Mood effects on the ERP processing of emotional intensity in faces: A P3 investigation with depressed students

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Abstract

This study examined mood-relevant emotion processing in depression using event-related potentials (ERPs). Cognition in depression has been characterized as having attention and memory biases for negative (or mood relevant) information and away from positive (or mood incongruent) information, however, the time course and specificity of this processing during the perception of emotional expressions is not well known. In order to index specific information processing stages a visual oddball task with facial stimuli was utilized, with neutral expressions as the standard and targets varying on valence (happy and fear) and intensity (40%, 70% or 100% emotive) dimensions. Participants were 36 university students grouped according to their BDI-II scores; 18 non-depressed controls (BDI-II $\leq$ eight; $M=4.1$) and 18 depressed (BDI-II ≥ 15, $M=25.5$), age- and sex-matched between groups. Mixed model ANOVAs revealed interactions between control and depressed participants with happy and fearful stimuli showing significantly reduced P3 amplitudes and P3 latencies for happy faces as well as significantly delayed P3 latencies specifically for 40% happy faces in depressed participants. These findings are interpreted as evidence for a diminished cognitive processing ability during emotion discrimination for low-intensity mood incongruous (happy) faces in depression.
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1. Introduction

Cognitive processing in depression is characterized with negative schemata that distort one's view of the world, the self, and the future (Beck, 1976) along with a concurrent information processing bias that affects attention to and memory for negative information (Gotlib and Krasnoperova, 1998). These schemata affect information processing by increasing the salience of negative events and even diminishing the salience of positive events, as experiments using attention and memory tasks have shown (Gotlib and Neubauer, 2000). Many studies use emotional stimuli but little is known about how affective characteristics of stimuli are influenced by regional brain activity during perceptual

processes and how these may contribute to a mood-related attention bias in depression. An understanding of the detailed time course of cognitive processing during the perception of emotional stimuli could help delineate which specific cognitive processes are affected by mood-related biases.

Investigations into information processing time courses have frequently utilized event-related potential (ERP) measurements to examine discrete stimulus processing on a temporal scale of milliseconds. Using stimuli that differ from each other elicits a late cognitive waveform component, the P3, which represents stimulus evaluation, attentional allocation and context updating (Polich and Comerchero, 2003; Polich and Kok, 1995; Coles et al., 1990). ERPs provide a temporally accurate measurement to investigate the time course and specificity of emotionally biased perceptual processing in depression, yet few studies have utilized ERP measurements for this purpose.

Depressed patients have shown reduced P3 (Roschke and Wagner, 2003) and N2 amplitudes (Ogura et al., 1993) in

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auditory oddball paradigms, indicating specific deficits in neuronal resource allocation and mismatch detection. Pierson et al. (1996) found reduced amplitudes and delayed latencies at P3a and P3b in blunted-affect depressed patients, yet faster latencies and larger amplitudes at P3b for anxious-agitated patients between control and depressive subtype groups in an active choice task. These studies have provided evidence for the phenomenon of differential cognitive processing in depression, also indicating how subtypes can influence perceptual processes.

However, in order to capture emotion-specific cognitive biases, affective stimuli are often utilized. Depressed patients have shown lower P3 amplitudes for negatively valenced words (Blackburn et al., 1990) and also higher frontal amplitudes in the 250–500 ms temporal range for negatively valenced words (Dietrich et al., 2000), both interpreted as indexes of a negativity bias. A similar negativity bias was evidenced with depressed patients displaying lower right parietal P3 amplitudes to pictures of diseased dermatological faces (Kayser et al., 2000), supporting Heller's (1990, 1993) theory of a hypoactive right parietotemporal cortex in depression. Although these studies provide some electrophysiological evidence for negativity biases in depression, region-specific effects and affective dimensions need to be further dissociated for a thorough understanding of this phenomenon.

Deldin et al. (2000) found a reduced N2 amplitude in the right parietal cortex in depressed participants when viewing already seen happy faces (but not positive words), also supporting the theory of a hypoactive parietotemporal cortex (Heller, 1990, 1993) and providing evidence of region-specific diminished processing for happy faces. A similar mood-relevant effect was also observed in a reduced P3 amplitude for control (but not depressed) participants for previously viewed happy faces and words (collapsed together), interpreted as a positivity bias in controls that is not present in depressed participants (Deldin et al., 2001). Since facial stimuli have been shown to elicit region- and valence-specific ERP effects in depressed populations, the current study will utilize facial stimuli to examine the time course of biased perceptual processing.

In order to examine the effect of cognitive biases on emotion perception, this study used facial stimuli with varying intensities (40%, 70%, and 100% emotive) of positive (happy) and negative (fear) expressions. Emotional faces functioned as target stimuli in a visual oddball design with neutral expressions as the standards. Latencies and amplitudes of the P3 component provided measurements of processing speed and neuronal resource allocation during evaluative stages of biased emotion perception. It was hypothesized that depressed participants would have slower and lower P3s to emotional stimuli, especially happy faces. The use of different levels of intensity of expressions should indicate if multiple dimensions of emotion interact in mood-relevant biased processing. Accordingly, data were analyzed to primarily investigate

the interaction between stimulus intensity and valence, and secondarily to identify any cortical areas with differential processing.

2. Methods

2.1. Participants

Participants were students from San Francisco State University that received course credit or volunteered their time. All participants were informed about the study procedures and signed a written informed consent prior to participation in the study. Participants were grouped according to scores on the Beck Depression Inventory-II (BDI-II: Beck et al., 1996) with the control group scoring eight or below and the depressed group scoring 15 or higher. It is important to note that these groups were only differentiated by their scores on the BDI-II and the participants in the 'depressed' group have not been diagnosed with Major Depressive Disorder or any other affective disorders reported to the experimenters. Each group included 12 females and six males from 18 to 30 years old who were right handed, fluent in English and free of any past neurological trauma. Controls were free of psychoactive drugs, and three participants from the depressed group were using prescription drugs: (1) Wellbutrin, (2) Celexa and Wellbutrin, and (3) Lithium and Zoloft.

2.2. Materials and task

Prior to ERP testing, participants took the BDI-II, the State-Trait Anxiety Inventories (STAI-S, STAI-T: Spielberger et al., 1970), the Behavioral Inhibition System/Behavioral Acquisition System scales (BIS/BAS: Carver and White, 1994) and a demographic questionnaire that included a Likert scale asking how depressed do you feel right now, at this moment on a scale of 0 (not depressed) to 10 (very depressed). See Table 1 for questionnaire means and correlations with BDI-II scores.

Facial stimuli were taken from the Facial Expression of Emotion: Stimuli and Tests (FEEST: Young et al., 2002)

Table 1
Means (standard deviations) for demographic and questionnaire variables and Pearson's r correlations with the BDI-II ($n = 36$)

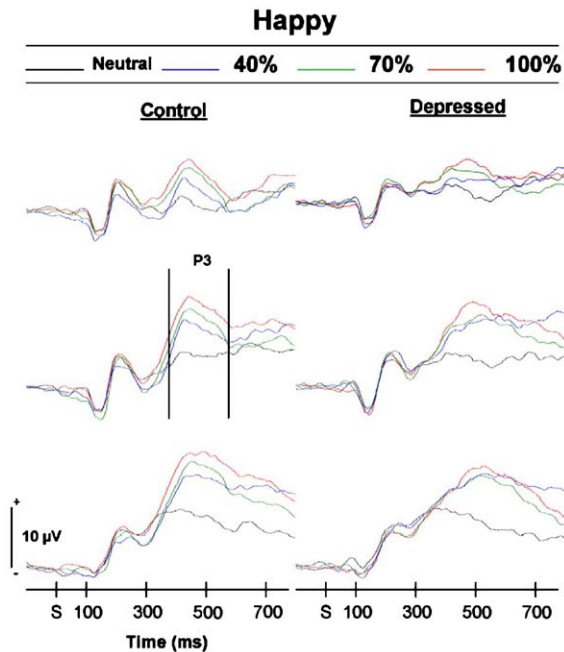| Variable	Control ($n = 18$)	Depressed ($n = 18$)	Correlation (r) with BDI-II
Age	22.3 (3.0)	21.6 (3.4)	–
BDI-II	4.1 (2.9)	25.5 (9.0)	–
State Depression	1.1 (1.9)	4.0 (2.3)	+0.72[*]
STAI-T	33.7 (8.2)	53.5 (10.6)	+0.77[*]
STAI-S	32.1 (9.6)	48.3 (8.2)	+0.74[*]
BIS	19.6 (3.0)	22.2 (3.5)	+0.33
BAS	43.0 (5.1)	38.3 (5.4)	−0.49[*]

[*] $p < 0.01$.

Fig. 1. Grand averaged event-related potential waveforms for neutral face standards and happy face targets (40%, 70% and 100% intensities) over midline electrode sites for control and depressed groups. The analysis time window of the P3 (350–550 ms) is shown at the Cz electrode.

series set where faces from the Ekman and Friesen (1976) series have been morphed from a neutral expression to fully emotive in 10% intervals. Black and white pictures (monitor display of 13 cm × 9.5 cm) of fear and happy expressions were used from the same actress and were presented separately using an oddball sequence with neutral faces as the standard and intensities of 40%, 70% and 100% as targets (7% probability each). Each emotion condition consisted of two pseudo-random blocks (for every six stimuli, at least the first three were neutral, with equal probability of a target occurring in the 4th, 5th or 6th position) with 10 presentations of each target stimuli. The emotion condition blocks were randomly presented as ABAB or BABA. An average of 18–20 artifact-free epochs were available for analysis for each condition for both groups.

The faces were presented using Superlab Pro software on a monitor for 750 ms with an inter-stimulus interval of 1000 ms. Since the aim of the study was not to investigate the threshold of detection but the full cognitive processing of the subtle discriminations between stimuli, the stimuli were presented long enough to facilitate accurate behavioral responses and uninterrupted cognitive evaluation. Participants were instructed to press '1' on the keyboard with their index finger if the face was neutral and '2' with their middle finger if the face was emotional.

2.3. Electrophysiological recording

EEG activity was recorded from Fz, Cz, Pz, F3, F4, P3 and P4 recording sites according to the International 10/20

System using Grass gold-plated electrodes and referenced to linked earlobes. EOG activity was recorded from the outer canthus and below the right eye and stored off-line. Data were sampled at 400 Hz for 1100 ms (100 ms pre-stimulus baseline) and amplified 20,000 times using the Biopac MP150 acquisition unit and Grass Model 12 Neurodata Acquisition System amplifiers, with band pass settings of 0.01–30 Hz. Impedances were kept below 10 KΩ. Trials contaminated with ocular activity or muscular artifact greater than 100 μV were rejected before averaging.

2.4. Data analysis

The amplitudes and latencies of the P3s were measured within the time window of 350–550 ms post-stimulus onset around the grand average display of the waveform at the Pz electrode (see Figs. 1 and 2 for grand average ERPs). P3 latencies and amplitudes were separately investigated in two analysis clusters: a midline cluster (Fz, Cz, Pz) and a global cluster (F3, F4, P3, P4). To examine the affective dimensions of the stimuli, a four-way ANOVA was utilized for the midline cluster. The within subjects factors: valence (happy, fear) × intensity (40%, 70%, 100%) × electrode (Fz, Cz, Pz) × the between subjects factor: group (control, depressed). The significant ($p < 0.05$) three-way valence × intensity × group interactions within the four-way ANOVA were analyzed with separate three-way ANOVAs (valence × electrode × group) split by intensity.

A similar five-way ANOVA was used for the global cluster, splitting the electrode sites into two region-specific variables.
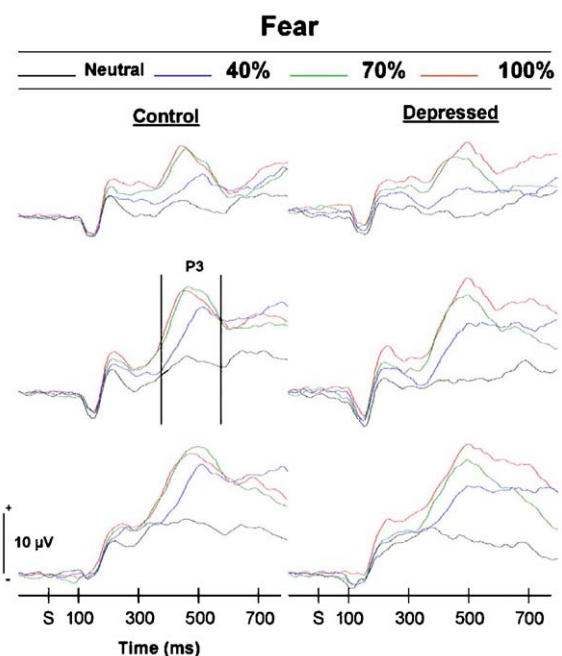

Fig. 2. Grand averaged event-related potential waveforms for neutral face standards and fear face targets (40%, 70% and 100% intensities) over midline electrode sites for control and depressed groups. The analysis time window of the P3 (350–550 ms) is shown at the Cz electrode.

The within subjects factors: valence (happy, fear) × intensity (40%, 70%, 100%) × hemisphere (left, right) × caudality (frontal, posterior) × the between subjects factor: group (control, depressed). The same midline criterion was applied to the global ANOVA, with the addition that region-specific differences were examined. Any significant five-way interactions were followed by intensity specific four-way (valence × hemisphere × caudality × group) ANOVAs and if warranted, intensity-specific hemisphere (valence × hemisphere × group) and intensity-specific caudality (valence × caudality × group) models. Greenhouse–Geisser corrections were used for multiple within-subjects testing.

3. Results

3.1. Behavioral data

Both control and depressed groups averaged over 90% accuracy for responses identifying neutral, 70%, and 100% intensities for fear and happy faces. In the 40% targets, controls and depressed participants averaged over 75% accuracy. Reaction times (RTs) were faster for controls in nearly all emotional conditions, with independent sample t-tests revealing with significantly faster RTs for controls in fear 70% $t(34)=2.10$, $p<0.05$ and happy 100% $t(34)=2.23$, $p<0.05$ faces.

3.2. ERP data

Depressed participants showed slower P3 latencies (as demonstrated in Fig. 3) and varying amplitudes compared to controls for all stimuli (as can be seen in Fig. 4). The four-way

midline ANOVA revealed a significant three-way interaction (valence × intensity × group) both for P3 amplitudes $F(1,34)=3.60$, $p<0.05$ and P3 latencies $F(1,34)=3.91$, $p<0.05$ with the depressed group having lower and slower P3s to happy faces. A follow up three-way midline ANOVA revealed a significant P3 latency interaction (valence × group) at 40% intensities with depressed participants displaying slower P3s for happy faces $F(1,34)=4.21$, $p<0.05$.

The global ANOVA revealed a significant three-way interaction (valence × intensity × group) for P3 latency $F(1,34)=3.74$, $p<0.05$ but not for P3 amplitude. It is important to note that the P3 amplitude three-way interaction approached conventional significance levels $F(1,34)=3.26$, $p=0.051$, however, due to the conservative a priori hypothesis criterion, follow up tests were not run. Similar to the midline ANOVA, a four-way (valence × hemisphere × caudality × group) ANOVA yielded a significant interaction (valence × group) only at 40% intensities with depressed participants displaying slower P3s for happy faces $F(1,34)=7.92$, $p<0.01$.

There was also a significant five-way (valence × intensity × hemisphere × caudality × group) interaction for P3 latency $F(1,34)=3.20$, $p<0.05$. Follow up tests to the five-way interaction revealed a significant four-way interaction (valence × hemisphere × caudality × group) only at 40% intensities $F(1,34)=5.04$, $p<0.05$. No significant hemispheric (valence × hemisphere × group) or caudality (valence × caudality × group) differences were found.

3.3. Medication effects

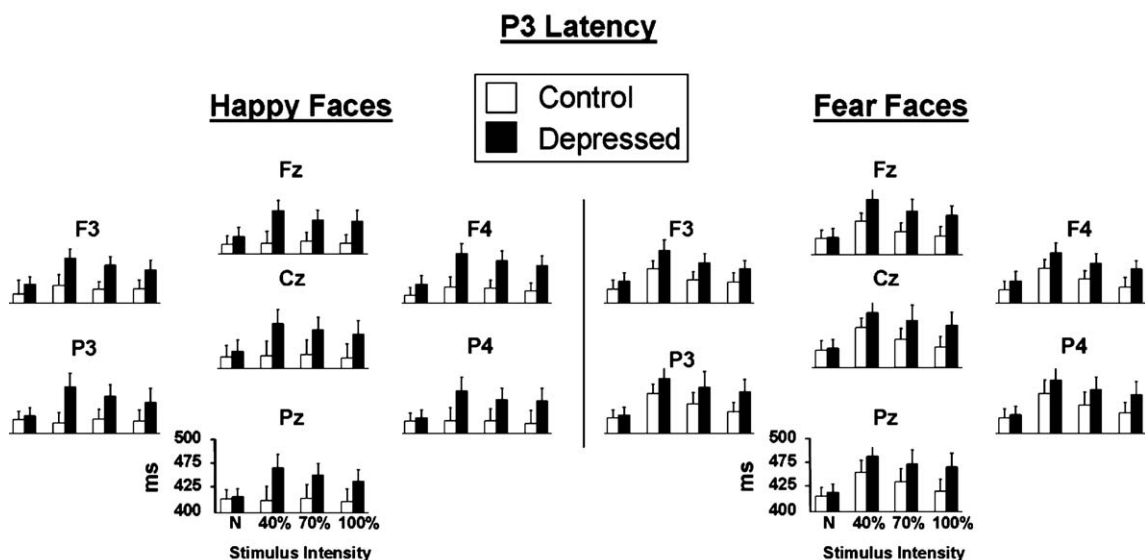To investigate the possible confounding effects of medication on these data, all the ANOVAs were run without

Fig. 3. P3 latency (Means ± SE) in milliseconds for neutral face standards (N) and emotional face (happy and fear) targets. Intensities (40%, 70%, 100%) for each target stimulus are represented at each electrode site for control and depressed groups. This figure shows how non-depressed controls processed happy faces at a similar latency regardless of intensity, where depressed participants display an inverse relationship between P3 latency and intensity of happy facial expression.
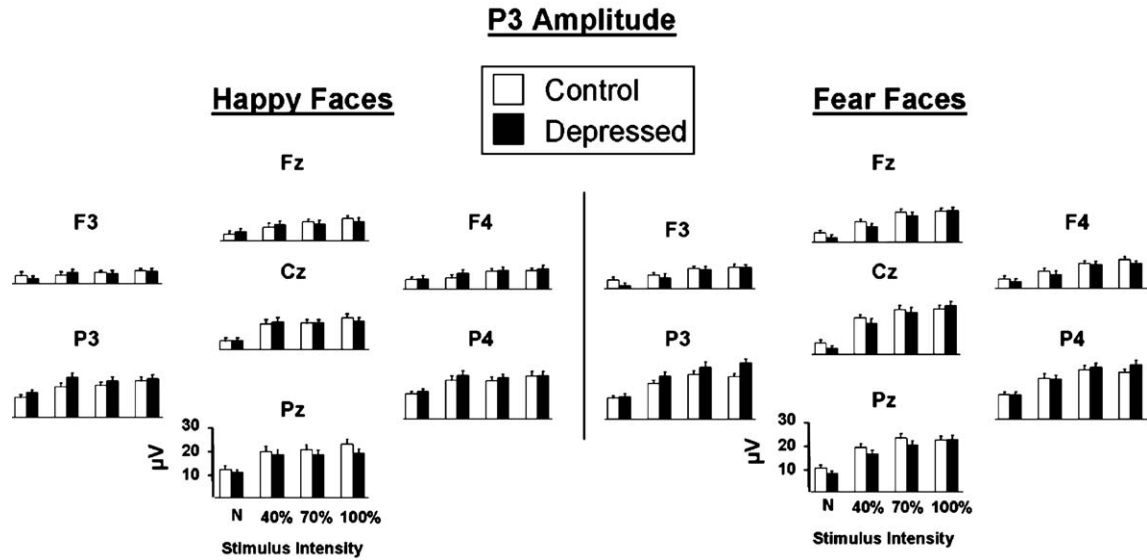
Fig. 4. P3 amplitude (Means ± SE) in microvolts for neutral face standards (*N*) and emotional face (happy and fear) targets. Intensities (40%, 70%, 100%) for each target stimulus are represented at each electrode site for control and depressed groups.

the three medicated depressed participants and three random age- and sex-matched controls. The depressed subjects taken out were at the 39th, 89th and 100th percentile ranges of BDI-II score in their diagnostic group. Three statistical tests did not meet statistical significance at the $p < 0.05$ level. The midline three-way amplitude interaction, the midline 40% latency interaction and the global five-way

interaction all failed to meet conventional levels of significance, yet they all remained under $p < 0.10$. These three analyses were run again with all 18 control subjects and duplicates of three depressed participants in the remaining closest percentiles to the excluded cases. All statistical tests met significance thresholds of $p < 0.05$, providing evidence that lack of statistical power, not medication status explains the null results.

Pearson's r correlations were performed for each participant between BDI-II score and P3 latencies for each stimulus at each electrode site to further examine the relation between depression severity and stimulus evaluation for neural and emotional stimuli. There was a direct correlation with each stimulus with the greatest differences between stimuli valence over posterior sites, with high correlations for happy faces and low correlations for fear faces as observed in Fig. 5.
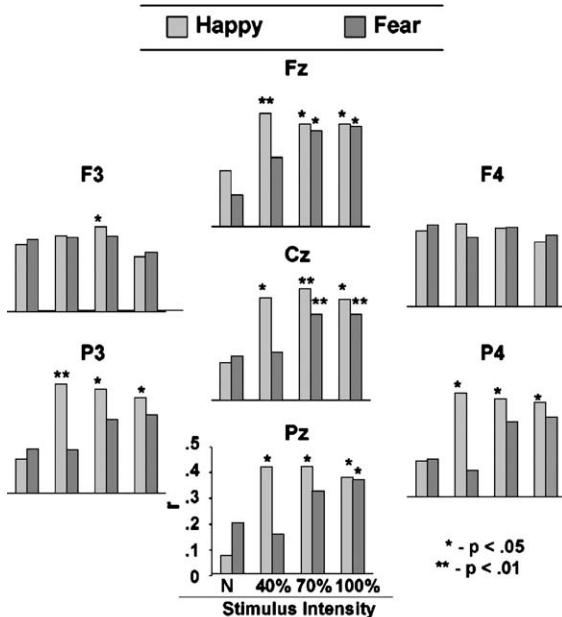

Fig. 5. Pearson's r correlations between P3 latencies for each intensity of target emotion and BDI-II scores (*n* = 36), showing processing speed of stimulus categorization is decreased as depression severity increases. This effect is most pronounced between valences for 40% intensities over posterior electrode sites.

4. Discussion

These results indicate a cognitive processing difference between depressed and non-depressed control participants for emotional expressions, especially in low intensities of happy faces. Significantly delayed or reduced P3s in the depressed group could be affected by a number of different factors relating to symptoms of depression, but when analyzed with the valence specific differences within the groups, these data indicate that depressed participants were different than controls in the cognitive evaluation and processing of happy faces specifically. The midline ANOVA yielded findings that showed lower P3 amplitudes for depressed participants when viewing happy faces. The amplitude of the P3 is directly related to the amount of information provided by a novel stimulus (Polich, 1991),

and it increases with the amount of attention resources allocated for a stimulus (Polich, 1999). Lower P3 amplitudes may indicate that depressed participants use less attentional resources than controls when viewing happy faces, and that happy faces are not as salient to depressed participants. Although the exact relationship between the cognitive processes indexed by P3 amplitude and P3 latency is hard to dissociate, this effect may help explain the longer latencies during stimulus evaluation, especially for slightly happy faces.

P3 latency reflects the speed of stimulus classification with faster latencies reflecting better mental performance (Polich, 1999). Control participants had similar latencies for all happy faces regardless of intensity but depressed participants took increasingly longer to process happy faces as the intensity decreased (as shown in Fig. 3), indicating an increasing difficulty in classification. This mood incongruent effect is further evidenced by the significant valence × group interaction whereby depressed participants display slower P3 latencies for 40% happy faces over all electrode site areas, regardless of topography. This effect provides the strongest evidence in the present study that valence and intensity dimensions interact during mood-biased processing, particularly for mood incongruent faces that are difficult to discern from a neutral expression.

Pearson's r correlations revealed a more sensitive relation between depression score and stimulus evaluation time particularly over posterior brain areas. This effect further supports a specific positive valence × low-intensity processing difference in depression that may be most pronounced in posterior brain areas. It is possible that either the conservative nature of the global ANOVA or the BDI-II grouping criterion was not adequate to reveal region-specific differences in the analyses; null effects in the global interactions do not mean that regional differences do not exist. It will be important that further studies of cognitive functioning in depression vary both the valence and the intensity dimensions of emotional stimuli as well as utilize more intensive region-specific measurements. These data provide further evidence of the diminished salience and delayed evaluation for mood incongruent stimuli in depression, and that these effects depend on the difficulty of emotion discrimination. This provides evidence that mood incongruent processing in depression may not only be the lack of a positivity bias, but also the presence of a deficit in positive stimuli perception due to a lack of attentional resources recruited for stimulus evaluation, necessitating longer cognitive evaluative processes.

The use of a psychometric assessment on a population of students does not discriminate samples into distinct clinical groups and the current designation of 'depressed' could indicate anything from general negative mood to full clinical depression in any individual. However, ERPs have proven to be a useful methodology for examining the time course and regional specificity of emotional information processing even in non-clinical populations. The present data provide evidence for an interaction between a mood incongruent valence and low-intensity arousal cognitive processing deficit in depression for happy faces.

Acknowledgements

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