

NEGATIVE VERSUS POSITIVE IMPLICIT FACIAL EXPRESSION PROCESSING IN DEPRESSIVE MOOD. THE LINK BETWEEN THE SEVERITY OF DEPRESSION AND AUTOMATICAL FACIAL EXPRESSION PROCESSING.

R. Cserjési^{a,b*}, N. Vermeulen^{b,c}, L. Lénárda, and O. Luminet^{b,c}

^a *Institute of Physiology and Neurophysiology Research Group of the HAS, Pécs University Medical School, Pécs, Hungary,*

^b *Research Unit for Emotion, Cognition and Health, Faculty of Psychology, Université catholique de Louvain, Louvain-la-Neuve, Belgium,*

^c *Belgian National Fund for Scientific Research (F.N.R.S.-FRS)*

ABSTRACT

Facial expressions of emotions are innate and universal across the cultures. The neural mechanisms underlying the expression and perception of facial emotion are “hard-wired” such as happy expressions innately appetitive (i.e. positive) stimuli, while angry or sad expressions innately aversive (i.e. negative) for humans. Accurate recognition of facial emotions is necessary for appropriate human social interaction. The implicit or automatic emotional priming effect can take place outside of the awareness and influence the behaviour differently than explicit presentation of emotional stimuli. Previous studies confirmed that different neural substrates responsible for the processing of implicit and explicit facial emotional stimuli. It was suggested that mood disorders are characterised by the higher sensitivity to negative stimuli and automatic thoughts. Automatic thoughts are cognitive products and they occur rapidly and the individual has limited control over them. In our studies we investigated the implicit processing of facial emotion stimuli in different psychopathology where commonly negative mood was reported on the explicit level. We used the affective priming task in order to compare implicit treatment of different emotions (sad, angry, and happy) represented on a schematic face. In the presentation we will discuss the differences between implicit emotional processing and explicit self report on emotions.

* Corresponding author: Renata Cserjesi, PhD

Clinical and Developmental Neuropsychology, University of Groningen
Grote Kruisstraat 2/1, 9712 TS, Groningen, The Netherlands
Email: rcserjesi@hotmail.com

Keywords: implicit emotion, depression, facial expression

1. INTRODUCTION

Facial expressions can carry important information about individuals' own and the others' emotional state. The neural mechanisms underlying the expression and perception of facial emotion are "hard-wired" such as happy expressions innately positive stimuli, while angry or sad expressions innately negative for humans (Morris et al. 1998). Therefore, the accurate recognition of and the appropriate responses to these stimuli are critical for successful interpersonal function in social environment and for emotion regulation (Surguladze et al. 2005). Empirical researches demonstrated that emotion processing may occur automatically or purposely and further can be conscious or unconscious (Richards and Gross, 2000). Besides explicit (conscious) processing, facial expressions are also processed rapidly and automatically (unconscious) in the absent of explicit awareness. Implicit processing is suggested to operate subcortically, while explicit processes include cortical areas that are more accessible through conscious attention (Scheuerecker et al. 2007). A relatively long history of research has shown that mood disorders are associated with abnormalities in both explicit and implicit processing of facial emotional stimuli (Leppanen, 2006). Depression is a common emotional illness that varies widely in its intensity. Typical symptoms of depression of mild to moderate severity include depressed mood, lack of energy, sleep problems, anxiety, appetite disturbance, difficulty concentrating, and poor stress tolerance. More severe depression includes symptoms such as slowed speech, slowed or agitated responses, impaired memory and concentration, poor sleep, significant weight change, intense feelings of guilt, recurrent thoughts of suicide, and lack of interest in pleasurable activities (APA, 1994). Depression is more common among women than men (Rubinow et al. 1998). Depressive mood often co-exists with other mental illnesses such as anxiety, post-traumatic stress disorders, obsessive-compulsive disorder, eating disorder and addictive disorders (Regier et al. 1998). Intersection of depression and other illnesses differ for every person and situation, however as a common primary feature of depressive mood in this illnesses is a preoccupation with negative ideation (Drevets et al. 2008). Many behavioural studies have documented an attention bias toward negative emotional cues (e.g. sad faces), and an enhanced memory for negative emotional material in depressive mood (Mogg et al., 1995, Yoon et al., 2009). The recent development of neuroimaging techniques that allows in vivo examination of the anatomical, physiological and neurochemical characteristics of mood disorders, revealed significant advances toward the understanding of the psychopathology background of depression, however these results are rather ambiguous (Kan et al., 2004). It is still not clear that self-reported negative ideations whether are linked to more sensitivity to negative emotions or they are linked to less sensitivity to positive emotions. The role of hypersensitivity to mood congruent negative emotions, namely sadness has been confirmed by neuroimaging studies (Keedwell et al., 2005). Behavioural study showed that explicit recognition problem of happy facial expressions is linked to the severity of depression (Csukly et al. 2009). A few fMRI studies have been carried out on implicit emotional processing in depressive mood (Sheline et al. 2001, Fu et al. 2007, Fu et al. 2008). Confirming the hypersensitivity to negative emotion in depression, elevated amygdala activity has been found in patients with major depression when they were exposed to negative stimuli presented outside of conscious awareness (Sheline et al. 2001, Fu et al. 2008).

Until now most of the studies aimed to explore the explicit facial emotion processing differences between healthy control and patients with severe depression (Kan et al. 2004, Surguladze et al. 2005, Keedwell et al., 2005, Fales et al. 2008). These studies were focusing less on the different intensity of

depressive mood (from mild to severe) in the patients. On the other hand previous studies paid not less attention on the implicit processing of facial emotions. Therefore, our aim was to investigate whether there are associations between the severity of depression and the emotional facial expression processing on the implicit level.

2. METHODS

2.1. Participants

The 116 women between 18-60 years old participated in our study. The patients with depression were recruited from several Belgian clinics. Healthy volunteers were recruited through friends or academic staff. Subjects with a neurological disease, substance dependence, organic impairments, psychotic disorders (e.g. schizophrenia), subnormal intelligence, or electroconvulsive therapy were excluded. Table 1 shows the personal and demographical data of all participants. Based on the subcategories of the Beck Depression Inventory (no, mild, moderate, and severe depression), we have divided the participants into four groups (see table1).

There were significant differences in groups for depression scores ($F(3, 112)=73.760, p<0.001$), state ($F(3,112)=41.310, p<0.001$) and trait ($F(3,112)=38.276, p<0.001$) anxiety scores. There was no significant difference for age in the first three groups. Only the fourth “severely depressed” group was significantly younger than the other groups ($F(3,112)=3.113, p=0.030$). Two patients from the severely depressed group were under regular antidepressant medication. All participants were born in Belgium, and their native language was French. All participating subjects had normal or corrected-to-normal vision. Examinations were performed in accordance with institutional and international (Declaration in Helsinki, 1964; European Union Council Directive 86/609/EEC) ethical standards. Prior to their inclusion into the study, participants gave their informal consent and written permission of participants was documented.

Table 1 Demographical and personal data of the participants

	No depression BDI score 0-9 N= 47	Mild depression BDI score 10-18 N=28	Moderate depression BDI score 19-29 N=23	Severe depression BDI score 30-49 N=18
BDI	4.65 (± 2.67) ^a	13.88 (± 2.54) ^b	24.19 (± 2.99) ^c	36.61 (± 6.27) ^d
STAI state	30.51 (± 7.81) ^a	41.47 (± 10.55) ^b	48.47 (± 11.09) ^b	60.00 (± 7.95) ^c
STAI trait	39.63 (± 7.91) ^a	47.44 (± 6.95) ^b	50.74 (± 8.34) ^b	65.60 (± 9.14) ^c
Age	39.23 (± 16.95)	38.89 (± 14.26)	38.52 (± 15.13)	25.30 (± 8.53) [*]

(a, b, c, d) $p<0.01$, (*) $p<0.05$, BDI= Beck Depression Inventory, STAI = State-trait Anxiety

2.2. Materials

2.2.1. Beck Depression Inventory II

Beck Depression Inventory II (BDI-II) is 21 item self-report inventory measuring the characteristic attitudes and symptoms of depression (Beck et al., 1996). Each item rated on a 4 point scale (from 0 “I do not feel sad” to 3 “I am so sad or unhappy that I can’t stand it”). The score is calculated by summing each item (range 0–63). Individuals scoring from 10 – 18 are classified as having mild to moderate depression, those scoring between 19 - 29 are classified as having moderate to severe depression, and severe depression is evidenced by scores ranging from 30 - 63. We used the validated French version of the BDI-II (Beck et al. 1998).

2.2.2. State-Trait Anxiety Inventory

State-Trait Anxiety Inventory (STAI, Spielberger, 1983) is a self-rating measure of anxiety and it consists of two parts: the State (describing the actual situation) and the Trait (general measure of anxiety). Participants indicated their degree of approval on 20 items for each form of anxiety (e.g. “I am satisfied”, “I have thoughts that disturb me”) on a 4 –point Likert scale ranging from 1 “No” to 4 “Yes”. Possible scores range from 20 to 80 for each form. We used the validated French language version of the STAI questionnaire (Bruchon-Schweitzer, Paulhan, 1993).

2.3. Affective priming

The affective priming paradigm is used to assess implicit cognition, leaning on the procedure of early attention allocation and automatic reactions when affective information is presented (Fazio et al., 1986). The principle of the task is to measure whether the preliminary presented emotional stimuli (prime) modify the processing speed and accuracy of subsequent target evaluation on positive or negative valences. When the affective valence of the prime is similar to that of the target stimuli (positive-positive; negative-negative) a congruence or facilitation effect occurs, which leads to faster and more accurate responses than incongruent prime-target combination (positive-negative; negative-positive). The difference in reaction time (RT) or error percentage (accuracy) between congruent and incongruent trials is called the priming effect. Affective priming task was used to examine the main hypothesis about automatic facial emotion processing.

Primes: Schematic faces representing happiness, sadness and anger were used as the prime stimuli. A happy face was considered as positive prime, while the sad and the angry faces as negative primes. The neutral schematic face was used as a baseline to measure the differences in emotional information processing.

Targets: Twelve positive (e.g., joyful) and 12 negative (e.g., sorrowful) adjectives were selected as target stimuli based on the previous study (Vermeulen et al. 2006). No differences were found in the frequency of usage in the language or familiarity measured on 7 -points scale from 0 to 6 ($F [1,11] < 1$, N s positive: $M = 4.93$, $S.D. = 0.52$; negative $M = 4.80$, $S.D. = 0.40$), and for the length of the target

words ($F [1,11] < 1$, Ns., positive: $M = 7.16$, $S.D. = 1.83$; negative: $M = 6.83$, $S.D. = 0.98$) There was a significant difference between positive and negative targets for valence ($F [1,11] = 157.63$, $p < 0.001$; positive $M = 2.17$, $S.D. = 0.31$; negative: $M = -1.91$, $S.D. = 0.26$).

2.4. Procedure

Participants were tested individually both on the questionnaires and the Affective priming task. First, they were asked to fill up the questionnaires and then they performed the task. During the Affective priming task the participants were invited to evaluate and categorize each target word according to its value (positive vs. negative) as fast and accurately as possible by pressing the left (L) or right (S) keys of an AZERTY Belgian French type of keyboard. Each target word was anticipated by a very shortly displayed schematic face prime stimulus. The schematic faces were presented in a random order on the computer display. Participants were asked to perform the task as quickly and as accurately as possible. The task started with 12 training trials in order to become familiar with the task. The experimental part was divided into three blocks of four positive and four negative adjective targets. Each of the three blocks consisted of 80 trials, resulting in a total of 240 trials. The task procedure and the timing of the trials were arranged after a previous study (Vermeulen et al. 2006). Each trial sequentially included a fixation point, the prime lasting for 100 ms (the stimuli onset asynchrony [SOA] was 100 ms) directly replaced by the target word appearing for 500 ms.

Priming effect was calculated in the following way: Congruent prime-target combinations (mean RT [negative prime/negative target]; RT [positive prime/positive target]) were subtracted from the incongruent prime-target combinations (mean RT [negative prime/ positive target]; RT [positive prime/negative target]). The interactions between the prime stimuli (happy, angry, and sad) x targets (positive versus negative) and the overall priming effect were analyzed by applying ANOVA repeated measure within group test. Group differences were analyzed with ANOVA repeated measures between subject methods, t-test and Pearson's correlation.

3. RESULTS

RT associated with correct responses either too fast (300 ms) or too slow (1000 ms) or erroneous ones were excluded from analysis (Ratcliff, 1993). The Affective Priming effects were analyzed by examining the interaction between the valence of target and prime stimuli in the different groups. Our analysis focuses on RT (speed), as no significant differences were found on response accuracy (number of errors) for the groups. Both the prime x target interaction ($F (2, 112) = 5.151$, $p = 0.05$) and the overall priming effect ($F (1, 112) = 3.456$, $p = 0.035$ figure 1) were significant. It means that the different facial expression primes influenced the participants performed on the categorization task. While there was no significant difference for the groups ($F (3,110) = 0.988$ $p = 0.401$).

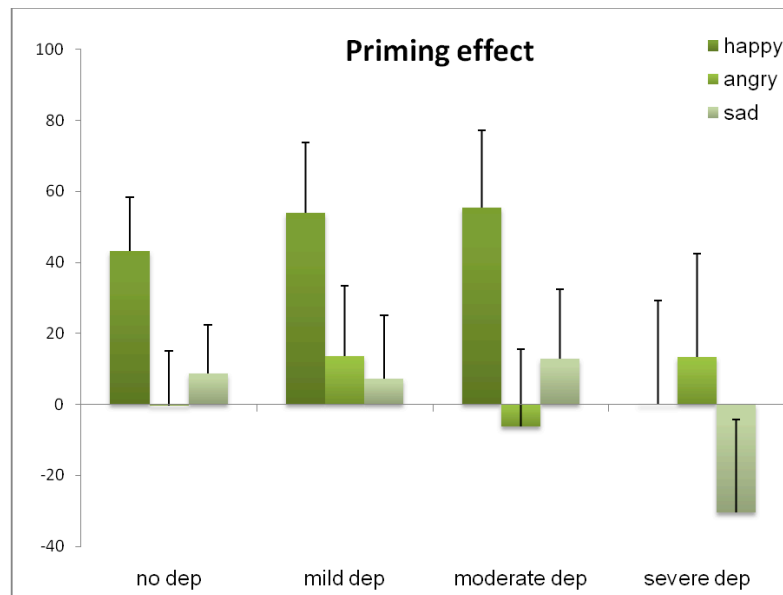


Figure 1 Priming effect calculated from the incongruent- congruent prime target combination for the happy, angry and sad facial expression. No dep=individuals with no depression, mild dep=with from mild to moderate depression, moderate dep=from moderate to severe depression, severe dep= with severe depression

Using paired sample test we have examined the difference between the congruent and the incongruent prime/target combination in each group. We have found significant difference for the happy face in the not depressed ($t[46]=3.541$, $p<0.001$), mildly depressed ($t[27]=2.430$, $p=0.022$) and moderately depressed groups ($t[22]=1.995$, $p=0.048$), but not in patients with severe depression ($t[17]=0.010$, $p=0.992$). Partial Pearson's correlation controlling for the groups have confirmed that there was no relationship between State-Trait anxiety and implicit affect processing (priming effect).

4. DISCUSSION

Difficulties in facial emotion processing may underlie the interpersonal difficulties frequently reported in people with depression. Previous reports have emphasized in explicit attention biases toward sad and away from happy stimuli. In the present study we aimed to examine the negative versus positive facial expression processing in the absent of conscious attention control in healthy participants and patients with different intensity of depression (from mild to severe). Our findings showed that patients with severe depression did not process positive face (happy) implicitly compared to individuals with no depression and patients with mild and moderate depression. However, no difference has been found for negative facial emotion expression (sad and angry). These findings are in line with the previous researches, which showed that impairments in the neural processing of happy

facial expressions in depression were important marker in the pathology (Fu et al., 2007, Csukly et al., 2009). These findings are also consistent with previous studies using full-intensity facial expressions that reported that participants with depression tended to interpret happy faces as neutral (Suslow, et al., 2001).

On the other hand our results showed that patients with severe depression differed from patients with mild or moderate depression. Uniquely, patients with severe depression were not sensitive to happy face compared to the control and the other patient groups. This result suggests that there is a qualitative change, a cut-off point between moderate and severe depression based on positive facial emotion processing. This selective impairment may underlie one of the hallmark features of severe depression: anhedonia. However, we have found no significant between-group differences using the ANOVA test, which may due to the enhanced sensitivity of the Affective priming test toward individual differences, or/and the small size of the severely depressed group.

In conclusion, our findings contribute to the growing literature indicating that only severe depression, but not mild or moderate depression is characterized by impaired implicit processing of positive affect, perhaps even more so than by biases in the processing of negative affect. Our results are consistent with findings of previous research demonstrating that depression is related to biases in the processing of positive stimuli (Yoon et al. 2009). It may also have important consequence for the understanding of interpersonal difficulties that are associated with severe mood disorders (Gotlib and Hammen, 1992).

ACKNOWLEDGEMENT

We would like to thank the participants for their time and their availability. We thank also the health care team for their help in patient recruitment. Special thank to Anne-Sophie Poncelet for her help in the patient recruitment and data collection.

REFERENCES

1. Morris J.S., Friston K.J., Buechel C., Frith C.D., Young A.W., Calder A.J., et al Aneuromodulatory role for the human amygdala in processing emotional facial expressions. *Brain*, Vol. 121, pp. 47–57, 1998. Mogg K., Bradley B.P., and Williams R. Attentional bias in anxiety and depression: The role of awareness. *British Journal of Clinical Psychology*, Vol. 34, No.1, pp. 17–36, 1995.
2. Surguladze S., Brammer M.J., Keedwell P.A., Giampietro V., Young A.W., Travis M.J., Williams S.C. and Philips M.L. A differential pattern of neutral response toward sad versus happy facial expressions in major depressive disorder. *Biological Psychiatry*, Vol. 57, No. 1, pp. 201-209, 2005.
3. Richards J.M. and Gross J.J. Emotion regulation and memory: The cognitive costs of keeping one's cool. *Journal of Personality and Social Psychology*, Vol. 79, pp. 410-424.
4. Scheuerecker J., Frodl T., Koutsouleris N., Zetzsche T., Wiesmann M., Kleemann A.M., Brückmann H., Schmitt G., Möller H.J., and Meisenzahl E.M. Cerebral Differences in Explicit and Implicit Emotional Processing –An fMRI Study. *Neuropsychobiology*, Vol. 56, pp. 32–39, 2007.
5. Leppänen J.M. Emotional information processing in mood disorders: a review of behavioural and neuroimaging findings. *Curr Opin Psychiatry*, Vol. 19, No. 1, pp. 34-39, 2006.
6. APA Diagnostic and statistical manual of mental disorders (DSM-IV). APA Press, Washington, DC, 1994.

7. Rubinow D.R., Schmidt P.J. and Roca C.A. Estrogen-serotonin interactions: implications for affective regulation. *Biological Psychiatry*; Vol.44, No.9, pp.839-850, 1998.
8. Regier D.A., Rae D.S., Narrow W.E., Kessler C.T., Schatzberg A.F. Prevalence of anxiety disorders and their comorbidity with mood and addictive disorders. *British Journal of Psychiatry*, Vol. 173 No.34, pp. 24-28, 1998.
9. Drevets W.C., Price J.L. and Furey M.L. Brain structural and functional abnormalities in mood disorders: implications for neurocircuitry models of depression. *Brain Struct Funct*, Vol. 213, pp. 93–118, 2008.
10. Mogg K., Bradley B.P. and Williams R. Attentional bias in anxiety and depression: the role of awareness. *British Journal of Clinical Psychology*, Vol. 34, No. 1, pp. 17-36, 1995.
11. Yoon K.L., Joormann J., and Gotlib I.H. Judging the intensity of facial expressions of emotion: depression-related biases in the processing of positive affect. *Journal of Abnormal Psychology*, Vol. 118, No 1, pp. 223-228, 2009.
12. Kan Y., Mimura M., Kamijima K., and Kawamura M. Recognition of emotion from moving facial and prosodic stimuli in depressed patients. *J Neurol Neurosurg Psychiatry*, Vol. 75, pp.1667–1671, 2004.
13. Keedwell P.A., Andrew C., Williams S.C., Brammer M.J. and Philips M.L. A double dissociation of ventral prefrontal cortical responses to sad and happy stimuli in depressed and healthy individuals. *Biological Psychiatry*, Vol. 58, No. 6, pp. 495-503, 2005.
14. Csukly G., Czobor P., Unoka Z., Takacs B. And Simon L. Associations between symptom severity and emotion recognition in depressed patients. *Psychiatria Hungarica*, Vol. 24, No. 1, pp. 68-73, 2009.
15. Sheline YI, et al. Increased amygdala response to masked emotional faces in depressed subjects resolves with antidepressant treatment: An fMRI study. *Biological Psychiatry*, Vol. 50, pp. 651–658, 2001.
16. Fu C. H.Y., Williams S.C.R., Brammer M. J., Suckling J., Kim J., Cleare A. J., Walsh N.D., Mitterschiffthaler M.T., Andrew C.M., Pich E.M., and Bullmore E.T. Neural Responses to Happy Facial Expressions in Major Depression Following Antidepressant Treatment. *American Journal of Psychiatry*, Vol. 164, pp. 599-607, 2007.
17. Fu C.H.Y., Williams S.C., Cleare A.J., Scott J., Mitterschiffthaler M.T., Walsh N.D., Donaldson C., Suckling J., Andrew C., Steiner H., Murray R.M. Neural responses to sad facial expressions in major depression following cognitive behavioral therapy. *Biol Psychiatry*, Vol. 64, No. 6, pp. 505-12, 2008
18. Fales C.L., Barch D.M., Rundle M.M., Mintun M., Snyder A.S., Cohen J.D., Mathews J., and Sheline Y.I. Altered emotional interference processing in affective and cognitive-control brain circuitry in major depression. *Biol Psychiatry*, Vol.63, No. 4, pp. 377–384, 2008.
19. Beck, A.T., Steer, R.A., Ball, R., and Ranieri, W.F. Comparison of Beck Depression Inventories - IA and -II in Psychiatric Outpatients. *Journal of Personality Assessment*, Vol. 67, No. 3, pp. 588-597, 1996.
20. Beck AT, Steer RA, Brown GK Manual of the Beck Depression Inventory, 2nd edition. (Manuel de l'inventaire de dépression de Beck, 2eme édition). The Psychological Corporation, Toronto, Ontario, Canada, 1998.
21. Spielberger C.D. Manual for the State-Trait Anxiety Inventory (STAI). Consulting Psychologists Press, PaloAlto, CA, 1983.
22. Bruchon-Schweitzer, M.L., and Paulhan, I. STAI-Y manual of CD Spielberger, French version. ECPA, Paris, 1993.
23. Fazio R.H., Sanbonmatsu D.M., Powell M.C. and Kardes, F.R.. On the automatic activation of attitudes. *Journal of Personality and Social Psychology*, Vol. 50, pp. 229–238, 1986.

24. Vermeulen N., Luminet O., and Corneille O. Alexithymia and the automatic processing of affective information. Evidence from an affective priming paradigm. *Cognition & Emotion*, Vol. 20, pp. 64-91, 2006.
25. Ratcliff, R. Methods for dealing with reaction time outliers. *Psychological Bulletin*, Vol.114, pp. 510-532, 1993.
26. Suslow T., Junghanns K., and Arolt V. Detection of facial expressions of emotions in depression. *Perceptual & Motor Skills*, Vol. 92, pp. 857-868, 2001.
27. Gotlib I. H., and Hammen C. L. Psychological aspects of depression: Toward a cognitive-interpersonal integration. Oxford, England, Wiley, 1992.