INTERVENTION EFFECTS TO PREATTENTIVE PROCESSING OF EMOTIONAL INFORMATION IN DEPRESSED INDIVIDUALS: A STUDY OF ERPs AND REACTION TIMES

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CONTRIBUTIONS

Professor Raimo Lappalainen was responsible for designing, gathering and analysing the clinical data, as well as organizing the therapy treatment including training the therapists and supervising them during the treatment. Docent Piia Astikainen was responsible of the brain research in the project: she planned the EEG-experiments and supervised the analysis and reporting of the data. The following authors have used or are partly using the same data in their Master`s theses:

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Their studies are executed from different viewpoints.

ABSTRACT

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In the present study, the positive effects of a brief intervention, based on acceptance and commitment therapy (ACT), were investigated in 21 depressed participants that benefited from the treatment. The 21 subjects that benefited from the intervention were selected from 42 depressed participants that underwent the intervention on the grounds of their BDI scores, so the scrutiny was limited to subjects with good treatment-response. The stability of the treatment effect was examined seven months after the intervention. Similar data was also collected from 23 non-depressed participants and compared with the baseline measurement of the depressed group. The treatment effects were examined in the context of reaction times and event-related potentials (EPRs). The groups underwent an experiment utilizing the affective priming paradigm. Affective priming refers to an automatic phenomenon in which the valence of a previously presented prime stimulus effects the processing of the following target stimuli. If the valences of the prime and the target are congruent (e.g. positive-positive), the reaction times are expected to be shorter and if the valences of the prime and target are incongruent (e.g. positive-negative) the reaction times are expected to be longer. In the experiment, the participants evaluated the valences (positive, negative, neutral) of happy, sad or neutral facial expressions that were preceded by affectively pleasant, unpleasant or neutral pictures. 160 affectively congruent, 160 incongruent and 160 neutral prime-target pairs were presented in the experiment. The EEG recording was conducted with 14 electrodes placed on the scalp of the participants. The extensively studied ERP component N170 was under scrunity since it is shown to be associated with the processing of faces. As for behavioral results, both groups demonstrated only positive priming effects in their reaction times, and no differences between the groups or treatment effects were found. As for electrophysiological results, the localisation of the priming effect differed between the groups at baseline measurement: as the non-depressed group demonstrated both positive and negative effects at the right-hemispheric parietal electrode P8, the depressed group demonstrated the same but also a positive priming effect at the left-hemispheric parietal electrode P7. This indicates for a laterality difference between the groups, when considering positive valence. After the treatment, the positive priming effect did not appear in the left-hemispheric parietal electrode P7 in the depressed group. This treatment effect remained in the follow-up seven months afterwards. The results indicate that ERPs are a more sensitive meter to detect changes in the automatic priming effect than reaction times. The results are in line with previous literature that indicates for differences in the preattentive processing of emotional information in depressed individuals related to non-depressed individuals. The results also show that the positive effects of a brief therapeutic intervention can alter the function of the brain, and that these changes can be relatively stable.

Keywords: visual affective priming, facial expressions, event-related potentials (ERPs), N170, depression

TIIVISTELMÄ

Intervention vaikutukset automaattiseen emotionaalisen informaation käsittelyyn masentuneilla: herätevaste- ja reaktioaikatutkimus

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Tutkimuksessa tutkittiin hyväksymis- ja omistautumisterapiaan pohjautuvan lyhyen intervention positiivisia vaikutuksia 21 masentuneella jotka hyötyivät hoidosta. 21 hoidosta hyötynyttä koehenkilöä valittiin BDI -pisteidensä perusteella 42 masentuneesta joille interventio toteutettiin, joten tutkimus rajoittui koehenkilöihin joilla oli hyvä hoitovaste. Hoitovaikutuksen pysyvyyttä tutkittiin seitsemän kuukautta intervention jälkeen. Vastaava data kerättiin myös 23 ei-masentuneelta ja sitä verrattiin masentuneiden alkumittaukseen. Hoitovaikutuksia tutkittiin herätevasteiden (ERPs) ja reaktioaikojen kontekstissa. Ryhmille toteutettiin koe, jossa käytettiin affektiivista priming –paradigmaa. Affektiivinen priming viittaa automaattiseen ilmiöön, jossa aiemmin esitetyn alustusärsykkeen valenssi vaikuttaa sitä seuraavan kohdeärsykkeen prosessointiin. Jos alustus- ja kohdeärsykkeen valenssit ovat kongruentteja (esim. positiivinen-positiivinen), reaktioaikojen odotetaan olevan lyhyempiä ja jos alustus- ja kohdeärsykkeen valenssit ovat inkongruentteja (esim. positiivinen-negatiivinen) reaktioaikojen odotetaan olevan pidempiä. Kokeessa koehenkilöt arvioivat iloisten, surullisten ja neutraalien kasvokuvien valensseja (positiivinen, negatiivinen, neutraali), joita edelsivät emotionaalisesti miellyttävät, epämiellyttävät tai neutraalit kuvat. Kokeessa esitettiin 160 emotionaalisesti kongruenttia, 160 inkongruenttia ja 160 neutraalia alustusärsyke-kohdeärsyke-paria. EEG-mittaus toteutettiin 14 elektrodilla, jotka asetettiin koehenkilöiden kallon pinnalle. Laajasti tutkittu ERP -komponentti N170 oli tarkastelun kohteena, sillä sen on osoitettu olevan yhteydessä kasvojen prosessointiin. Behavioraalisten tulosten osalta kummallakin ryhmällä näkyi vain positiivinen priming –ilmiö reaktioajoissaan, eikä ryhmien välisiä eroja tai hoitovaikutuksia havaittu. Elektrofysiologisten tulosten osalta priming -ilmiön lokalisaatio erosi ryhmien välillä alkumittauksessa: kun ei-masentuneiden ryhmällä näkyivät sekä positiivinen että negatiivinen priming –ilmiö oikean hemisfäärin parietaalisella elektrodilla P8, näkyi masentuneiden ryhmällä vastaavan lisäksi positiivinen priming –ilmiö vasemman hemisfäärin parietaalisella elektrodilla P7. Tämä indikoi lateralisaatioeroa positiivisen valenssin osalta ryhmien välillä. Positiivinen priming -ilmiö ei näkynyt vasemman hemisfäärin parietaalisella elektrodilla P7 hoidon jälkeen masentuneiden ryhmässä. Tämä hoitovaikutus säilyi seurannassa seitsemän kuukautta myöhemmin. Nämä tulokset osoittavat, että herätevasteet ovat reaktioaikoja herkempi mittari havaitsemaan muutokset automaattisessa priming –ilmiössä. Tulokset ovat linjassa tutkimusta edeltävän kirjallisuuden kanssa, joka viittaa eroihin masentuneiden emotionaalisen informaation automaattisessa prosessoinnissa suhteessa ei-masentuneihin. Tulokset osoittavat myös, että lyhyen terapeuttisen intervention positiiviset vaikutukset voivat muuttaa aivojen toimintaa, ja että nämä muutokset voivat olla suhteellisen pysyviä.

Avainsanat: visuaalinen affektiivinen priming, kasvojenilmeet, herätevasteet (ERPs), N170, masennus

CONTENTS

1. INTRODUCTION

The aim of the present study was to investigate the effects of a brief acceptance and commitment therapy based intervention to both the reaction times and the event-related brain potentials of depressed individuals that benefited from the intervention, as measured in BDI scores. The participants took part in a task of visual affective priming before and after the intervention. The reaction times and the event-related potentials of the participants were examined again seven months after the treatment in a follow-up to investigate the stability of the treatment effect. Similar data was also collected from non-depressed controls and compared to the pre-treatment measurement of the benefited depressed group.

1.1 Affective priming

1.1.1 The paradigm

Priming refers to an implicit phenomenon where previously presented stimuli bias the processing of the following stimuli. The emotional priming paradigm, developed by Fazio, Sanbonmatsu, Powell & Kardes (1986), is a widely used approach that enables the evaluation of automatic information processing. In emotional priming, the valence of the prime stimuli, preceding the target stimuli, interacts with the processing of the latter target stimuli. Shorter response latencies are expected when the valence of the prime and the target stimuli is congruent (positive-positive, negative-negative), whereas with incongruent valence of the prime and the target, longer response latencies are expected (positive-negative, negative-positive). Fazio (2001) states that this kind of attitude activation has functional value as it influences the processes of attention, categorization, judgement, decision making and behaviour.

At least two mechanisms have been proposed to be involved in affective priming. Fazio et al. (1986) suggested that the activation in memory would spread from the primed object to the associated evaluation. Another mechanism proposed is so-called response competition, where the prime creates a response tendency – in other words prepares the individual to answer in a certain way (Wentura, 1999). According to Wentura, the attitude activations of the prime and target may either enhance each other (in case of congruency), which results as a faster response, or conflict (in case of incongruency), which results in inhibition of the prime. There is evidence for both of the

mechanisms, partly depending on methodological differences in different studies, especially variability in applied tasks (Fazio, 2001).

The effects of affective priming have been studied using different types of stimuli as primes and targets, including words and pictures, and multiple tasks (Fazio, 2001). The phenomenon appears to be facilitated by physical similarity of the target and the prime stimuli, but it occurs even if the target stimulus and the prime stimulus vary in their perceptual or semantical features (Avero & Calvo, 2006). It also seems that the arousal dimension of the presented stimuli has an effect on affective priming (Hinojosa, Carretie, Me´ndez-Be´rtolo, Mı´guez & Pozo, 2009). Overall, the effect is stronger for stronger prime stimuli (Fazio et al., 1986).

Affective priming seems to be very automatic, since it appears extremely fast and is short-lived. There is evidence that affective priming shows effects under very brief exposures of the prime stimulus (Murphy & Zajonc, 1993). The time course of the effect is also well known because of experiments that have manipulated the stimulus onset asynchrony (SOA), the interval between the presentation of the prime stimulus and the presentation of the target stimulus. The affective priming effect seems to appear very early, even when the prime and the target stimuli are presented at the same time (Hermans, De Houwer & Eelen, 2001). The effect seems to have its most powerful appearance typically around the SOA of 150 to 200 ms and decrease in intensity already in SOA of 300 ms, diminishing in SOA of 1000 ms (Avero & Calvo, 2006; Hermans et al., 2001).

1.1.2 The electrophysiology of affective priming

Affective priming has proven to be a robust, general phenomenon when studied by behavioral means, mostly reaction times. The research for the neural mechanisms of the effect has been relatively moderate in numbers, but increasing in the recent years. Particularly, electrophysiological methods are an effective way of studying automatic cognitive processes due to their excellent temporal accuracy. Electroencephalography, or EEG, is a technique in which the electrical activity of human brain is recorded with electrodes that are attached to the scalp (Kolb & Wishaw, 2009). Event-related potentials (ERPs) are time-locked responses to discrete sensory stimuli, appearing as brief changes in slow wave EEG signal (Kolb & Wishaw, 2009).

The ERP results concerning the priming effect seem to differ regarding the stimulus type used. Semantic or linguistic priming appears to be associated with a negative component around 400 ms

after target onset. This N400 component increases in amplitude when target words are unrelated, compared to related target words (e.g. Brown, Hagoort & Chwilla, 2000). Zhang, Lawson, Guo & Jiang (2006) also found the increased N400 effect of incongruent stimulus pairs in a study of affective priming, using evaluative categorization task with words and pictures as primes, but only words as targets. They also detected a larger N200 component for incongruent trials than to neutral trials when words were used as primes. In a study using facial expressions as stimuli in a priming experiment, attenuated frontal ERP amplitudes were obtained for 100-200 ms after stimulus onset for congruent stimulus pairs, and augmentation of the late positive potential, LPP, at 500-600 ms after stimulus onset, was found for incongruent stimulus pairs (Werheid, Alpay, Jentzsch & Sommer, 2005). The authors concluded that the earlier priming effect reflects the emotion-specific facial configuration and the latter an enhanced arousal in response to a changing expression.

Pictures of facial expressions, indicating different emotions, are overall an optimal way of studying the affective priming effect (Aguando, Garcia-Gutierrez, Castañeda & Saugar, 2007; Murphy and Zajonc, 1993). The perception of facial expressions is suggested to be a product of evolution, serving the development and regulation of interpersonal relationships (Ekman, 1992). It indeed seems that there is also a specialized neural system for face perception in human brains, consisting of multiple bilateral regions (for a review, see Haxby, Hoffmann & Gobbini, 2000). An electrophysiological indication of the processing of facial expressions is a face-specific ERP component, N170.

1.2. The ERP component N170

N170 is a bilateral negative component of ERP (Bentin, Allison, Puce, Perez & McCarthy, 1996). It appears between 130 and 200 ms and peaks mostly in 170 ms after stimulus onset (for a review, see Rossion & Jacques, 2007). N170 is localised in the occipito-temporal area of the brain and appears stronger in the right hemisphere (Bentin et al., 1996). Overall, N170 corresponds to a time-locked increase in the EEG amplitude, and it also seems to have a counterpart of equivalent dipoles, a vertex positive potential, or VPP (Rossion & Jacques, 2007).

Pictures of faces elicit significantly larger N170 responses than other stimuli of multiple categories, and this seems to be the case even when the faces are inverted or their inner components are dislocated (Bentin et al., 1996). It seems, however, that N170 appears stronger when the facial stimuli are optimal for recognition: front and profile views of faces, both internal and external

features present, elicit the component (Eimer, 2000). N170 seems to be unaffected by the familiarity of faces, which suggests the component to be linked with the automatic structural coding of faces, not identification of them (Bentin & Deouell, 2000). Jemel et al. (2003) applied a gradual presentation procedure of facial pictures, and as a result the N170 gradually emerged from the EEG activity of the participants as the face configuration became clearer. The authors suggested that the N170 reflects a perceptual integration process, where low-order visual information is integrated into a higher-order visual representation of a face.

There is contradicting evidence of the modulation of N170 in relation to the emotional expression of faces. Studies have shown that facial expressions do not have an effect on N170, even if the task is to discriminate between neutral and emotional stimuli or even if six basic emotions (anger, disgust, fear, happiness, sadness, surprise) are employed (e.g. Eimer, Holmes & McGlone, 2003). Nevertheless, some recent results conflict with this. Blau, Maurer, Tottenham and McCandliss (2007) found that fearful expressions enhanced N170 when compared to neutral faces in a non-explicit task condition. Krombholz, Schaefer and Boucsein (2007) used a priming procedure, requiring the attention of the participant to the emotional expressions of the faces, and found that angry faces elicited larger N170 amplitudes than happy faces.

1.3. The processing of emotional information in depression

Depression is a mood disorder characterized by several symptoms, including, for example, lack of positive affect, feelings of guilt, constant self-criticism, exhaustion, problems with concentration and changes in psychomotoric function, sleeping and appetite (Isometsä, 2007). It is estimated that about 5 % of the population is affected by the disorder in Finland (Isometsä, 2007), which makes it a relevant phenomenon for research. Several studies have indicated biases in the processing of emotional information in depressed individuals. The cognitive model of depression emphasizes the automatic cognitive bias of the disorder that appears, for example, as selective attention to negative aspects of perception (Beck, 2008). The bias is assumed to be based on dysfunctional attitudes: constantly activating negative views about the world, self and the future.

The cognitive bias in depression seems to appear also in the processing of emotional information. In a study of pupillary and reaction time measures (Siegle, Gronholm, Ingram & Matt, 2001) depressed participants were particularly slow to respond to a valence identification task and

displayed greater sustained emotional processing. Difficulties in processing emotional information seem to appear, among other things, as general deficits in the recognition of facial expressions (Mikhailova, Vladimirova, Iznak, Tsusulkovskaya & Sushko, 1996). Since the perception of faces provides significant information about social communication (e.g. Ekman, 1992; Haxby et al., 2000) the inability to process facial expressions accurately in depressed individuals is suggested to contribute to interpersonal difficulties related to the disorder (e.g. Surguladze et al., 2004).

1.3.1 Behavioral perspective

Some of the evidence concerning the precise nature of abnormal processing of facial expressions in depressed individuals are inconsistent, but the overall general lines are starting to clarify (for a review, see Leppänen, 2006). The different results might be attributed to methodological variability in the studies, including, for example, differences in the populations, stimuli or categories of expressions used (Surguladze et al., 2004).

There is evidence for a mood-congruency hypothesis, where negative mood elicits the perception of negative stimuli: a bias towards negative affect in depression has been detected (e.g. Dannlowski, 2006) . For example, in a study of Hale (1998) depressed patients judged significantly more sadness in schematic facial expressions than healthy control subjects. In addition, the severity and persistence of the condition of the depressed patients correlated significantly with the disposition to judge the expressions negatively. The perception of negative emotions in ambiguous faces is also found to be associated with relapse in depressed individuals (Bouhyus, Geerts & Gordijn, 1999).

Consistent with the bias towards negative affect, it also seems that depressed individuals have a bias away from positive stimuli. Surguladze et al. (2004) found an impaired recognition accuracy (when stimuli were presented for 100 ms), and a reduced response bias away from labeling happy facial expressions as happy (when the stimuli were presented for 2000 ms), in depressed patients compared to healthy controls. In addition, it seems that depressed individuals lack a positivity bias that is found in healthy controls (Joormann and Gotlib, 2007). In a study of Yoon, Joormann & Gotlib (2009) the subjects had to judge which of two faces presented expressed stronger emotion. Depressed individuals were less likely to choose subtle happy expressions over neutral expressions as more intense. The authors concluded this to reflect difficulties in processing positive affect in depressed individuals. Furthermore, depression seems to bias the recognition of neutral expressions

as well. Leppänen, Milders, Bell, Terriere & Hietanen (2004) found that depressed individuals recognized neutral expressions more inaccurately and slowly than healthy controls. In addition, depressed individuals recognized sad expressions faster than neutrals, when controls recognized neutral expressions faster than sad ones, which reflects the negativity bias in depressives. The authors suggested that depression might enhance the interpretation of emotionally neutral cues as emotionally meaningful.

1.3.2 Neurophysiological and –anatomical perspective

Studies have provided relatively lots of behavioral evidence for biases in the processing of emotional stimuli in depressed individuals. Another line of research, concerning the neuropsychological and -anatomical basis of this phenomenon, has also arisen in recent years. As to ERP studies of the biases, a reduction in the right-posterior N200 component (the face-specific N170) has been found in depressed individuals, especially to positive facial expressions (Deldin, Keller, Gergen & Miller, 2000). In addition, it seems that depressives have generally smaller amplitudes to positive than to neutral stimuli (Shestyuk, Deldin, Brand & Deveney, 2005). At the same time, non-depressed controls seem to have smaller amplitudes to negative facial stimuli than to positive stimuli, which supports the evidence for the controls` bias away from negative that depressives seem to lack (Deveney & Deldin, 2004). A frequently investigated ERP component in depressed individuals is also the P300, which is suggested to reflect the allocation of attentional resources when stimuli of high informational salience are perceived (Ilardi, Atchley, Kwasny & Garratt, 2007). Ilardi et al. (2007) found that negative stimuli elicited larger P300 amplitudes in depressives when compared to non-depressed controls, or, interestingly, remitted depressives. In addition, Deldin, Keller, Gergen and Miller (2001) found that P300 was enhanced in healthy controls to positive stimuli in an explicit recognition task, including faces as stimuli. Depressed participants lacked this bias for positive information.

There are also studies concerning the brain structures involved in the biases of facial processing in depressed individuals. Fu et al. (2008) found excessive amygdala activity to sad facial expression processing in depressed individuals in a fMRI study. This exaggerated amygdala-hippocampal activity reduced to the level of healthy control subjects in these depressed individuals after an episode of cognitive behavioral therapy. The amygdala is suggested to be a part of an extended system of the neural system for face perception, processing particularly emotion (Haxby et al.,

2000). In another fMRI study of Suslow et al. (2010) a priming task with masked facial expressions as stimuli was applied. In the study, stronger right amygdala activation to sad facial expressions than happy facial expressions was found in depressives, whereas the right amygdala activation of healthy controls showed stronger responses to happy than sad facial expressions. The outcome is consistent with the results indicating a negativity bias in depressives and supports as well the assumption of a positivity bias in healthy controls, and moreover suggests these biases to function already in an automatic level. In a third fMRI study, Surguladze et al. (2005) applied a task involving implicit evaluation of emotion. Significant decreases in the neural response of several areas to happy expressions and significant increases of neural response of several areas to sad expressions were found in depressed individuals, compared with healthy controls. Interestingly, significant correlations between the severity of depression and neural responses to happy expressions were also detected. The authors conclude that, in depressed individuals, there are differential neural patterns of neural regions important for the response to visual presentations of happy and sad facial expressions, compared to controls.

Studies propose several neural dysfunctions to contribute to depression in general (for a review, see Beck, 2008). Neuroimaging and neuropathological studies have found, for example, structural and functional abnormalities in orbital and medial prefrontal cortex, amygdala and related parts of the striatum and thalamus in depression (for a review, see Drevets, 2001). The dysfunctional regions in prefrontal and striatal systems normally modulate the limbic and brainstem structures, so impairment in the inhibition of responses to emotional stimuli was suggested by the author to correspond to mood disorders in general. Increased amygdala activity in the limbic system together with the decreased prefrontal control has been suggested to relate to the sustained emotional reactivity in depression (DeRubeis, Siegle & Hollon, 2008). In addition, Heller & Nitschke (1998) suggest that activation of the right posterior hemisphere, modulating the autonomical effects of emotional arousal, would be decreased in depression. Several studies have supported the model (e.g. Deldin, Keller, Gergen & Miller, 2000), but some results remain inconsistent. There are no findings of enhanced left-hemispheric activation in depression, but in some forms of anxiety this kind of phenomenon has been found (Heller & Nitschke, 1998).

1.4. The effects of remission and treatment

1.4.1 The effects of remission

Few studies have also integrated the aspect of remission from depression to the research of the biases in the facial processing related to the disorder – namely, the question of the biases being trait- or state-like. The perception and judgement of negative emotions in facial expressions seem to be associated with the persistence of the symptoms (Hale, 1998) and relapse (Bouhuys et al., 1999) in depressives. Deficits in the processing of neutral faces seem also to remain in formerly depressed individuals (Leppänen et al., 2004). There is also evidence that both currently and formerly depressed patients selectively attend to sad faces, as opposed to the healthy controls that had instead a bias towards happy faces (Joormann & Gotlib, 2007). A difficulty in processing subtle happy faces in remitted depressives has also been demonstrated after negative mood instruction (LeMoult, Joormann, Sherdell, Wright & Gotlib, 2009). Therefore, these studies indicate that the cognitive biases of the depressed individuals are relatively lasting or at least difficult to change. On the other hand, in a study of Mikhailova, Vladimirova, Iznak, Tsusulkovskaya & Sushko (1996) depressed individuals recognized schematic facial expressions more accurately after remission.

Some studies have utilised the affective priming paradigm. Goeleven, Raedt, Baert & Koster (2006) applied a priming task with facial stimuli and found that the depressed patients showed a specific failure to inhibit negative information, whereas formerly depressed individuals demonstrated, surprisingly, impaired inhibition of both negative and positive information. Other studies have examined the automatic processing of facial expressions with implicit tasks of affective priming. Koschack, Höschel and Irle (2003) found an emotional priming effect in healthy and partially remitted depressives, but not in subjects experiencing acute major depressive disorder. In addition, subjects with partially remitted depression demonstrated a negative response bias: they judged neutral faces as negative significantly more often than healthy controls. In an analogous study, Dannlowski et al. (2006a) also found a negative evaluative shift especially with neutral faces as primes before an episode of therapy in depressives. This evaluative shift was not detected after the episode of therapy. In another study of Dannlowski et al. (2006b) the results were repeated with words as stimuli: depressives had an interference effect of negative primes before the therapy but not after the therapy, as healthy controls demonstrated a reversed affective priming effect. According to these studies, it seems that post-treatment changes in the negative response bias of the depressives concerning the evaluation of facial expressions can be detected at behavioral level when priming tasks are used.

1.4.2 Treatments, neurobiology and depression

Overall, the effectiveness of therapies for depression has often been examined with self-evaluative inventories. In recent years the treatment-related changes in the function of the brain have been observed with neuroimaging studies, also concerning depression (for a review, see Roffmann, Marci, Glick, Dougherty & Rauch, 2005). The effects of cognitive therapy have been suggested to relate to the increase in the controlled inhibitory processing of the prefrontal cortex, leading to reduction in the increased activation of the limbic system, as perceived in depression (DeRubeis, Siegle & Hollon, 2008). There is even proof of treatment effects related to the dysfunctional facial processing in depression: in a fMRI study of Fu et al. (2009) the effects of cognitive-behavioral therapy (CBT) on neural responses to sad faces in depressed individuals were examined. The treatment effects were evident in the areas involved in affective facial processing, and anterior cingulate activity appeared to predict the treatment response to CBT. Overall, in addition to the frequently-used self-evaluations, these neurophysiological and –anatomical studies, examining the effects of therapies, may provide useful ways of measuring the effects of treatment, as well as means for predicting the onset and reoccurence of depression.

Overall, cognitive therapy, based on the cognitive model of depression (Beck, 2008) has been proven to be effective, with more enduring results than antidepressant medication provides alone (DeRubeis, Siegle & Hollon, 2008). The present study investigated the effects of acceptance and commitment therapy or ACT (for a meta-analysis, see Hayes, Luoma, Bond, Masuda & Lillis, 2006), that is part of larger modern development of cognitive-behavioral therapy. In the approach, larger psychological flexibility is aimed at through mindfullness and acceptance processes, and commitment and behavioral change processes. There is a growing base of promising empirical support for the model and its components, although some limitations exist: many studies have relied on self-evaluative measures. In a preliminary controlled effectiveness trial, symptom improvement was shown concerning depression, when student therapists with very limited training used ACT (Lappalainen et al., 2007). The symptom changes were significant also from pre-treatment to follow-up.

1.5. The objectives of the present study

In the present study, the affective priming paradigm was utilized to examine the positive effects of a short-term acceptance and commitment -based therapeutic intervention of four sessions, carried out by nonprofessional student therapists with limited training, to the event-related potentials of depressed individuals. We examined reaction times and amplitudes of N170/VPP components of the depressives that benefited from the intervention. The depressed participants took part in a task of affective priming, with pictures of facial expressions as target stimuli, before and after the treatment, and again seven months after the treatment. Data from non-depressed control subjects, performing a similar task, was also collected.

In our study, several hypotheses were tested. Firstly, we examined if the affective priming effect, investigated in the reaction times and in the event-related potentials, differed between the depressed and the non-depressed group before the intervention. Secondly, it was premised that the affective priming effect of the depressed group would alter to resemble the affective priming effect perceived in the non-depressed group after the therapeutical intervention. This change would appear in both the reaction times and the event-related potentials. Thirdly, it was hypothesized that these changes would remain in the follow-up seven months after the intervention.

2. METHODS

2.1 Experimental design

The depressed individuals were randomized in two groups, of which the experimental group received the intervention first, as the wait-list control group waited with no intervention. Afterwards, the wait-list control group was treated as well, as we were interested in the treatment effects in the group as well. In the present study, the second measurement was applied as the baseline for the wait-list control group. The groups were united and the subjects that did not benefit from the therapy were excluded from the analysis. The subjects whose BDI points rose at least 40 % from pre-treatment to post-treatment were considered to have benefited. This separation was conducted to limit the scrunity to the positive effects of the therapy. Another master`s thesis, concerning only the pre- and post-treatment measurements of the whole depressed group, is in the making. The data collection was carried out in two consecutive years so that approximately a half of the participants participated to the study in 2008 and the other half in 2009. Figure 1 illustrates the experimental design.

Repeated measures design was utilised in scrunities due to serially presented stimuli and the investigation of effects presumed to change in time. The measurement of the non-depressed control group and the pre-treatment measurement of the benefited depressed group were compared. The within-subject variables included congruency (congruent, incongruent), valence (positive, negative) and electrode (P7, P8, O1, O2, F3, F4). In addition, the effects of the intervention were investigated in the benefited depressed group. The within-subject variables included measurement (pre-treatment, post-treatment, follow-up), congruency, valence and electrode.

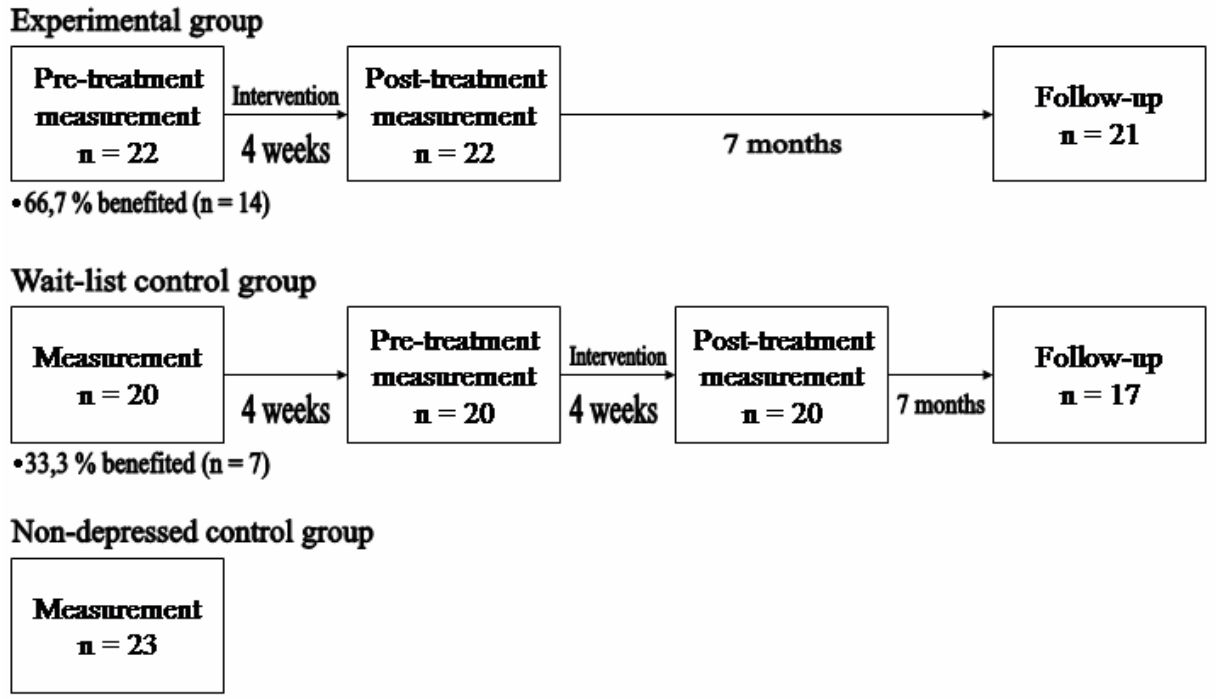

Figure 1. ***The experimental design***

2.2 Subjects

The depressed participants were recruited with a newspaper advertisement. In the advertisement, a brief therapy and a measurement of EEG was offered for volunteers with depressive symptoms. The participants were briefly screened in the phone. Suitable participants were scheduled for a visit to undergo a diagnostic interview and filling of self-evaluative inventories. Exclusion criteria included neurological disorders; history or current substance abuse; electroconvulsive therapy; suicidal behaviour; or psychosis level psychiatric disorders. All of the entered participants were treated for ethical reasons even if they were excluded from the study. Beck Depression Inventory, or BDI was used to evaluate the level of depression in the study. Only the participants that fulfilled the criterion of a BDI score of over 10 were included in the EEG measurement, since it was premised that changes in milder depressive states would not appear in the event-related brain potentials of the subjects that explicitly. The participants with a BDI score lower than 10 are not included in the descriptive statistics here.

When considering all of the fourty-two depressed participants (35 women, 7 men), the mean of pre-treatment BDI scores were 25,4 (SD=8.76), the mean of the post-treatment BDI scores 14.95 (SD=10.41) and mean of the follow-up BDI scores 10,50 (SD=9.45). Four of the subjects dropped out from the follow-up. The depressed participants were 28 to 66 years old, with a mean of 47.6

years (SD=10.03). Before the study, most of the depressed subjects had a diagnosis of only depression (n=20), some had no diagnosis of any kind (n = 18), a few had diagnosis of depression with some other diagnosis (n=3) and one had a diagnosis of some other psychiatric disorder than depression. 74 % (n=31) were not medicated during the study, the remaining 26 % (n=11) were.

Twenty-one depressed participants (19 women, 2 men) that were considered to have benefited from the intervention were included in the analysis. The BDI change from pre-treatment to post-treatment was remarkable in these participants, with an increase of 40 % or more in the BDI scores. The mean pre-treatment BDI score of this group was 23.48 (SD=8.59), as the post-treatment mean BDI score of the group was 6.9 (SD=4.19) and the follow-up mean BDI score 6,42 (SD=6.14). The age of the benefited participants varied from 29 to 58 years, with a mean of 46,76 years. 66,7 % (n=14) of them were from the experimental group that received the intervention first, and 33,3 % (n=7) from the wait-list control group that received the intervention later. Ten of them had no diagnosis of any kind, ten had a diagnosis of only depression and one had a diagnosis of depression and some other psychiatric diagnosis. 33,3 % (n=7) of these participants were medicated during the study, as the remaining 66,7 % (n=14) were not. Two of these participants that benefited from the intervention dropped out before the follow-up seven months after the therapy, so they are not included in the follow-up analysis.

The remaining twenty-one participants (16 women, 5 men) were considered not to have benefited enough or not to have benefited on the whole from the intervention. If the BDI scores of these subjects increased after the intervention, the rising did not reach 40 % or more. The mean of the pre-treatment BDI scores for this group was 27.24 (SD=8.41), the mean of post-treatment BDI scores 23 (SD=8.28) and the mean of the follow-up BDI scores 14.58 (SD=10.53). Two of these participants also dropped out so they are not included in the follow-up descriptive statistics or t-tests. The not-benefited group was aged 28 to 66 years (mean = 48.43). 38.1 % (n = 8) of them were from the experimental group, receiving the intervention first, and 61.9 % (n = 13) from the wait-list control group, receiving the intervention later. Eight of them had no diagnosis of any kind, ten had only the diagnosis for depression, one had no diagnosis of depression but some other psychiatric diagnosis and two had both diagnosis for depression and for some other psychiatric disorder. 81 % (n=17) of this non-benefited group was not medicated during the study, as the remaining 19 % (n=4) was.

Independent samples t-test indicated that these two groups of depressed patients, the one with subjects that were considered to have benefited from the intervention and the other with subjects that were considered not to have benefited, did not differ in their pre-treatment BDI scores significantly: $t(40)=1.409$, $p=0.167$. As to the post-treatment BDI scores, the group that was considered to have benefited had significantly lower BDI scores than the group that was considered not to have benefited, $t(40)=7.949$, $p=0.001$. This was the case also for the follow-up BDI scores of the groups, $t(36)=2.918$, $p=0.001$. The BDI scores indicate, in other words, that the groups were both equally depressed in pre-treatment measurement, but the group that was considered to have benefited had significantly more improvement when considering BDI scores, compared to the group that was considered not to have benefited. The third test also indicates that this difference in the BDI scores remained in the follow-up.

Twenty-three non-depressed control subjects (20 women, 3 men) were recruited through advertisements or acquaintances of the students in the project. They reported no diagnosed psychiatric or neurological disorders. They filled the same questionnaires than the depressed participants, and their BDI scores varied from 0 to 6, with a mean of 2,13 (SD=1.91). One-way ANOVA indicated that they had significantly lower BDI scores than the depressed subjects, $F(1)=156.657$, $p=0.001$. The healthy control subjects were aged 24 to 58 years, with a mean of 46.83 years. According to one-way ANOVA, their age did not differ significantly from the age of the benefited depressed group that was under scrunity in this study, $F(43)=0.001$, $p=0.980$.

All of the participants gave informed consent, were right-handed and had a normal or a corrected-to-normal vision. The experiment was undertaken in accordance with the Declaration of Helsinki. The ethical committee in the University of Jyväskylä approved the research protocol.

2.3 Intervention

We examined the effects of a brief intervention that was based on acceptance and commitment therapy. Every subject underwent four 60-minute sessions of treatment. The subjects were given different exercises (e.g. mental practices) and homework according to their individual situation. These treatment sessions were executed by Master`s students in the Department of Psychology, University of Jyväskylä. The student therapists were trained by experts and received weekly supervision.

2.4 Stimuli

Fourty-eight affectively unpleasant, pleasant or neutral pictures from the International Affective Picture System (IAPS), with 16 pictures of each valence, were used as prime stimuli. In a 1-9 scale of valence (with 9 being the most positive dimension), neutral pictures varied from 4.5 – 5.5, positive pictures were higher than 5.5 and negative pictures were lower than 4.5. The prime pictures were presented in multicolour, against dark backround, with a resolution of 600 x 450 pixels. They provided a visual angle of 10° x 6°.

Twenty-four pictures of happy, sad and neutral facial expressions from Pictures of Facial Affect (Ekman & Friesen, 1976) served as target stimuli. Pictures of 4 male and 4 female models with the expressions were selected. These target pictures were presented in black and white colour, against dark backround, with a resolution of 348 x 570 pixels. They provided a visual angle of 6° x 6°.

From these prime and target pictures, 160 affectively congruent, 160 incongruent and 160 neutral prime-target pairs were created. The stimuli were presented with the program E-prime that also controlled the timing. Prime-target pairs were presented in a random order. There was a total of 480 trials. In each trial, the affective prime picture was presented for 200 ms, following a 100 ms interval without stimulus presentation, and finally the target picture of facial expression was presented for 200 ms. That is, there was a stimulus onset asynchrony (SOA) of 300 ms, which is frequently used in experiments utilizing the affective priming effect. After the target picture there was 2000 ms of black screen, during which the individual reaction times were registered.

2.5 Procedure

The participants sat in a comfortable chair in a dimly-lit room, with a light of approximately 10 lux, during the experiment. Stimuli were presented on a computer screen (Eizo Flexscan, inch CRT display) approximately one meter away from the subject. The participants received a keypad with two buttons, one for the positive evaluation of the facial expressions and the other for the negative. The positive evaluation button was marked with a sticker of a smiling face to remind the participant. The assignment of the buttons was counterbalanced across subjects by removing the sticker. The participants were instructed not to pay attention to the first appearing picture of emotional content (the prime) and to focus on the following facial picture that was to be evaluated

in valence. The participants were told to press the button with the sticker of a smiling face if they considered the presented expression to be happy, to press the other button with no sticker if they considered the expression to be sad and not to press any button if the expression was neutral. The participants practiced shortly with 18 trials before the experiment under the supervision of an experimeter to make sure that they understood the task. If necessary, feedback was given and instructions repeated. The experiment was divided in two sections of 10 minutes (80 + 80 trials), with a short break in the middle, so the whole session lasted about 20 minutes. The experimeters were present in the next room. The experiment was part of a larger test battery, and the whole measurement lasted about an hour.

2.6 Data recording

2.6.1 Behavioral data

The mean reaction times from the congruently primed and incongruently primed positive and negative targets were calculated for each subject individually. Responses were registered by the E-prime software between 200 and 2200 ms after target stimulus onset.

2.6.2 Electrophysiological data

Electroencephalogram (EEG) was recorded from 14 tin electrodes mounted in an electrode cap (Easy Cap) at scalp positions FZ, F3, F4, CZ, C3, C4, PZ, P3, P4, P7, P8, OZ, O1, O2 with the Brain Vision Recorder software (Brain Products GmbH, Munich, Germany). Two bipolar electrodes were utilised to record the electro-oculogram (EOG) in order to control the interference of eye-blinks to EEG-signal. One electrode was placed above the left eye and another to the right side of the right eye. An average reference was applied.

The EEG data was analyzed with Brain Vision Analyzer software (Brain Products GmbH, Munich, Germany). The signals were filtered first and then segmented with a bandpass of 0,1 – 30 Hz. The data of eight subjects was exceptionally segmented with a bandpass of 0.1 – 18 Hz, due to some noise in the higher frequencies. Segments including amplitudes greater than -100 μv – 100 μv in any channel were removed with artifact rejection. Ocular correction was made with Gratton & Coles

algorithm. Trials were averaged separately for different prime-target stimulus types (positive-negative, negative-positive, negative-negative, positive-positive), and the peak amplitudes of the N170 component were searched between 130 - 210 ms. Neutral prime-target pairs were not examined in this study (unlike to the other stimulus types, reaction times were not measured to them). The post-treatment and follow-up data of one depressed subject and partly the pre-treatment data of one depressed subject were not included in the electrophysiological analysis, due to large amount of the artefacts in the ERP recording.

Six electrode sites were chosen for the final analysis. Previous studies (e.g. Bentin, Allison, Puce, Perez & McCarthy, 1996) have shown that the N170 component is present bilaterally in the parietal and occipito-temporal area of the brain, so channels P7, P8, O1 and O2 were chosen for analysis to investigate the component. Channels F3 and F4 were examined to detect the frontal positive counterpart of N170, *the vertex positive potential, VPP.*

2.7 Statistical analysis

2.7.1 Behavioral data

Reaction times were first compared between the measurement of the non-depressed controls and the pre-treatment measurement of the depressed participants. Firstly, the reaction times were compared between depressed and non-depressed groups using one-way ANOVA, with Group as factor and the original variables (congruent positive, incongruent positive, congruent negative, incongruent negative) as dependent variables. For the investigation of the priming effect, repeated measures ANOVA was applied for both groups, with factors Congruency (congruent, incongruent) and Valence (positive, negative).

As to the analysis of the pre- and post-treatment, and in addition the follow-up measurements of the depressed group, a repeated measures ANOVA was applied, with factors Measurement (pre-treatment, post-treatment, follow-up), Congruency (congruent, incongruent) and Valence (positive, negative). Paired samples t-tests (two-tailed) were used for post hoc tests. Huyhn-Feldt adjusted degrees of freedom were used in case of violated spherecity assumption.

2.7.2 Electrophysiological data

In the analysis of non-depressed controls and the pre-treatment measurement of the depressives, repeated measures ANOVA was applied, with factors Congruency (congruent, incongruent), Valence (positive, negative) and Electrode (P7, P8, O1, O2, F3, F4).

Repeated measures ANOVA was applied also to investigate the treatment effects in the data of depressives, including factors Measurement (pre-treatment, post-treatment, follow-up), Congruency (congruent, incongruent), Valence (positive, negative) and Electrode (P7, P8, O1, O2, F3, F4).

Paired samples t-tests (two-tailed) were used for post hoc tests. For the depressed group, a priming variable was created by reducing the amplitudes to incongruent stimulus pairs from the amplitudes to congruent stimulus pairs to investigate the possible time-related change in the priming effect in the N170/VPP amplitudes for both valences at different electrodes. The values of the priming variables of the pre-treatment measurement, post-treatment measurement and the follow-up measurement were then compared for different valences and electrodes. Huyhn-Feldt adjusted degrees of freedom were used in case of violated spherecity assumption.

3. RESULTS

3.1 Behavioral results

3.1.1 Baseline measurement: the non-depressed control subjects and the pre-treatment measurement of the depressives

For the reaction times, one-way ANOVA showed that differences between the groups were significant for every dependent variable (see Tables 1 and 2): for positive congruent $F(1,42)=9.370$, $p=0.004$; for positive incongruent $F(1,42)=7.497$, $p=0.009$; for negative congruent $F(1,42)=10.697$, $p=0.002$ and for negative incongruent $F(1,42)=9.470$, $p=0.004$ stimulus pairs. The non-depressed group demonstrated significantly smaller reaction times than the depressed group.

Repeated measures ANOVA showed an interaction of Congruency × Valence for both groups (for controls, $F(1,22)=15.853$, $p=0.001$; for depressed group, $F(1,20)=14,296$, $p=0.001$), and main effects for Valence (for controls, $F(1,22)=52.917$, $p=0.001$; for the depressed group, $F(1,20)=41.819$, $p = 0,001$) and Congruency (for controls, $F(1,22)=29.253$, $p=0.001$; for the pre-treatment measurement of the depressed group, $F(1,20)=35.706$, $p=0.001$) for both groups, indicating faster responses to congruent than to incongruent stimulus pairs, differing in different valences. Paired samples t-test showed only the positive priming effect to be significant for both groups, with significantly faster reaction times to positive congruent than to positive incongruent stimulus pairs (see Table 1): for controls, $t(22)=6.388$, $p=0.001$ and for the pre-treatment measurement of depressed group $t(20)=7.364$, $p=0.001$). Thus, the groups demonstrated only a positive priming effect in their reaction times and did not differ in the actual priming effects nor in the valence of the priming.

Group	Congruent stimulus pairs	Incongruent stimulus pairs
Non-depressed group	597,41	669,48
Depressed group	709,10	807,24

Table 1. ***Mean reaction times (ms) of both groups for positive stimulus pairs***

Group	Congruent stimulus pairs	Incongruent stimulus pairs
Non-depressed group	743,13	759,17
Depressed group	871,45	888,69

Table 2. ***Mean reaction times (ms) of both groups for negative stimulus pairs***

3.1.2 The treatment effects

It was hypothesized that the reaction times of the depressive group would come closer to the ones of the non-depressed control group in post-treatment and follow-up measurements. Repeated measures ANOVA showed the priming effect to remain constant in all three (pre-treatment, post-treatment, follow-up) times of measurement. There were neither significant effects for the factor Measurement, nor any interaction effect with it.

3.2 Electrophysiological results

3.2.1 Baseline measurement: the non-depressed control subjects and the pre-treatment measurement of the depressives

In controls, repeated measures ANOVA showed that Congruency × Valence × Electrode interaction did not reach significance, $F(5,18)=0.804$, $p=0.562$, but Valence × Electrode interaction did, $F(5,18)=3.128$, $p=0.033$, as did Congruency × Electrode interaction, $F(5,18)=4.044$, $p=0.012$. It has to be also notified that the p-value for the interaction Congruency × Valence was small: $F(1,22)=3.176$, $p=0.089$. The main effects for factor Electrode with $F(5,18)=21.295$, $p=0.001$, for factor Valence $F(1,22)=8.428$, $p=0.008$ and for factor Congruency, $F(1,22)=4.894$, $p=0.038$, indicated different amplitudes in different electrodes for congruent and incongruent stimulus pairs. Paired samples t-test indicated that, in non-depressed controls, only the channel P8, located in the right hemisphere, showed larger amplitudes to the congruent (mean = -6,42) than to the incongruent (mean = -5,84) stimulus pairs, regardless of the valence: $t=(23)-5.610$, $p=0.001$ (see Figures 2 and 3). In other words, there were positive and negative priming effects in the N170 amplitudes of non-depressed control group, localising in the right-hemispheric electrode P8. In addition, channel O2

showed larger amplitudes for congruent (mean = -5,53) than for incongruent (mean = -5,12) stimulus pairs almost significantly: t(23)=-1,987, p=0.059, regardless of the valence.

As for the depressives, there were significant interactions of Congruency × Valence × Electrode, F(5,15)=8.410, p=0.001, and Congruency × Electrode, F(5,15)=7.020, p=0.001. In addition, the main effects of Electrode, F(5,15)=16.597, p=0.001 and Congruency, F(1,19)=6.718, p=0.018, were significant. The pairwise comparisons indicated that, for happy expressions, both channels P7 and P8 showed significantly larger negative N170 amplitudes for congruent than for incongruent stimulus pairs (see Figures 2 and 4): for channel P8, t(19)=4.930, p=0.001; for channel P7, t(19)=4.258, p=0.001. For sad expressions, the priming effect was absent in channels P7 and P8, but channels F3 and O2 showed significantly larger ERP amplitudes for congruent than the incongruent stimulus pairs (see Figure 3): for channel F3, t(19)=-2.426, p=0.025; for channel O2, t(19)=-2,858, p=0.010. Additionally, in channel P8, negative congruent stimulus pairs (mean = -5,31) elicited nearly significantly larger amplitudes than for negative incongruent stimulus pairs (mean = -4,86): t(19)=1.976, p=0.063. In sum, the depressed group showed a positive priming effect not only in the same right-hemispheric P8 channel as the non-depressed controls, but also in the left-hemispheric P7 channel, which indicates laterality difference between the groups (see Figures 2 and 4). Negative priming effect did also show in the depressed group, but partly in different channels: in the left frontal F3 and the central occipital O2 (Figure 3), and moderately in the right-hemispheric P8.

Valence of the priming	Channel	Congruent pairs	Incongruent pairs
Positive	P7	-4,52	-3,44
	P8	-5,97	-4,84
Negative	F3	3,83	3,24
	O2	-4,94	-4,12

Table 3. ***Means of amplitudes (μv), channels and the associated valences of the depressed group that showed significant priming effects in pre-treatment measurement, as used in the statistical examination of the baseline measurements***

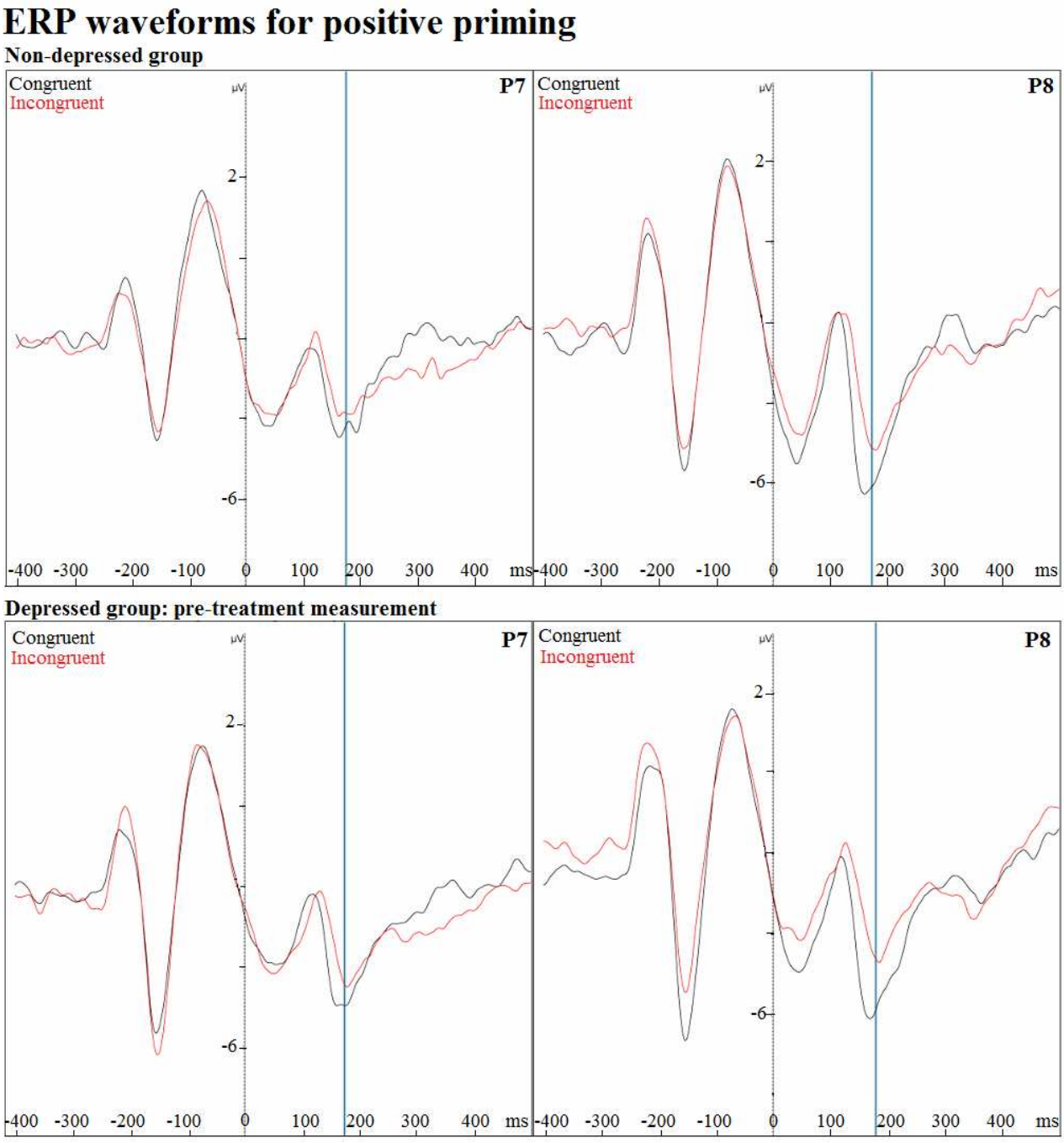

Figure 2. ***The ERP waveforms for positive stimulus pairs at channels P7 and P8 in both groups. The black wave represents the ERPs associated with the congruent stimulus pairs, as the red wave represents the ERPs associated with the incongruent stimulus pairs. The vertical blue line represents the ERP component N170. In the present study, latencies were not under investigation, and the images represent grand averages, so these pictures do not directly reflect the results as they are statistically calculated.***

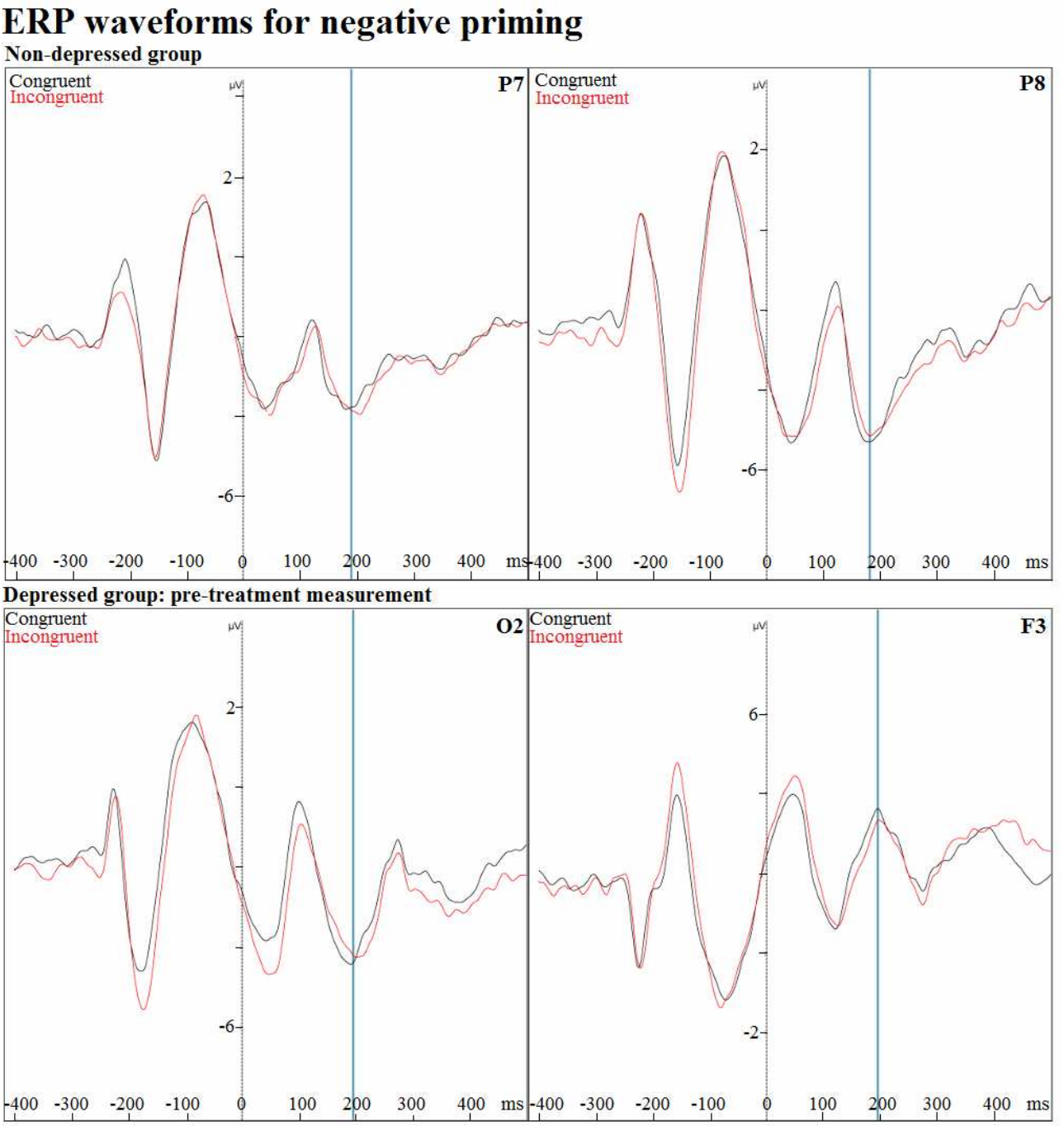

Figure 3. ***The ERP waveforms for negative stimulus pairs in both groups at channels that showed significant priming effects. The black line represents the ERPs associated with the congruent stimulus pairs, as the red line represents the ERPs associated with the incongruent stimulus pairs. The vertical blue line represents the ERP component N170 in images of channels P7 and P8 (non-depressed group) and 02 (pre-treatment measuremet of the depressed group); in image of channel F3 (pre-treatment measuremet of the depressed group), the line represents the vertex positive potential (VPP) . In the present study, latencies were not under investigation, and the images represent grand averages, so these pictures do not directly reflect the results as they are statistically calculated.***

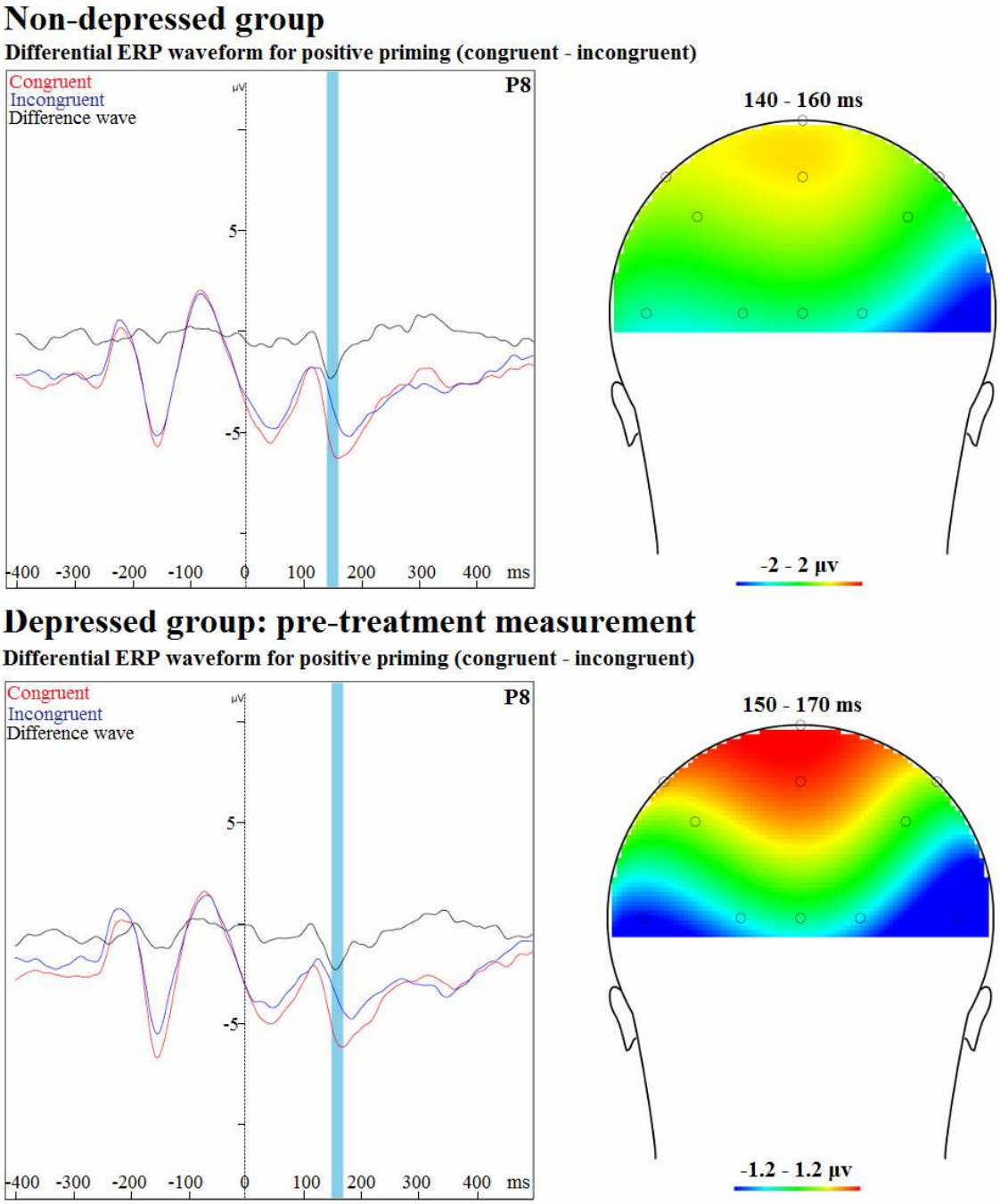

Figure 4. ***The positive priming effect in non-depressed controls and the pre-treatment measurement of the depressives. The black wave represents the difference waveform, red wave the waveform to congruent stimulus pairs and the blue wave the waveform to incongruent stimulus pairs. The vertical blue line represents the time window that is drawn in the topography. In the present study, latencies were not under investigation, and the images represent grand averages, so these pictures do not directly reflect the results as they are statistically calculated.***

3.2.2 The treatment effects

It was hypothesized that the post-treatment and follow-up ERPs would differ from the pre-treatment ERPs in the depressed group. It was expected that the priming effects in the N170/VPP amplitudes would start to resemble of those of the non-depressed controls` as the mood of the depressed participants would improve. Repeated measures ANOVA showed multiple interactions: Congruency × Valence × Electrode ($F(5,12)=4.033$, $p=0.022$), Valence × Electrode ($F(5,12)=4.760$, $p=0.013$), Congruency × Electrode ($F(5,12)=4.056$, $p=0.022$ and Congruency × Valence ($F(1,16)=11.420$, $p=0.004$). Main effects included Electrode ($F(5,12)=44.106$, $p=0.001$), Valence ($F(1,16)=8.220$, $p=0.011$ and Congruency ($F(1,16)=25.449$, $p=0.001$). These indicate larger ERP amplitudes for positive congruent than for positive incongruent stimulus pairs in depressed group at different locations. The main effect for factor Measurement or any of its interactions were not significant, but the interaction Measurement × Congruency × Valence × Electrode almost reached significance: $F(10,7)=3.505$,$p=0.055$.

Consequently, the values of the calculated priming variables of the pre-treatment measurement and the post-treatment measurement were compared in a paired samples t-test. In the left-hemispheric channel P7, there was a significantly larger priming effect in the N170 amplitudes ($t(18)=-2,288$, $p=0,034$) in the pre-treatment measurement (mean = -1,12) than in the post-treatment measurement (mean = -0,45) for positive expressions. For negative expressions, there was no such difference between the values of the priming variable in pre- and post-treatment measurements. Thus, as shown in Figure 6, the left-hemispheric activation in the channel P7 was diminished after the treatment, although this regarded only the positive priming effect. When comparing the values of the priming variables between the post-treatment measurement and the follow-up measurement with a paired samples t-test ($n=17$), there were no significant differences for either valence. This indicates that the effects of the treatment remained in these subjects: the kind of left-hemispheric P7 activation that was present in the pre-treatment measurement was still relatively absent seven months after treatment (see Figures 5 and 6). When concerning positive priming effect in channel P7, the values of the priming variable of the pre-treatment measurement (mean = -1,21) were also significantly larger, $t(16)=-2.909$, $p=0.010$), than those of the follow-up measurement (mean = -0,55). In addition, channel F3 showed larger priming variable values for positive valence in pre-treatment measurement (mean = 0,26) than in the follow-up (mean = -0,38), $t(16)=2.224$, $p=0.041$. In sum, these paired sample t-tests indicate for a significantly larger positive priming effect in the N170 amplitudes at left-hemispheric P7, and also at left frontal F3, in the pre-treatment

measurement than in the follow-up measurement. Consistent with the hypothesis, the localisation of the depressives` activation resembles that of the non-depressed group after the treatment, and this remains in the follow-up.

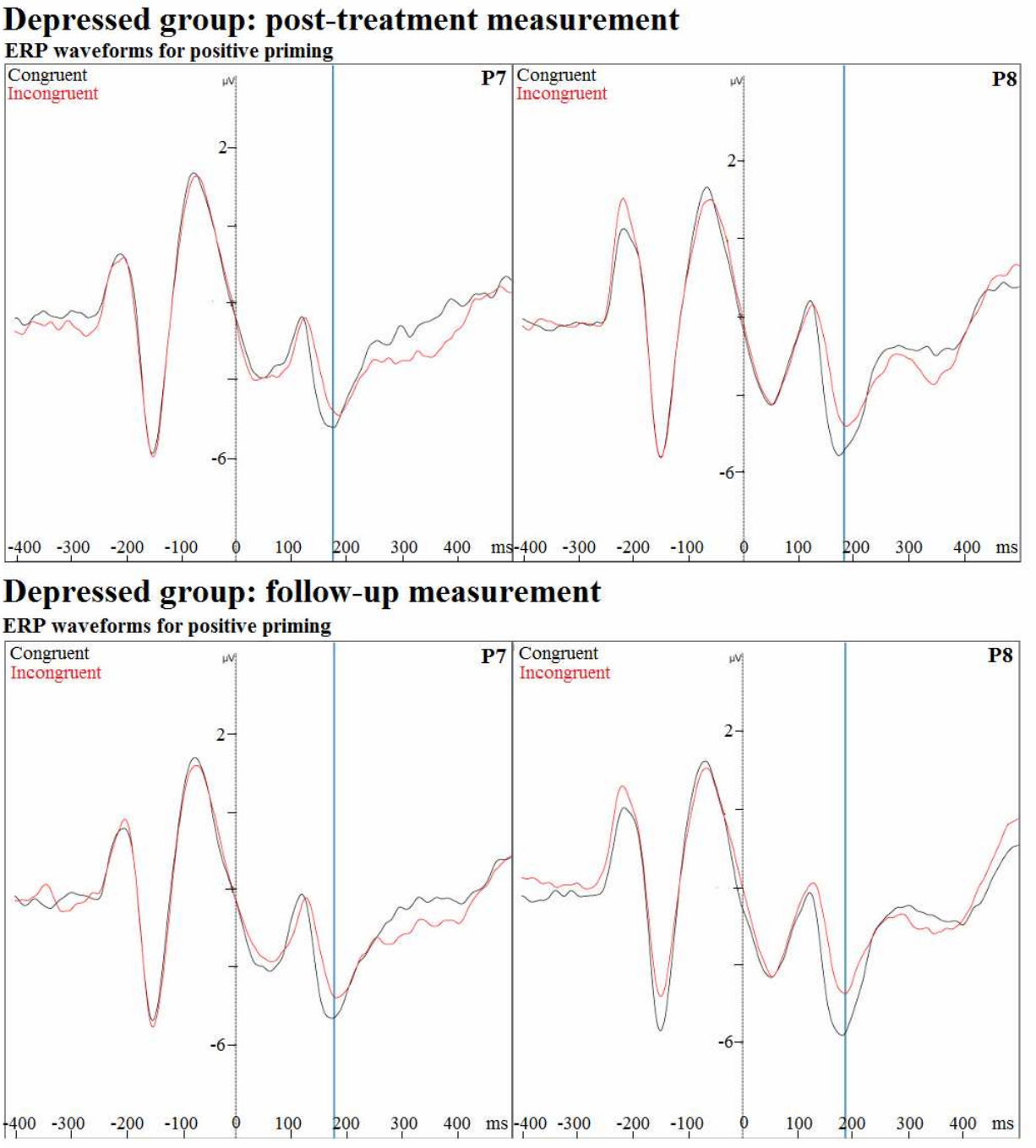

Figure 5. ***The ERP waveforms for positive stimulus pairs at channels P7 and P8 in the depressed group at post-treatment and follow-up measurements. The black line represents the ERPs associated with the congruent stimulus pairs, as the red line represents the ERPs associated with the incongruent stimulus pairs. The vertical blue line represents the ERP component N170. In the present study, latencies were not under investigation, and the images represent grand averages, so these pictures do not directly reflect the results as they are statistically calculated.***

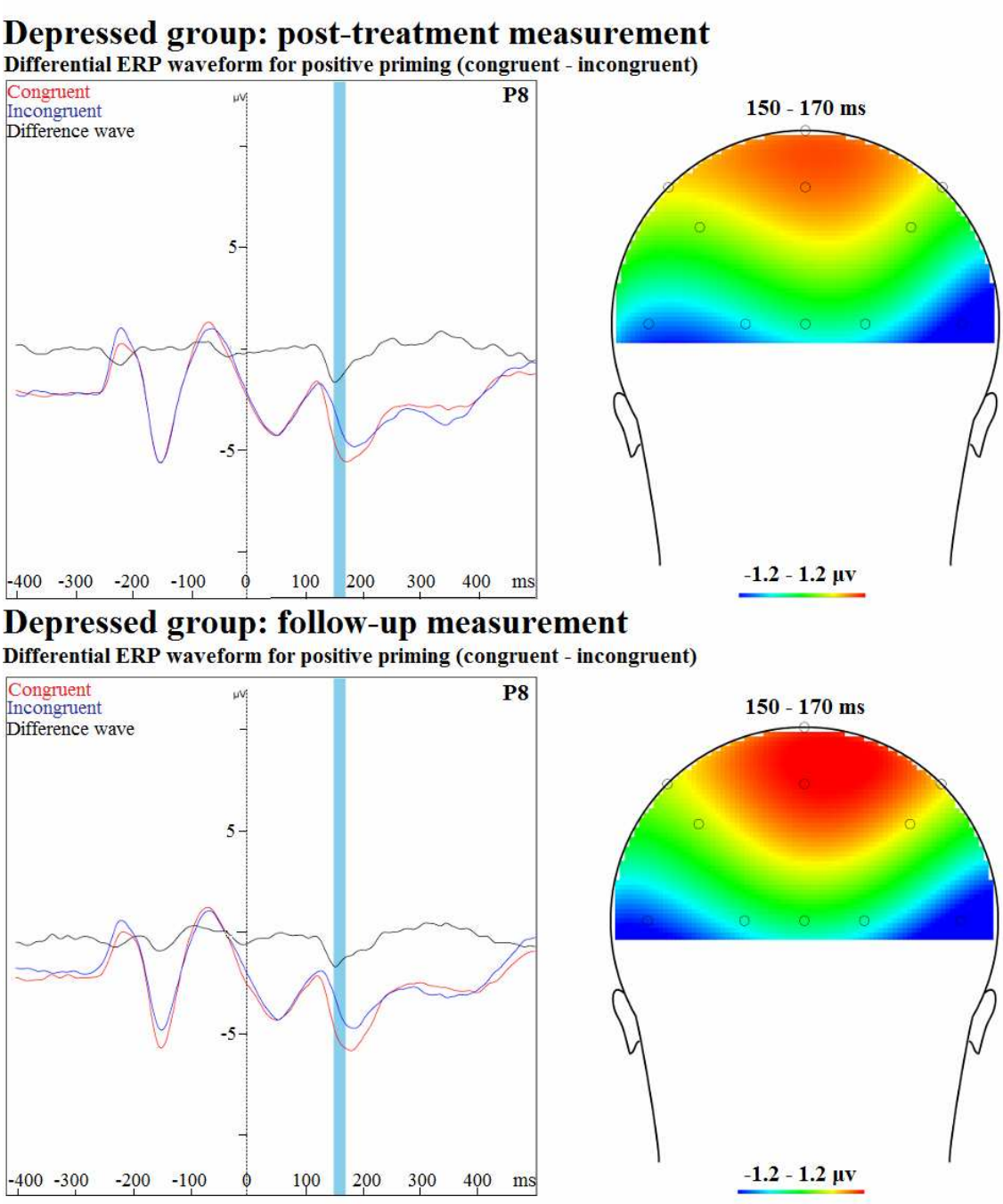

Figure 6. ***The positive priming effect in post-treatment and follow-up measurements of the depressed group. The black wave represents the difference waveform, red wave the waveform to congruent stimulus pairs and the blue wave the waveform to incongruent stimulus pairs. The vertical blue line represents the time window that is drawn in the topography. In the present study, latencies were not under investigation, and the images represent grand averages, so these pictures do not directly reflect the results as they are statistically calculated.***

Electrode	Valence of the priming	Congruency	Pre-treatment n = 19	Post-treatment n = 19	Follow-up n = 17
P7	Positive	Congruent	-4,70	-4,89	-5,23
		Incongruent	-3,58	-4,45	-4,68
	Negative	Congruent	-3,61	-4,34	-4,98
		Incongruent	-3,63	-4,06	-4,97
P8	Positive	Congruent	-6,20	-6,03	-6,47
		Incongruent	-5,00	-5,08	-5,51
	Negative	Congruent	-5,40	-5,33	-5,83
		Incongruent	-5,00	-5,05	-5,33
O1	Positive	Congruent	-5,02	-5,62	-5,27
		Incongruent	-4,81	-5,30	-5,41
	Negative	Congruent	-4,74	-5,06	-5,01
		Incongruent	-4,27	-4,76	-5,20
O2	Positive	Congruent	-4,93	-5,48	-5,55
		Incongruent	-5,13	-5,17	-5,31
	Negative	Congruent	-5,11	-5,11	-5,07
		Incongruent	-4,17	-4,65	-4,80
F3	Positive	Congruent	4,26	4,34	4,49
		Incongruent	4,02	4,41	4,87
	Negative	Congruent	3,95	4,19	4,96
		Incongruent	3,30	3,76	4,19
F4	Positive	Congruent	4,53	4,71	5,11
		Incongruent	4,34	4,87	5,08
	Negative	Congruent	3,90	4,38	4,98
		Incongruent	3,82	4,49	4,76

Table 4. ***The mean N170/VPP amplitudes (μv) of the depressed group at all times of measurement, as used in the statistical examination of the treatment effects***

4. DISCUSSION

The aim of the study was to investigate how positive effects of a brief therapeutic intervention, measured as an increase in the BDI scores of the subjects, would appear in the reaction times and event-related potentials of the depressed group. We were also interested in the differences in the reaction times and event-related potentials between the non-depressed and the depressed group at baseline measurement.

At behavioral level, the depressed group demonstrated longer reaction times to all of the stimulus pairs when compared to the non-depressed group. Additionally, both groups demonstrated only positive priming effects, that is, smaller reaction times to congruent than to incongruent stimulus pairs in positive valence. There were no differences between the groups. In addition, no changes were observed in the reaction times of the post-treatment and the follow-up measurements of the depressed group in relation to the reaction times of the pre-treatment measurement.

At electrophysiological level, both groups demonstrated both positive and negative priming effects, but in different brain locations. The non-depressed group demonstrated larger amplitudes to congruent than to incongruent stimulus pairs for both valences in the right-hemispheric electrode P8. The depressed group demonstrated also larger amplitudes to congruent than to incongruent stimulus pairs for positive valence at right-hemispheric electrode P8, but also at left-hemispheric electrode P7. For negative valence, the depressed group demonstrated larger amplitudes to congruent than to incongruent stimulus pairs at left frontal electrode F3 and right occipital electrode 02, and moderately in the right parietal P8. The perceived treatment effect in the ERPs concerned the location of the positive priming: after the intervention, the positive priming effect that was earlier perceived in the pre-treatment measurement at left-hemispheric electrode P7, was diminished, and this was still the case in the seven-month follow-up. When examining the treatment effect by comparing the pre-treatment and the follow-up measurements, the positive priming effects found at electrodes P7 and F3 in the pre-treatment measurement were absent in the follow-up measurement.

The longer reaction times of the depressed group can be attributed to the psychomotoric retardation related to depression in general (Isometsä, 2007). The results concerning the affective priming effect measured in reaction times could be explained in multiple ways. Given that there was no difference between the groups at the baseline and no treatment effects found in the depressed group,

the results could indicate that the reaction times might simply not be sensitive enough to measure the between-group or treatment-related differences in this phenomenon – especially when the ERPs did show negative priming in both groups and both between-groups and treatment-related differences in the localisation of the priming. When compared to the early stages of facial processing that the ERP component N170 is suggested to reflect (e.g. Bentin & Deouell, 2000), the reaction times might be considered expressing a more conscious process, involving decision-making, and therefore differences in the automatic level of facial perception might not show in them that explicitly. Previously, Zhang et al. (2006) have discussed the same and suggested that RTs might be sensitive to conscious task-dependent strategies, and thus reflect different cognitive operations than ERPs.

Anyhow, the results concerning the reaction times of the non-depressed group are in line with previous studies, indicating that positive expressions are recognized faster by healthy subjects – also called the happy face advantage (e.g. Kirita & Endo, 1995). Given that there is this kind of positivity bias, one could suggest that the relatively weaker negative priming effect might also have shown if the size of the groups would have been larger. There is also several studies that have found an orientation towards happy faces and an orientation away from sad faces in never-depressed controls (e.g. Joormann & Gotlib, 2007), and these are often referred to as a protective bias.

Kirita and Endo (1995) suggest in their study that happy faces are processed holistically as sad faces are processed analytically – one can consider if these different ways of processing could affect in our experiment that was based on priming effect. Could the affective state, supposively elicitated by the prime picture, influence or confuse the analytic process concerning the target picture of a sad face more than the holistic process concerning the target picture of a happy face? Werheid and colleagues (2005) found no priming effect in angry faces as opposed to happy faces in the reaction times of their healthy subjects, and they present that the structural similarity of facial features might have an effect, since a happy face is constituted more uniformly than an angry face. This could be the explanation for the present results as well: a sad face is not that uniform and clear than a happy face.

It is also known that affective priming appears to be facilitated by physical similarity of the target and the prime stimuli, although it appears even if these two differ in their features (Avero & Calvo, 2006). In this study, affectively positive, negative and neutral pictures from the International Affective Picture System (IAPS) were used as prime stimuli, and happy, sad and neutral pictures of

emotional expressions from Pictures of Facial Affect (Ekman & Friesen, 1976) were used as target stimuli. It could be that a priming experiment like ours, with a picture of affective content as a prime, and a picture of an emotional facial expression as a target, might not reflect the affective priming effect in its fullest possible appearance. A priming experiment using pictures of facial expressions as both primes and targets might have produced clearer results, but this remains as an interesting question.

Finally, Fazio (2001) has pointed out that in an affective priming condition the subjects` level of consciousness of the prime stimuli matter. In this study, the participants were simply told to ignore the prime pictures of the affective content, and according to Fazio (2001), there is evidence that this kind of procedure might lead to automatic tendencies to correct the influence of the primes. This kind of reversed priming is supposed to appear especially when the prime stimuli are extreme. In addition, the task that is given to the participants can be too easy and as the procedure can reach ceiling effect of facilitation, and therefore the results will not reach differences between the groups. However, the final reason for the RT results of the present study remains ambiguous, and more elucidating research is needed, utilizing the priming procedure with the processing of facial expressions in non-depressed and depressed individuals.

At electrophysiological level, we found both positive and negative priming effects in both groups at the baseline measurement. Surprisingly, larger ERP amplitudes to congruent than to incongruent stimulus pairs were discovered in both groups. Formerly, ERP studies of the emotional priming effect have demonstrated larger ERP amplitudes to incongruent than to congruent stimulus pairs (e.g. Brown, Hagoort & Chwilla, 2000; Zhang, Lawson, Guo & Jiang, 2006). Again, there can be multiple explanations for this. The type of the experiment could account for the differing results: we presented affective pictures as primes, and pictures of facial expressions as targets, when the previous studies used words (Brown, Hagoort & Chwilla, 2000) or pictures and words (Zhang, Lawson, Guo & Jiang, 2006), as primes and targets. Because of this, partly different ERP components have been investigated in the previous studies: the N400 (Brown, Hagoort & Chwilla, 2000; Zhang, Lawson, Guo & Jiang, 2006) and the N200 (Zhang, Lawson, Guo & Jiang, 2006). Given that the ERP component examined in the present study, the N170, reflects the early processing of faces, the effects of the affective prime picture might simply appear in it in a different way when compared to the previously investigated components that also have different neural backgrounds.

The actual reason for the larger N170 amplitudes to congruent than to incongruent stimulus pairs in this study remains indefinite. Possible explanations could rise from the experiment: as mentioned above, pictures of affective content and pictures of facial expressions were used in the study, so there was a stimulus mismatch between the primes and the targets. Additionally, this kind of composition of stimuli may provide a situation where the affectively incongruent stimuli might not evoke such perceived link between them as the affectively congruent stimuli. In other words, the emotional connection that the brain perceives between the affectively congruent stimuli might show as an increase in the amplitude of the ERP component N170 in this kind of experiment. Thus, the peculiar amplitude differences of N170 perceived in this study might reflect the perceived affective similarity of the prime and the target stimuli. Clearly more research of the effects of affective priming in the modulation of N170 is needed to clarify the mechanisms behind this phenomenon.

There were differences between the depressed and non-depressed groups in the localisation of the brain activation related to the priming. Non-depressed group demonstrated both positive and negative priming effects in the N170 at the right-hemispheric electrode P8, which is in line with the previous studies: the N170 is perceived larger in amplitude in the right hemisphere in healthy subjects (Bentin et al., 1996). The depressed group demonstrated positive priming in right-hemispheric electrode P8 and also in the left-hemispheric electrode P7, and negative priming in electrodes F3 and O2. Negative priming at right parietal channel P8, reflecting the component N170, almost reached significance. With a larger sample size it probably would have done so, given that significant effects were shown in the left frontal channel F3, reflecting the vertex positive potential that is the positive counterpart of the equivalent dipoles generating the N170. The significant results that appeared also in the central occipital channel O2 reflect activation at the electrodes located at the back of the skull. Anyhow, this activation did not reach the electrodes at the left hemisphere. One can conclude that the depressed group demonstrated priming of negative expressions lateralized at the right, similarly as the non-depressed group.

For positive expressions, the primed processing seems to be bilateral in the depressed group. Formerly, a decrease in the activation of the right posterior hemisphere has been linked with depression (Heller & Nitschke, 1998), and clearly the depressed group demonstrates some kind of abnormality in the right-side lateralization also in this study, when concerning positive expressions. The right hemisphere has consistently been linked to the processing of emotional information – and, specifically, the processing of facial expressions (e.g. Shenal, Harrison & Denaree, 2003). A general view that a dysfunction in the other hemisphere results in the overactivation of the other

hemisphere has often been discussed (e.g. Shenal, Harrison & Denaree, 2003), and the results of the present study could be also explained with the account. Some studies have also demonstrated left-hemispheric activation in anxious individuals (Heller & Nitschke, 1998), so the possible effects of comorbidity can be considered here, as it is known that depression and anxiety appear often together (Isometsä, 2007). Because the perceived activation in the left-hemispheric channel P7 decreased after the intervention and remained absent seven months afterwards, one can conclude that this kind of activation is associated with the depressive state.

It is also interesting that this bilateral processing did indeed concern only the positive priming effect. Yoon, Joormann & Gotlib (2009) suggest a deficit in the processing of positive stimuli in depression. One can ask if this kind of deficit, shown previously mostly behaviorally, has something to do with the abnormal bilateral activation in the brain of the depressed group that was shown during the positive priming effect before the treatment.

The precise effects of the therapeutic intervention might also account for the post-treatment change in the localisation of the activation concerning only the positive priming effect. The intervention was based on acceptance and commitment therapy, and the approach emphasizes, for example, the ability to accept one`s feelings. One might suggest that the effects could concern especially the handling of positive emotions, or change in how they are experienced. This could then show in the primed processing of positive stimuli. Anyhow, conclusions must be made carefully: although the subjects were treated with the same pattern, the exercises and homework that were given when the intervention was executed varied with the individual situations of the subjects. The experience might have differed a lot between subjects, and the precise mechanisms behind the recoveries might not be possible to attribute to anything specific. In any case, it seems that change occurred in both the mood and the localisation of the priming effect in the brains of the depressed group – but the link between these two remains vague.

The bilateral processing of positive expressions in the pre-treatment measurement also raises the question of the depressive state being state- or trait-like, and the result of the activation diminishing from the left supports naturally the state perspective. It is anyhow possible that the matter is not that straightforward. In the future, it would be interesting to investigate whether the activation concerning the primed expressions was in the left to begin with or whether it shifted to the left hemisphere as a result of the onset of the disorder. Does this kind of bilateral processing of primed happy faces – or possibly, positive stimuli in general - expose one to depression?

A challenge for the future studies is to solve whether this kind of change in the location of the electrical activation in the brain, related to remission from depression, reflect change in the processing of positive affect, or change in the processing of facial expressions - or the combination of these two, possibly in different relations. It has to be also noted that some other unknown factors might confuse the findings. Additionally, when interpreting the results of this study, one must keep in mind that electroencephalography reflects only the actions of the cerebral cortex, and the localisation of the activation is only an approximation in the method. Other methods of neuroscience could shed light to specific mechanisms involved in the priming of the positive facial expressions at the left hemisphere in depressives, and its absence after remission, as perceived in the present study.

Overall, the examination of the post-treatment and follow-up measurements of the depressed group showed that an intervention of only four sessions can influence the electric activation of the brain. This supports the previous literature that shows therapy effects to show in the level of the brain function (e.g. Roffmann et al., 2005). In this study the results are also relatively stable even after seven months from the intervention. One may speculate that in the future, these kind of studies might provide means for predicting the onset and reoccurence of depression.

Finally, it is necessary to consider the limitations of the study that are associated with the design, the experiment and the subjects. The depressed participants underwent the same experiment multiple times, and some kind of learning effect could have affected both the behavioral and the electrophysiological results. Naturally, this kind of effect cannot be separated from the actual treatment effect. In the other Master`s thesis that is in the making, the treatment effects will be investigated with a full factorial model, including the wait-list control group, so the effect of time will be controlled.

When considering the depressed participants that benefited from the intervention, most of them were women and the overall group size was quite small. 66,7 % of the benefited group also came from the experimental group that underwent the intervention first, which indicates that the participants that did not have to wait for the treatment benefited more. These factors should be considered when generalizing the results. Additionally, the types of the original depressive states of the benefited participants varied notably, as some of them suffered from chronic disorders and some demonstrated more of a reactive depression, related to their life situations.

Moreover, the use of anti-depressant medication in the benefited group that was examined in the study is a factor worth of taking especially into account. 33,3 % of the benefited group were medicated during the experiments, as only 19 % of the non-benefited group were medicated during the experiments. Firstly, this supports the previous literature showing that therapy and medication together equal the best result (Pampallona, Bellini, Tibaldi, Kupelnick & Munizza, 2004). Secondly, the medication might have affected to the electrophysiology of the medicated subjects in benefited group investigated in this study, which is to be notified when interpreting the results.

When considering the challenges for future studies, the examination of non-benefited participants might also bring forth important information. The benefited and non-benefited groups did not differ significantly in their pre-treatment BDI scores in this study, but in the future, more objective indicators like reaction times and ERPs could reveal original differences between these types of groups. Other measurements beside BDI might also provide more information. Obviously different interventions are suitable for different people and for different types of depressive states – and with these kind of experiments, the means of neuroscience might even serve possibilities to solve this puzzle further. In the future this kind of separation for the benefited and non-benefited groups in studies might lead to practical applications concerning the suitability of certain therapeutic methods for certain types of depressive disorders.

5. CONCLUSION

The results of the present study indicate that there are differences in the affective priming effect of non-depressed and depressed participants, as measured in the localisation of the event-related potentials. The results also show that the positive effects of a brief intervention can alter the ERP localisation of depressed participants to resemble the ERP localisation that was demonstrated by non-depressed participants. This change remained in the follow-up seven months after the intervention. The results bring support the literature showing that psychological intervention can influence the function of the brain relatively permanently. Reaction times turned out not to be sensitive enough to present these differences in this study. In the future, more research is needed to elucidate whether the kind of electrophysiological differences in the preattentive processing between the groups, as perceived in this study, are linked to facial expressions or to positive valence. Furthermore, the precise mechanisms behind the effects of the intervention need to be examined more closely.

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