarly, in the study of Leppänen, Moulson, Vogel-Farley and Nelson (2007), larger amplitude for the N170 was found for fearful faces, compared with happy and neutral faces, in passive observation of different emotional facial expressions. Another study investigated emotional facial expression discrimination in a visual oddball task by morphing two emotional expressions, fear and sadness (Campanella et al. 2002). The expressions of fear and sadness were used as a standard stimulus with half of the deviants depicting the same emotion as the standard, and the other half either fear or sadness, depending on standard stimulus. Campanella et al. found that response for deviants compared to standard stimuli was more negative in the period of 200-400 ms after stimulus onset.

It remains undefined whether the N170 component is sensitive to emotional facial expressions. However, according to the studies reviewed, it seems plausible to suggest that N170 may be sensitive to different facial expressions when a certain design is applied.

1.4. Depression and the processing of emotional facial expressions

Several studies have shown that depressed people process emotional information dysfunctionally. According to Beck's (1967) model of depression, three sets of cognitive concepts can help explain the psychological aspects of depression. The first component is the pattern of construing experiences in a negative way. Depressed individuals have thought patterns that lead them to consider their interactions with their environment as representing defeat, deprivation or disparagement. The second component of the triad is the pattern of viewing oneself in a negative way. The depressed individual regards himself as deficient or unworthy. Finally, the third

component entails that one will perceive their future in a negative way. Depressed individuals anticipate that their current difficulties or suffering will continue indefinitely. The cognitive triad is consisted of these thinking patterns and these cognitive schemas provide the basis of the depressed individual's approach toward their environment. Dysfunctional schemas cause distorted experiences in depression.

The findings from studies on the processing, during depression, of emotional facial expression are somewhat inconsistent. Some researchers have found biases in the processing of negative, positive or neutral information whereas others have not. For example, Joorman and Gotlib (2006) have found depression-associated bias in the identification of facial expressions of emotion for subjects diagnosed with major depression (MDD). Subjects with MDD, when compared with non-depressed control group, require a markedly greater intensity of emotional expression to correctly identify happy expressions. Also, MDD -participants require less intensity to identify correctly sad faces as opposed to angry faces. However, MDD -participants do not identify sad expressions at a lower intensity compared to non-depressed participants. The results of the study by Gotlib et al. (2004) indicates that, persons diagnosed with MDD selectively attend to the sad face when presented with a sad face and matched neutral face. Leyman et al. (2007) have demonstrated that individuals with MDD show a stronger attententional engagement with angry faces compared with non-depressed individuals.

Depression has also been shown to disrupt the recognition of neutral faces. In the study by Leppänen et al. (2004), depressed people were able to recognize neutral expressions significantly less accurately than happy or sad expressions. This impairment was still apparent after symptom remission. Frewen and Dozois (2005) failed to demonstrate a difference in dysphoric and nondysphoric women's abilities to accurately recognize and label other's' emotions from their facial expressions. However, they did find that dysphoric woman made more negative interpretations of the meaning of other's expressions. For example, dysphoric woman were more likely to attribute themselves as the cause of other's negative emotions. Moreover, Ridout et al. (2003) investigated both the identification and recognition memory of the facial expression of emotion in people with clinical depression compared to healthy controls. No differences were detected in the emotion recognition task for depressed compared to non-depressed subjects. On

the contrary, groups' results differed significantly with the memory task. Depressed subjects remembered remarkably more sad facial expressions than happy expressions, while the control group remembered remarkably more happy facial expressions than sad expressions.

Non-depressed individuals have a tendency to shift attention away from negative faces (Bradley et al. 1997; Leyman et al. 2007; Joorman and Gotlib, 2007). This could be perceived as a "protective bias" because it diverts the individual's attention away from negative information, and thus may help maintain non-depressed state and avoidance of engaging in dysfunctional cognitions (McCabe et al. 2000). Nondysphoric people have an attentional bias towards happy faces (Bradley et al. 1997).

Mood also affects the processing of emotional facial expressions measured by ERPs. The P300 component is thought to reflect an information processing cascade when attentional and memory mechanisms are engaged (Polich, 2007). According to Cavanagh and Geisler (2006), a cognitive processing difference exists between depressed and non-depressed subjects especially in low intensities of happy faces: the depressed subject group have lower and delayed P300s to happy faces. As the intensity of facial expressions decreased, depressed participants took increasingly longer to process happy faces. Cavanagh and Geisler argue that lower P300 amplitudes may indicate that happy faces are not as salient to depressed participants and that they may use less attentional resources when viewing happy faces. Deldin, Keller, Gergen and Miller (2001) also investigated the P300 component in major depression. Depressed subjects did not show a bias towards negative information whereas non-depressed controls showed bias towards positive stimuli. The P300 component was interpreted by the authors as reflecting resource allocation. Depressed participants allocated equal amounts of resources when recognizing positive and negative stimuli. The control group, however, allocated more resources when first viewing positive information and fewer resources when recognizing positive information. Additionally, P300 was enhanced for the control group during encoding and reduced during recognition of positive stimuli.

Deveney and Deldin (2004) investigated the slow wave ERP amplitudes in response to emotional facial expressions during a working memory task in individuals with and without MDD. The

results suggested that controls and individuals with MDD differentially maintained valenced information in their working memory. Non-depressed individuals exhibited smaller slow wave ERP amplitudes in response to negatively valenced faces. This was perceived as an avoidance of elaborative processing of negative stimuli in the control group. Individuals with MDD showed equivalent slow wave amplitudes for negative and positive facial expressions. In addition, results of this study implicated the left parietal region in the differential slow wave amplitudes for facial stimuli in the working memory task. The study by Deldin, Keller, Gergen and Miller (2000) showed that a region-specific group difference between individuals with MDD and nondepressed controls exists. The results suggested a reduced right-posterior functioning in depression. The group with MDD exhibited a reduction in the N200 component of ERP to facial stimuli which were most mood incongruent (happy faces).

1.5. Psychotherapy and EEG

Psychotherapy's effects on brain functioning have been investigated with, for example, an fMRI, but an EEG has not been extensively used to study the treatment-effects of psychotherapy. Some studies exist where depressed participants have undergone EEG sleep recordings before and after psychotherapy treatment. For example, the study by Nofzinger et al. (1994) suggests that cognitive-behavioral therapy can alter the sleep patterns of depressed participants when measured with an EEG. Similar results were found in a study by Thase et al. (1997) where depressed individual's' abnormal EEG sleep patterns were normalized after psychotherapy. Deldin and Chiu (2005) investigated individuals with major depression. Their interest was the predictive utility of EEG alpha power with regard to mood improvement. The techniques of cognitive therapy were applied as an intervention. Deldin and Chiu stated that most striking was the presence and predictive utility of overall greater cortical activity for identifying those who reporter greater post- than pre-intervention happiness compared with those who did not report such an improvement.

These studies, along with other studies with similar results, suggest that psychotherapy can modulate brain's electrical activity and possibly normalize it.

2. Method

2.1. Participants

Participants of this study consisted of non-depressed and depressed people.

2.1.1. Depressed group

Twenty-four depressed people participated in this study but only seventeen performed all the parts. Twenty people participated in the first recording. Therefore only twenty participants qualified to be included for statistical analysis concerning the phase before onset of treatment. Seventeen participants who performed all the parts of the study were chosen for the treatment-effect analysis. Of those who qualified, twenty (n=20) were 28-59 years of age (M=43.1 years), fifteen were female, one was left-handed, and ten had been diagnosed with depression. Participants were recruited via add in local newspaper. Those feeling depressed were encouraged to contact the research group. Subjects were then interviewed by phone and the following rejection criteria was applied: drinking problems, diagnosis of schizophrenia, suicidal tendencies, past or present neurological illness or injury, and an age over 60 or less than 18 years. Subjects who qualified were randomly assigned to either group 1 or group 2. All participants had normal or corrected-to-normal vision. Eight subjects reported taking medication for their depression. Subjects were informed of the study and they signed a written informed consent. The ethical committee of the University of Jyväskylä approved the study and the experiment was undertaken in accordance with the Declaration of Helsinki. Group 1 received therapy after the first EEG recording whereas group 2 received therapy six weeks after the first EEG recording.

Although participants were randomly assigned to the groups, the groups differed in by age and the level of depression. On group 1, the mean BDI-score before the onset of treatment was 29.3. On group 2, the mean BDI-score was 23.3. The participants on group 1 were also older than the participants on group 2 (M=46.9 versus M=39.4).

2.1.2. Non-depressed group

Ten healthy, non-depressed volunteers were qualified to participate in this study; they were between 21 and -50 years of age (M=28.8 years), 7 were female and all were right-handed.

Participants were recruited via an e-mail post among the students of University of Jyväskylä, as well as from acquaintances of members of the research group. All participants had normal or corrected-to-normal vision. None reported a history of past or present neurological illness. Participants reported taking no medication affecting the nervous system. Participants were informed of the study and they signed a written informed consent form. After EEG recordings, participants were given self evaluation forms in order to control for their level of depression. The forms were to be taken home and returned via post. Those participants who returned the forms and did not show signs of depression were included for analysis (10 out of 14). The group of non-depressed participants will be referred as group 3 further in this paper.

The EEG recording procedure and stimuli were identical for all groups. Only depressed people received therapy.

2.2. Stimuli and procedure

Stimuli were black and white pictures of faces expressing emotions. The pictures were taken from a standard set of pictures of facial affect (Ekman & Friesen, 1976). They were presented in the middle of a computer screen on a black background. Four different identities were used; two female and two male. Pictures depicting neutral, happy, or fearful facial expressions were included from each identity.

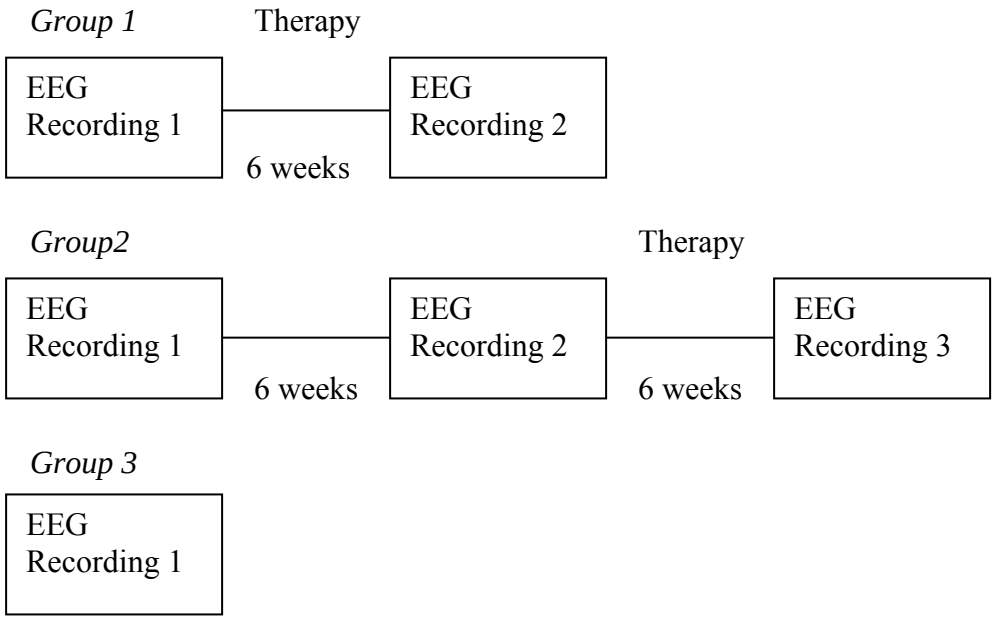


Figure. 1. A diagram of recordings and therapy for groups 1, 2 and 3.

Participants were seated in a comfortable chair in recording room. A computer screen, with a visual angle of 6,2° × 8,6°, was placed (100 cm) in front of the participant. A white cross in the middle of the screen was used as a fixation point. An oddball paradigm was applied ($P_{standard}$=0.8, $P_{deviant}$=0.1 for both expressions). Pictures with neutral facial expressions served as the standard stimulus. Two emotional expressions, fear and happiness, were used as deviants. Pictures of emotional faces were shown in a quasi-randomized order: there were at least two standard stimuli between deviant stimuli. Participants were instructed to fixate to the middle of the screen and remain their fixation throughout the whole recording period. No task was given concerning the pictures. A radio play was played to direct the participant's attention away from conscious observation of photos, and therefore ensuring passive viewing. The experiment consisted of two parts and two paradigms were utilized: oddball and priming. The first part (oddball) lasted approximately 20 minutes and was always carried out before the second part (priming). However, because this paper concentrates on the first part of the experiment (oddball), the second part of the study will not be discussed any further here.

Each photo was presented for 200 ms, followed by 700 ms break before the next stimulus. A neutral facial expression was presented 1600 times. Happy and fearful facial expressions were presented 160 times each.

Fourteen electrodes (F3, F4, Fz, C3, C4, Cz, P3, P4, Pz, P7, P8, O1, O2, Oz) were inserted to record ERPs using an electrode cap (Easy Cap). Brain Vision Recorder (Brain Products) software was used to record the data. Impedances were kept below 5 kΩ. Eye movements were recorded with two bipolar electrodes from the right outer canthus and below the left eyebrow. An average reference was used.

EEG recordings were carried out before the onset of therapy and both groups (1 and 2) were recorded at the same time. Over the following six weeks, only group 1 received therapy. After the end of therapy, EEG recordings were again carried out for both groups. After this round of recordings, group 2 received the same amount and type of therapy as group 1. Again, after therapy, the EEG recoding were carried out for group 2. Group 1 was not recorded for a third time. The therapy utilized was Acceptance and Commitment Therapy (ACT). The treatment

consisted of four sessions with each lasting 45-60 minutes. Therapy was given by students (majoring in psychology) who had received training for the method and had the opportunity to consult with their supervisor during the whole process.

2.3. Data analysis

Data were analyzed with the Brain Vision Analyzer (Brain Products) software. A 100 ms pre-stimulus baseline was applied. The data was segmented, filtered with a band pass of 0.1-30 Hz, and an artefact correction was carried out. All segments with more than -100 μV or 100 μV were considered to be contaminated by ocular activity or muscular artefact and thus were deleted. For those participants (two people) who had more than half of the segments deleted, an ocular correction method (Cratton & Coles) was applied instead of the regular artefact correction.

The peak amplitudes of the N170, P240 and N240 components were measured within the time windows of 140-210 ms and 210-270 ms after stimulus onset. Different electrodes were chosen for analysis of different components: P7, P8, O1, O2, Oz for N170; P7, P8, Pz, O1, O2, Oz, P3, P4 for P240; and F3, F4, Fz, C3, C4, Cz for N240. The electrode sites were chosen after ocular inspection of grand averaged event-related potential waveforms. The electrodes were chosen where standards and deviants seemed to differ from one another. In order to investigate whether groups differed from one another, a four-way ANOVA was utilized. Huynh-Feldt corrections were used. The within subjects factors were: expression (happy, fear) × stimulus type (standard, deviant) × electrode × the between subjects factor: group (1, 2, 3). Another ANOVA was carried out to investigate the treatment effect: measurement × stimulus type × expression × group. If ANOVA could not be utilized or it did not reveal significant interactions, correlation analyses were carried out. Correlation coefficients were calculated for both expressions' amplitude difference score and either a BDI Score Change score (Beck's Depression Inventory), Mood Score Change score (Visual Analogue Scale Measuring Mood) or AAQ2 Score Change score (Acceptance and Action Questionnaire-II) for previously determined electrodes. For example, the BDI Score Change score was calculated as such: BDI score after treatment – BDI score before treatment. Spearman's rho was used as the correlation coefficient.

BDI measures depression, Mood questionnaire measures mood and AAQ2 measures psychological flexibility and acceptance. All are self-evaluation questionnaires.

3. Results

3.1. ERP data

3.1.1. The N170 component

Three groups (1, 2 and 3) were compared with repeated measures ANOVA before the onset of treatment. A significant four-way interaction was detected for N170 amplitude F(5.09, 68.73)=3.033, p=0.015 (electrode × stimulus type × expression × group). Further analysis revealed that groups 1 and 2 differed significantly from each other F(2.72, 48.98 = 4.955, p = 0.006. Also groups 1 and 3 almost differed significantly from one another, F(2.251, 40.524)=2.939, p=0.058. However, groups 2 and 3 did not differ from one another, F(2.45, 44.22)=1.099, p=0.352. Paired samples t-tests were carried out to investigate further the differences among groups. In group 1, one significant difference between ERPs to standards and ERPs to deviants emerged, t(9)=3.579, p=0.006, for the expression of *fear* at electrode P7. With group 2, a corresponding difference was found for the expression of *happy* at Oz t(9)=6.245, p=0.000; for *happy* at P7 t(9)=2.607, p=0.028; for *fear* at P8 t(9)=3.986, p=0.003; for *happy* at O1 t(9)=4.404, p=0.002; for *happy* at O2 t(9)=4.176, p=0.002. In group 3, a difference was found for *happy* at Oz t(9)=2.331, p=0.045; for *happy* at P7 t(9)=4.675, p=0.001; for *happy* at P8 t(9)=2.804, p=0,021; for *happy* at O1 t(9)=4.178, p=0.002; for *fear* at O2 t(9)=2.438, p=0.038. Also, the expression of *fear* at electrode P8 almost reached a level of significance, t(9)=2.221, p=0.053.

Group 1

	Oz	P7	P8	O1	O2
Fear	p=0.108 0.303 µV	**p=0.006** **0.99 µV**	p=0.142 0.244 µV	p=0.062 0.352 µV	p=0.120 0.359 µV
Happy	p=0.181 0.495 µV	p=0.463 0.144 µV	p=0.098 0.751 µV	p=0.269 0.419 µV	p=0.238 0.704 µV

Group 2

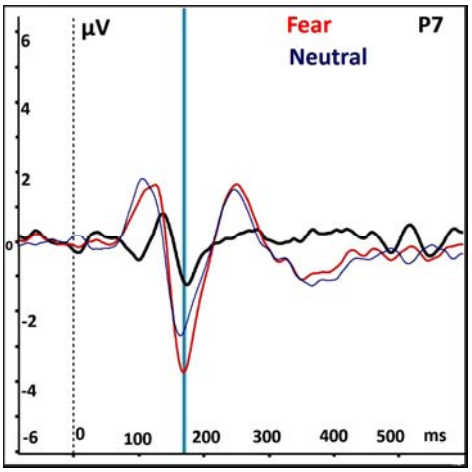
	Oz	P7	P8	O1	O2
Fear	p=0.147 0.303 µV	p=0.270 0.323 µV	**p=0.003** **0.738 µV**	p=0.142 0.414 µV	p=0.082 0.328 µV
Happy	**p=0.0001** **1.24 µV**	**p=0.028** **0.725 µV**	p=0.239 0.453 µV	**p=0.002** **1.33 µV**	**p=0.002** **1.08 µV**

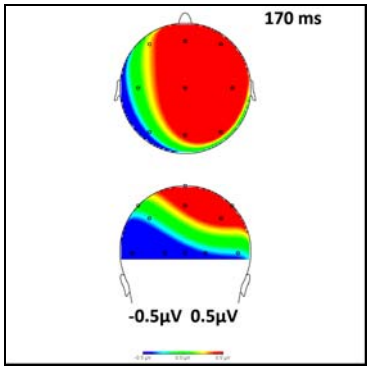
Group 3

	Oz	P7	P8	O1	O2
Fear	p=0.836 0.046 µV	p=0.147 0.465 µV	p=0.053 0.898 µV	p=0.768 0.067 µV	**p=0.038** **0.419 µV**
Happy	**p=0.045** **0.56 µV**	**p=0.001** **0.766 µV**	**p=0.021** **0.872 µV**	**p=0.002** **0.696 µV**	p=0.089 0.490 µV

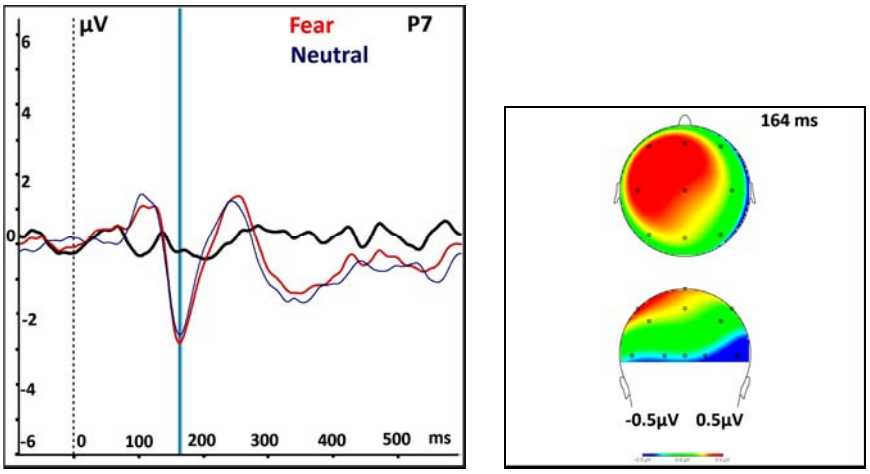
Table. 1. Tables of significant p-values obtained from paired samples t-test and the mean amplitudes (absolute values) of difference ERPs of component N170 (ERPs to neutral expression subtracted from ERPs to emotional expression) for groups 1, 2 and 3.

Group 1





Group 2



Group 3

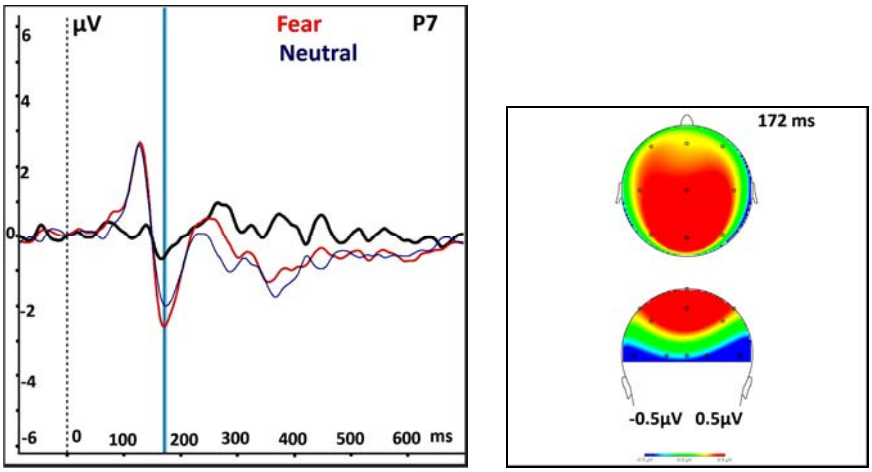


Figure. 2. Difference ERPs (black line) to fearful and neutral expressions at electrode P7. The red line represents fearful expression. The blue line represents neutral expression. The scalp potential maps illustrate the time window marked with the blue color.

Also, an analysis was carried out where only electrodes P8 and O2 were chosen. This was done because of the special role of the right hemisphere in emotional information processing. However, the depressed groups (1 and 2) still differed significantly from one another, and other groups did not.

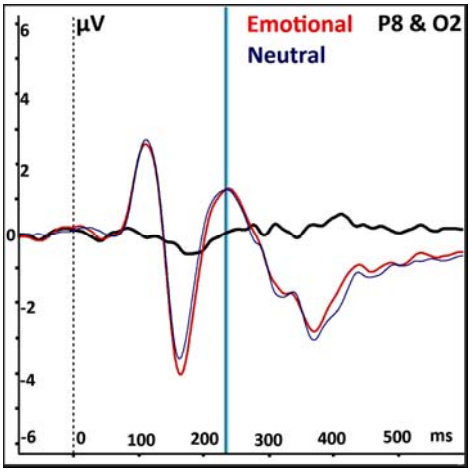
Correlation analysis was utilized. Correlations were calculated between BDI Score Change and difference ERPs. This was done for all five electrodes and for both expressions. No correlations

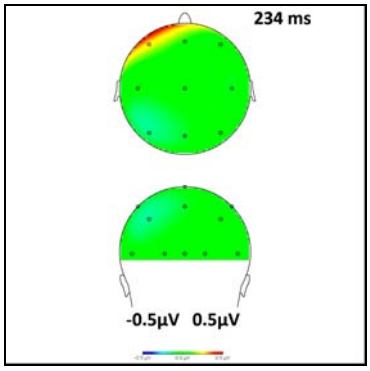
were detected. The same procedure was carried out for other self-evaluation questionnaire change scores (Mood, AAQ2). No correlations were detected.

3.1.2. The P240 component

Three groups (1, 2 and 3) were compared before the onset of treatment with repeated measures ANOVA. No statistically significant differences were detected between groups when eight electrodes (P7, P8, Pz, O1, O2, Oz, C3, C4) were included for analysis. However, when groups 1 and 2 were joined and only two electrodes, O2 and P8, were chosen for analysis, a significant two-way interaction emerged for P240 amplitude (stimulus type × group) $F(1, 28)=5.175$, $p=0.031$. Investigation was continued with a paired samples t-test. Electrode and expression factors were eliminated because they had no main effect. The mean difference in amplitudes to emotional and neutral expressions in the group 3 was 0.53 µV, ($p=0.011$). The corresponding difference in depressed group was 0.05 µV ($p=0.66$).

Depressed group





Non-depressed group

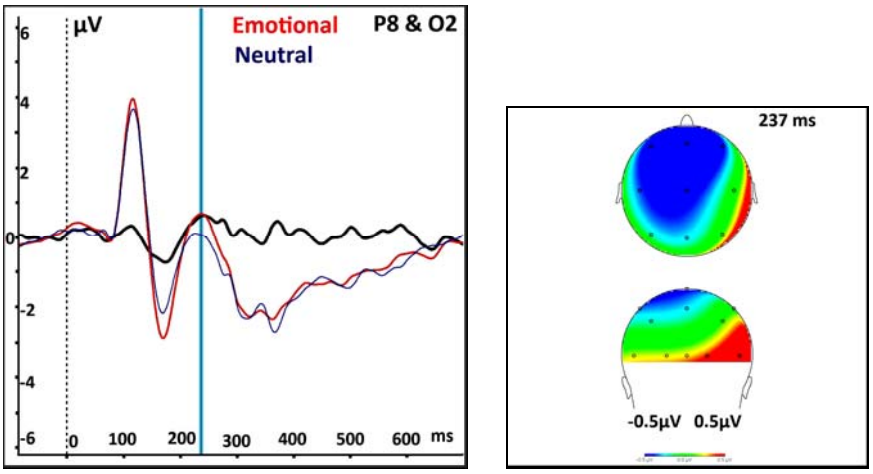


Figure. 3. Difference ERPs (black line) to emotional and neutral expressions for the depressed and non-depressed groups. Electrodes P8 and O2 are grouped together. The red line represents emotional expression. The blue line represents neutral expression. The scalp potential maps illustrate the time window marked with the blue color.

No treatment effect was detected with repeated measures ANOVA. All eight electrodes were included and groups 1 and 2 joined. No treatment effect was detected when groups were analyzed separately.

In the next stage of analysis, correlation analysis was utilized. Groups were joined. Six significant correlations were detected: electrode P4 for BDI Score Change and ERP amplitude change for fearful expressions ($p=0.001$; $\rho= -0.730$), Mood Score change and ERP amplitude change for fearful expressions ($p=0.034$; $\rho=0.517$), Mood score change and ERP amplitude change for happy expressions ($p=0.044$; $\rho= -0.493$) and AAQ2 Score Change and ERP amplitude change for fearful expressions ($p=0.006$; $\rho=0.637$); electrode Pz for Mood score change and ERP score change for happy expressions ($p=0.048$; $\rho= -0.486$) and AAQ2 Score Change and ERP score change for happy expressions ($p=0.028$; $\rho= -0.531$).

P4 ERP amplitude change

	BDI Score Change	Mood Score Change	AAQ2 Score Change
Fear	p=0.001 ρ= -0.730	p=0.034 ρ=0.517	p=0.006 ρ=0.637
Happy		p=0.044 ρ= -0.493	

Pz ERP amplitude change

	BDI Score Change	Mood Score Change	AAQ2 Score Change
Fear			
Happy		p=0.048 ρ= -0.486	p=0.028 ρ= -0.531

Table. 2. Tables of ERP amplitude change correlations between expressions and questionnaire scores at electrodes P4 and Pz. P-values and correlation coefficients (Spearman's rho) are displayed.

3.1.3. The N240 component

All three groups were compared using repeated measures ANOVA for component N240 before the onset of treatment. No significant differences between groups were detected. Again, when groups 1 and 2 were joined and then compared with group 3, a significant three-way interaction (electrode × stimulus type × group) was obtained $F(3.34, 80.53)=2.978$, $p=0.03$. All six electrodes (F3, F4, Fz, C3, C4, Cz) were included for analysis. Paired samples t-tests were carried out to investigate which electrodes differed among groups. No significantly differing responses were found in the depressed group. In group 3, two electrodes reached the level of significance: Fz ($p=0.023$) and Cz ($p=0.002$). Also, electrode F4 almost reached a level of significance ($p=0.058$). The mean amplitude of difference ERPs at electrode Fz in group 3 was -0.56 μV and 0.02 μV in the depressed group. The mean amplitude of electrode Cz in group 3 was -0.46 Vμ and -0.08 μV in the depressed group.

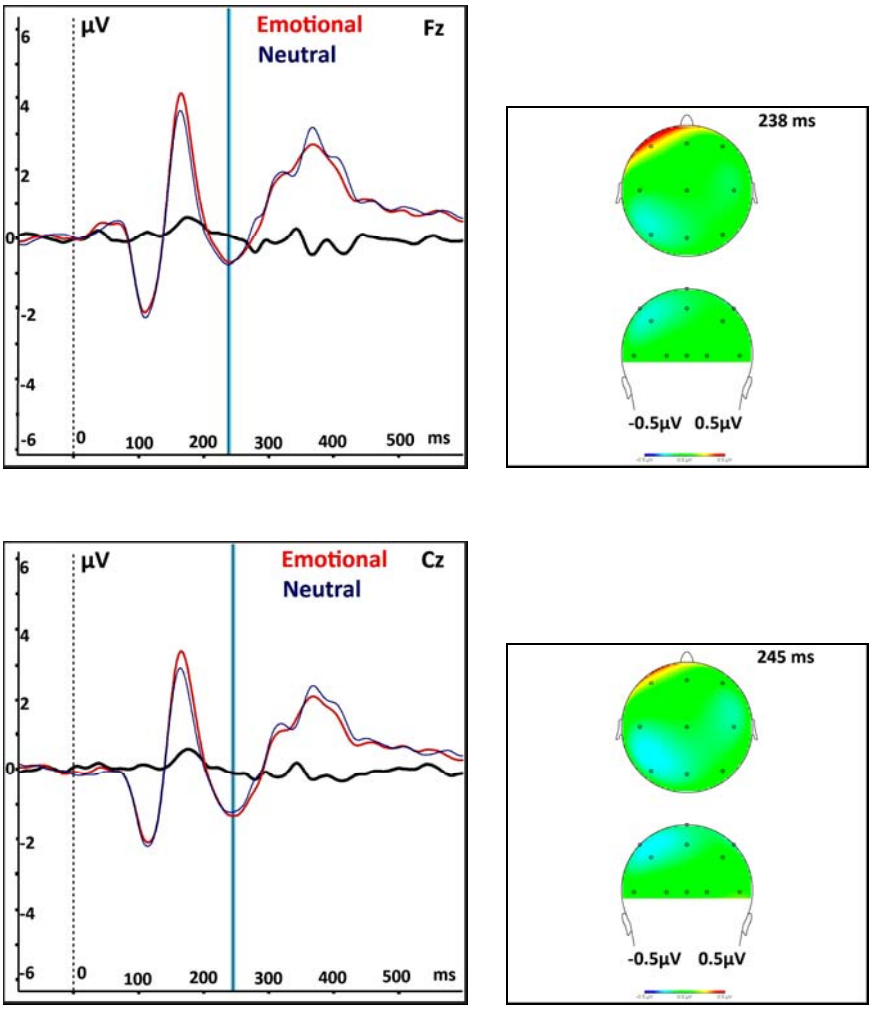


Figure 4. ***The depressed group.*** *Difference ERPs (black line) to emotional and neutral expressions at electrodes Fz and Cz. The red line represents emotional expression. The blue line represents neutral expression. The scalp potential maps illustrate the time window marked with the blue color.*

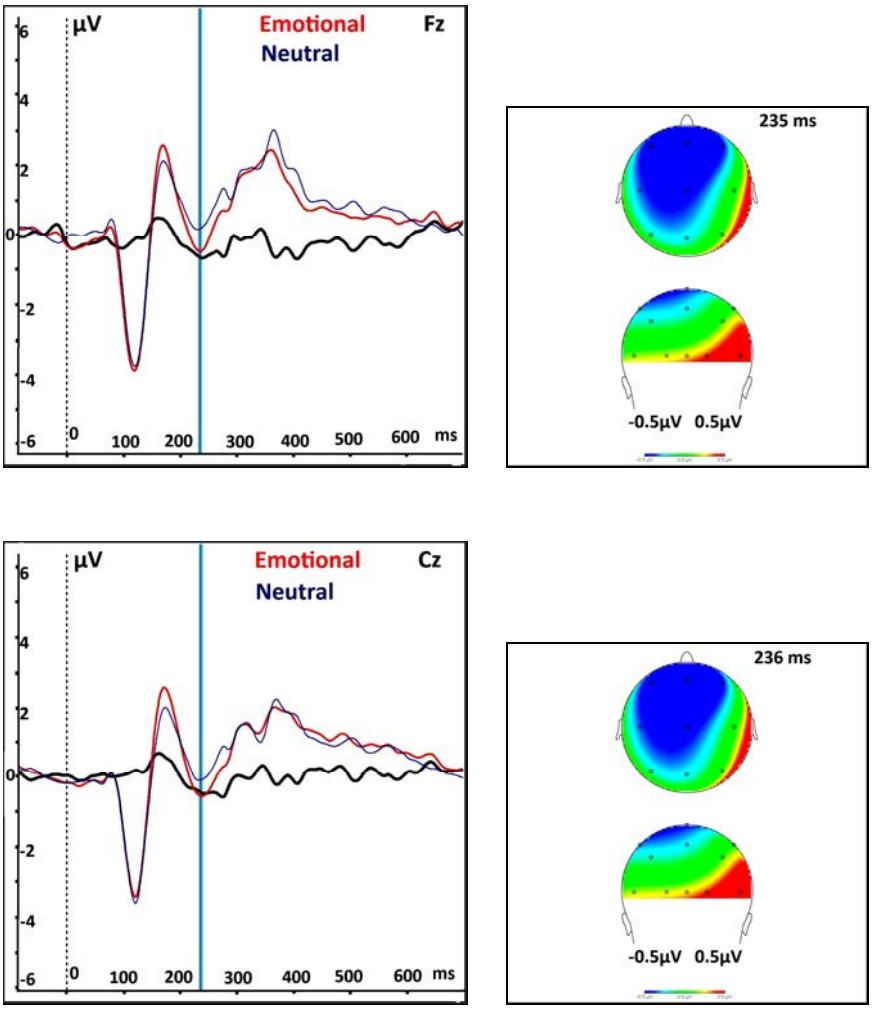


Figure. 5. ***The non-depressed group.*** *Difference ERPs (black line) to emotional and neutral expressions at electrodes Fz and Cz. The red line represents emotional expression. The blue line represents neutral expression. The scalp potential maps illustrate the time window marked with the blue color.*

No correlation between BDI Score Change, AAQ2 Score Change or Mood Score Change was detected for either expression for any electrode.

4. Discussion

The objective of the present study was to investigate the possible appearance of visual mismatch negativity reflected by components N170, P240 or N240 in an oddball design when emotional facial expressions were used as stimuli. Also, depressed and non-depressed individuals'

responses were compared and the effect of brief psychotherapy to the depressed individual's brain activity was investigated.

It seems that visual mismatch negativity is best reflected by the N170 component although the components P240 and N240 also reflect change detection. The results indicate that depressed compared to non-depressed individuals process emotional visual information differently. This finding is in line with previous research (e.g. Cavanagh & Geisler, 2006; Deldin et al. 2001; Deveney & Deldin, 2004). The results indicate that components P240 and N240 can detect and differentiate emotional and neutral facial expressions from one another in the non-depressed group but not different emotional expressions (fear and happiness). In the depressed group such change detection was not found. It seems that depressed individuals were not able to distinguish emotional and neutral facial expressions from one another as measured by ERPs.

In component P240, the difference between standard and deviant stimuli became significant when only electrode sites P8 and O2 were chosen for analysis. The discriminative power of P240 therefore seems to be lateralized to the posterior regions of the right hemisphere. The mean amplitude of difference ERPs between standard and deviant stimuli were significantly smaller in the depressed group as opposed to the control group. Also, the component N240 seems to have discriminative power. In group 3, two electrode sites reached significance, Fz and Cz. The electrode site F4 almost reached significance. This indicates that the discriminate power of N240 is localized in the parieto-frontal regions, with a possibly slight emphasis on the right. There are no signs of such asymmetry or change detection in the depressed group.

These results suggest that depressed individuals are not able to distinguish neutral and emotional facial expressions from one another. The findings are in accordance with previous research on hemispheric asymmetry in depression. It is widely accepted that depression is characterized by distinctive patterns of lateralized brain activity (e.g. Henriques and Davidson, 1997). This is evident despite the nature of the stimuli used (emotional or non-emotional). Depression has found to cause deficits in right-hemisphere functioning, for example, during spatial task performance (Henriques et al. 1997), dichotic pitch discrimination tasks (Bruder et al., 1995), and during the processing of emotional chimeric faces (Jaeger, Borod and Peselow, 1987). Heller (1993) states

that there seems to be a link between variations in emotional states and variations in the activity level of the right hemisphere.

Some studies can be found concerning, for example, hemispheric lateralization in depression and auditory processing measured by an EEG (e.g. Bruder et al., 1995; Sumich, Kumari, Heasman, Gordon & Brammer, 2006). Also, several studies exist concerning hemispheric asymmetry in depression not measured by an EEG (e.g. Moretti, Charlton & Taylor, 1996). However, studies like ours, where emotional facial stimuli were used and combined with EEG-recordings in depressed versus non-depressed individuals, are not many. One example is the study by Deldin et al. (2000). They found reduced right-posterior activity in depression. They used positive, negative, and neutral face and word stimuli in a recognition-memory task. The N200 was reduced in right-posterior brain regions in the group that was diagnosed with major depression. The depressed persons showed reduction in N200 to facial stimuli that were most mood-incongruent (happy faces). In the study by Zhao and Li (2006), standard stimuli evoked more positive P250 than the deviant stimuli used in their study. In our study, the findings were different: deviant stimuli evoked more positive P240 and more negative N240 than standard stimuli in the non-depressed group.

Shenal, Harrison and Demaree (2003) have suggested a preliminary neuropsychological model of depression in their literature review. The model is composed of four neuroanatomical divisions: left-frontal, right-frontal, left-posterior and right-posterior. Depressive symptomology may result from dysfunction within three of four divisions; Shenal et al. do not suggest any hypotheses for left-posterior dysfunction. According to the model, the right-posterior division may be specialized for emotional information processing. Deficits in this area may produce impaired emotional perception and inappropriate responsiveness. Shenal et al. continue that the individual would experience less arousal and a generalized reduction of brain activation as the right-posterior region becomes less activated. These symptoms, combined with impaired emotional perception and responsiveness, may lead to the diagnosis of depressive syndrome. A dysfunction in right-frontal division may also lead to a depressive disorder. Activation of the right hemisphere is associated with avoidance, withdrawal from aversive stimuli, and the experience of negative affects (e.g. Davidson, 1998). The model suggests, leaning on the findings of Davidson (1995),

that anterior regions of left and right hemisphere are especially key components for an affective regulatory system. Decreased right-frontal activation may therefore result in a lack of avoidance and withdrawal from aversive stimuli. This, according to the model, can be construed as depression and a type of learned helplessness. The findings of the present study can be viewed through and explained by the model proposed by Shenal et al. The two types of findings, lack of asymmetry in the right posterior and right parieto-frontal regions may reflect two different types of depressive symptomology in the depressed individual's group. It is possible that also the left-frontal dysfunction would have appeared with a larger group size. The model is in accordance with Beck's (1967) model of depression. Deficits in emotional information processing and in emotion regulation fit with Beck's cognitive triad: viewing oneself and the future in a negative way, and construing experiences in a negative way. In further research, it might be suggested to test Shenal et al.'s model by grouping depressed participants according to their recorded hemispheric asymmetries and then compare the nature of group's depressive symptoms.

The component N170 seems to be able to detect change between different expressions. It therefore seems to be the best of the three components to reflect visual mismatch negativity. The severity of depressive symptoms seems to be related to the processing of expressions. However, the interpretation of results is not straightforward. Although the depressed participants were randomly assigned into two groups, the groups differed by depressiveness and age. The participants in group 1 were more depressed and older than participants in group 2. This might be one reason why the results obtained from component N170 are contradictory. The two depressed groups (1 and 2) differed from each other significantly with regard to component N170. Groups 2 and 3 did not differ from each other. However, groups 3 and 1 almost differed significantly, p-value reaching 0.058.

This finding is especially puzzling. The reason why the difference between groups 1 and 3 did not reach significance may be due to small group sizes. The interaction between groups 1 and 3 might have reached a level of significance with larger groups. Also, the nature of depressive symptomology in groups 1 and 2 may differ or be generated in different brain regions. An explanation that needs to be considered as well is the possibility that the nature or severity of

depressive symptoms or age is not a factor causing differences between groups. Instead, the factor might be something that was not controlled in this experiment.

In group 1 there is only one significant difference. That is for the expression *fear* at electrode P7. The finding gives support for Beck's (1967) idea that depressed individuals are more prone to perceive information that is mood-congruent. Also, the finding suggests that group 1 might lack a "protective bias", an attentional bias towards happy faces. In groups 2 and 3, there were more electrode sites activated by happy rather than by fearful expressions. This may be an indication of the protective bias. Similarly, Cavanagh and Geisler (2006) found that depressed participants showed significantly reduced P3 amplitudes and P3 latencies for happy faces compared to controls. In the present study, according to the scalp topography maps, the electrical activation in group 1 is lateralized to the left whereas in group 2 it is lateralized to the right. In group 3, the activation seems to be lateralized more bilaterally, but emphasis is on the right. This finding again gives support for the hypothesis that right hemisphere dysfunction characterizes depression.

Similar results were obtained by Lynn and Salisbury (2008) with schizophrenia patients. Control subjects showed significant differences in N170 amplitude elicited by facial expressions. Schizophrenic patients did not exhibit such a modulation. The right hemisphere tended to show greater differentiation than the left. Valence specific theories (for review, see Wager, Phan, Liberzon and Taylor, 2003) suggest that the right hemisphere is specialized for the processing of negative emotions, while the left hemisphere is specialized for positive emotions. In groups 2 and 3, the fearful expressions were processed in the right hemisphere, at electrodes O2 and P8 in group 3 and at electrode P8 in group 2. Krolak-Salmon, Fischer, Vighetto and Mauguière (2001) obtained similar results with later latencies. In the time window of 550-750 ms after stimulus onset the processing of fear was predominantly performed in the right hemisphere in occipito-temporal areas.

In our study (in groups 2 and 3) the processing of happy faces was centered at occipito-temporal regions of the brain. The activation was localized bilaterally with an emphasis on the left. This result may be understood in the light of hemispheric specialization to negative and positive emotions as discussed above. Zhao and Li (2006) obtained differing results. Both expressions

(sad and happy) distributed over posterior areas and covered larger areas in the right rather than in the left hemisphere sites.

However, the valence specific theories must be applied with caution. Wager et al. (2003) have criticized them in their review and state that the emotional brain is much more complex than simple hemisphere-level predictions. The method used in the present study, EEG, does not provide precise information of the brain location generating the activity. Rather, it gives an approximation and is thus a coarse method for analyzing the location of the brain regions which generate the activity. Also, the small group sizes, differences between groups 1 and 2, and the fact that some of the subjects were medicated for their depression set limitations for the interpretation of results. In further research, the groups should be larger and more similar in at least by age and depressiveness. Preferably, the depressed and non-depressed groups would be matched by age and sex.

The treatment effect was investigated using correlations and indeed six significant correlations emerged for component P240. It seems that the change detection of expressions alters during therapy. However, when the correlation data was investigated more thoroughly, with for example scalp potential maps, no straightforward interpretation of the treatment effect could be made. The findings were too vague and uncertain. The results did not offer a sufficient base for creating a hypothesis. To avoid making faulty hypotheses, the findings of the correlation analysis will not be discussed any further.

Our results need to be confirmed by future research. In particular, the relationship between visual mismatch negativity and component N170, and the existence of a possible treatment effect need more clarification.

5. Conclusion

Despite the limitations of our study, it seems that there exists a real difference between depressed and non-depressed individual's brain mechanisms concerning the processing of emotional facial expressions. Depressed individuals are not able to distinguish emotional and neutral expressions from one another. All components, N170, P240 and N240, reflect the brain's change detection in

the non-depressed group. The discriminative power of P240 seems to be lateralized to the posterior regions of right hemisphere, whereas the discriminative power of N240 is localized in the parieto-frontal regions. Such asymmetry of activation was not found in the depressed group. Component N170 seems to be the best to reflect visual mismatch negativity. It was able to make a differentiation between different emotional expressions. These are interesting findings which are in line with the results of other studies. Prior studies have shown that differences in information processing exist between depressed and non-depressed individuals.

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