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Midline theta dissociates agentic extraversion and anhedonic depression

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ABSTRACT

The agency facet of extraversion is related to individual differences in reward anticipation and has been linked to the neurotransmitter dopamine. Dopamine has also been associated with components of anhedonia, which is one of the cardinal symptoms of depression and refers to lack of responsiveness to pleasurable stimuli. This raises the question whether low agency is associated with anhedonia symptoms in depression and if agency and anhedonia are characterized by similar neurobiological mechanisms. To address this hypothesis, we tested whether questionnaire measures of agency and anhedonia are correlated within depressed ($n = 20$) and non-depressed ($n = 22$) participants. Further, we investigated whether dopamine-related signatures in the EEG recorded during a gambling task (feedback-evoked theta activity, and frontal versus posterior theta activity) similarly relate to agency and anhedonia. Results indicated that anhedonia was significantly elevated in the depression group, and negatively correlated with agency. However, while theta activity evoked by negative vs. positive feedback was sensitive to anhedonia and depression status but unrelated to agency, frontal versus parietal theta activity predicted agency, but was unrelated to anhedonia and depression. Together, this double dissociation suggests that in spite of considerable phenotypical overlap, anhedonia and agency may be linked to partially distinct neurobiological markers.

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1. Introduction

Depression is a debilitating condition with a high prevalence and economic burden for society across cultures (Kessler & Bromet, 2013). In order to better understand its etiology and pathophysiology it has been recommended to study more narrowly defined phenotypes or symptoms of this complex and relatively heterogeneous mental illness. A core symptom of depression that has received increasing interest in this regard is anhedonia – the lack of responsiveness to pleasurable stimuli (Hasler, Drevets, Manji, & Charney, 2004; Meehl, 1975; Pizzagalli, 2014). Although the personality dimensions that are specifically associated with an increased risk for anhedonia have not been systematically explored, early theories suggested that low extraversion relates to depression (e.g. Fig. 1 of Eysenck, 1944), or anhedonia in particular (Clark & Watson, 1991); in line with these early theories negative associations between extraversion and depression are well established (e.g. Jylha & Isometsa, 2006). However, extraversion is a relatively broad and heterogeneous construct capturing individual differences in agency, affiliation and impulsivity (Depue & Collins, 1999) and it is not known which of these facets are linked to anhedonia or depression. Furthermore, little is known about biological mechanisms that may link extraversion facets to anhedonia.

For several reasons, it could be hypothesized that it is particularly the agency facet of extraversion that is related to anhedonic symptoms in depression. The agency facet comprises individual differences in one's sense of accomplishing goals, assertiveness, social dominance, levels of activity, well-being and positive affect and these differences are presumably related to the motivational salience of positive incentives. Thus, both low agency (Depue & Collins, 1999) and anhedonia (Meehl, 1975; Pizzagalli, 2014) are conceptually related to reduced sensitivity for positive incentives or potential rewards and there is some evidence relating behavioral indices of reward processing to questionnaire measures of agency-related constructs (Gupta & Shukla, 1989) and to anhedonia (Pizzagalli, Jahn, & O'Shea, 2005). Second, consistent with reward processing being influenced by the neurotransmitter dopamine (DA), both agency- and anhedonia-related constructs have been theoretically (Depue & Collins, 1999; Dunlop & Nemeroff, 2007; Gray, 1982; Pizzagalli, 2014; Wise, 2008) and empirically (Lambert, Johansson, Agren, & Friberg, 2000; Reuter & Hennig,

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2005; Wacker, Chavanon, & Stemmler, 2006) associated with the DA system. Third, in line with DA having a strong association with neurobiological processing of performance-feedback and rewards (Holroyd & Coles, 2002; Schultz, 1998), anhedonia (Liu et al., 2014) and agency (Lange, Leue, & Beauducel, 2012; Mueller, Burgdorf, Chavanon, Schweiger, Wacker, et al., 2014) have been linked to altered electrophysiological signatures of feedback processing, which are known to be sensitive to DA levels (Mueller, Burgdorf, Chavanon, Schweiger, Hennig, et al., 2014; Santesso et al., 2009). Finally, a correlation of anhedonia and agency in a small healthy sample of an unpublished study was noted (Wacker, Chavanon, & Stemmler, 2010), suggesting that the two constructs covary at the level of behavioral self-reports in non-depressed participants. In spite of these converging lines of evidence, it has not yet been explicitly tested whether agency relates to symptoms of anhedonia in currently depressed individuals.

Moreover, it is unclear, whether agency and anhedonia only relate to each other at the level of questionnaire measures or if they also show similarities at the neurobiological level. Based on the common link to DA and reward processing, it could thus be hypothesized that agency and anhedonia show similar modulations of neural activity evoked by reward-related feedback. A recently discovered neural correlate of reward-related feedback processing is feedback-evoked frontomedial theta (4–8 Hz) activity as measured with EEG. Of relevance, it has been shown that feedback-evoked theta (1) is lower for positive "reward" feedback vs. negative "loss" feedback (Cohen, Elger, & Ranganath, 2007) and (2) is associated with individual differences in agency and DA (Mueller, Burgdorf, Chavanon, Schweiger, Wacker, et al., 2014). To our knowledge, whether feedback-evoked theta also relates to anhedonia or depression has not yet been tested.

In addition to feedback-evoked theta, the FzPz index, a feedback-independent measure of frontal versus parietal theta topography is a potentially relevant marker as it has been consistently associated with agency and shown to be modulated by DA (Chavanon, Wacker, & Stemmler, 2011; Wacker et al., 2006, 2010). Whether the FzPz index relates to anhedonia has not yet been tested, although there is some evidence for altered theta activity in healthy individuals with high vs. low anhedonia (Wacker, Dillon, & Pizzagalli, 2009).

Taken together, anhedonia and low agency share a number of features but their correlation has not yet been tested in a clinical sample. Further, it is unknown if agency and anhedonia are characterized by the same electrophysiological correlates. The aim of the current study was to address these issues by performing secondary analyses of a dataset recently described (Mueller, Panitz, Nestoriuc, Stemmler, & Wacker, 2014).

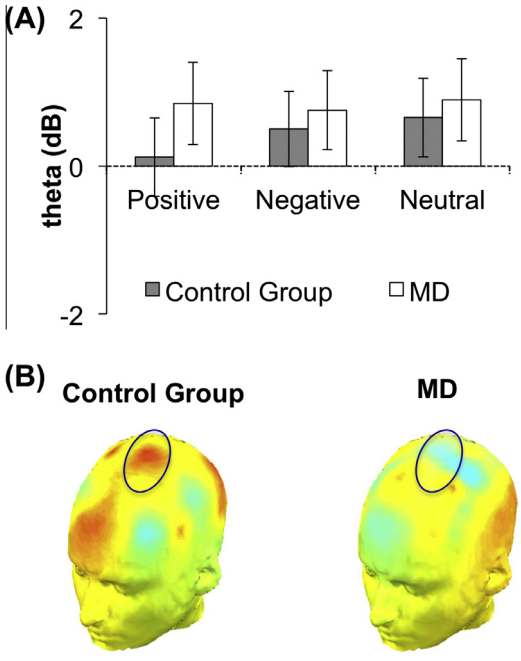

Fig. 1. (A) Barplot indicating mean feedback-evoked theta at channels FCz and Cz for the control (grey) versus MD (white) group for the three types of feedback valence. Error-bars indicate SEM. (B) Topographic heat maps of the difference in theta power for neutral vs. positive feedback for control (left) and MD (right) group. Ellipse indicates the location of centromedial electrodes FCz and Cz used for the present analyses.

2. Methods

2.1. Sample

Data from $N = 21$ participants with depression and $N = 23$ control participants were analyzed for the present study. These participants constitute a subsample of a larger study that also included $N = 22$ participants with panic disorder and investigated brain–heart coupling in panic disorder rather than theta oscillations (Mueller, Panitz, et al., 2014). Due to missing questionnaire data, 1 participant from each group had to be removed, yielding a final sample of $N = 42$ participants. Sample characteristics are provided in Table 1.

2.2. Participants

Participants were invited to a first session where they signed informed consent and a brief standardized clinical interview was conducted (Margraf, 1994). If participants met DSM-IV criteria for a major depressive episode (MD group) or no criteria for any DSM-IV diagnosis (control group) they were sent home with a series of questionnaires to complete (see below) and re-invited to the EEG session within seven days after the interview.

The experimenter of the EEG session was blind to the participants' diagnoses. After an initial 10-min resting phase, participants completed the gambling task including a 15-trial practice block. After the gambling task participants were debriefed and compensated with 35 € (about $46). The study was approved by the Ethics Committee of the Psychology Department of Marburg University.

2.3. Paradigm

The paradigm was a 360-trial gambling task which is described in more detail elsewhere (Mueller et al., 2010; Sato et al., 2005). At the beginning of each trial the amount of money to win or lose was displayed (0, 10, or 50 cent). Subsequently, participants were presented a card showing a number (from 2 to 7) and were asked to guess by button press if a second card drawn by the computer would have a lower or higher value. 3000 ms after the button press, participants received positive (green circle), negative (red cross), or uninformative (blue question mark) feedback to inform them if they had won or lost the amount. Unbeknownst to the participants, presentation of feedback was quasi-randomized with balanced frequencies for the different feedback types. Participants were told in advance that they could win a total between 10 and 15 €, however, every participant received 15 € at the end of the session.

2.4. Questionnaire measures

Anhedonia was measured using a German adaptation of the 10-item anhedonic depression subscale from the 30-item version

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