



Intensified emotion perception in depression: Differences in physiological arousal and subjective perceptions



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ABSTRACT

People suffering from depression perceive themselves and their surroundings as more negative than healthy ones. An explanation might be that depressed individuals experience negative information as more stressful than non-depressed subjects and, consequently, respond in an amplified manner on a subjective and physiological level. To test this proposition, we presented 41 patients with recurrent depressive episodes and 42 controls with stimuli from the International Affective Picture System split into three valence categories while different parameters of physiological arousal (e.g., heart rate variability) and subjective perceptions of valence and arousal were assessed. Furthermore, we examined social skills and emotional competence. Results regarding physiological arousal revealed an elevated skin temperature and a more accentuated respiratory frequency in depressed subjects. Furthermore, depressed subjects rated the stimuli as more negative and arousing, which was associated with reduced social and emotional competence. Variation in antidepressant medication, menstrual cycle and other factors that have an impact on HRV are a potential bias. Our findings suggest an intensified perception of negative emotion in depressed individuals as compared to controls that manifests itself in an increased physiological arousal as well as on a subjective level. This intensified emotion perception is further associated with deficits in social and emotional competence.

1. Introduction

Major depressive disorder (MDD) is defined as a condition that includes key symptoms such as depressed mood and loss of interest or pleasure in daily activities as well as at least four others (e.g., changes in appetite or sleep) over a period of two weeks or longer (American Psychiatric Association, 2013). However, it is not fully understood, yet, which underlying mechanisms lead to these symptoms. Multifactorial models of mental disorders emphasize the importance of the way we deal with stress as one vital factor that determines psychological health or sickness (Nemeroff and Vale, 2005). Since depressed individuals have been shown to exhibit a *negativity bias* (Bourke et al., 2010; Gollan et al., 2016), i.e., to selectively emphasize and better remember negative information (Ridout et al., 2003; Watkins et al., 1996) one could assume that this bias leads them to perceive negative information as more stressful and respond to it in an amplified manner as compared to non-depressed individuals. Such an intensified reaction

might consequently lead to an elevated stress level in depressed individuals.

Stress can be defined as a stimulus that disrupts the physiological or psychological homeostasis of an individual when encountering internal or external harmful events (Kyrou and Tsigos, 2009). On a biological level the autonomic nervous system (ANS) is responsible for an organism's stress response. It regulates visceral functions through its sympathetic and parasympathetic arms, which act antagonistically to preserve a dynamic equilibrium of vital functions (Xhyheri et al., 2012). For example, the sympathetic arm can induce excitation by increasing heart rate, blood pressure and the skin conductance level within seconds (Ulrich-Lai and Herman 2009, Braithwaite et al., 2013, Guerra et al., 2012). A dysregulation of ANS activity impairs a person's ability to cope with stress and might, thus, be an important factor in the development and perpetuation of MDD. In fact, MDD has been repeatedly linked to altered patterns of ANS functioning (Bassett, 2015; Branković, 2008; Brunoni et al., 2013).

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There are various signals that reflect ANS activity. Heart rate variability (HRV), among others, describes fluctuations between intervals of consecutive heartbeats as a result of ANS dynamics on cardiovascular control that maintain the dynamic equilibrium of vital functions (Xhyheri et al., 2012). Reductions in HRV have not only been found in MDD but in various psychiatric disorders such as schizophrenia (Moon et al., 2013), bipolar disorder (Bassett, 2015; Bassett et al., 2016; Henry et al., 2010) or post-traumatic stress disorder (Moon et al., 2013). Kemp et al. (2010) conducted a meta-analysis examining HRV in 18 samples of altogether 673 patients suffering from MDD and 407 healthy controls. He found that MDD subjects showed a reduced HRV in time domain measures and lower HF power. A literature review of Stapelberg et al. (2012) came to a similar conclusion. Further evidence was added by Wang et al. (2013) who found reduced SDNN, SDANN, RMSSD, pNN50 and HF power in depressed patients as well as by Ha et al. (2015) who found lower SDNN, RMSSD and pNN50 in first-episode depressed female subjects. However, according to Kemp et al. (2014a), heterogeneous samples and confounding variables might confound results. For example, he found lower RMSSD levels in subjects suffering from melancholia than in a nonmelancholic sample (Kemp et al., 2014b) and reduced RMSSD and HF values in depressed subjects with comorbid generalized anxiety as compared to MDD subjects without comorbid anxiety (Kemp et al., 2012). On the other hand, Bassett et al. (2016) reported reduced SDNN scores in a sample of patients suffering from recurrent depressive disorder in remission. Antidepressant medication is considered another confounding variable since it has been reported to reduce HRV. While the exact influences of antidepressant medication are not completely understood, yet, it appears that tricyclic substances influence HRV the most (Kemp et al., 2010; Udupa et al., 2011) whereas, e.g., selective serotonin reuptake inhibitors (SSRIs) seem to have less impact or no impact at all (Kemp et al., 2010; Van Zyl et al., 2008)."

Furthermore, the human stress response includes shifts in the skin conductance level, the galvanic skin response (GSR). Research concerning GSR at rest reveals a heterogeneous picture. Some publications suggest reduced GSR levels as a biomarker of depression (Storrie et al., 1981; Straub et al., 1992; Ward et al., 1983). However, others found increases (Branković, 2008; Toone et al., 1981).

While baseline levels of HRV and GSR in depression have been examined by a number of research groups, few studies have assessed how those signals change in response to negative, emotionally arousing stimuli. Shinba (2014) found that HF power and the LF/HF-ratio of HRV was increased in depressed subjects as compared to a control sample while performing a cognitive challenge task. However, Shinba's sample was quite small ($N=22$ depressed subjects). Schneider et al. (2012) reported elevated levels of GSR in MDD during the presentation of short video clips depicting actors expressing emotions by face, voice and prosody. Gehricke and Shapiro (2001), however, did not find changes in GSR while imagining sad and happy situations. Finally, Lin et al. (2011) measured GSR, heart rate, RSP and TEMP and their association to depressive symptoms during an arithmetic stress test. They found no variations in GSR but noted a correlation between TEMP and depressive symptoms. Yet, they did not use emotionally stressful material and merely measured depressive symptoms in a healthy student sample using the Beck Depression Inventory II (BDI-II; Hautzinger et al., 2009).

Prior research does not give a comprehensive picture of how depressed subjects react to emotional stress. Studies differ largely in their choice of stress induction paradigm oftentimes using stimuli that have not been validated according to their arousal or valence. Most researchers focus on one indicator of ANS activity only. Respiratory frequency (i.e., breaths per minute; RSP) and skin temperature (TEMP) as measures of ANS activity have - to our knowledge - received very little attention in research on depression so far. Also, we note differences in the diagnostic inclusion criteria for the depressed subjects. Furthermore, various symptoms associated with MDD were

not assessed in prior studies. For example, depressed individuals are often limited in their ability to master social situations (Quintana et al., 2012). Consequently, they exhibit symptoms such as social withdrawal, which restrict them in their social relationships. Those deficits in social skills might be caused by poor ANS regulation (e.g., reduced ANS) according to Quintana et al. (2012) and the *polyvagal theory* (Porges, 2007).

Thus, the aim of the current study was to provide a comprehensive overview of how depressed individuals perceive negative information on a biological as well as behavioral level as compared to healthy control subjects. We focused on assessing four different markers of ANS activity. Moreover, we used stimuli that are well validated and were proven to elicit negative emotions. We included only depressed patients that were diagnosed by a trained clinician and were currently experiencing an acute depressive episode into our sample. We hypothesized that depressed subjects would not only perceive the negative information as more intense but would also show higher levels of arousal indicated by increased ANS activity (RSP, TEMP, GSR) as compared to healthy control subjects and that HRV will be reduced. Furthermore, we expected that these differences in physiological arousal would be associated with symptoms of depression, such as reduced emotional and social competence.

2. Methods and material

2.1. Participants

41 patients diagnosed with recurrent depressive disorder (25 females, 16 males; $M_{\text{age}}=43.85$ years, $SD=12.18$) and 42 healthy controls (29 females, 13 males; $M_{\text{age}}=40.67$ years, $SD=14.28$) were included in the study. Patients were recruited as inpatients of the Department of Psychiatry, Psychosomatic Medicine and Psychotherapy of the University Hospital of Frankfurt/Main, Germany or via announcements in newspapers. In order to confirm the diagnosis in the patient group and to rule out any mental illness in the control group we conducted the Structured Clinical Interview for DSM-IV (SCID-I and SCID-II; German version by Wittchen et al., 1997).

Patients had suffered from depressive episodes for up to 43 years ($M=12.75$, $SD=31.11$) and were currently experiencing a depressive episode as diagnosed by a treating clinician, based on DSM-V criteria. Patients suffering from comorbid axis I or II disorders were excluded from the study with the exception of anxiety disorders. Depressive symptom severity assessed using the BDI II (retest-reliability: $r=0.78-0.93$; construct validity: $\alpha=0.92-0.93$) was moderate to severe ($M=22.66$; $SD=10.60$). Thirty-three patients were currently taking antidepressant medication. Medication substance and dose had been stable for at least two weeks prior to testing. To compare different medication doses, scores equivalent to antidepressant and antipsychotic medication were calculated (Bollini et al., 1999; Woods, 2003).

Control subjects had no current drug-abuse, addiction or neurological illness. In order to assure comparable visual attention abilities we applied the Trail Making Test A (TMT-A; Nuechterlein and Green, 2006). Since they were shown to affect HRV (Xhyheri et al., 2012), the body mass index (BMI; kg/m^2) and nicotine consumption (cigarettes/day) were assessed as control variables (see Table 1). All participants received a description of the experiment and gave written informed consent before participating. The ethical board of the Medical School of the University Hospital of the Goethe-University, Frankfurt/Main, Germany, approved experimental procedures.

2.2. Assessment of indicators of ANS activity

We used the "NeXus-10 MKII"-system and the software Biotrace+ to record and analyze the function of the ANS (HRV, GSR, TEMP, RSP). In order to have a stationary and representative baseline HRV sample, a resting period of 2–5 min time preceded the baseline

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