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Review article

Social functioning in major depressive disorder

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ABSTRACT

Depression is associated with social risk factors, social impairments and poor social functioning. This paper gives an overview of these social aspects using the NIMH Research and Domain Criteria 'Systems for Social Processes' as a framework. In particular, it describes the bio-psycho-social interplay regarding impaired affiliation and attachment (social anhedonia, hyper-sensitivity to social rejection, competition avoidance, increased altruistic punishment), impaired social communication (impaired emotion recognition, diminished cooperativeness), impaired social perception (reduced empathy, theory-of-mind deficits) and their impact on social networks and the use of social media. It describes these dysfunctional social processes at the behavioural, neuroanatomical, neurochemical and genetic levels, and with respect to animal models of social stress. We discuss the diagnostic specificity of these social deficit constructs for depression and in relation to depression severity. Since social factors are importantly involved in the pathogenesis and the consequences of depression, such research will likely contribute to better diagnostic assessments and concepts, treatments and preventative strategies both at the diagnostic and transdiagnostic level.

Contents

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1. Introduction

Major depressive disorder (MDD) frequently affects psychiatric patients in both inpatient and outpatient treatment settings, and ranks among the leading mental health causes of the global burden of disease (Ferrari et al., 2013). Although depressed patients are generally less severely impaired in everyday lives than patients with other mental disorders such as autism and schizophrenia (Bazin et al., 2009; Derntl and Habel, 2011; Lee et al., 2004; Weniger et al., 2004), the impairment of social functioning, defined as "an individual's ability to perform and fulfill normal social roles", is considered an important sign of depression (Hirschfeld et al., 2000). The dysfunctions in social interactions remain persistent even after three years of recovery from depressive symptoms (Rhebergen et al., 2010) and are correlated with unemployment, disability and decreased work performance (Rizvi et al., 2015). However, the US National Comorbidity Survey reported that, in the US, the social role impairment associated with depression was greater in the social role domain (43.4% severe or very severe), leading to impaired interpersonal (Hirschfeld et al., 2002) and marital functioning (Fink and Shapiro, 2013), than in the work role domain (28.1% severe or very severe) (Kessler et al., 2003). The social impairments could partly result from social emotional dysfunction, like difficulties in understanding and controlling social emotions, or a deficit in the ability to read signals of interpersonal threat and safety (Tse and Bond, 2004). Further, the interpersonal difficulties might be a result of reduced motivation, altered empathic responding in social interactions and a reduced capacity to come up with effective solutions for interpersonal problems. Furthermore, the lack of expressiveness in the nonverbal behavior of depressed individuals, such as a reduced tendency to smile (Rehman et al., 2008), is likely to be interpreted as impolite, uninterested, or inattentive by dyadic partners who are attempting to interact with them.

Epidemiological and neurobiological studies have suggested that interactions between biological (e.g. neurotransmitter system dysfunctions, genetic vulnerabilities), psychological (e.g. impaired emotion recognition) and social factors (e.g. bullying) over time explain the risk of developing MDD. For example, genetic factors contribute to the risk of stress exposure (Kendler et al., 2003), and stressful social events are capable of serving as triggers for epigenetic alterations at specific gene loci, potentially causing long-term changes in brain functioning (Lohoff, 2010). Thus, the National Institute of Mental Health has proposed a new, etiology-based framework for psychiatric diseases – the Research Domain Criteria (RDoCs) – which includes genes, neurotransmitter systems, brain circuitry, behaviour and self-report as units of analysis. Using the framework offered by the RDoC's domain 'Systems for Social Processes', this review provides an overview of pathogenic pathways underlying social impairments associated with MDD. Specifically, we describe the bio-psycho-social interplay regarding social impairments in the four constructs contained in this domain: "Affiliation and Attachment", "Social Communication", "Perception and Understanding of Self" and "Perception and Understanding of Others" (for an overview, see Table 1). Because animal models are crucial in dissecting the causal role of circuit level changes in disease models, we mention translational aspects for each deficit construct. At the end, we discuss the social impact of MDD-related social impairments regarding social networks in real life and

Table 1
Schematic overview of the *subconstructs of systems for social processes* domain of RDoC framework.

	Units of Analysis				Experimental paradigms
Subconstructs	Impairments	Brain systems	Brain regions	Molecules	
Affiliation and attachment	Social anhedonia	Reward system, evaluative system	NAcc, mPFC, insula, amygdala	Mu opioid receptor, dopamine	Social acceptance paradigm
	Increased sensitivity to social rejection	Evaluative system, cognitive control system	Amygdala, insula, DLPFC, subACC	Mu opioid receptor, oxytocin	Chatroom task, cyberball game, social feedback task
	Increased altruistic punishment	Reward system	NAcc, caudate nucleus	Dopamine serotonin	Ultimatum game, dictator game
	Abnormal use of social media	Reward system	NAcc, caudate nucleus	Dopamine	Viewing stimuli related to social network sites
Social communication	Mood-congruent bias in processing of emotional expressions	Cognitive control system, evaluative system	Amygdala, mPFC, DLPFC	Serotonin	Emotional face expression tests
	Stronger response to emotional expressions of shame, fear and social threat	Evaluative system, cognitive control system	Insula, amygdala DLPFC	Serotonin	Shame and fear recognition from pictures
	Diminished cooperativeness	Reward system	NAcc, caudate nucleus	Serotonin, oxytocin	Prisoners dilemma, public goods game
	Increased competition avoidance	Reward system, evaluative system	NAcc, amygdala, insula	Dopamine, testosterone	Social competition paradigm
Perception and understanding of others	Mentalizing deficits	Cognitive control system, empathic system	DLPFC, mPFC, subACC	Oxytocin	Second-order false belief test, RMET test, understanding jokes, sarcasm and metaphors
	Reduced empathy	Empathic system	mACC, somatosensory cortex	Oxytocin	Empathic accuracy tasks, evaluating videos depicting painful stimuli

Notes: The table was adapted from: http://www.nimh.nih.gov/research-priorities/rdoc/nimh-research-domain-criteria-rdoc.shtml#toc_matrix. RDoC's domain'Systems for Social Processes' is conceptualized as a two-dimensional matrix with rows representing its subconstructs and the columns representing various units of analysis that can be used to assess these subconstructs. The cells at the intersections of the rows and columns are populated by research findings related to social impairments in MDD. The last column contains an overview over the experimental paradigms designed to measure the alterations in social functioning. This table is not intended to capture the full range of psychopathology in MDD, but rather focuses on findings for which there is solid evidence resulting from ongoing research. Further, the "Perception and Understanding of Self" subconstruct is not included in the table. Competition avoidance could also be understood as affiliation and attachment problem.

abnormal use of social media. Finally, we discuss how treatment of social deficits in MDD can profit from accumulating knowledge.

2. Social anhedonia

Developmental deficits in the ability to form social bonds and affiliate with someone increases the risk for reduced interest in social activity, which we refer to as social anhedonia. Social anhedonia manifests as a significantly reduced drive for social affiliation (Brown et al., 2007; Kwapil et al., 2009), characterised by the absence of the need to belong to a social group (Brown et al., 2007; Kwapil et al., 2009) and social disinterest (Silvia and Kwapil, 2011). In addition, social anhedonia is related to lower levels of social functioning (Blanchard et al., 2011) and decreases the potential to engage in positive social interactions later in life. Social anhedonia has also been related to outcomes such as being unmarried, having a low number of friends, and social isolation (Brown et al., 2007). Apart from being associated with depression severity and the melancholic subtype of MDD (Atherton et al., 2015; Pelizza and Ferrari, 2009; Rey et al., 2009), social anhedonia is believed to predict non-response to antidepressants (Gorwood, 2008) and psychotherapy (Hasler et al., 2004a, 2004b).

Social anhedonia also occurs in the depressed phases of bipolar disorders and seems to be more developed in recovered BD I than BD II patients (Akiskal et al., 2006). In schizophrenia, it is a typical negative symptom, although its pathogenesis may be different from the one in affective disorders (Dodell-Feder et al., 2014; Kollias et al., 2008). Comorbid disorders such as substance use disorder and borderline personality disorder can increase social anhedonia and the risk of social impairment in MDD (Davis et al., 2008). For example, social interaction might be even less rewarding in depressed patients who consume cocaine because cocaine use leads to a markedly blunted activation of the social reward system (Preller et al., 2014).

2.1. Findings from behavioural studies

The social skill deficits in MDD such as increased speech latency during dialogue (Yang et al., 2013), lack of eye contact (Ellgring, 2007) and reduced ability to concentrate on the topic of conversation (Schwartz-Mette and Rose, 2015) can result from the increased self-focus, in which depressed individuals may not have sufficient cognitive resources to process incoming signals (Mor and Winquist, 2002). Due to their failure to disengage from their own thoughts and feelings (Grimm et al., 2011; Isen, 2000), self-focused individuals with depressive symptoms might be perceived by friends as especially annoying and abrasive, leading to difficulties in interpersonal relationships (Schwartz-Mette and Rose, 2015). Apart from increased self-focus, the anhedonic social behaviors of depressed individuals also include decreased initiation and responsiveness to social contacts, possibly giving their dyadic partners an impression of having lack of interest to engage in social interactions. Further, MDD patients show little adaptive behavior in response to facial emotional expressions (Radke et al., 2014), increased withdrawal behavior during observation of emotional facial expressions (Derntl et al., 2011), and less smiling during social interaction (Girard et al., 2013). Possibly, patients with MDD withdraw from others in order to protect themselves from anticipated disappointment, rejection,

scorn, and social exclusion (Allen and Badcock, 2003; Girard et al., 2013). Indeed, it has been shown that depressed individuals were less likely to see their partner's behavior as loving and supportive compared with non-depressed subjects (Overall and Hammond, 2013). Further, in a social interaction, depressed participants also rated themselves as less competent than non-depressed participants on all measures of social competence (Gable and Shean, 2000).

2.2. Findings from functional neuroimaging studies

2.2.1. Decreased pleasure from social interactions due to reduced response from the social reward system

An important reason for diminished social interest in MDD is the reduce in gaining pleasure from interpersonal interactions paralleled by a decreased responsivity of the social reward system (Germine et al., 2011). Thus, during a social acceptance experiment, in comparison to healthy individuals, patients with MDD showed a lower activity in the ventral striatum (nucleus accumbens, NAcc) (see Fig. 1), an important social reward structure in the brain (Hsu et al., 2015). An activation of this structure was positively correlated with increases in the desire for social interaction in the healthy controls but not in MDD patients. Another study showed that during approach of friends in contrast to approach of peers and celebrities, NAcc was more strongly active in healthy subjects than in patients with MDD, suggesting that dysfunction of the social reward system may be particularly disadvantageous with respect to maintenance of relationship with friends, which is consistent with social deficits observed in MDD (Güroğlu et al., 2008). The inability to gain reward from social contact can even be a reason for parenting deficits in depressed mothers. Thus, the hypoactivity of striatal areas (caudate nucleus, NAcc) during presentation of their own infant's cry in the MDD may indicate the depressed mothers' inability to experience reward and motivation for approaching their crying infants (Laurent and Ablow, 2011).

Rodent models of stress-induced depressive-like behavioral abnormalities have provided an opportunity to dissect the underlying cellular and circuit-based mechanisms linking the mesolimbic reward system to social anhedonia (Heshmati and Russo, 2015). Social stress induced depressive behaviors have been studied using a chronic social defeat stress (CSDS) paradigm in which a young adult male mouse is exposed to a larger, aggressive mouse over the course of 10 days. Individual differences in response to this social stress lead to a group of mice that continue to display normal social approach and interaction, termed 'resilient' and a group that demonstrate social avoidance, termed 'susceptible' (Krishnan et al., 2007). Susceptible and resilient mice show distinct cellular and circuit alterations between the NAcc and other brain regions within the reward circuitry, demonstrating a causal role for plasticity in this circuit in mediating social anhedonia in a mouse model of depression (Heshmati and Russo, 2015; Russo and Nestler, 2013). In the mesolimbic dopamine pathway projecting from the ventral tegmental area (VTA) to the NAcc, phasic firing is increased in mice that are susceptible to CSDS, and inducing phasic firing in resilient mice leads to a social avoidant phenotype characteristic of susceptible mice. Conversely, inhibition of this pathway promotes resilience after social stress (Chaudhury et al., 2013). In addition to dopaminergic effects on social avoidance, Brain Derived Neurotrophic Factor (BDNF) signaling in the mesolimbic pathway also plays an important role in susceptibility to social anhedonia following social stress: BDNF signaling from the VTA to the NAcc promotes susceptibility, while blocking BDNF action within the NAcc leads to a resilient phenotype (Koo et al., 2015).

The NAcc receives glutamatergic projections from the prefrontal cortex (PFC), thalamus, amygdala, and hippocampus and each of these projections has been shown to have a distinct contribution to modulating social anhedonia following social stress. For example, susceptible mice show increased synaptic strength at thalamic inputs to NAcc, and modulating thalamo-NAcc projections bi-directionally alters social interaction after social stress (Christoffel et al., 2015). While increased activity of thalamo-NAcc projections leads to increased social avoidance after social stress, PFC-NAcc afferents may promote resiliency, as high-frequency optogenetic stimulation of this pathway reverses social avoidance (Covington et al., 2010; Vialou et al., 2014). Optogenetic activation of ventral hippocampal projections to the NAcc promotes susceptibility, while optogenetic induction of synaptic depression at NAcc synapses from the ventral hippocampus promotes resiliency and increased social interaction (Bagot et al., 2015). These findings demonstrate that reduced social interest in depression is related to modulation of the NAcc through interactions with the larger brain reward network, including the VTA, thalamus, PFC, and hippocampus.

2.2.2. Altered μ-opioid receptor activation and hyperactivity of the medial prefrontal cortex leading to striatal hypoactivity

One additional molecular mechanism for the decreased activation of the NAcc during social interaction seems to be an altered endogenous opioid activity in the brain of depressed individuals. The μ-opioid receptor (MOR) deactivation in NAcc during social acceptance in MDD might lead to decreased motivation to seek out positive social interaction (Hsu et al., 2015). Apart from opioid involvement, another reason for the NAcc deactivation and thus decreased pleasure derived from positive interactions might be greater medial prefrontal cortex (mPFC) activation and stronger positive connectivity between NAcc and the mPFC (Healey et al., 2014). Thus, individuals with higher social anhedonia and stronger depressive symptoms showed a stronger response of mPFC to mutual liking (i.e., being liked by someone they also liked) relative to received liking (i.e., being liked by someone whom they did not like) (Healey et al., 2014). Possibly, the increased mPFC function during social reward reflects signaling to enhance neural response from the ventral striatum, which is not responding with typical magnitude or duration in MDD (Forbes and Dahl, 2012) (see red arrow in Fig. 1).

2.2.3. Loss of emotional reactivity to positive social stimuli

Another important hypothesis for a low desire to engage in social interactions in MDD might be the loss of emotional reactivity to positive social stimuli (Rottenberg and Hindash, 2015; Rottenberg et al., 2005), resulting from reduced salience attribution to socially rewarding information at early processing levels (Stuhrmann et al., 2013). Thus, when examining the neuronal basis of social approach and withdrawal, Derntl et al. (2011) demonstrated significantly decreased amygdala activation in MDD patients in comparison to controls, especially during approach to happy faces.

3. Increased sensitivity to social rejection

Impaired social affiliation and attachment frequently manifests as increased sensitivity to rejection and leads to social withdrawal. A recent study has demonstrated that almost 50% of patients with MDD and bipolar disorders experience an increased rejection sensitivity (Ehnvall et al., 2014). Although rejection sensitivity is an important aspect of MDD, it might be fundamental to other psychiatric disorders such as schizophrenia (Silvia and Kwapil, 2011), anxiety disorders (Lau et al., 2011) and represents a core psychopathology in borderline personality disorder (Staebler et al., 2011). Rejection sensitivity relates to the tendency to anxiously or angrily expect and readily perceive rejection (Zimmer-Gembeck

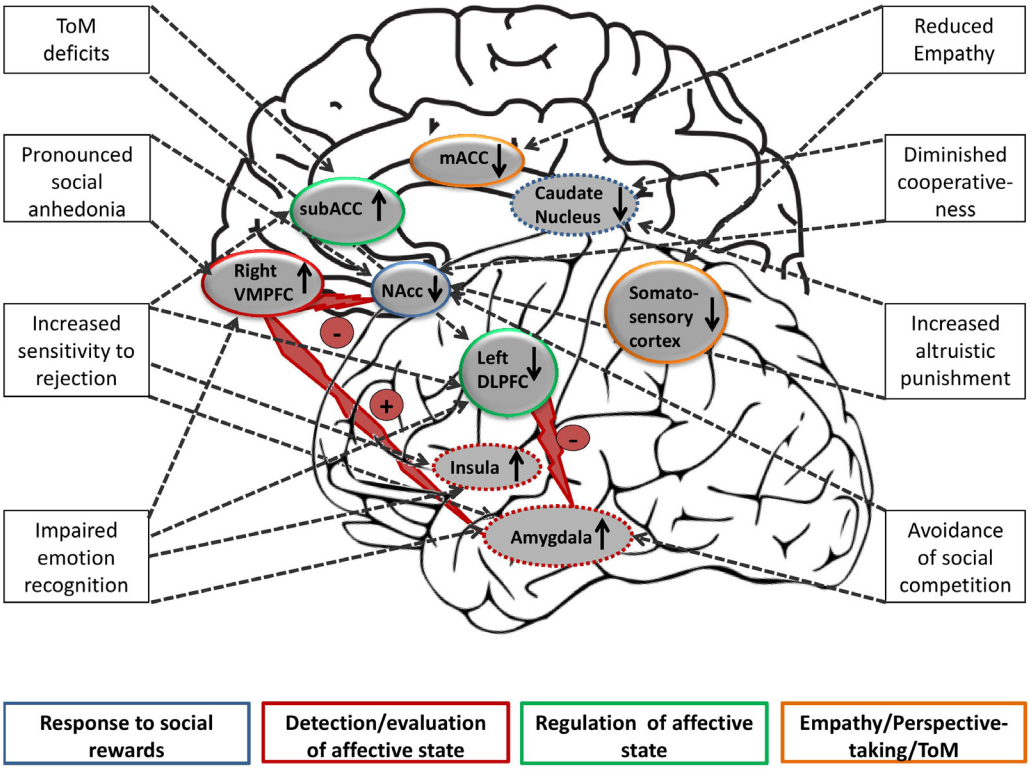

Fig. 1. Schematic model for the neural basis of social impairments. The figure depicts neural structures important for the processesunderlying social impairments and shows the relationship between functional abnormalities in certain brain regions and the observed social deficits in perception and behaviour. A predominantly ventral system is important for the identification of the emotional significance of a stimulus, the production of an affective state and emotional appraisal (depicted in red), whereas a predominantly dorsal system (depicted in green) is important for the effortful regulation of the resulting affective states. Other two systems associated with social impairments in depression are the social reward system (depicted in blue), which is related to socially competitive and cooperative behaviours, and the mentalizing/emphatic system (depicted in orange), which is related to social attribution and perspective taking. A functional relationship (facilitation or inhibition) may exist between certain brain regions (depicted by red arrows). Abbreviations: DLPFC: dorsolateral prefrontal cortex; mPFC: medial prefrontal cortex; mACC: medial anterior cingulate cortex; subACC: subgenual anterior cingulate cortex; NAcc: nucleus accumbens. (For interpretation of the references to colour in this figure legend and the text, the reader is referred to the web version of this article.)

et al., 2016). (Zimmer-Gembeck et al., 2016). It co-varies with a heightened emotional reaction to rejection and maladaptive behavioral responses such as social withdrawal (or isolating oneself from others) and aggressive retribution.

There are a number of tasks to measure the effects of peer rejection and assess its consequences on affective outcomes. One of this paradigms, "Cyberball" (Williams et al., 2000), is a virtual ball-throwing game where participants play a ball game online with two unknown fictitious partners (the participant is informed that these partners are real peers). After several trials, these fictitious partners appear to exclude the participant from play, leading to the feeling of social exclusion in the participant. Whereas Cyberball elicits peer rejection in a gaming context, the "Chatroom" paradigm (Guyer et al., 2009) has been developed to measure the effects of peer rejection during online social communication. In this task, participants are told that they are taking part in a study of online social interaction, and based on a photo, indicate their preference about which unknown peers they would like to chat with. Then, these peers make a decision as to whether to reject or accept the participant's request, based on a photo taken of the participant. Another task to measure social rejection is the "social feedback" task (Hsu et al., 2013). In this task, subjects complete an online personal profile that includes age, major/occupation, a list of their interests, a short paragraph of their positive qualities, and a picture of themselves. Then the subjects select online profiles of preferred-sex individuals with whom they would be most interested in forming an intimate relationship. During the task, subjects are presented with their highest-rated profiles along with feedback that they were not liked (rejection), or liked (acceptance).

3.1. *Findings from behavioural studies*

Social rejection is one of the strongest proximal risk factors for depression (Slavich et al., 2010) and there are indications that rejection prospectively predicts depression (Nolan et al., 2003). Peer rejection can be seen as a threat to self-preservation because it is associated with negative social evaluation and therefore a risk of social exclusion. Thus, rejection may give rise to self-conscious emotions like shame and lead to social withdrawal.

Administration of a "social feedback" task in the MDD has shown that ratings of the magnitude of feeling "sad and rejected" returned toward baseline levels after 5 min following a rejection trial for healthy controls, whereas MDD patients still had an elevated rating for that feeling (Hsu et al., 2015). Increased rejection sensitivity has been shown to predict higher rates of internal life stressors, which are at least partially influenced by the behavior and cognition of the individual (Liu et al., 2014). This internal generation of stress functioned as a mediational mechanism for the link between rejection sensitivity and subsequent depressive symptoms (Liu et al., 2014). A recent review has suggested that higher-level cognitive biases, such as attributional style and negative expectations may increase adolescents' depressive responses to peer rejection (Platt et al., 2013).

3.2. Findings from functional neuroimaging studies

3.2.1. Hypersensitivity to social rejection due to reduced endogenous opioid release in the amygdala

On the neuronal basis, a mechanism for impaired emotion regulation during social rejection might be an altered endogenous opioid activity in the amygdala of depressive individuals. For example, during peer rejection in the chatroom task, individuals with MDD show an increased activation in the amygdala (Silk et al., 2014). Consistent with that, administration of social feedback task in healthy volunteers (Hsu et al., 2013) and depressed patients (Hsu et al., 2015) has shown that amygdala response to social rejection is regulated by endogenous opioids and the μ-opioid receptor (MOR), which is involved in alleviating physical and emotional pain, including the effects of social rejection. The MRO activation in the amygdala of the healthy individuals is protective and may reduce the negative impact of stressors, since a greater predisposition for resiliency predicted a greater magnitude of MOR activation during rejection in the amygdala (Hsu et al., 2013). MDD patients in contrast to healthy participants showed MOR deactivation in the amygdala, which may contribute to blood-oxygen-level-dependent hyperactivity in this region in response to negative social cues such as peer rejection (Hsu et al., 2015). Recently, a polymorphism in the MOR gene has been found to influence neural and psychological responses to rejection, likely by affecting opioid receptor expression and signalling efficiency (Slavich et al., 2010).

3.2.2. MDD patients demonstrate increased emotional pain to rejection due to hyperactivity of the insula

Depressed individuals exhibit increased activation of the insula in response to peer rejection, suggesting that they might experience rejection as more affectively and motivationally salient/painful than healthy controls (Silk et al., 2014). This is supported by the finding that, during the cyberball game, individuals who showed greater activity in the insula reported greater feelings of social distress in response to social exclusion (Masten et al., 2009).

3.2.3. Failure to override negative emotions due to hypoactivity of the DLPFC

It has been proposed that vulnerability to depression is characterized by a failure to engage the PFC in response to a personally significant psychosocial threat of rejection and thus a failure to override other more automatic negative emotional responses of the amygdala and the insula (Hooley et al., 2005). Supporting this hypothesis, participants with a history of depression, compared with healthy controls, failed to activate DLPFC when they heard critical remarks (Hooley et al., 2005).

3.2.4. Negatively biased interpretation of peer rejection due to hyperactivity of the subACC

The subACC has also been implicated in altered emotion regulation following social rejection in the cyberball game (Masten et al., 2011). In online social interaction, greater subACC activity related to social exclusion was correlated with increased parent-reported depressive symptoms during the following year. This finding suggests subACC activity predicts depressive symptoms prior to disorder onset (Masten et al., 2011). The greater subACC sensitivity to peer rejection might result in more acute emotional responses and more negative interpretation of peer rejection due to the inability to properly regulate emotions resulting from such negative events (Masten et al., 2009). The absence of a link between subACC activity and concurrent depressive symptoms could indicate that this heightened activity in response to peer rejection represents a vulnerability among certain individuals that is cumulative over time (Masten et al., 2011). Thus, the activity in the subACC during peer rejection may also be predictive of individuals' risk for developing depression during late adolescence or adulthood, before symptoms potentially reach a clinical level (Masten et al., 2011).

3.2.5. Oxytocin and social exclusion

A recent study has shown that patients with chronic depression react to social exclusion during the cyberball game with pronounced negative emotions and reduction in plasma oxytocin levels which may contribute to interpersonal dysfunction and difficulty in coping adequately with aversive social cues (Jobst et al., 2015). Since oxytocin has been shown to facilitate the sensation of social stress (Eckstein et al., 2014), the reduction in oxytocin following social exclusion might act as a protection mechanism at the price of social isolation.

4. Avoidance of social competition

Freud's theory of the Oedipus complex assumes dysfunctional imbalances between attachment and competition in the pathogenesis of neurotic disorders. As a result, RDoC's attachment and affiliation construct might be associated with competition avoidance in depression. Alternatively, Price's social competition hypothesis of depression relates competition avoidance to RDoC's social communication construct. Based on an evolutionary theory, Price predicts that depressed individuals avoid competition when chances of winning are low and thus reduce the damage caused by a potential loss (Price et al., 2004). The involuntary subordinate strategy promotes the communication of a submissive, non-combative status of the defeated individual and thus inhibits aggressive behavior toward rivals. Further, the subjective sense of incapacity encourages acceptance of rank loss (Hagen, 2011). Indeed, depressed people often feel that they are losing competitions for support, acceptance and care, and believe that this is due to personal undesirable qualities (e.g. being boring, a failure, stupid, incompetent, weak, or unattractive) (Gilbert et al., 2009).

4.1. Findings from behavioural studies

In a recent study, we investigated competitive behavior in depressed patients using a simple motoric paper folding task (Kupferberg et al., 2016). In this study, each participant was matched with an opponent and could make the decision to choose between a cooperative and a competitive payment scheme before performing the actual task, which involved folding as many DIN-A4 papers as possible and then inserting them into envelopes within a 5-min period. The results demonstrated that depressed participants avoided competition much more often than did healthy controls, even although there were no significant differences in task performance (Kupferberg et al., 2016). Further, this study showed that the tendency to avoid competition was positively associated with the severity of depressive symptoms. We did not find competition avoidance in patients with borderline personality disorder and adult patients with autism (unpublished data), suggesting that this abnormality in social behaviour may be specific for MDD.

4.2. Findings from functional neuroimaging studies

To our knowledge, there are no studies, which investigated the neural correlates of competitive behavior in MDD. The few studies on social competition have provided preliminary evidence for the involvement of the inferior parietal cortex (Decety et al., 2004) during competition in healthy subjects. A recent study has demonstrated focal deactivation of bilateral inferior parietal cortex when a participant performing a skilled motor task was evaluated socially in real-time (Yoshie et al., 2016), indicating that this region might

be a possible target for neural substrates of competitive behavior, where one's own performance is evaluated by others.

Since competition is often associated with social rewards but also with the fear of losing, high reactivity of the stress system, involving insula, amygdala and ACC, in response to fear of losing and social exclusion may explain competition avoidance in patients with depression (Dedovic et al., 2009; Eisenberger et al., 2011; Onoda et al., 2010; Waugh et al., 2012) (see Fig. 1).

Competition avoidance in MDD might also be caused by the inability to experience pleasure from entering a competition and obtaining rewarding social stimuli. Depression is associated with reduced responding of the brain reward pathways to social and financial rewards, such that there is an attenuated activation of the NAcc (see Fig. 1) in response to the social reward stimuli (Schaefer et al., 2006; Smoski et al., 2009).

Further, an altered activity in the habenula (a structure located at the most caudal and dorsal part of the thalamus) might explain the reduced motivation to compete and reduced sensitivity to social reward by exerting a powerful inhibition on dopamine neurons (Hikosaka, 2010). Abnormal activity in the lateral dorsal habenula is an important correlate of clinical depression (Proulx et al., 2014). Interestingly, there is recent evidence from the preclinical research that this region plays an important role in the regulation of hierarchy-related behaviour and thus competition (Chou et al., 2016). In mice, projections from the ventral striatum to the lateral habenula control the rewarding effects of aggressive behavior, suggesting a link between altered NAcc circuitry in depression and alterations in competitive and aggressive behaviors (Golden, 2014).

Given that testosterone increases competitive tendencies (Mehta et al., 2009; Mehta and Josephs, 2006), low plasma testosterone levels in the depressed patients (Shores et al., 2005) might also be associated with competition avoidance.

5. Increased altruistic punishment

Altruistic behaviour is a form of prosocial behaviour and is related to strengthening social bonds and attachment. A widely experimental task for studying prosocial behavior is called the "Ultimatum Game" (UG). The UG is a well-studied decision task which is used as a paradigm to study fairness by investigating how people sanction non-cooperative behaviour in experimental neuroeconomics (Emanuele et al., 2008). It represents a simple two-person bargaining scenario between a proposer and a responder. The proposer offers the responder a proposal about how to split a sum of money. The responder then decides whether to accept or reject the proposal. If the responder accepts the proposal, each player earns money according to the proposer's offer. If the offer is rejected, neither player receives any money. The rejection of low offers is considered to be an expression of costly punishment of unfair behavior and is motivated by the individuals' prosocial preferences for fairness. The responder is willing to impose the cooperation norm on the proposer and punishes him/her for the violation of this social norm (Fehr and Fischbacher, 2003). The rejection of "free money" is costly and is therefore called "altruistic punishment" (Emanuele et al., 2008). Although irrational economic decision making has been consistently shown in MDD (Radke et al., 2013; Scheele et al., 2013; Wang et al., 2014) with a positive relation to depression severity (Wang et al., 2014), alterations in the acceptance of unfair offers have also been demonstrated in individuals with bipolar disorder (Duek et al., 2014) and schizophrenia (Csukly et al., 2011). Further, individuals with psychopathy showed more altruistic punishment than controls (Masui et al., 2011. Thus, over-sanctioning of unfair offers is not specific for MDD.

5.1. Findings from behavioural studies

Using UG, Radke et al. (2013) and Wang et al. (2014) demonstrated that patients with depression rejected more unfair offers than did healthy controls, and patients with recurrent depression even over-sanctioned unfair proposals in the UG both at the beginning and after 6 weeks of inpatient treatment (Scheele et al., 2013). The theory that depression is associated with increased fairness concerns and altruism was further confirmed by studies that involve hyperfair offers, which present a beneficial prospect for the individual. Thus, Radke et al. (2013) demonstrated that, when compared to healthy controls, depressed patients did not only reject more unfair offers, but also rejected hyperfair offers. Similarly, patients with severe depression offered more money in the UG (Destoop et al., 2012). There are two possible explanations for this kind of prosocial behavior. First, the MDD patients might offer significantly higher amounts in order to avoid rejection from their opponent. Alternatively, anhedonia might lead to attenuated responses to monetary gains and blunted reward responsiveness (Henriques and Davidson, 2000). Both alternatives are related to the RDoC construct "Attachment and Affiliation".

The finding of reduced acceptance rates in depression appeared to be causally related to the sad mood (Harlé and Sanfey, 2007). Thus, in a depression induction experiment, Harlé and Sanfey (2007) have shown that induced sadness resulted in lower acceptance rates (41% in contrast to 56%) of unfair offers, even when the alternative was no gain at all. The authors suggest that sadness may focus the responder's attention on the negative emotional consequences of unfair offers rather than the positive impact of accepting such offers (i.e., monetary reward), thereby prompting lower acceptance rates of unfair offers. However, the increased feeling of fairness in the depressed patients might have also facilitated imposing the cooperation norm by punishing the proposer.

Although reduced acceptance rate in the UG has been consistently related to depression (Destoop et al., 2012; Harlé and Sanfey, 2007; Scheele et al., 2013), there is one study applying UG in depression that has yielded conflicting results. Thus, Harlé et al. (2010) have shown that depressed participants accepted significantly more unfair offers (61%) than controls (41%). However, this study had several methodological limitations, which makes this conflicting finding questionable. For example, the study sample was derived from a student population and classified based on BDI scores, which tend to be lower than those obtained from the clinical patients.

5.2. Findings from functional neuroimaging studies

5.2.1. Reduced response to social reward due to hypoactivity of the striatum

One reason for lower acceptance rate of unfair offers might be a decreased responsiveness to social rewards. Supporting this explanation, depressed participants reported decreased levels of happiness in response to 'fair' offers in comparison to healthy controls (Gradin et al., 2014). In this study, the control group showed a positive correlation between the fairness of offers and the activation of both NAcc and dorsal caudate nucleus – regions, both of which have been shown to process social information and responses to social rewards. In contrast, the participants with depression did not show this activity correlation with increasing fairness, which may contribute to an increased rejection rate.

5.2.2. Serotonin deficiency

A potential physiological mechanism for the decreased acceptance rate of unfair offers may be a serotonin deficiency (Hasler, 2010). Serotonin deficiency possibly leads to a decreased activity in the striatum by affecting dopaminergic terminal function

(Navailles and De Deurwaerdère, 2011) and thus gaining less pleasure from social rewards. In line with this hypothesis, lowering serotonin levels by depleting of the serotonin precursor tryptophan produced increased rejection of the unfair offers (Crockett et al., 2010, 2008). On the other hand, enhancing serotonin levels with citalopram led to more acceptance of unfair offers, indicating that citalopram increased the aversion to harm others by increasing serotonin (Crockett et al., 2010). A more recent study showed that individuals with a low level of serotonin transportation in the dorsal raphe nucleus were more likely to be honest and trustful and could not tolerate unfair offers (Takahashi et al., 2012).

6. Impaired emotion recognition and mood-congruent emotional bias

The RDoC domain "Social Communication" refers to a dynamic process that includes both receptive and productive aspects used in social interactions. In depression, there is growing evidence that impaired emotion recognition and a negative emotional bias contributes to deficits in the receptive communication aspect. Facial communication deficits have been particularly well studied. Impaired emotional facial expression recognition is related to the risk of developing MDD, as well as to its maintenance and relapse (Foland-Ross and Gotlib, 2012; Mathews and MacLeod, 2005). Specifically, the recognition of happy facial expressions is negatively correlated with the severity of depressive symptoms (Csukly et al., 2009). However, the impaired emotion recognition from socially relevant stimuli in MDD is not unique for MDD, being typical also for anxiety disorders (Bourke et al., 2010; Demenescu et al., 2010), autism spectrum disorder (Baribeau et al., 2015) and bipolar disorder (Phillips et al., 2003). In bipolar disorder, the deficits in the ability of emotion recognition were more pronounced in patients suffering from Bipolar I Disorder (Derntl et al., 2009). Schizophrenic patients have also been shown to have difficulties in recognizing fear expressions (Baez et al., 2013).

6.1. Findings from behavioural studies

Most studies investigating emotion recognition and perception in MDD have focused on the processing of neutral facial expressions and of basic affective expressions such as sadness, happiness, anger, fear and disgust (Maniglio et al., 2014; Tse and Bond, 2004). Although some older studies showed similar accuracy of facial emotion recognition for depressed patients and healthy controls (Hertel et al., 2009; Kan et al., 2004; Maniglio et al., 2014), taken together, results indicate a general deficit in emotion recognition of basic facial emotional expressions in patients with major depression (Dalili et al., 2015). Further, there is a bias toward the identification of emotional information as negative or sad so that positive (happy), neutral or ambiguous facial expressions tend to be evaluated as more sad or less happy compared with healthy control group (Bourke et al., 2010). Furthermore, compared with healthy volunteers, depressed subjects rated the intensity of negative emotions as higher (Naranjo et al., 2011) and the intensity of positive facial expressions as lower (Yoon et al., 2009). Additionally, depressed patients required significantly greater intensity of emotion to correctly identify happy expressions as happy both in acute stages (Joormann and Gotlib, 2006; Münkler et al., 2015) and in remission (LeMoult et al., 2009). Finally, they showed impaired recognition accuracy for emotional expressions of subtle intensity (Gollan et al., 2010).

The negative emotional bias in recognition of social information becomes obvious not only when it comes to recognition of facial expressions, but also during perception to emotional body language (e.g., posture or speed of movement). For example, a recent study has shown that MDD patients demonstrated poorer emotion recognition accuracy for happy body language stimuli that did not include facial expressions (Loi et al., 2013). Additionally, the negative emotional bias was demonstrated when emotions were expressed vocally. In comparison to healthy controls, depressive patients showed impairments in recognition of fear, happiness and sadness from emotional words spoken by actors and rated happy stimuli as more fearful and sad (Péron et al., 2011).

6.2. Findings from functional neuroimaging studies

Functional imaging studies suggest that emotion depend on the functioning of two neural systems: a ventral system and the dorsal system. The ventral system, encompassing the ventral PFC, amygdala and insula, is important for the identification of the emotional significance of a stimulus and the production of affective states. The dorsal system, including the dorsolateral prefrontal cortex (DLPFC) and anterior cingulate cortex (ACC), is important for the effortful regulation of affective states (Phillips et al., 2003).

6.2.1. Hyperactivity of the amygdala and ventromedial prefrontal cortex lead to mood-congruent processing bias of emotional facial expressions

There is evidence that during emotion processing, MDD patients showed abnormalities in a common face processing network, indicating mood-congruent processing bias (hyperactivity to negative and hypoactivity to positive stimuli) particularly in the amygdala, insula, parahippocampal gyrus, fusiform face area, and putamen (Stuhrmann et al., 2011). Since amygdala has connections with prefrontal regions and striatum (Roy et al., 2009), its abnormal response might lead to alterations in productive aspects of communication.

Functional imaging studies have consistently reported higher metabolism in ventral PFC during emotional processing (Phillips et al., 2003) and hyperactivity of amygdala in response to sad and fearful facial expressions in depressed individuals (Godlewska et al., 2012; Greening et al., 2013; Mingtian et al., 2012; Stuhrmann et al., 2011). This hyperactivity includes sustained amygdala responses to emotional stimuli (25s in comparison to 10s in healthy controls) (Siegle et al., 2002). Additionally, an increased connectivity between subgenual parts of the ACC (subACC) and the amygdala, causing mutually enhancing abnormal emotion processing during observation of fearful faces, may represent a neural mechanism for the abnormally increased representation of social threat, which is particularly prevalent in depressed in patients with a comorbid social anxiety disorder (de Almeida et al., 2011; Stuhrmann et al., 2011).

6.2.2. Amygdala and insula hyperactivity during processing of subtle emotional expressions

Our understanding about the neural mechanisms underlying emotion attribution to complex social emotions such as pride, guilt and shame is scarce, but would be very relevant to understanding MDD. Using fMRI, it has been shown that recognition of the social emotional expression of shame leads to an enhanced neuronal response within the right amygdala and posterior insula in depressed patients (Pulcu et al., 2014). This is in line with the notion that shame requires an imagined critical observer and thus is associated with brain activity in regions linked to sensory perception of emotionally relevant stimuli. In addition, reduced bilateral anterior insula volumes have been associated with pathological guilt in preschool depression ant the risk for MDD later in life (Belden et al., 2015).

6.2.3. Serotonin deficiency associated with amygdala hyperactivity

Lowering brain serotonin using acute tryptophan depletion leads to an increased negative affective bias as a result of a change in the connectivity between mPFC and amygdala (Robinson et al., 2013), which has been associated with the risk of depression (Roiser et al., 2012). In addition, evidence from neuroimaging-genetics studies indicates that amygdala response to fearful faces depends on allelic variation in the promoter region of the serotonin transporter (5-HTT) gene (Hariri et al., 2002). Individuals carrying one or two copies of the short allele of the 5-HTT-linked polymorphic region (5-HTTLPR), which is associated with reduced 5-HT transporter expression, are termed 's-carriers'; they display greater levels of trait anxiety and are at greater risk for depression following stress (Caspi et al., 2003). However, although there may indeed be a very small effect, this early finding of association between carrying the short form of the allele and depressive disorder has not been uniformly replicated as shown in a more recent meta-analysis by Clarke et al. (2010). Corroborating human findings, mice lacking the gene encoding 5-HTT show impaired stress coping and increased spine density in the basolateral amygdala (Wellman et al., 2007). 5-HTT knockout mice also show reduced aggressive behavior (Holmes et al., 2002) and diminished social interaction (Kalueff et al., 2007; Moy et al., 2009), which may reflect a social anxiety phenotype (Calcagnoli et al., 2015; Neumann and Slattery, 2016).

6.2.4. Impaired top-down control of emotional processing by the DLPFC

Studies employing functional neuroimaging techniques during emotional processing in MDD have consistently demonstrated reductions in metabolism within the DLPFC but increased metabolism and blood flow within the ventral PFC (Phillips et al., 2003). A meta-analysis has reported hypoactivity in the DLPFC (Groenewold et al., 2013) during emotional processing in MDD (see Fig. 1). Specifically, the right DLPFC has been shown to be less active in individuals with depression than in healthy controls during observation of threat-related facial expressions (Mingtian et al., 2012) and of fearful faces, even if these were not explicitly attended to (Fales et al., 2008). The hypoactivity of DLPFC could lead to a failure to override the more automatic negative responses of the amygdala to emotional faces (Zhong et al., 2011), leading to more negative evaluation of emotional expressions during interaction and thus social withdrawal and feeling of rejection. The decreased connectivity between amygdala and DLPFC has also been found in social anxiety disorders and other stress-related conditions, suggesting it is not a unique feature of MDD (Prater et al., 2013).

7. Diminished cooperativeness

Social cooperativeness is a construct derived from behavioral economics. It does not relate to a specific RDoC construct and is rather the higher-order consequence of various constructs, including affiliation and social communication. Diminished cooperativeness is prevalent in depressed individuals but not specific for MDD at all, occurring in almost all major psychiatric disorders. It may be clinically most prominent in psychopathy (Mokros et al., 2008) and borderline personality disorder (Saunders et al., 2015).

Behavioral economics has provided a theoretical framework that allows for the development of experiments to study cooperative behavior in the laboratory setting (Clark et al., 2013). One of such game-theory based experiments is referred to as the Prisoner's Dilemma (PD), which measures the reciprocity in a situation in which cooperative actions lead to the best outcome for all participants, but where free riders or non-cooperative individuals can benefit the most individually. Another measure of cooperativeness is the "public goods game", which confronts individuals with the temptation to defect, i.e., to take advantage of the public good without contributing to it. Thus, the public goods game measures the willingness to contribute to the public good, which reflects cooperation and adherence to social norms.

7.1. Findings from behavioural studies

Using PD to investigate cooperative behavior in MDD has shown that depressive symptoms were linked to difficulty sustaining reciprocal cooperation (Clark et al., 2013). Similarly, another recent study showed that depressive patients defected significantly more in a hypothetical survival situation while interacting with forgiving partners (Pulcu et al., 2015). Further, it has been shown that depressed patients make fewer contributions in the public goods game (Clark et al., 2013).

7.2. Findings from functional neuroimaging studies

In healthy individuals, mutual cooperation has been linked to neural activation of the reward system (Rilling et al., 2002). In comparison to healthy individuals, depressed patients did not show an increased activation in the NAcc and dorsal caudate when contrasting the mutual cooperation condition vs. the condition where the partner defects (Gradin et al., 2016). This finding indicates that MDD patients gain less pleasure from social rewards in comparison to the healthy individuals.

In mice, formation of social reward requires coordinated activity of oxytocin and serotonin within the NAcc (Dölen et al., 2013). In humans, intranasal oxytocin increased prosocial behavior (donate money, reduction in non-verbal behaviors that cut off social contact) and improved social cognition (increased accuracy in a task measuring the understanding of mental states) in individuals with MDD (MacDonald et al., 2013; Marsh et al., 2015). Allelic variation in the oxytocin receptor gene has been associated with increased symptoms of depression (Thompson et al., 2011).

8. Reduced empathy

Following the RDoC concept, deficits in the perception and understanding of others, particularly action perception and understanding mental states, leads to reduced empathy. Empathy plays a central role in successful interpersonal engagement by serving as a means for establishing rapport and building a basis for trustworthy communication (Baron-Cohen and Wheelwright, 2004). In depression, reduced awareness of others' emotions (Donges et al., 2005) may impair social abilities and lead to misunderstandings in social interaction, potentially reducing the quality of interpersonal relationships (Kronmüller et al., 2011). Impaired affective responsiveness as well as high empathic distress (an emotional state characterized by the inability to tolerate the perceived pain or suffering of another) correlates with the severity of depression (Donges et al., 2005). Similar to impaired emotion recognition, deficits in empathic abilities and perspective taking are not specific for MDD, but have also been demonstrated in patients with borderline personality disorder (Roepke et al., 2013) as well as bipolar disorder and schizophrenia (Baez et al., 2013; Cusi et al., 2010; Seidel et al., 2012). In fact, schizophrenic patients were shown to have the strongest impairment in emphatic abilities followed by bipolar patients and MDD (Derntl et al., 2012).

8.1. Findings from behavioural studies

In experimental studies on empathy, depressed individuals expressed lower pain ratings when evaluating painful videos

compared with healthy controls (Fujino et al., 2014). This finding suggests MDD patients experienced diminished feelings of compassion, which may lead to miscommunication with others. Interestingly, depressive symptoms have been associated with lower levels of empathic accuracy among women, but not among men (Gadassi et al., 2011). For example, depressed mothers were less responsive to crying of their newborn babies (Field et al., 2009) and showed reduced affective touching, such as slow caress or stroke, of their babies than healthy mothers (Young et al., 2015).

An older study has indicated that seemingly reduced empathy in depressed individuals might be caused by higher personal distress when facing another's stressful situation (Batson et al., 1997). In line with this hypothesis, depressed patients scored significantly higher on ratings of personal distress when presented with emotional situations, indicating a decreased ability to tolerate the perceived pain or suffering of another (O'Connor et al., 2007). Thus, although people who are depressed most often have normal or elevated levels of empathy, their affect-directed, automatic causal interpretations of pain in others are often disturbed, leading to non-conscious assertions of blame, usually placed on themselves (O'Connor et al., 2007). This personal distress may motivate depressed individuals to withdraw from stressful social situations and avoid similar situations in the future (Seidel et al., 2010), which can be perceived by others as low empathic concern (Schreiter et al., 2013).

8.2. Findings from functional neuroimaging studies

To our knowledge, there is only one study, which investigated the neural correlates of empathy in MDD. This study found that the deficit in the identification of pain of others in MDD was associated with reduced cerebral activation in the right somatosensory-related cortices and the left middle anterior cingulate cortex (mACC) (Fujino et al., 2014). One interpretation of these data is that altered activation of the somatosensory-related cortices led to difficulties using social cues for understanding emotional states of others (Keysers et al., 2010), and abnormal activation of the left mACC led to deficits in the ability to evaluate pain processing (Guo et al., 2013; Jackson et al., 2006).

9. Theory-of-mind deficits

The RDoC constructs "Perception and Understanding of Self" and "Perception and Understanding of Others" relate to social processes and representations involved in awareness, knowledge, and reasoning of oneself and other people, making judgments about other animate entities, including information about cognitive and affective states, traits and abilities. The ability to infer mental states including intentions, desires and pretending to oneself and others, of other individuals, is called theory of mind (ToM) or "mentalizing" (Premack and Woodruff, 1978). In the RDoC, ToM is also included in the "social perception" domain.

ToM deficits typically occur in patients with schizophrenia (Brüne, 2005), autism (Kana et al., 2014) and across the mood states of bipolar disorder (Kerr et al., 2003; Mitchell and Young, 2016). However, a recent meta-analysis has shown that ToM deficits also contribute to social impairments in depression, since the ability to mentalize is essential for effective and adaptive interpersonal functioning and communication (Bora and Berk, 2016).

Impaired ToM has not only been associated with increased risk for depression (Inoue et al., 2006) but also with the severity of acute and remitted depressive symptoms as indicated by a recent meta-analysis (Bora and Berk, 2016).

The clinically relevant measures of ToM and knowledge about thinking include clinical interviews, second order false-belief tests, understanding intentions, metaphor, sarcasm, jokes and "Reading the Mind in the Eyes" (RMET) test (Sprung, 2010). The RMET-test is an advanced test of theory of mind involving mental state attribution and complex facial emotion recognition from photographs where only the eye region of the face is available (Baron-Cohen et al., 2015).

9.1. Findings from behavioural studies

Using a semi-structured clinical interview designed to elicit thoughts, feelings, and memories about early attachment experiences, one study demonstrated a significantly lower capacity for mentalization in depressed female inpatients compared with the healthy controls (Fischer-Kern et al., 2013). Additionally, patients with depression performed worse than healthy controls in guessing the intentions of protagonists in short stories (Wang et al., 2008). Further, studies which examined the second-order beliefs in patients with MDD showed a ToM deficit even in the state of remission (Inoue et al., 2004). Second-order beliefs refer to the ability to infer what one person thinks about another person's thoughts (Astington et al., 2002). This ability is strongly correlated with skilful social relationships. Since interpersonal conflict is a strong precipitating factor in relapse, it is not surprising that depressed patients with a ToM deficit in second-order false beliefs during remission may be at higher risk for recurrence, and have lower social function one year after recovering from a major depressive episode (Inoue et al., 2006).

Although some basic aspects of ToM, like the pure decoding of mental states, might be preserved in depression, more complex mechanisms, such as reasoning about mental states, involving the integration of contextual information about the other person and the situation, seem to be more markedly impaired (Wolkenstein et al., 2011). It seems that the deficit lies not so much in general decoding of mental states from observable cues, but in drawing valid conclusions about the mental states of other people in social contexts (Wolkenstein et al., 2011). The lack of understanding of others' mental states in specific contexts can contribute to inadequate social reactions. For example, in more complex tasks, such as understanding humour, Uekermann et al. (2008) demonstrated that depressed patients performed below the control group. Further, individuals with MDD showed an overall lower interpretation quality of others' sarcastic remarks (Thoma et al., 2015). Finally, Ladegaard et al. (2014) reported a deficit for the interpretation of paradoxical but not of simple sarcasm in their MDD cohort. Paradoxical sarcasm only makes sense if the participant understands that one of the speakers is being sarcastic.

9.2. Findings from functional neuroimaging studies

Although ToM plays a fundamental role in social cooperation, its functional aspects and corresponding neural mechanisms are not fully understood. Findings from a review of imaging studies investigating the neural correlates of ToM indicate that there are several "core" regions which support the ToM reasoning (Carrington and Bailey, 2009). These regions are part of a widely distributed network including parts of the PFC, temporoparietal junction and superior temporal sulcus as well as several more "peripheral" regions such as supplementary motor area, ACC and insula. ToM deficits in depression might be related both to functional abnormalities and dysconnectivity in brain networks that play a role in ToM. There is well established evidence indicating structural and functional impairments in brain networks in depression that include abnormalities in regions that play a role in ToM such as ventromedial prefrontal and temporoparietal regions (Cusi et al., 2012). However, there are few studies which directly investigated neural correlates of ToM-abilities in MDD.

Using transcranial electrical stimulation, a recent study suggested dysfunctional left DLPFC activity during perspective taking (Conson et al., 2015). Supporting this finding, Berlim et al. (2012) showed an improved performance in the RMET- task and the alleviation of depressive symptoms after a 10 Hz-rTMS to the left DLPFC. Additionally, using a new method of near-infrared spectroscopy (NIRS), which detects regional cerebral blood volume changes in superficial brain regions, Takei et al. (2014) reported a decreased continuous activation in the left DLPFC in MDD compared with healthy subjects during face-to-face conversations, indicating an impaired adaptation during communication with others (Takei et al., 2014). Further, one study demonstrated that participants with remitted depression showed attenuated mPFC response relative to controls for both positive and negative images depicting social interactions (Elliott et al., 2012). Reduced responses to these images might reflect ToM deficits.

Another reason for impaired mentalizing abilities in MDD might be an increased activity in anterior subACC, which is involved in the recognition of emotional states (Phillips et al., 2003). During RMET, depressed subjects show elevated subACC activity, indicating a more primary, impulsive way of mentalizing (Pincus et al., 2010). Consistent with the increased activity, a meta-analysis on volumetric fMRI studies has revealed a volume reduction of ACC compared with healthy control subjects (Drevets et al., 2008; Zhao et al., 2014). Interestingly, in mice, observational fear learning requires activity of the ACC (Jeon et al., 2010), suggesting a conserved network for social learning that may allow us to study neurobiological mechanisms of depression-induced alterations in response to emotional states of others.

10. Abnormal use of social media

Many adolescents consider the internet a highly important medium in their everyday social life and use it to form and maintain social relationships (Valkenburg and Peter, 2007; Wolak et al., 2003). Thus, studying the use of social media provides psychological researchers with an important tool to assess virtual social interactions.

Apart from MDD (Katikalapudi et al., 2012; Kim et al., 2006; Pantic et al., 2012), alterations in social media have been demonstrated in other psychiatric populations, for example patients with schizophrenia were increasingly connected to and engaged with social media (Torous and Keshavan, 2016). In bipolar mania, more facebook friends predicted more clinical symptoms (Rosen et al., 2013) and addiction to social media is associated with social anxiety in young adults (Ko et al., 2012; Weinstein et al., 2015).

10.1. Findings from behavioural studies

Traditional social contacts are often formed and upheld through religious activity and marriage, which are strongly associated with a reduced risk of depression (McKenzie et al., 2013). In contrast, the results of several cross-sectional survey studies have indicated that individuals with more psychosocial risk factors or detrimental health behaviors were more likely to use email and chatrooms rather than more traditional means of creating social bonds (Sun et al., 2005). In both high-school (Kim et al., 2006; Pantic et al., 2012) and college students (Katikalapudi et al., 2012), depression was associated with more frequent usage of social media.

Currently, the causal relationship between depressive symptoms and excessive use of social media remains unclear. On the one hand, levels of depression and loneliness predicted the level of preference for online social interaction over real-life face-to-face interaction (Caplan, 2003; Weiser, 2001). Excessive online social interaction might prevent individuals from engaging in in-person social activities and negatively impact the development and maintenance of social relationships (Amichai-Hamburger and Ben-Artzi, 2003; Caplan, 2003; Weiser, 2001), thus leading to depressive symptoms. This suggests a reciprocal relationship between using social media and depressive symptoms. One reason for this link may be cyberbullying – a form of bullying using electronic devices such as computers and mobile phones, which becomes more prevalent with the growing use of social media by the society (Hinduja and Patchin, 2010). Cyberbullying victimization led to an increase in depressive symptoms, which in turn increased the probability of cyberbullying (Gámez-Guadix et al., 2013). Ten out of thirty-five studies found a statistically significant association between being cyberbullied and reports of depression (Hamm et al., 2015), indicating that cyberbullying can be relentless and discouraging, especially if the bully is anonymous.

In contrast to the example of cyberbullying, studies have shown that the use of social media can lessen depressive symptoms if the variable of envy is controlled for (Tandoc et al., 2015). In this case, it was not the quantity of social networking site usage per se that was associated with depressive symptoms, but rather the negative quality of peer interactions such as social comparison and feedback-seeking (Nesi and Prinstein, 2015). In fact, studies have shown that social media may present an effective compensatory strategy for social deficits in MDD (Campbell et al., 2006; Shaw and Gant, 2002). For example, people who are depressed may try to cheer themselves up by going online and talking to their friends, which increases perceived social support and decreases feelings of loneliness and depression (Shaw and Gant, 2002). One reason depressed individuals may choose to interact through social media instead of in person, is that the negative emotional affect when being rejected in an online interaction is not as high (Caouette and Guyer, 2016).

10.2. Findings from functional neuroimaging studies

One explanation for the increased use of social media in MDD might lie in the fact that individuals with MDD experience problems maintaining offline relationships, in part due to a fear of rejection. This fear might be less pronounced in online social relationships with anonymous individuals. On the other hand, depressed participants might search social rewards and gratification from usage of social networking sights. Social online interaction might improve their mood, since it might be associated with the activation of the social reward system. Thus, it has been shown that, for example, use of facebook (Meshi et al., 2013; Turel et al., 2014) and sharing information about oneself (Tamir and Mitchell, 2012) both activate NAcc. These findings indicate that individuals who compulsively use facebook exhibit similar brain patterns to people suffering from drug addiction, although, in contrast to drug addiction, they retain adequate activity in the PFC.

11. Dysfunctional social networks

Dysfunctional social networks and lack of social support is a prevalent consequence of impaired social processes including attachment and affiliation, social communication and deficits in social perception. The lack of emotional control typical of MDD presents as crying (Rottenberg et al., 2002), verbal aggression (Kahn et al., 1985), irritability (Pasquini et al., 2004), and impulsivity (Hur and Kim, 2009), which can have a substantial effect on social relationships through impaired emotion-regulation in various interpersonal situations (Schreiber et al., 2012). The interaction between depressed individuals and their family or friends appears to be characterized by specific patterns of communication, such as burdening or alienating romantic partners (Benazon and Coyne,

2000). For example, in social interactions with family and friends, individuals with MDD often demand support in a hostile manner (Rehman et al., 2010) and have a tendency to seek excessive reassurance from others to relieve their self- doubts and deficits (Evraire and Dozois, 2011). Although in the beginning others may provide support, the depressed person may doubt the authenticity of this support and may be unable to use the feedback signal of reassurance. Thus, friends and relatives may frequently become annoyed and feel burnt out by repeatedly having to provide reassurance, which can ultimately lead to rejection of the depressed individual.

The potentially severe consequences of social behavior impairments described above have also been demonstrated by studies investigating real-life social networks in the context of depression. The occurrence of depression is twice as high in people with the lowest overall quality of social relationships in comparison to those with highest quality (Teo et al., 2013). Similarly, dysfunctional social networks are common in schizophrenia (Harley et al., 2011) and bipolar disorder (Michalak et al., 2006), with manic episodes having a more deleterious effect on social relationships than depressive episodes (Romans and McPherson, 1992). Social isolation is associated with and predicts the development of depression (Chou et al., 2011; Teo et al., 2013). Thus, having very few or no friends correlates with risk of developing depressive symptoms (Brendgen et al., 2000; Ueno, 2005). For example, findings from a US national survey (2005–2008) demonstrated a linear relationship between the number of close friends and the presence of depression, where the lowest prevalence of depression occurred in individuals with 10 or more close friends, and the highest was seen among those who self-reported no close friends (McKenzie et al., 2013). However, it has to be noted, that individuals with both a very low (between 1 and 7) a very high number of acquaintances and friends (between 15 and 21) tend to experience more depressive symptoms than individuals with less extended social networks (between 7 and 15) (Falci and McNeely, 2009). The authors suggested that this may be due to excess of feelings of duty or obligation to a high number of peers, since friendship entails a set of behavioral expectations, such as providing comfort or assistance and spending time together. In girls, a large, fragmented network consisting of friends who were not friends with each other further increased risk of depression because the care of maintaining such networks involved even more work (Bearman and Moody, 2004). In addition, the perceived quality of social relationships significantly interacted with depressive symptoms, such that an increase in perceived quality of social relationships ameliorated the harmful effect of depressive symptoms (Lee, 2015).

Rosenquist et al. (2011) have shown that depressive tendencies seem to travel along social networks, and that depression scores are strongly correlated with depression measures in one's friends and neighbors. A similar result was found by another study, showing that, over time, adolescents' depressive symptoms increasingly converged toward the average levels of their peers, independent from the level of the peers' depressive symptom severity (Kiuru et al., 2012). Further, adolescents tended to select friends with similar levels of depression, and friends might increase each other's depressive symptoms as relationships endure (Van Zalk et al., 2010).

An important methodological tool for unraveling social complexity at the individual, relationship, and group levels offers the social network analysis, which examines social behaviors of the individuals within the context of their own direct relationships with others as well as their indirect relationships. The application of social network analyses in large cohorts of mice living in complex environments (So et al., 2015; Williamson et al., 2016) potentially provides an opportunity to mechanistically study neural correlates relating to social network formation and maintenance. These experiments have revealed stable linear dominance hierarchies that establish within a few days (Williamson et al., 2016). Interestingly, a study by So et al. (2015) demonstrated the presence of distinct social networks for different social behaviors, including aggressive interactions and affiliative behaviors such as grooming. These paradigms have pointed to stress related hormones, such as corticotropin-releasing factor (CRF), acting within brain regions such as the medial and central amygdala and the medial preoptic area of the hypothalamus as predictors of dominance in large social networks (So et al., 2015). In the future, it will be interesting to use these paradigms to assess alterations in social networks in mice with genetic trait or experience-dependent vulnerability to depression.

12. Currently available and potential interventions for addressing social impairments

Reliable assessments of social impairments in MDD and the neurobiological understanding of such impairments will allow for the development and application of stratified treatments adjusted to specific social dysfunctions. Such advances have the potential to improve prognosis and functional outcomes for depressed patients.

Our findings indicate that treatment approaches targeting problematic aspects of social processing may be of particular benefit. However, most psychotherapy and pharmacotherapy trials in depression have focussed on the reduction of depressive symptoms, rather than improvement of social functioning (Silva et al., 2013). Unfortunately, reduction in symptom severity in depressed patients does not denote normal quality of life or social functioning (Cohen et al., 2013).

The wide-spread occurrence of social impairments in mental disorders like MDD, schizophrenia, BPD, and social anxiety as well as high comorbidity and similar genetic, familial, and environmental risk factors underlying them, suggest transdiagnostic strategies for the improvement of social functioning, without tailoring the treatment approach to specific diagnoses. The transdiagnostic approach is also compatible with the RDoC, since RDoC focuses on the underlying mechanisms that cut across multiple disorders.

12.1. *Effects of psychotherapy on improving social deficits*

Since interpersonal deficits are common to a number of mental disorders, one of the transdiagnostic strategies could be a cognitive behavioral analysis system of psychotherapy (CBASP). In this approach, the therapist and patient collaboratively and systematically analyse brief, distressing interpersonal interactions in order to show the patient new perspectives on how to interact with others that may result in more satisfying interpersonal interactions (Hames et al., 2013). Using a transdiagnostic, emotion-focused cognitive-behavioral treatment (CBT), applicable across anxiety and mood disorders, (Ellard et al., 2010) has shown improvement in social adjustment and social relationships in depressed patients.

Another approach to cope with social deficits is interpersonal therapy (IPT), which is based on the premise that depression affects individual's relationships to family and peers, and these relationships affect his or her mood. A recent study has shown significant improvements in interpersonal functioning and changes in attachment style following IPT in depressive adolescents (Spence et al., 2015). The long-term goal of interpersonal therapies is to not only reduce the depressive symptoms, but primarily to enable people with depression to make their own appropriate social adjustment and better cope with social situations, which is supposed to have a positive effect on depressive symptomatology. Interestingly, reduction of depressive symptoms alone is insufficient to fully explain

improvement in social functioning as shown by a meta-analyses on psychotherapeutic effects on social functioning (Denninger et al., 2011; Renner et al., 2014).

Although most psychotherapies used for the treatment of depression address social conflicts, a recently published meta-analysis indicated that the positive effect of psychotherapy on social functioning was only small to medium (Renner et al., 2014). Further, although interpersonal therapies specifically aim to improve social skills compared with other forms of psychotherapy, a meta-analysis in adult and older adult depressed patients showed no differences in improvements in social functioning between different types of psychotherapeutic interventions (CBT vs. others; IPT vs. others) after controlling for publication bias (Renner et al., 2014). However, these results must be interpreted with caution since there is a low consensus on the measurement of social functioning between different studies, with usage of different scales. Furthermore, in some studies, social functioning was assessed by self-report, and in others it was evaluated by clinicians. Therefore, only standardized assessment of social functioning will enable us to evaluate the effectiveness and compare the effects of different types of treatments on social functioning. In addition, more basic research is needed to develop psychological interventions that target specific social impairments in depression.

12.2. Effects of pharmacotherapy on improving social deficits

The reported effects of pharmacotherapy on social functioning vary considerably. A meta-analysis showed that the effect of norepinephrine reuptake inhibitors and selective serotonin reuptake inhibitors on improvement of psychosocial functioning is moderate (Papakostas et al., 2008). Briley and Moret (2010) reported that selective noradrenaline reuptake inhibitors and dual antidepressants inhibiting both noradrenaline and serotonin seem to be rather more effective in improving social functioning than selective serotonin inhibitors. Additionally, the two neurotransmitters may affect different aspects of social functioning. Thus, the selective noradrenaline reuptake inhibitor, reboxetine, increased social engagement and cooperation and reduced self-focus (Tse and Bond, 2006, 2003, 2002a), whereas selective serotonin reuptake inhibitor, citalopram, had less effect on cooperative behaviour but enhanced the social dominance in healthy individuals in a social interaction paradigm (Tse and Bond, 2002b). Further, the personalized application of oxytocin could improve interpersonal problems by compensating for the deficit in endogenous oxytocin in certain individuals with MDD (Jobst et al., 2015). In healthy men, intranasal oxytocin administration can increase empathic concern (Hurlemann et al., 2010), enhance empathic accuracy (Bartz et al., 2010) and improve ToM (Domes et al., 2006). In depressed patients, intranasal oxytocin administration led to enhanced neuronal activation within the insula during presentation of emotional faces (Pincus et al., 2010) and decreased the ability to ignore sad faces (Ellenbogen et al., 2013), suggesting that oxytocin can enhance neural representation of affective states and enhance empathic concern. Additionally, MacDonald et al. (2013) demonstrated that oxytocin can improve mind reading abilities as tested by RMET in a depressed patient group.

In addition to monoaminergic antidepressants, sex hormones with antidepressant properties such as testosterone might also directly influence social behavior. For example, in healthy males, testosterone increase after losing led to higher motivation to compete again (Mehta and Josephs, 2006). Further, it has been shown that testosterone may increase competitive behavior when social challenges are high (Boksem et al., 2013). One might even speculate that the antidepressant properties of testosterone (Khera, 2013; Peixoto et al., 2014; Schmidt et al., 2005; Zarrouf et al., 2009) might be partly based on improved social behavior.

12.3. Effect of deep brain stimulation on improving social deficits

Apart from pharmacotherapy and psychotherapy, there have recently been indications that a surgical technique called deep brain stimulation (DBS) has direct positive effects on social functioning in individuals suffering from depression by leading to an alteration in function of a specific neural network. In a pilot study using deep brain stimulation, Schlaepfer et al. (2013) have shown an improvement of social functioning from serious to mild impairment after 1 week of stimulation of the nucleus accumbens, which is involved in the processing of reward. The deep brain stimulation led to an increase of metabolism in the ventral mPFC (an area related to social stress) and NAcc (an area related to social rewards). Another study showed progressive improvement in the domain of social functioning up to three years after deep brain stimulation (Kennedy et al., 2011).

13. Conclusions

The results of this review suggest the social disturbance of patients with MDD is pervasive and encompasses almost every aspect of one's social capabilities. The main disturbances in the social processing domain of the RDoC in MDD patients relate to reduced desire to communicate, increased sensitivity to peer rejection, diminished cooperativeness, competition avoidance, alterations in social decision-making as well as problems in identifying emotions and in understanding how others think and feel. Deficits in performing and fulfilling normal social roles are a major reason for high levels of stigma and social withdrawal in patients suffering from depression. For example, the inability of depressed individuals to accurately identify subtle changes in facial, vocal and bodily expressions of others, and excessive responding to negative emotions and body postures while ignoring positive ones leads to social withdrawal and impairments in understanding others' intentions and humour. Apart from increased self-focus and diminished psychomotor skills such as reduced smiling and body movement, depressive patients demonstrated a decreased interest in social interactions, which can lead to difficulties in initiating, establishing, and maintaining satisfying relationships with other people. Further, in experimental economic games, depressed patients showed a decreased desire to cooperate and an increased altruistic punishment of unfair offers and avoidance of social competition. These altered patters of social behavior combined with pronounced impulsivity and reassurance-seeking may often lead to the social rejection of depressive individuals.

On the neuronal level, deficits and the negative bias in perception of social information might be triggered by a hyperactivity of the ventral system and a hypoactivity of the dorsal neocortical structures to social stimuli. The neural basis of increased social withdrawal might lie in the diminished reward system activation and therefore reduced salience attribution to socially rewarding stimuli. Reduced motivation to cooperate and elevated motivation to punish unfairness altruistically might both stem from serotonin deficiency, which was shown to diminish the reward value of cooperative interactions and financial gains. Further, depressed patients showed an opioid receptor deactivation in the nucleus accumbens during social acceptance, which could at least partly explain a low motivation for initiating social interaction. The increased rejection sensitivity might be caused by the reduced endogenous opioid release in the amygdala, leading to its hyperactivity. Additionally, social exclusion is accompanied by strong negative emotions and

reduced oxytocin levels, which may lead to avoidance of social interactions.

Taken together, findings from recent studies demonstrate that recovery from depression requires not only a significant decrease of depressive symptoms but also an improvement in domains such as empathy, mentalizing, social decision making and social skills. Therefore, we recommend that when screening for depression, treatments and therapeutic approaches should target not only core depressive symptoms but also should integrate an assessment of social functioning and social network analysis.

Our results further indicate an urgent need to study the psychological risks and benefits of social media use and to explore how to maximize the potential of digital social networking to strengthen social bonds while minimizing its negative effects. For example, efforts aimed at reducing cyberbullying have the potential to reduce the risk of depression. The increase of quality in social relationships and social support from peers can also help to ameliorate the effect of depressive symptoms. Additionally, we would like to emphasize the advantages of economic social games such as the Prisoner's Dilemma, Ultimatum Game, Public Goods Game and competition games for studying behavioral and neuronal abnormalities in cooperative behaviour and the social decision-making process in MDD. These paradigms further allow for testing the modulatory effects of antidepressants on social preferences in realistic interactive settings where players make up their decisions depending on social norms and the behavior of their opponents in real time. Further, they give researchers the opportunity to measure abnormal behaviours and correlate them with biological markers, such as brain activity measured by fMRI or NIRS, which can help to identify biological markers of certain behaviors and determine targets of pharmacological or psychological therapies. An enhanced and detailed understanding of social impairments and their underlying neuronal correlates in patients with MDD may contribute to new advances in the treatment of this illness.

Finally, the measurement of social impairments in depression should integrate Research Domain Criteria (RDoC) – a strategic plan developed by NIMH, which proposes ways of classifying mental disorders based on dimensions of observable behavior and neurobiological measures incorporating current information from integrative neuroscience research (Cuthbert, 2014). In contrast to prior assessment of depression based on standardized self-report or clinician-administered measure of depressive symptoms, the measurement of depression at the symptom level must become more focused, including measures specifically related to RDoC's social domain (The National Institute of Mental Health Strategic Plan). The RDoC plan considers psychopathology in terms of maladaptive extremes along a continuum of normal functioning, to promote a translational emphasis (Cuthbert, 2014). Recently, a number of suggestions have been made for integrating RDoC into depression research, emphasizing the negative valence system (Woody and Gibb, 2015). Our review further suggests taking into account neural systems for social processes when constructing a new diagnostic framework for depression. Clarification of the interacting genetic, neural and environmental mechanisms through integrative approaches offers opportunities for therapy, and possibly prevention.

Conflict of interest disclosures

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