EFFECTS OF SHORT INTERVENTION FOR THE PROCESSING OF FACIAL EXPRESSIONS IN DEPRESSION

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Master's thesis

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ABSTRACT

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Effects of short intervention for the processing of facial expressions in depression
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The aim of the study was to examine the differences in the automatic processing of information between the depressed and the non-depressed by measuring brain's event-related potentials (ERPs). Also, the effects of short psychological intervention for the ERPs were studied. There were 40 depressed and 23 non-depressed participants in the study. The electroencephalogram (EEG) was measured from the depressed subjects before and after the intervention and also in the follow-up after seven months. For the non-depressed subjects the EEG was performed once. The depressed subjects were divided into the benefitted and the not-benefitted groups based on their self-evaluation after the intervention. In the experiment, the subjects were shown repeatedly a *neutral* facial expression (p=0.8) on a computer screen which was occasionally replaced by a *fearful* or a *happy* expression (p=0.1 for each). The effects of depression for the processing of expressions and the brain's change detection mechanism were studied. The analyzed ERP components were N170/P170 and P230/N230. The P230 responses between the depressed and the non-depressed differed; however, the type of expression or its frequency did not affect on the P230 responses. The non-depressed had right-sided hemispheric asymmetry of activation which the depressed lacked. The intervention did not affect the P230 responses in the depressed although the response to the *happy* expression increased in the follow-up. Other differences between the processing of *happy* and *fearful* expressions were not detected between the depressed and the non-depressed or between the benefitted and the not-benefitted. Differences between the groups were found when the responses to *neutral* and *emotional* (the mean of *happy* and *fearful*) expressions were examined. The P170 responses were larger to the *emotional* expression than to the *neutral* expression in the non-depressed and the not-benefitted groups while in the benefitted group the P170 responses to *neutral* and *emotional* expressions were similar. The N170 or P170 responses did not change between the measurements. In regard to P230, the brain of the not-benefitted differentiated between *neutral* and *emotional* expressions unlike the brain of the benefitted and the non-depressed. The differences between expressions measured for the not-benefitted were no longer visible in the follow-up. The results confirmed prior findings of impaired processing in emotional information during depression. Further research is needed to determine if the ERPs of the depressed differ from the non-depressed already before the onset of the symptoms.

Keywords: depression, electroencephalography, event-related potentials, facial expressions, N170, P230, visual mismatch negativity.

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Tutkimuksen tarkoituksena oli selvittää automaattisen tiedonkäsittelyn eroja masentuneiden ja mielialansa hyväksi kokevien välillä mittaamalla aivojen herätevasteita. Lisäksi tutkittiin lyhyen psykologisen intervention vaikutusta näihin vasteisiin. Tutkimukseen osallistui 40 masentunutta ja 23 kontrollikoehenkilöä. Masentuneiden koehenkilöiden aivosähkökäyrä mitattiin ennen interventiota, sen jälkeen sekä seitsemän kuukauden seurannassa. Henkilöt, jotka eivät kokeneet masennusta, mitattiin vain kerran. Masentuneet koehenkilöt jaettiin hyötyneisiin ja hyötymättömiin sen perusteella, miten he arvioivat mielialansa intervention jälkeen. Kokeessa kuvaruudulle esitettiin toistuvasti *neutraali* kasvonilme (todennäköisyys 0.8), jonka korvasi ajoittain *pelokas* tai *iloinen* kasvonilme (molempien todennäköisyys 0.1). Näin tutkittiin masennuksen vaikutusta kasvonilmeiden prosessointiin sekä aivojen muutoksenhavaitsemismekanismiin. Analysoitavia vasteita olivat N170/P170 sekä P230/N230. P230-vasteet kasvokuviin erosivat masentuneiden ja kontrollikoehenkilöiden välillä, mutta ilme tai sen yleisyys ei vaikuttanut P230-vasteisiin. Kontrollikoehenkilöillä huomattiin oikeanpuoleinen aivopuoliskojen aktivaation epäsymmetria, jota ei havaittu masentuneilla. Interventio ei vaikuttanut P230-vasteeseen masentuneilla, vaikkakin vaste *iloiseen* kasvonilmeeseen suureni seurannassa. Muita eroja *iloisen* ja *pelokkaan* kasvonilmeen prosessoinnissa ei havaittu masentuneiden ja kontrollikoehenkilöiden tai hyötyneiden ja hyötymättömien välillä. Ryhmien välisiä eroja löytyi, kun tarkasteltiin *neutraaliin* ja *emotionaaliseen* (*iloinen* ja *pelokas* keskiarvostettuna) kasvonilmeeseen syntyvien vasteiden välisiä eroja. Kontrollikoehenkilöiden ja hyötymättömien P170-vasteet olivat isompia *emotionaaliseen* kuin *neutraaliin* ilmeeseen. Hyötyneiltä mitatut P170-vasteet olivat samanlaiset *neutraaliin* ja *emotionaaliseen* ilmeeseen. N170- tai P170-vasteet eivät muuttuneet mittausten välillä. P230-vasteen mukaan hyötymättömien aivot erottelit *neutraalin* ja *emotionaalisen* ilmeen toisistaan, toisin kuin hyötyneiden ja kontrollikoehenkilöiden aivot. Hyötymättömiltä mitattu erottelu ilmeiden välillä ei ollut näkyvissä enää seurannassa. Tulokset vahvistavat aiempaa tietoa, jonka mukaan masennus vaikuttaa emotionaaliseen prosessointiin ja näkyy aivopuoliskojen aktivaation epäsymmetrian puuttumisena. Lisätutkimusta tarvitaan selvittämään, ovatko poikkeavuudet aivotoiminnassa piirteenomaisia ominaisuuksia masentuneille vai kehittyvätkö ne vasta oireiden puhjettua.

Avainsanat: aivosähkökäyrämittaus, herätevasteet, kasvonilmeet, masennus, N170, P230, poikkeavuusnegatiivisuus.

CONTRIBUTIONS

Professor Raimo Lappalainen and **docent Piia Astikainen** were responsible for organizing and supervising the present study. Lappalainen planned the intervention, instructed the trainees who gave the intervention, and was responsible for the gathering and analyzing of the survey data. Astikainen planned the EEG experiments and supervised the gathering, the analysis and the reporting of the ERP data.

In addition to the present master's thesis, the following authors have completed or are preparing their master's theses using the data from this study, but with different research problems:

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CONTENTS

INTRODUCTION

Depressive affect is an appropriate response to losses and disappointments, and is part of normal emotional life (Lönnqvist & Aalberg, 2007). However, when the depressive affect becomes more permanent state, it can be described as depressive mood. The depressive mood is one of the symptoms of major depressive disorder (MDD), also known as major depression, in the diagnostic and statistical manual of mental disorders (DSM-IV) criteria. MDD is diagnosed when a person has suffered from at least five different symptoms of the disorder during the last two weeks. The DSM-IV criteria include for example loss of pleasure, sleeping problems, fatigue, sense of worthlessness, and thoughts of death. Due to the symptoms, the depressed are in high risk to commit suicide. Often depression is prompted by a negative life event and the risk of relapse is considerable after symptom remission. Different explanations about the causes of depression include alterations in neurotransmitter or hormone levels and structural and/or functional changes in the brain.

Approximately 3–10 per cent of European population suffers from depression (Lönnqvist & Aalberg, 2007). In Finland, the prevalence of MDD is about five per cent for women and about three per cent for men (Isometsä, Aro, & Aro, 1997). Depression complicates the everyday life and it can lead to working disability (Lönnqvist & Aalberg, 2007). Therefore, it is essential to acquire information on this debilitating disorder that causes both individual suffering and enormous costs to the society.

The thought content of a depressed person can be explained by a cognitive triad (Beck, 1976). The three elements of the cognitive triad are (1) negative conception of the self, (2) negative interpretation of life experiences, and (3) nihilistic view of the future. These thinking patterns lead to emotions typical for depressed individuals: sadness, disappointment, and apathy. As a consequence, the cognitive schemas that serve as automatic information processors are negatively biased in depression (Beck, 2008).

Brain abnormalities and recognition of emotions in depression

The depressed have structural and functional abnormalities in the brain, for example in the amygdale, prefrontal cortex, hippocampus, and thalamus, which cause them to interpret stimuli emotionally within a negative context (for a review, see Phillips, Drevets, Rauch, & Lane, 2003). These abnormalities could be connected to the difficulties that the depressed have in recognizing facial expressions. The depressed make more errors in labeling facial expressions (Persad & Polivy, 1993), and are slower in recognizing facial expressions (Cooley & Nowicki, 1989; Surguladze et al., 2005) compared to the non-depressed. Normally, people across the cultures share the same interpretations of basic facial expressions (Ekman, Sorenson, & Friesen, 1969; Ekman & Friesen, 1971; Ekman et al., 1987). Obviously, the correct recognition of facial expressions is vital for sustaining social relationships.

Depression affects perception of emotions. Especially the processing of positive information is impaired with the depressed (Deldin, Keller, Gergen, & Miller, 2000; Shestyuk, Deldin, Brand, & Deveney, 2005; Suslow, Junghanns, & Arolt, 2001). The depressed need more intensity to identify happy expressions (Joormann & Gotlib, 2006; Yoon, Joormann, & Gotlib, 2009) and subtle low arousal facial expressions (Csukly, Czobor, Szily, Takacs, & Simon, 2009). In addition, there is evidence of abnormal processing of negative stimuli in depression (Kayser, Bruder, Tenke, Stewart, & Quitkin, 2000). Positive bias causes people to concentrate on positive information instead of negative; however, the depressed do not have positive nor negative bias (Bradley et al., 1997; Deldin, Keller, Gergen, & Miller, 2001). Similar results were obtained when facial expressions were studied (Surguladze et al., 2004), though, according to another study, the depressed have an attentional bias towards mood-congruent sad faces (Gotlib, Krasnoperova, Yue, & Joormann, 2004).

There is controversy on what causes the differences in processing of emotions between the depressed and the non-depressed. The problems in the recognition of emotions could be caused by a perceptual deficit (Asthana, Mandal, Khurana, & HaqueNizamie, 1998). On the other hand, the slower responses to positive faces could result from comorbid anxiety disorder, which means that depression alone does not impair automatic facial processing (Suslow et al., 2004). However, depression seems to slow down the detection of neutral faces (Leppänen, Milders, Bell, Terriere, & Hietanen, 2004; Suslow et al., 2004).

The asymmetry of brain activation differs between the depressed and the non-depressed. For instance, electroencephalography (EEG), which measures the activity of pyramidal neurons in neocortex, has revealed that depressed individuals have decreased right posterior activity during a spatial task (Henriques & Davidson, 1997) and a recognition-memory task (Deldin et al., 2000). In addition, according to an auditive oddball study the activity of the right central brain area is lowered in the subclinically depressed (Sumich, Kumari, Heasman, Gordon, & Brammer, 2006). The right hemisphere of the brain is important for understanding, expressing, and feeling emotions, and the functioning of this hemisphere seems to be impaired in depression (for a review, see Heller, Nitschke, & Miller, 1998). In short, MDD patients lack the normal asymmetry where the right hemisphere elicits more substantial responses than the left hemisphere. By contrast, resting EEG data shows that depression is also associated with relative right-sided frontal asymmetry in which the right frontal lobe elicits stronger responses than the left (for a review, see Thibodeau, Jorgensen, & Kim, 2006).

The lack of asymmetry is also visible with responses to facial expressions. Mikhailova, Vladimirova, Iznak, and Tsusulkovskaya (1996) found that the non-depressed have right hemisphere superiority for happy expressions unlike the depressed. In addition, Moratti, Rubio, Campo, Keil, and Ortiz (2008) noticed decreased arousal from emotional pictures in the right temporoparietal cortex with the depressed. Unlike the depressed, the non-depressed have bigger amplitudes at the right parietotemporal region, for negative images when compared to neutral images (Kayser et al., 2000). In conclusion: the depressed differ from the non-depressed in hemispheric asymmetry of the brain.

The ERP component N170

Event-related potentials (ERPs), which are graded potentials elicited by sensory stimuli, are measured using EEG. When measuring signal ERPs, it is necessary to average a large amount of stimuli. The face-specific ERP component N170, which is elicited automatically in response to human face, was first found 172 ms post-stimulus (Bentin, Allison, Puce, & Perez, 1996). N170 is a negative potential and especially sensitive to the human eyes, and it is more extensive in the right than in the left hemisphere (Bentin et al., 1996). N170 is elicited by upright and inverted faces but

not by other objects (Itier & Taylor, 2004). The component is not affected by the familiarity of faces (Bentin & Deouell, 2000).

It is controversial whether N170 merely reflects the structural coding of human face (Ashley, Vuilleumier, & Swick, 2004; Bentin et al., 1996; Eimer & Holmes, 2002; Eimer, Holmes, & McGlone, 2003; Holmes, Vuilleumier, & Eimer, 2003; Santesso et al., 2008) or whether there is modulation in the component within different emotions (Batty & Taylor, 2003; Blau, Maurer, Tottenham, & McCandliss, 2007; Krombholz, Schaefer, & Boucsein, 2007; Leppänen, Kauppinen, Peltola, & Hietanen, 2007; Schupp et al., 2004). Those studies that favor the modulation suggest that fearful and angry facial expressions induce greater ERPs than neutral or positive expressions.

Another ERP component, vertex positive potential (VPP), which is also known as P200, is elicited simultaneously with N170 in response to human face (Jeffreys, 1983; Jeffreys, 1989). Campanella et al. (2000) determined VPP at 158 ms and located it at the electrode site Cz. According to Ashley et al. (2004) VPP is enhanced for fearful expressions frontocentrally, which backs up the theory that VPP might be modulated by facial expressions in the same manner as N170.

Visual mismatch negativity

Mismatch negativity (MMN) is an ERP component that reflects a change detection mechanism in the brain. MMN is automatically elicited 100-250 ms post-stimulus and is not affected by stimulus predictability, significance, or attention (Näätänen, 1992). It was first discovered in the auditive modality (for a review, see Näätänen & Michie, 1979). In order to produce auditive MMN there has to be a change for example in the frequency, intensity, or duration of the sound. MMN is commonly studied with an oddball paradigm in which the standard stimulus is occasionally replaced with a deviant stimulus. Recent studies suggest that a similar mechanism to auditive MMN for detecting changes exists in the visual modality as well. Visual mismatch negativity (vMMN) has been reported for changes in colour (Czigler, Balazs, & Winkler, 2002; Stagg, Hindley, Tales, & Butler, 2004), orientation (Astikainen, Lillstrang, & Ruusuvirta, 2008; Fu, Fan, & Chen, 2003), motion direction (PazoAlvarez, Amenedo, & Cadaveira, 2004), combinations of features (Winkler, Czigler, Sussman, Horvath, & Balazs, 2005), quantity (Stagg et al., 2004; Tales,

Newton, Troscianko, & Butler, 1999), and spatial frequency (Heslenfeld, 2003). vMMN-like ERPs have also been found in rabbits (Astikainen, Ruusuvirta, & Korhonen, 2000).

In addition, vMMN is evoked by changes in facial expressions. Susac, Ilmoniemi, Pihko, and Supek (2004) found that happy and neutral expressions elicit vMMN around 280 ms post-stimulus. Expression mismatch negativity (EMMN), which is likely the same phenomenon as vMMN, has been found in an oddball study using neutral expressions as standard stimuli and happy/sad expressions as deviant stimuli (Zhao & Li, 2006). EMMN was more pronounced for sad facial expression than for happy expression. In accordance, Chang, Xu, Shi, Zhang, and Zhao (2010) noticed that sad facial expressions elicit larger vMMN than happy expressions. EMMN or vMMN is located at the posterior brain areas and it is more vigorous at the right hemisphere than at the left (Chang et al., 2010; Zhao & Li, 2006). Astikainen and Hietanen (2009) also found vMMN at two latency ranges, 150-180 ms and at 280-320 ms after stimulus onset, when they used fearful, happy, and neutral expressions in an oddball paradigm. Similarly, Chang et al. (2010) found early vMMN at 120-200 ms (reflecting the component N170) and late vMMN at 220-320 ms (reflecting the component P250). Therefore, vMMN for facial expressions is elicited simultaneously with the component N170. However, because it is controversial whether N170 is modulated by different expressions, it is difficult to define which differences between the responses to different expressions are caused by vMMN and which by modulation of N170.

There are differences in vMMN between the depressed and non-depressed which might reflect impaired preattentive processing of information during MDD. Chang et al. (2010) determined that the early vMMN reduces in depression; moreover, they did not detect late vMMN at all with the depressed. The usage of variations in schematic expressions to mimic different identities altered the vMMN results slightly. The depressed produced smaller early vMMN at the right hemisphere although the left hemisphere elicited equal responses with the depressed and the non-depressed. In addition, the auditive MMN has been reported to be affected by depression (Takei et al., 2009).

Effects of intervention for depression

Besides the personally experienced improvement in the quality of life, psychological interventions in treating depression have been proven to elicit measurable changes also in the brain function (Costafreda, Khanna, MouraoMiranda, & Fu, 2009; Fu et al., 2008). Cognitive therapy is as

effective as antidepressant medication in treating depression; furthermore, the likelihood of reoccurrence of depression is considerably lower with therapeutical intervention (for a review, see DeRubeis, Siegle, & Hollon, 2008). Therefore, it is plausible that the effects of psychological intervention could be measureable with brain imaging methods.

It is however debatable if the depressed could become more accurate at recognizing different expressions after successful intervention. Mikhailova et al. (1996) noticed that the recognition of emotions improved along with the mood. Other studies suggest that the subjects that had been depressed in the past were still impaired in the recognition of neutral (Leppänen et al., 2004) and happy facial expressions (LeMoult, Joormann, Sherdell, Wright, & Gotlib, 2009). Bouhuys, Geerts, Mersch, and Jenner (1996) suggested that the impairments in emotion recognition are trait-like qualities for the depressed rather than an acquired condition. In conclusion, although it is probable that some processes change after depressive periods, some of the processes might remain somewhat constant.

Aims of the study

Purpose of this Master's thesis was to find out if there exist differences in the processing of facial expressions between the depressed and non-depressed participants. Two ERP components, N170 and P230, were studied. Both positive (P170 and P230) and negative (N170 and N230) polarities of these components were analyzed. The brain's change detection mechanism in the visual modality, i.e. visual mismatch negativity was examined using an oddball paradigm. This allowed us to analyze whether depression affects the processing of *happy* and *fearful* expressions. More importantly, the effects of short psychological intervention were determined. It was also studied if there were differences between those who benefitted from the intervention and those who did not. The follow-up was performed to find out the long-lasting consequences of the intervention.

METHODS

Participants

There were 40 depressed (6 men) and 23 non-depressed (4 men) participants in this study. The depressed participants were recruited with a newspaper advertisement, while the non-depressed participants found out about the study from advertisements sent to different communities, such as schools and organizations. Originally, more participants were measured, but some of them were discarded due to e.g. young age, poor data quality or non-filled inventories. The data was gathered during the years 2008-2009. All participants gave written informed consent and the study was approved by the ethical committee of the University of Jyväskylä.

All subjects were right-handed and had normal or corrected-to-normal vision. They did not suffer from drug abuse or neurological illnesses. The depressed participants were interviewed by psychology majors to attain information about the severity of their depression according to the DSM-IV-TR criteria. The participants did not report any psychiatric diagnoses other than depression. Eleven depressed participants were currently on medication. The participants in the non-depressed group did not consider themselves as depressed and their Beck's Depression Inventory (BDI) scores were below 10. The depressed participants were on average 47.4 years old (standard deviation; SD 9.700) ranging from 28 to 61 years and the non-depressed participants were on average 46.6 years old (SD 7.721) with the age range of 30-58.

Experimental design

The depressed participants were randomly assigned into (1) the experimental group and (2) the waiting list group. The experimental group received intervention after the first measurement while the waiting list group was measured twice before the intervention (Figure 1). The intervention was given by final stage psychology majors and it consisted of four sessions, each lasting about sixty minutes. The students were provided with a short training on the methods of the intervention and weekly supervision during the intervention.

The intervention that was applied followed the principles of acceptance and commitment therapy (ACT). ACT is part of the third wave of behavioral and cognitive therapy (for a review, see Hayes, Luoma, Bond, Masuda, & Lillis, 2006). The theoretical roots of ACT stem from functional contextualism and relational frame theory. ACT aims to increase the client's psychological flexibility by affecting six core processes: (1) acceptance, (2) cognitive defusion, (3) being present, (4) experiencing self as context, (5) choosing personal values, and (6) committing to action towards values. The ACT intervention is typically short.

Seven months after the intervention there was a follow-up measurement in which 36 depressed participants were included. The experimental group had their EEG recorded three times in total and the waiting list group four times while the non-depressed participants were measured only once (Figure 1). The subjects received movie tickets after each EEG recording.

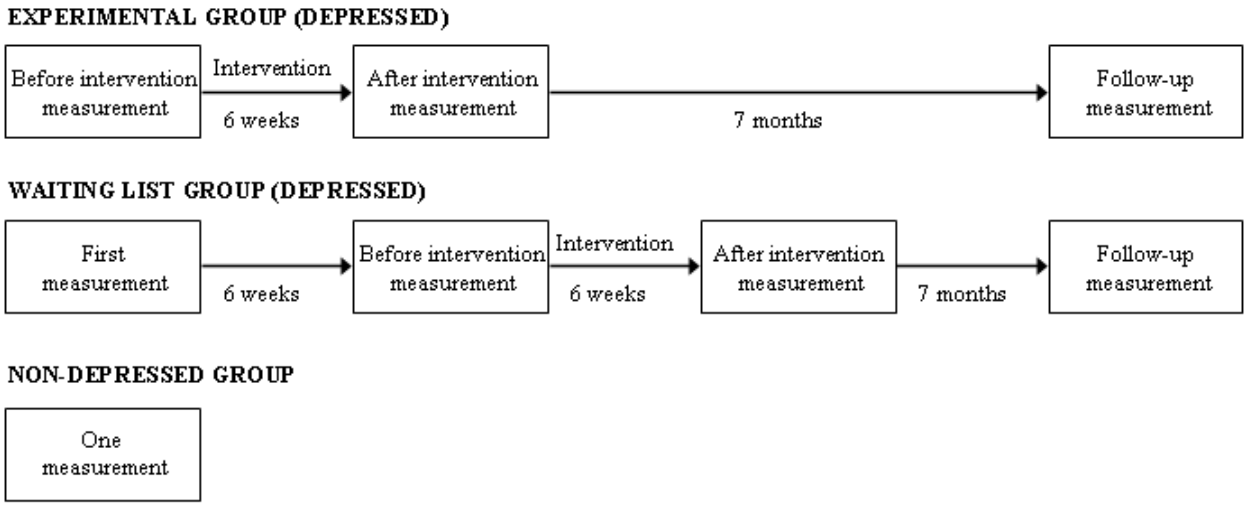

Figure 1. The experimental design of the study. Members of the waiting list group received intervention after the experimental group. The first measurement for the waiting list group was not used in the thesis.

Group division

Vanhatalo analyzed the differences between the experimental group and the waiting list group in her master's thesis (Vanhatalo, 2009). Therefore, in this thesis the experimental group and the waiting list group were combined as one depressed group. Three measurements were analyzed from the depressed participants: (1) the measurement before the intervention, (2) the measurement after the intervention, and (3) the follow-up measurement. Note that the measurements before and

after the intervention are from different time points in the experimental and waiting list groups (Figure 1).

The research subjects were divided into groups in two ways: 1) the depressed/the non-depressed group and 2) the benefitted/the not-benefitted/the non-depressed group (Figure 2). In the latter group division the depressed participants were subdivided into two groups based on the change in their BDI scores after the intervention. If the BDI score decreased 40 per cent or more, the participant was allocated into the benefitted group and if the score decreased less than 40 per cent or increased, the participant was a member of the not-benefitted group.

In the benefitted group (n=20, 1 man) the average age was 47.3 years (SD 9.033) and in the not-benefitted group (n=20, 5 men) 47.6 years (SD 10.560). Seven of the participants in the benefitted group and four in the not-benefitted group were medicated. In the benefitted group there were 15 participants from the experimental group and five participants from the waiting list group and in the not-benefitted group there were eight participants from the experimental group and twelve participants from the waiting list group.

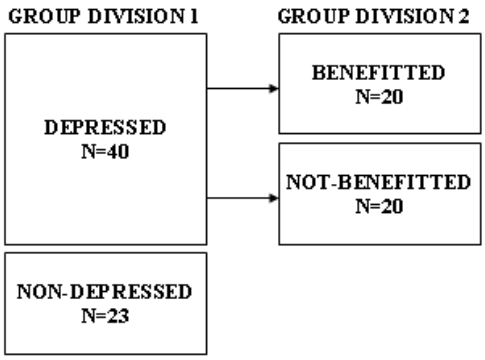

Figure 2. Illustration of the two group divisions used in this thesis.

Survey data

In addition to EEG measurements participants were asked to fill in nine inventories in every meeting: BDI, Beck's anxiety inventory (BAI), Symptom check list–90 (SCL-90), Acceptance and action questionnaire 2 (AAQ-2), Kentucky inventory of mindfulness skills (KIMS), JES, which measures coping in every-day life, and Ojanen's Mood, Self-confidence, and Satisfaction to life questionnaires. In the depressed group the BDI scores decreased on average 10.8 points between the measurements before and after the intervention (Table 1). In the benefitted group the BDI scores lowered on average 70 per cent (17.6 points) and in the not-benefitted group 14 per cent (3.9 points).

Table 1
The mean Beck's Depression Inventory (BDI) scores, standard deviations (SD), minimum and maximum scores, and sample sizes (n) in different groups and measurements.

Group	Before intervention BDI	After intervention BDI	Follow-up BDI
Depressed	**25.9** (SD 8.485) 10-41, n=40	**15.1** (SD 10.350) 1-39, n=40	**10.7** (SD 9.495) 0-36, n=33
Benefitted	**24.9** (SD 8.589) 10-40, n=20	**7.3** (SD 4.253) 1-14, n=20	**6.4** (SD 5.966) 0-21, n=16
Not-benefitted	**26.9** (SD 8.478) 13-41, n=20	**23.0** (SD 8.488) 9-39, n=20	**14.7** (SD 10.570) 1-36, n=17
Non-depressed	**2.0** (SD 1.821) 0-6, n=23	-	-

Stimuli and procedure

The experiment consisted of a slide show in which pictures of facial expressions were shown with an interstimulus interval of 500 ms (offset to onset). Four different personalities (female actors NR and PF, male actors EM and JJ) and three expressions (neutral, happy, fearful) from Ekman's and Friesen's *Pictures of Facial Affect* (1976) were used. Each picture was shown for 200 ms. An oddball paradigm was applied in which the probability of the standard stimulus, i.e. the neutral face was 0.8. The probabilities of the happy and fearful expressions were 0.1 for each; thus, the probability of the deviant stimuli was 0.2. The pictures were presented in a quasi-random order so that there were at least two neutral expressions between *emotional* (*happy* or *fearful*) expressions. Totally 1600 *neutral*, 80 *happy*, and 80 *fearful* pictures were presented. The experiment lasted about twenty minutes and no breaks were taken unless necessary.

The stimuli were presented on a 15´ computer screen approximately one meter away from the subject at their eye level. The chair was comfortable and the room was dimly-lit. A radio play was played to keep the participants alert during the experiment. The participants were instructed to keep their gaze at the center of the screen and listen to the radio play or stay in their own thoughts if they preferred. They were also told that paying attention towards the pictures was not necessary.

EEG recordings and data analysis

The electroencephalogram (EEG) recordings were conducted with Brain Vision Recorder software at 14 scalp positions. The Ag-AgCl electrodes were placed on the electrode cap (Easy Cap) according to the international 10-20 system at Fz, F3, F4, Cz, C3, C4, Pz, P3, P4, P7, P8, Oz, O1, and O2. Electro-oculogram (EOG) was measured above the left eyebrow and on the right outer canthus to reveal the ocular artifacts. An average reference was used in the analysis but reference data was also gathered from both mastoids. Impedances were generally kept below 10 kΩ and the electrodes that had impedances larger than 20 kΩ were excluded from the analysis. The data was sampled with the rate of 1000 Hz. The online filter was 0.1-100 Hz and the offline filter was set to 0.1-30 Hz. Notch filter was used to erase the effects of main current.

Brain Vision Analyzer 2.0 software was used in the analysis of the EEG data. The data was segmented so that each segment started 100 ms before and ended 700 ms after the stimulus onset. 100 ms before the stimulus onset was used as a baseline. To prevent artifacts remaining in the data, signals larger than 50 μV or less than -50 μV were deleted with the time range of 200 ms in both directions. Ocular Correction was used in the analysis (Gratton-Coles method) for two subjects whose data was severely contaminated by blinks.

Four ERP responses were averaged for each participant: the response to (1) *neutral* expression before *happy* expression, (2) *happy* expression, (3) *neutral* expression before *fearful* expression, and (4) *fearful* expression. Six electrodes were chosen for statistical analysis because they had the most extensive responses: Fz, F3, F4, Cz, P7, and P8. Peak values for different responses were then identified from every participant separately by searching the most positive or negative peak value within given time interval. Responses of N170 and P230 were searched from the electrodes P7 and P8 and responses of P170 and N230 from the electrodes Fz, F3, F4, and Cz. The component N170 was searched within 120-200 ms while the component P230 was searched from 180-280 ms time interval. See Figure 3 for demonstration of the components at one frontal channel (F3) and one posterior channel (P8). The ERP components are referred to as N170 and P230, although both components were measured and analyzed as negative *and* positive, i.e. N/P170 and N/P230.

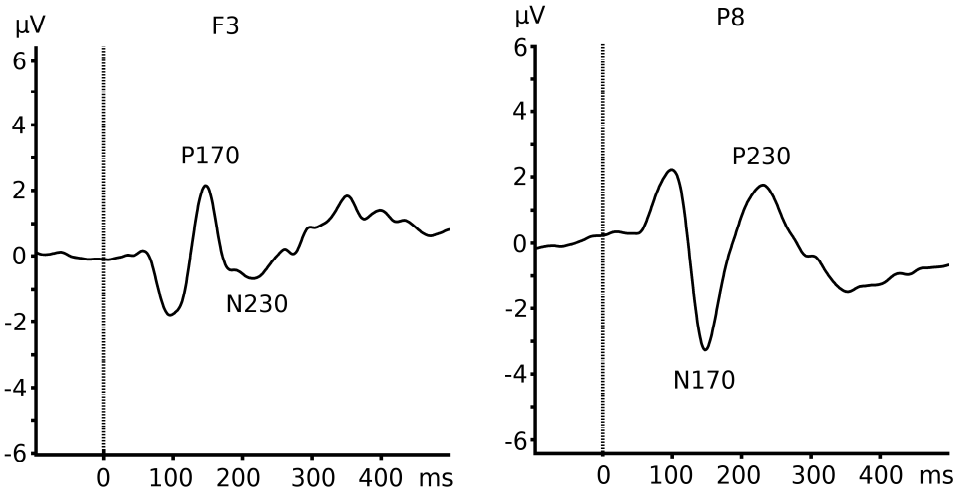

Figure 3. Demonstration of the components N170 and P230, and the two polarities that both of the components have. Figures are formed from the mean responses to facial stimuli in the non-depressed group: averages of the responses to *neutral before happy*, *happy*, *neutral before fearful*, and *fearful*. Left: the frontal electrode F3. Right: the posterior electrode P8.

Statistical analysis

The peak values from the responses to N170 and P230 were transported to SPSS (Statistical Package for Social Sciences 15.0 for Windows) as four variables: *neutral before happy*, *happy*, *neutral before fearful*, and *fearful*. The variables were tested with Multivariate analyses of variance (MANOVAs) in different groups and measurements. Whenever a significant interaction was found, separate MANOVAs and post hoc tests were performed for different groups to specify the nature of each interaction.

3-way MANOVAs were used to discover differences in the brain responses between depressed and non-depressed subjects. The within-subject factors were Stimulus type (standard, deviant), Expression (*happy*, *fearful*), and Electrode site (Fz, F3, F4, Cz, P7, and P8). Time of measurement (before intervention, after intervention, and follow-up) was an additional within-subject factor in the 4-way MANOVAs when responses of the benefitted and not-benefitted groups were compared after the intervention and the follow-up. The group was treated as a covariate called Group in the MANOVAs.

RESULTS

Component N170

The depressed and non-depressed groups did not differ significantly from each other in relation to the component N170. Nonetheless, when a different group division (benefitted/not-benefitted/non-depressed) was used, a Stimulus type × Electrode site × Group interaction ($F(5,56)=2.408$, $p=0.048$) was found. This interaction reflected the differences between the responses to the standard and deviant stimuli which were unequal between the electrode sites and the three groups.

Separate MANOVAs for the benefitted, not-benefitted, and non-depressed groups revealed that all three of them had a Stimulus type × Electrode site interaction ($F(5,14)=12.060$, $p<0.001$; $F(5,15)=4.661$, $p=0.009$; $F(5,18)=12.505$, $p<0.001$, respectively). That is to say, there were differences in the responses to the standard and deviant stimuli in each group. Expression × Stimulus type × Electrode site interaction was nearly significant both in the benefitted ($F(5,14)=2.772$, $p=0.061$) and in the not-benefitted group ($F(5,15)=2.862$, $p=0.052$).

To find out which of the electrodes recorded differential values to the standard and deviant stimuli, i.e. where significant difference ERPs could be detected, post hoc tests were conducted. Since the MANOVA indicated that Expression did not contribute to the interaction, two variables were averaged: 1) the standard (mean of *neutral before happy* and *neutral before fearful*) and 2) the deviant (mean of *happy* and *fearful*). The responses to the standard and deviant stimuli were then compared with paired samples *t*-tests within each group separately. The *t*-tests indicated that the non-depressed and the not-benefitted participants responded differently to the standard and deviant stimuli at every studied electrode site (Table 2). On the other hand, the responses to the standard and deviant stimuli did not differ at the Fz and F3 sites within the benefitted group.

Table 2
*The difference ERPs (deviant minus standard) of the component N170 in three groups. The amount of asterisks indicates the significance level of the difference between the responses to standard and deviant stimuli at each electrode site according to paired samples t-tests. *** p<0.001, ** p<0.01, * p<0.05.*

Electrode	Benefitted (n=19)	Not-benefitted (n=20)	Non-depressed (n=23)
P7	-0,527 µV *** *t*(19) = 5,615	-0,599 µV ** *t*(19) = 3,078	-0,512 µV *** *t*(22) = 4,357
P8	-0,403 µV ** *t*(19) = 3,402	-0,764 µV ** *t*(19) = 4,036	-0,708 µV *** *t*(22) = 5,614
Fz	0,248 µV *t*(19) = -1,801	0,708 µV ** *t*(19) = -4,124	0,663 µV *** *t*(22) = -5,391
F3	0,066 µV *t*(18) = -0,441	0,673 µV *** *t*(19) = -4,696	0,616 µV *** *t*(22) = -5,291
F4	0,336 µV * *t*(18) = -2,252	0,655 µV ** *t*(19) = -3,344	0,596 µV *** *t*(22) = -4,288
Cz	0,493 µV *** *t*(19) = -5,811	0,557 µV ** *t*(19) = -3,741	0,501 µV *** *t*(22) = -4,820

The difference ERPs were formed by subtracting the responses to the standard stimuli from those to the deviant stimuli (deviant minus standard; see Table 2). The difference ERPs at the electrodes P7 and P8 did not differ from each other in any group (benefitted: $t(19)=-0{,}936$, $p=0.361$; not-benefitted $t(19)=0{,}734$, $p=0.472$; non-depressed: $t(22)=1{,}096$, $p=0{,}285$). Independent samples *t*-tests were used to determine if the difference ERPs differed between groups. The *t*-tests showed that the difference ERPs were not similar between groups: the benefitted group differed at the Fz and F3 sites from the not-benefitted ($t(38)=-2.095$, $p=0.043$; $t(37)=-2.935$, $p=0.006$; respectively) and from the non-depressed group ($t(41)=-2.259$, $p=0.029$; $t(40)=-2.949$, $p=0.005$; respectively). The scalp potential maps of the difference ERPs and responses at the F3 electrode are shown in Figure 4. All in all, the *t*-tests revealed that the Stimulus type × Electrode site × Group (benefitted/not-benefitted/non-depressed) interaction was caused by the absence of significant difference ERPs in the benefitted group at two electrode sites, i.e. Fz and F3.

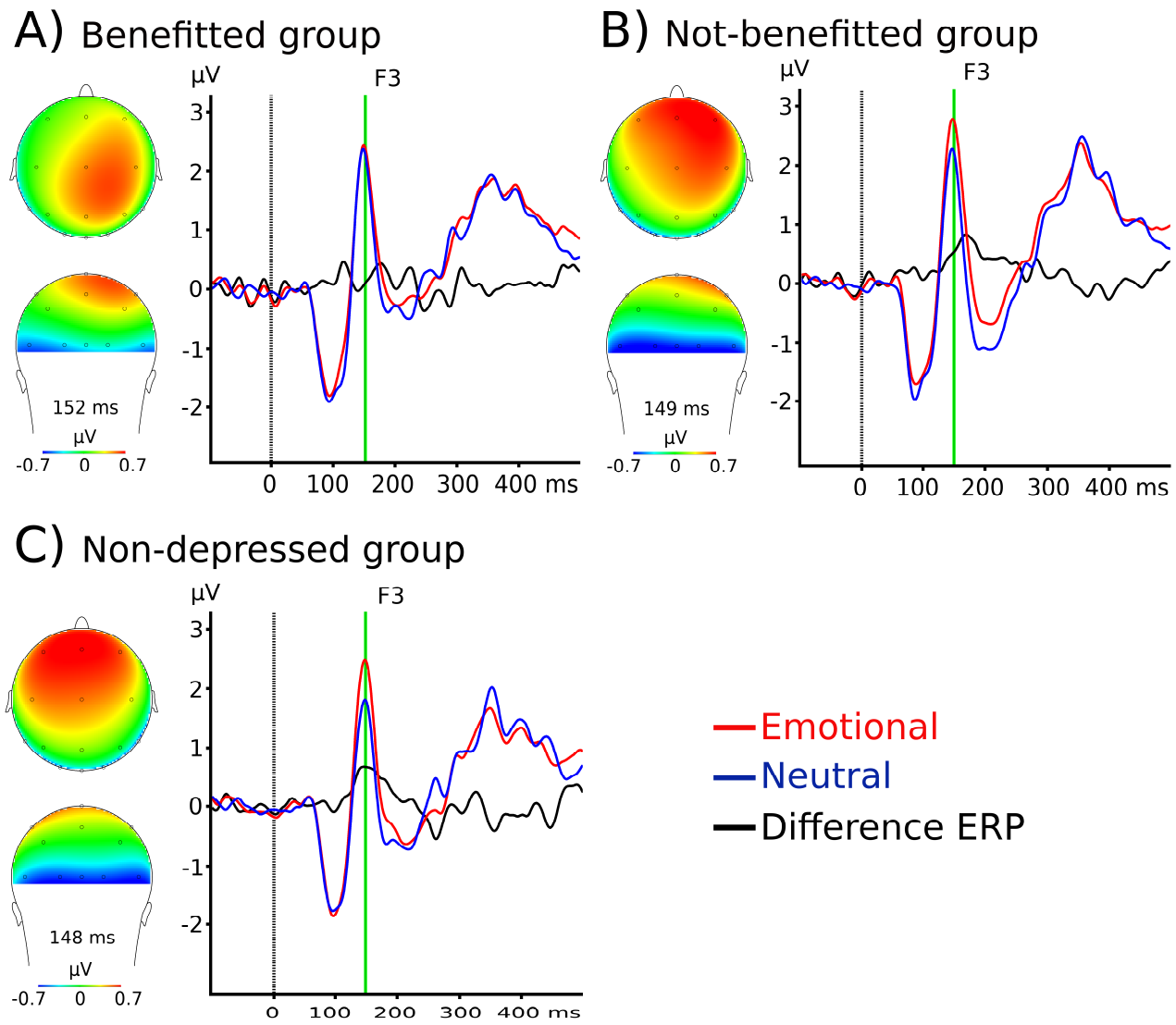

Figure 4. The scalp potential maps of the difference ERPs for the component N170 and the difference ERP curves for the F3 electrode: (A) the benefitted group, (B) the not-benefitted group, and (C) the non-depressed group. On the right is shown the average response to the standard stimuli (blue), the deviant stimuli (red), and the difference ERP between the standard and deviant simuli (black). On the left is shown the scalp potential map that represents a selected time point indicated with a vertical green line on the ERP curve illustration (right). The figures were formed by using averaged ERP waves instead of using the peak values of every participant individually as was done in the statistical analysis.

Because there were significant differences between the benefitted and not-benefitted groups already before the intervention (Table 2), it was tested whether the ERPs of either group had changed after the intervention or in the follow-up. The separate 4-way MANOVAs (Stimulus type × Expression × Electrode site × Measurement) for the two groups suggested that the responses remained the same in both groups in all three measurements.

Component P230

Next the component P230 was analyzed with MANOVAs to find out if there were differences between groups. At first the depressed and non-depressed groups were compared with a 3-way MANOVA (Stimulus type × Expression × Electrode site). An Electrode site × Group interaction ($F(5,56)=2.893$, $p=0.022$) was found. Because Expression and Stimulus type did not have an effect on the interaction, they were averaged into mean responses at each electrode site (mean of *neutral before happy*, *happy*, *neutral before fearful* and *fearful*). The interaction from the MANOVA suggested that the depressed and non-depressed participants responded differently to facial stimuli at some electrode sites. To find out which of the electrode sites differed between the depressed and the non-depressed groups, several post hoc tests were executed. Independent samples *t*-tests were unable to find differences between the depressed and non-depressed groups; therefore, paired samples *t*-tests were conducted to pinpoint the electrode sites that differed within either the depressed or the non-depressed groups.

In the component P230 the posterior electrode sites are positive and the frontal (and central Cz) electrode sites are negative; thus, the responses in these brain areas were not paired together in the *t*-tests. All the other possible combinations were tested as shown in Table 3. The paired samples *t*-tests indicated that the response was larger at the electrode P8 than at P7 in the non-depressed group (Table 3). Corresponding difference was not noticed in the depressed group. The non-depressed subjects had in general larger responses in the right hemisphere than in the left hemisphere while the depressed group had similar responses in the two hemispheres. The scalp potential maps for both groups are shown in Figure 5. In the depressed group, there was only one significant difference. The difference between the electrodes Fz and F4 was caused by a diminished response at the electrode F4 which is at the right hemisphere.

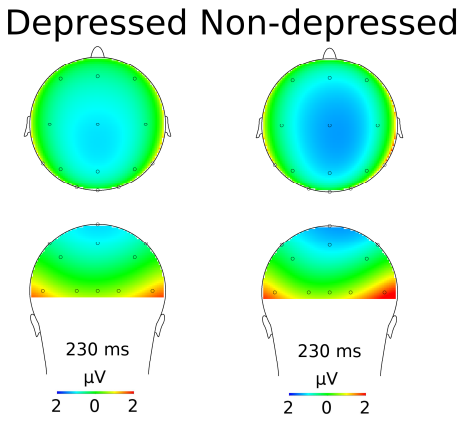

Figure 5. The responses of the component P230 to facial stimuli in the depressed (left) and the non-depressed (right) groups. The figures were formed by using averaged ERP waves instead of using the peak values of every participant individually as was done in the statistical analysis.

Table 3
The mean responses of the component P230 to facial stimuli in the depressed and the non-depressed groups. The values are the averages of four different variables: neutral before happy, happy, neutral before fearful, and fearful. Also shown are paired samples t-test comparisons of responses at seven electrode pairs for P230 within the depressed and the non-depressed groups separately. The significant p-values (<0.05) are marked with an asterisk.

Electrode pair	Depressed (n=39)		Non-depressed (n=23)	
	µV	*t*-tests	µV	*t*-tests
P7	1,8209	*t*(39)=-0.780	1,7298	*t*(22)=-3.931
P8	1,9675	*p*=0.440	2,2891	*p*=0,001*****
F3	-1,6312	*t*(38)=-1.770	-1,4803	*t*(22)=-3.553
Fz	-1,7297	*p*=0.085	-1,7544	*p*=0,002*****
F3	-1,6312	*t*(38)=-0.865	-1,4803	*t*(22)=4.136
F4	-1,5708	*p*=0.393	-1,7255	*p*<0.001*****
F3	-1,6312	*t*(38)=1.397	-1,4803	*t*(22)=2.519
Cz	-1,7936	*p*=0.171	-1,8553	*p*=0.020*****
F4	-1,5708	*t*(38)=-2.976	-1,7255	*t*(22)=-0.337
Fz	-1,7297	*p*=0.005*****	-1,7544	*p*=0.739
Cz	-1,7936	*t*(39)=0.659	-1,8553	*t*(22)=0.707
Fz	-1,7297	*p*=0.513	-1,7544	*p*=0.487
Cz	-1,7936	*t*(38)=1.890	-1,8553	*t*(22)=0.853
F4	-1,5708	*p*=0.066	-1,7255	*p*=0.403

4-way MANOVA was used to discover if the responses of the depressed group changed after the intervention or the follow-up. An Expression × Electrode site × Measurement interaction ($F(10,23)=2.643$, $p=0.026$) was found. Paired samples *t*-tests were used to find out what caused the interaction. It was tested if the response to either one of the expressions changed between different measurements at any electrode site. As the Stimulus type was not significant in the MANOVA, the variables were averaged into 1) *happy* (mean of *happy* and *neutral before happy*) and 2) *fearful* (mean of *fearful* and *neutral before fearful*).

The effect of the intervention was close to significant at the electrode P8 in relation to the happy expression ($t(39)=1.996$, $p=0.053$). The response diminished from 1.9479 µV to 1.7409 µV after the intervention. A significant increase was found in the happy expression at the same electrode between the measurement after intervention and the follow-up ($t(35)=-3.065$, $p=0.004$) where the response grew to 2.1276 µV. The response of the non-depressed group was on average 2.2509 µV.

Another difference close to significant in the depressed group was the difference between before and after intervention at the electrode site Fz ($t(39)=-1.869$, $p=0.069$), where the response became smaller, i.e. less negative. There were some suggestive results of changes in the fearful expression as well but none of the differences reached the significance level of 0.05. The *t*-tests indicated a nearly significant decrease at the electrode site P8 between before and after intervention ($t(39)=1.862$, $p=0.070$) and a nearly significant increase between after intervention and follow-up ($t(35)=-1.827$, $p=0.076$).

When using another group division, the 3-way MANOVA suggested that there were no differences in the component P230 between the benefitted, the not-benefitted and the non-depressed participants. Then, the effects of different measurements were studied. The 4-way MANOVA showed a Stimulus type × Electrode site × Measurement × Group (benefitted/not-benefitted) interaction ($F(10,22)=2.620$, $p=0.029$). This interaction was further analyzed with paired samples *t*-tests. Because Expression did not have an effect on the responses, averages of the responses to the standard and deviant stimuli were utilized. In the benefitted group there were no differences between the responses to standard and deviant stimuli at any electrode site or measurement. The responses at F3 differed almost significantly from each other in the measurement after the intervention ($t(18)=1.843$, $p=0.082$; Figure 6). On the other hand, in the not-benefitted group the responses to the standard and deviant stimuli differed significantly already before the intervention at electrode sites P8 ($t(19)=2.222$, $p=0.039$), Fz ($t(19)=-3.636$, $p=0.002$), F3 ($t(19)=-3.047$, $p=0.007$), and F4 ($t(19)=-2.791$, $p=0.012$; Figure 6). Similarly, the responses differed after the intervention at electrode sites P7 ($t(19)=2.653$, $p=0.016$) and Cz ($t(19)=-2.350$, $p=0.030$; Figure 6). In conclusion: the not-benefitted group had significant difference ERPs at four electrode sites (P8, Fz, F3, and F4) before the intervention and at two different sites (P7 and Cz) after the intervention.

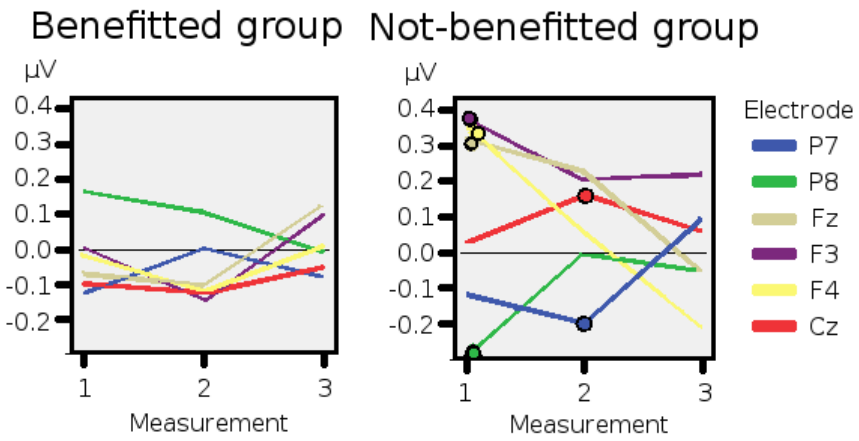

Figure 6. The difference ERPs (deviant minus standard) of the component P230 for three measurements: (1) before the intervention, (2) after the intervention, and (3) in the follow-up for the benefitted and not-benefitted groups. The significant difference ERPs are marked with a circle. The only significant change was observed between measurements 2 and 3 at the electrode P7 (blue) in the not-benefitted group.

Next, it was determined whether the difference ERPs changed after the intervention and/or the follow-up. The paired samples *t*-tests showed a significant change within the not-benefitted group at the electrode site P7 between the measurement after the intervention and the follow-up ($t(18)=-3.151$, $p=0.006$; Figures 6 and 7). Within the benefitted group statistically significant difference ERPs were not found (Figure 8). Therefore, the data indicated that (1) the responses to the component P230 differed between the depressed and non-depressed participants and (2) the not-benefitted group had some difference ERPs that were significant while the benefitted and the non-depressed groups had none.

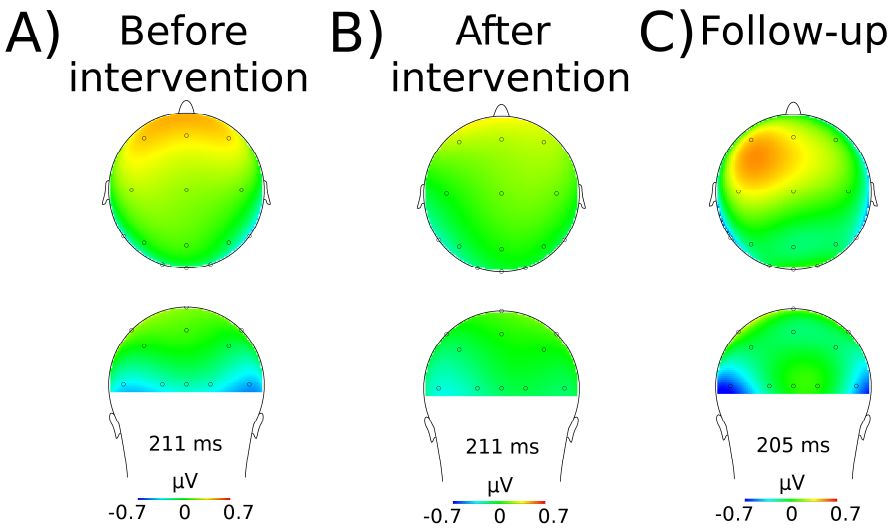

Figure 7. The scalp potential maps of the difference ERPs (deviant minus standard) in **the not-benefitted group** (A) before the intervention, (B) after the intervention, and (C) in the follow-up. A significant change was observed at the electrode P7 between measurements after the intervention and the follow-up. The figures were formed by using averaged ERP waves instead of using the peak values of every participant individually as was done in the statistical analysis.

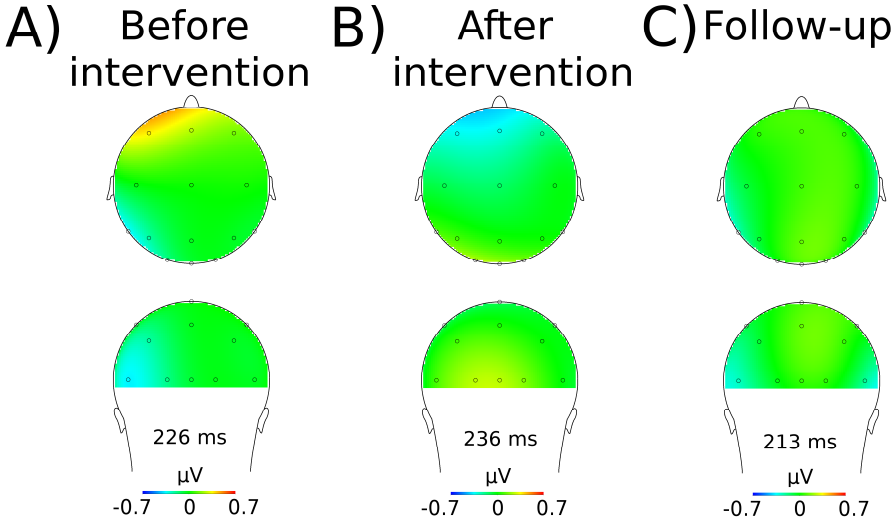

Figure 8. The scalp potential maps of the difference ERPs (deviant minus standard) in **the benefitted group** (A) before the intervention, (B) after the intervention, and (C) in the follow-up. There were no significant differences between the measurements. The figures were formed by using averaged ERP waves instead of using the peak values of every participant individually as was done in the statistical analysis.

DISCUSSION

The purpose of the study was to look into the ERPs elicited by *neutral*, *happy*, and *fearful* facial expressions in an oddball experiment with the depressed and the non-depressed groups. The brain's change detection mechanism (or MMN in the visual modality) was studied using the oddball paradigm. The effects of short psychological intervention were also determined. Two ERP components, N170 and P230, were analyzed in the thesis.

The preattentive processing of the human face differed between the depressed and the non-depressed groups. The non-depressed had more extensive responses for the component P230 in the right hemisphere than in the left. The depressed subjects lacked the hemispheric asymmetry that was seen with the non-depressed subjects. The right hemisphere is important for the processing of emotional information, and the results are in accordance with prior research involving the processing of emotional information in depression (for a review, see Heller et al., 1998). Accordingly, P230 distinguished effectively the depressed from the non-depressed. This might reflect impaired processing of emotional information in depression which is seen for example when recognizing facial expressions (Cooley & Nowicki, 1989; Persad & Polivy, 1993; Surguladze et al., 2005). However, the P230 responses between *happy* and *fearful* expressions did not differ either with the depressed or the non-depressed. Therefore, the results suggest that the depressed subjects have problems in the processing of emotional expressions in general and not only when processing some particular expressions.

Despite the similar responses for the expressions in the first measurement, the P230 responses to *happy* and *fearful* expressions differed between measurements at the right-posterior electrode site P8 with the depressed. The response decreased after the intervention and increased to the same level with the non-depressed in the follow-up for both *happy* and *fearful* expressions. Though, the only significant change was detected in response to the *happy* expression in the follow-up. The other changes were merely suggestive. However, the difference found between the measurements may not be related to the effects of intervention. Namely, one possible cause for the fluctuation is habituation to the stimuli. Familiarity with the procedure could have had a decreasing effect on the responses after the intervention. Furthermore, the seven month gap between the after intervention measurement and the follow-up might have been sufficiently long to cause the facial stimuli to seem novel for the brain. Because the non-depressed were measured only once, there is no way to

determine if the changes reported for the depressed occurred due to the repetition of measurements.

However, the increase of the P8 response to the happy expression in the depressed group might have been caused by the intervention. Depression affects especially the perception of positive emotions (Shestyuk et al., 2005; Suslow et al., 2001; Yoon et al., 2009), which might be reflected as attenuated responses to the *happy* expression during depression. Apparently, the intervention started a snowball effect and the average BDI score lowered from 25.9 to 15.1 after the intervention and further to 10.7 in the follow-up. This is a good result, because 10 is the cut-off point for depression in the BDI. Perhaps there is a level of improvement that has to be reached in order to detect significant ERP changes. On the other hand, the positive neurological impact of the intervention might appear after a short delay.

Furthermore, the results suggest that the positive polarity of the component N170, i.e. P170, could be a candidate for predicting successful outcome from short psychological intervention. Those participants who benefitted from the intervention differed from the not-benefitted and the non-depressed in regard to the difference ERPs between the *neutral* and the *emotional* stimuli. Before the intervention, the benefitted group lacked the difference ERPs at the electrodes Fz and F3, reflecting P170, which is also known as vertex positive potential (Jeffreys, 1983; Jeffreys, 1989). In other words, the benefitted processed *neutral* and *emotional* expressions similarly at the frontal brain areas while the not-benefitted and the non-depressed differentiated between the stimuli.

The difference between the ERPs to *neutral* and *emotional* expressions might be interpreted as visual mismatch negativity (vMMN). However, at the moment it is unclear whether N170 is only involved in the process of structural coding of the human face (Ashley et al., 2004; Eimer & Holmes, 2002; Eimer et al., 2003; Holmes et al., 2003; Santesso et al., 2008) or if it is modulated by distinct expressions (Batty & Taylor, 2003; Blau et al., 2007; Krombholz et al., 2007; Leppänen et al., 2007; Schupp et al., 2004). Therefore, the possibility, that any difference between the responses to the standard and the deviant stimuli (here referred to as *neutral* and *emotional*, respectively) might be elicited by the modulation of N170 and not by vMMN, should be considered. Even if the modulation of N170 or P170 does not affect the responses, it is difficult to confirm that the difference ERPs between *neutral* and *emotional* expressions are in fact a reflection of vMMN. Support for the interpretation is offered for example by Wei, Chan, and Luo (2002) who found a similar positive shift at 100-200 ms post-stimulus and by Czigler et al. (2002) who found vMMN at 120-160 ms.

However, it is puzzling, why the frontal vMMN-like ERPs were not detected for the benefitted group in the current study. There could be some fundamental differences between the benefitted and the not-benefitted groups that cause the dissimilar responses. For instance, the differential responses might be caused by variation in the speed of recovery after the intervention. The self-evaluated mood of the benefitted improved instantly after the intervention unlike the mood of the not-benefitted who reported improved mood only after the follow-up. Both the benefitted and the not-benefitted had similar average BDI scores before the intervention (24.9, 26.9, respectively). The score diminished more in the benefitted group than in the not-benefitted group immediately after the intervention (7.3, 23.0, respectively); however, the self-evaluated mood continued to improve in both groups in the follow-up (6.4, 14.7, respectively). The term "not-benefitted" might therefore be misleading since both of the groups benefitted from the intervention but the speed of the improvement varied. Because the two depressed groups did not differ in their BDI scoring before the intervention, the differences should not be caused by the severity of depression. On the other hand, it needs to be acknowledged that the participants can either exaggerate or downplay their symptoms or recovery in the self-evaluation. This might affect the division of participants into the benefitted and the not-benefitted groups and hence affect the results as a whole.

Nevertheless, the brain responses in the benefitted group did not differ between *neutral* and *emotional* expressions in any of the measurements. Therefore, it might be possible that the participants in the benefitted group were biologically different from the not-benefitted and the non-depressed groups. This abnormality could cause problems in preattentive processing which do not change after the intervention. The interpretation is in accordance with prior results suggesting that the differences in the brain function of the depressed are trait-like qualities that are not affected by the intervention (Bouhuys et al., 1996; Gotlib, Ranganath, & Rosenfeld, 1998). Alternatively, the follow-up might have been scheduled too early after the intervention to reveal changes in the ERPs.

The results of the vMMN-like ERPs are in conflict with the findings presented by Chang et al. (2010) as they reported diminished early vMMN (reflecting N170) posteriorly with the depressed. In accordance, diminished early vMMN responses were found in the benefitted group but only frontally. Chang et al. (2010) used schematic expressions while actual photographs of faces with different identities have been used here. This approach should exclude the possibility that the difference ERPs could merely reflect the physiological differences of the stimuli. In addition, the participants in the study by Chang et al. (2010) were all unmedicated, unlike some of the

participants in the present study. The medication might affect the results by changing the characteristics of MMN (Näätänen, 1992).

In addition to the early vMMN (reflecting N170) Chang et al. (2010) found a late vMMN (reflecting P250). Similarly, Zhao and Li (2006) found difference ERPs of P250 posteriorly which could reflect vMMN. In the present study, the difference ERPs that might correspond to the late vMMN mentioned by Chang et al. (2010) were found for the not-benefitted at 180-280ms (P230) frontally and right-posteriorly. Accordingly, the brain of the not-benefitted group differentiated between *neutral* and *emotional* expressions before the intervention. Curiously, there was a change after the intervention: significant difference ERPs were measured left-posteriorly and centrally. In the follow-up, ERPs resembling late vMMN were no longer present. The benefitted and the non-depressed did not have significant difference ERPs for P230 in any of the measurements.

The difference ERPs for P230 could predict unsuccessful outcome from short psychological intervention. Though, it is unclear why the location of the difference ERPs changed after the intervention and why they were missing in the follow-up. MMN should increase when the discrimination of the stimuli becomes more accurate (Näätänen, 1992). The depressed should have smaller vMMN compared to the non-depressed because their ability to recognize expressions is impaired (Persad & Polivy, 1993). Therefore, vMMN should increase if the intervention affects the ability to distinguish facial expressions but this was not observed in the present study.

The depressed and the non-depressed did not differ in the responses to the *happy* and *fearful* expressions. The depressed are impaired especially in recognizing low intensities of facial expressions (Csukly et al., 2009; Joormann & Gotlib, 2006; Yoon et al., 2009). The expressions used in this study were of high intensity. The problems in recognizing facial expressions could become apparent only with low intensities of expressions. To confirm if there is a correlation between depression and the ERPs elicited by facial expressions, further research with different intensities of expressions is required.

There are numerous factors that could affect the ERP data that need to be reviewed when interpreting the results of the study. First of all, with more participants both in the depressed and non-depressed groups, there might have been more statistically significant results. Some of the participants may have dropped out from the follow-up selectively. For example, those depressed subjects that benefitted least from the intervention may have been more prone to discontinue. Another concern is the wide age range of the subjects (28-61 years) which increases variation. On the other hand, because of the wide age range, the results can be generalized to a wider population. Additionally, the severity of symptoms varied notably and most of the participants did not have a

formal MDD diagnosis. The backgrounds of the subjects also varied in the age of onset for depression, duration of symptoms, and the existence of concurrent mental problems, e.g. anxiety. Furthermore, the results of this study cannot be generalized to represent all depressed persons because these particular subjects were volunteers. Due to the voluntariness the participants might be more motivated and eager to change than depressed people in general. Because there were only six men in the study the results cannot be generalized to depict depression for men. It is also noteworthy, that there were more medicated participants in the benefitted (7 participants) than in the not-benefitted (4 participants) group. Thus, medication likely accelerates the recovery from depression and may affect the brain response data as well.

The division of participants into the benefitted and not-benefitted from the original research groups was unequal. In the benefitted, there were considerably more participants from the experimental group (15 participants) than from the waiting list group (5 participants). On the contrary, in the not-benefitted group there were more participants from the waiting list (12 participants) than from the experimental group (8 participants). Possibly the participants in the waiting list group lost their motivation before the beginning of the intervention which might explain their fewer number in the benefitted group. Therefore, the effects reported for the benefitted and the not-benefitted groups might also be caused by the timing of the intervention. In addition, the BDI cut-off point had an effect on forming the benefitted and not-benefitted groups which influences the results.

Information gained with brain imaging methods is highly objective as the results are directly recorded from the research subject's brain and are not acquired by indirect methods such as questionnaires. The knowledge of the brain functioning in different clinical groups can have beneficial applications for diagnosing, preventing, and treating mental disorders in the future. The results from this thesis are in accordance with the Beck's cognitive triad (Beck, 2008) since the negative interpretation of life experiences could be related to the abnormal preattentive processing of emotions. However, the cause-effect relationship between the abnormalities in brain function and depressive mood is not clear. If the abnormalities exist already before the onset of the depressive symptoms, individuals more prone to depression could be screened out. Further research with more participants is needed to define the differences between the depressed and non-depressed more precisely. To conclude, the study offers new insight into depression; however, the limitations of the experimental design, methods, and the amount of participants need to be taken into account.

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