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SOCIAL COGNITION IN MAJOR DEPRESSIVE DISORDER: A NEW PARADIGM?

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Abstract

Social cognition refers to the brain mechanisms by which we process social information about other humans and ourselves. Alterations in interpersonal and social functioning are common in major depressive disorder, though only poorly addressed by current pharmacotherapies. Further standardized tests, such as depression ratings or neuropsychologic tests, used in routine practice provide very little information on social skills, schemas, attributions, stereotypes and judgments related to social interactions. In this article, we review recent literature on how healthy human brains process social decisions and how these processes are altered in major depressive disorder. We especially focus on interactive paradigms (e.g., game theory based tasks) that can reproduce daily life situations in laboratory settings. The evidences we review, together with the rich literature on the protective role of social networks in handling stress, have implications for developing more ecologically-valid biomarkers and interventions in order to optimize functional recovery in depressive disorders.

Keywords

• Social neuroscience • Social functioning • Game theory • Social dilemmas • fMRI • EEG

Introduction

Major depressive disorder (MDD) is a serious disabling illness of very high prevalence [1]. MDD is associated with high chronic physical disorder comorbidities and alterations in multiple other domains, including interpersonal and social functioning [2]. For example, individuals with mood disorders often experience a reduction in the frequency of social and leisure activities [3] as well as less fulfillment from social and family relationships [4]. Indeed, social anxiety disorder is an important and consistent risk factor for the development of severe depression [5]. Despite these well known findings, there are few studies addressing the underlying biological mechanism and possible therapeutic interventions aimed at improving social functioning.

Social cognition is a concept introduced to examine the underlying mechanisms of social impairment in neuropsychiatric disorders. It generally refers to the sum of those processes which allow individuals of the same species to interact with one another [6]. These processes include both understanding other people's emotions, intentions and actions, and acting in social settings. Key elements of social cognition include encoding, storage, retrieval, and processing of information relating to our social interactions. In classical social cognitive theory, information is represented in the brain as cognitive components that may include schemas (i.e., how ideas are categorized), attributes, stereotypes, etc. The use of this information enables us to understand others in a specific context. However, social cognition is more than figuring out other people; it also involves developing an understanding with others [7]. Thus, social cognition allows us to sustain interactions, develop relationships with others, understand each other, and act together [8].

Interestingly, this interactive aspect of social cognition has recently been enjoying renewed interest in neuropsychiatry with the advent of functional imaging tools. On the one hand, neurobiological studies in healthy people have revealed that several brain networks are consistently recruited when people face social decisions (see Figure 1) [9–12]. On the other hand, social skills that enable people to perform cooperative and altruistic behaviors seem to be altered in several psychiatric diseases, like MDD. These social impairments impact deeply the quality of life of these people [13] and can be a key factor in rehabilitation and the evaluation of treatments (see Figure 2) [14]. Nonetheless, the tasks used to study social cognition in laboratory settings, such as theory of mind (ToM), emotion recognition, empathy, and new interactive paradigms (e.g., game theory based tasks, see below), are most different from the typical tests administered in clinical settings (e.g., depression rating scales, neuropsychologic tests) and in clinical trials, generating a gap between neurobiological research and clinical practice.

In this article, we first review briefly the role of social support in wellness and the neurobiological bases of both MDD and social decision making in healthy people. We next discuss current literature that deals with social alterations in MDD patients, focusing on interactive paradigms. Finally, we propose interpretations of the findings as well as possible future directions of the research being carried out in the area.
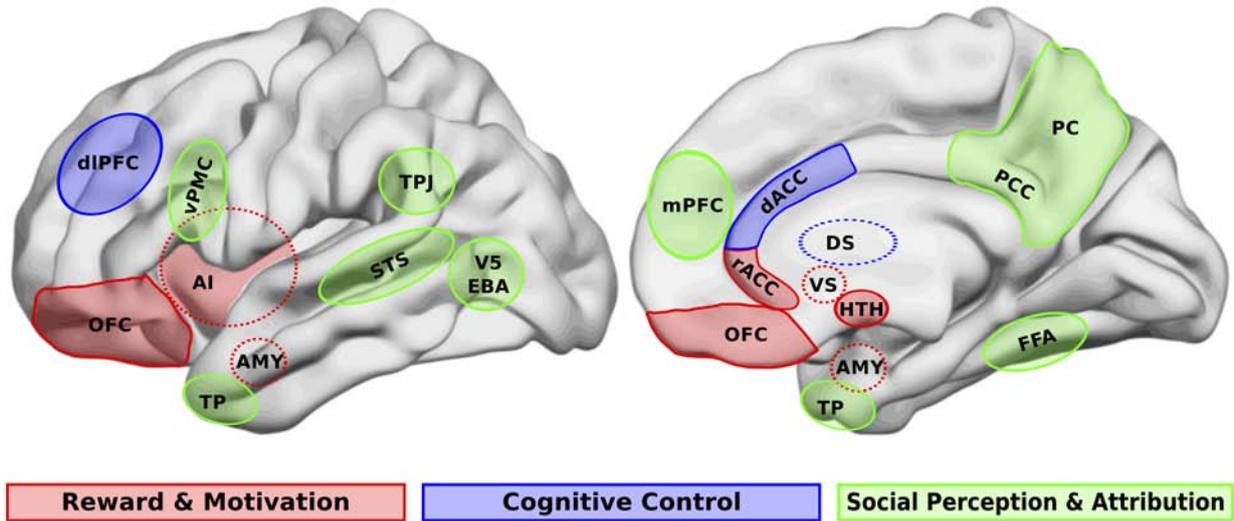
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Figure 1. Brain areas that participate in social processing. A simple classification of brain areas involved in social processing differentiates regions that participate in three related systems. The motivational and reward system (red) that includes cortical areas, such as the amygdala (AMY), the anterior insula (AI), the rostral anterior cingulate cortex (rACC), and the orbitofrontal cortex (OFC). These cortical structures interact with subcortical structures, such as the ventral striatum (VS) and the hypothalamus (HTH). The cognitive control system (blue) participates in goal-directed and adaptive behaviors. This system includes areas such as the dorsolateral prefrontal cortex (dlPFC), the dorsal anterior cingulated cortex (dACC), and the dorsal striatum (DS). Finally, the social attribution system includes areas that participate in the perception of social stimuli, such as the extra-striate body area (EBA) and the fusiform face area (FFA). There are other areas, such as the ventral premotor cortex (vPMC) and the cortex around the superior temporal sulcus (STS), that participate in the perception of intentions of the motor actions ("mirror system"). The attribution system also includes areas that participate in mentalizing processes, such as the posterior cingulate cortex (PCC), the precuneus (PC), the temporal pole (TP), the medial prefrontal cortex (mPFC), and the temporo-parietal junction (TPJ).
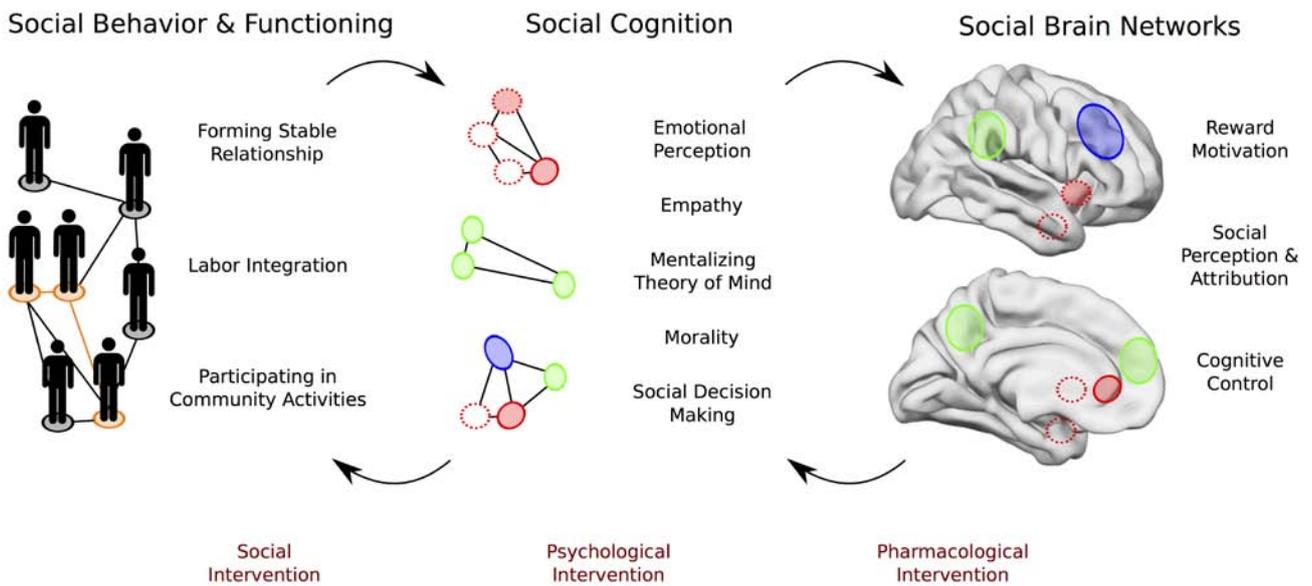

Figure 2. Schematic representation of the putative social alterations in major depressive disorder (MDD) across different levels of analysis. Even though the causal relations between these levels are complex, the interventions in one of the levels have repercussions in the others. For a discussion of the relations of these levels, see [145].

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Role of social support in wellbeing

Both common sense and empirical data tell us that high-quality relationships matter in everyday life and during periods of stress. Supportive others can, in fact, alter the perception of everyday events in such a way that they are not perceived as threats or stressors. For example, observers perceive a hill they have to climb as less steep if they are with a friend rather than alone [15]. Moreover, the presence or the physical contact of a loved one reduces the perception of physical pain and the neuronal response to a painful stimulus [16,17]. In addition, studies show that perception of social connections and positive emotions increase physical health, and these factors influence one another in a self-sustaining upward-spiral dynamic [18].

The link between close relationships and emotional wellbeing is, perhaps, best demonstrated by a study that followed 268 Harvard sophomores from the late 1930s over the course of their adult lives. The single most important predictor of successful aging, defined as being physically and mentally healthy, and satisfied with life at age 75 was neither cholesterol level, nor treadmill endurance, nor intelligence - it was close relationships. Based on the extensive data collected over seven decades, the authors concluded that the only things that matter in life are our relationships with other people [19]. Hence, experiences of social disconnection are processed as a survival threat, thus constituting a risk for physical and psychiatric diseases [20,21]. For example, low social support predicts high risk for both a first episode of major depression and recurrence [22–25]. Interestingly, using social network analysis, a study showed that people with looser ties have higher depression scores and the tendency to cut any remaining ties that they have left. Further the depression of these individuals is correlated with the future scores of their friends within the network. This suggests that isolation as well as clustering may have an impact on the spread of depressive symptoms [26]. So, understanding both the cognitive and biological mechanisms of successful social interactions and the specific alterations that are presented in depression may thus offer new

avenues for optimizing therapeutic outcomes in people with MDD (see Figure 2).

Neurobiology of depression: altered reward and punishment sensitivity

In recent years, the possible neural differences responsible for the symptoms of mood disorder have been extensively studied. These studies suggest that both the brain monoamine and frontostriatal system involved in motivation and reward [27–29], and the neuronal network related to resting state [30] play a causal role in the symptoms of depression.

Behavioral studies have shown that depressed subjects have maladaptive responses to punishment (negative feedback) and hyposensitive responses to reward (positive feedback). Patients with MDD do not improve their performance after a negative feedback and tend to increase the likelihood of making a subsequent error [31]. This tendency is specific to depressive patients, and is correlated with the severity of the symptoms, although it is also present in remitted subjects [32,33]. Further, patients with MDD tend to not choosing stimuli associated with reward [34,35]. The failure to learn from feedback of depressive patients might relate to frontostriatal dysfunction. For example, there is reduced ventral striatum activity during perception and anticipation of reward stimuli in adults and adolescents with depression [34,36]. Subjects with MDD have a hypersensitive response to the rewarding effects of dopaminergic drugs with altered brain activity in the striatum and the medial prefrontal cortex (PFC). Remitted depressive patients present a decrease of the striatum and anterior cingulate cortex (ACC) activities elicited by primary reward stimuli, and an increase of striatum activity elicited by aversive stimuli [37]. Interestingly, some studies have shown an uncoupling between PFC activity and both striatum and amygdala activities [38]. In addition, converging findings suggest abnormal increases of amygdala, ventral striatal, and medial PFC activities related to negative emotional and social stimuli [39–42]. Thus, anhedonia, maladaptation, and emotional dysregulation present in MDD

patients can be related to frontostriatal and amygdala dysfunctions.

On the other hand, excessive rumination and negative self-referential memory observed in depressed individuals have been linked to the function of the default mode network (DMN) [30]. The DMN consists of several brain regions that exhibit patterns of temporally coherent neural activity. These brain areas increase their activity when subjects are resting, whereas decrease their activity when subjects are engaged in an external task [43]. The most robust regions considered as part of this network are the medial PFC, the rostral part of the ACC, the precuneus, the posterior cingulate cortex, and the mediolateral temporal cortex and hippocampal formation. While individuals with depression are evaluating emotional stimuli, the DMN is overactive [44] and its activity is correlated with the level of depressive rumination [45]. Interestingly, MDD patients show alterations in the deactivation of the DMN in the transition from rest to task that can be improved by pharmacotherapy [46]. Patients with depression display increased metabolic activity in the rostral ACC, and deep brain stimulation of this brain area produces therapeutic effects [47]. Therefore, the functional coupling between the rostral ACC and other parts of the DMN, which is greater in patients with depression [48], might correspond to the interface between excessive self-referential thoughts and their negative emotional consequences.

Neurobiology of social decision making in healthy people

Social neuroscience studies have shown that there are three main brain systems implicated in maintaining a social interaction (Figure 1). One of these neural systems is the mesolimbic reward system that is consistently activated during decision making. The components of this system are involved in processing the possible outcomes of the decision, computing the probability and variability of the outcomes, and encoding the saliency of reward. This system also participates in the process of updating behavior, if the outcomes are not as expected [49]. The reward system involves dopaminergic

projections from the ventral tegmental area in the midbrain. The dopaminergic projections loop through the ventral striatum and connect to medial prefrontal areas, including the ACC [50]. This circuit is involved in affective and appetitive behaviors, and in motivation. Interestingly, the reward system may play a critical role in the process of evaluating whether expectations are met. Thus, dopamine neurons signal the prediction error, since they increase their activity in proportion to a reward that is better than expected, whereas decrease it when an expected reward is omitted [51].

In the context of social interaction, studies have shown that the reward system is activated in pro-social behaviors. For example, several works have used game theory based tasks, such as the Prisoner's Dilemma which has been used to investigate cooperation and altruism. In this game, the two participating players may independently choose to either cooperate or defect. Both will be awarded a sum of money in function of the choices made. Each player receives the highest payoff by defecting, if the other player chooses to cooperate. However, each player's payoff is higher for mutual cooperation than it is for mutual defection. Hence, a dilemma is created. If the game is played once and the players care only about their own payoffs, both players should defect. This is the dominant strategy because, heedless of the other player's strategy, a rational player has no incentive to deviate from this choice [52]. Nevertheless in laboratory experiments, these assumptions are frequently violated and humans often cooperate whether the game is one shot or repeated [53]. Interestingly, most participants report that they found mutual cooperation the most personal satisfactory outcome, despite the fact that this alternative is not the best paid one. Accordingly, neurobiological studies have shown that only reciprocal cooperation activates both the ventral striatum and the ventromedial PFC [54,55]. In addition, the activation of the ventral striatum is restricted to mutual cooperation in social interactions, and is not observed when participants play the same game with a computer partner. The former suggests that cooperation has a rewarding value over and above the material rewards obtained from unilateral defection [12,56].

In other experimental paradigms, cooperation strongly activates the reward system in spite of the fact that there are no monetary rewards involved [57]. The reward system is more activated when people receive money from a fair distribution than when they receive the same amount of money from an unfair distribution [58]. Finally, making charitable donations also activates the reward system together with the rostral ACC [59]. The preceding is an oxytocin-rich area connected to the mesolimbic dopamine reward system that is implicated in social attachment formation.

In spite of the fact that the reward system is necessary for developing social behavior, it is not enough *per se*. In addition to the medial dopaminergic system involved in the process of reward, there exists a lateral one that interconnects substantia nigra with the dorsal striatum and the PFC, including the dorsolateral PFC (dlPFC), the dorsal ACC and the lateral orbitofrontal cortex (OFC). The dlPFC is an important component of the working memory and executive functions [60], and participates in the impulse control in order to resist immediate selfish urges to realize greater cooperative benefits immediately or in a later time [61,62]. Interestingly, this area also participates when it is necessary to inhibit pro-social impulses in order to favor personal interests [63]. Further, the dorsal ACC is involved in conflict monitoring whenever there are competing motives, such as those presented in social dilemmas [55,63]. Finally, the lateral OFC participates in evaluating punishment threats that are strong incentives to maintaining cooperation [64]. Overall, during social dilemmas this system seems to register the presence of conflicting incentives and modulate decisions toward the rational best response in a specific context [65]. Thus, this system seems to compute the cognitive effort to make a rational decision whether it be a selfish or a pro-social one [12].

Using electroencephalography (EEG), studies have found that the medial frontal negativity, an event-related potential associated with prediction error and generated in the dorsal ACC [66,67], can be observed when the partner in an interactive game makes an unexpected or unfair social decision [68–70]. The theta activities in the medial region of the PFC and in the dlPFC are associated with the probability of receiving a negative feedback [68], and are greater in those subjects that expect a behavioral change in their partner. Thus, these activities are probably participating in the cognitive control system that enables us to both update our social expectations and adapt our behaviors concordantly.

Finally, there exists a social attribution system that involves several brain areas related to identifying social relevant stimuli, understanding the intentions of other humans, and enabling us to participate in on-line social interactions. There are specialized brain areas that are involved in identifying social relevant stimuli. As examples, the fusiform face area in the temporal lobule is activated by the presence of faces [71]; the cortex around the superior temporal sulcus is activated by biological coherent movements [72]. Recent meta-analyses reveal that two brain structures are crucial to and specific for mentalizing about others' intentions, beliefs, or moral traits, namely the temporoparietal junction (TPJ) and the medial PFC [73,74]. The TPJ is closely related to the "mirror system" and likely participates in a more perceptual level of representation, whereas the medial PFC integrates social information at a more abstract cognitive level [12,75]. This system influences cooperation and social decision-making mainly through processing trust/threatening signals [76]. Thus, the medial PFC is involved in explicit impression formation [77]; the more implicit evaluation (e.g., automatically assessing trustworthiness) relies on the TPJ [78,79]. Using interactive paradigms, several studies have found that games with other humans generate activity in these social attribution areas [80]. Interestingly, when people are engaged in a social interaction, TPJ activity is correlated with the subsequent decision only when this interaction is with another human [81]. In an EEG study, the fall in alpha activity (which likely reflects a neuronal activity increase [82]) in the TPJ is correlated with both the expectative of the other's behavior and the behavioral adaptations in the subsequent interaction [68]. Thus, the medial PFC and the TPJ play a key role in both the understanding of others' intentions and the maintaining of a social interaction.

Social cognition in major depressive disorder

There has recently been growing interest in the study of social skill alterations in MDD patients [13]. These alterations have been mainly observed during mood alteration periods, though some of them can persist during euthymic states. One of the social skills most studied in depressive patients is face perception. An important characteristic of the human face is the transmission of emotional states, and thus the ability to recognize the emotions displayed by others is crucial to social interactions. Patients with MDD present emotion recognition deficit that is mainly characterized by a bias toward the recognition of negative emotions. Thus, these patients tend to not recognizing happy faces and recognizing neutral faces as sad faces [83]. Notably, MDD patients also have an attentional bias toward sad faces [84,85].

Neurobiological studies have shown that patients with MDD present a special pattern of brain activity elicited by emotional facial expressions. They have an increased activity in the amygdala, the ventral striatum and the OFC, especially to sad faces [86–95]. The hyperactivity of the amygdala to negative emotions, especially sadness, is reverted by antidepressive drugs, and is absent in unmedicated euthymic patients [96–98]. Interestingly, euthymic patients show an increase in dlPFC activity that seems to be a compensatory cortical control mechanism that limits emotional dysregulation in limbic regions, like the amygdala. Nonetheless, patients with acute mood episodes show reduced dlPFC activity during tasks that require emotional regulation and emotional anticipation [99–102]. In addition, patients show reduced functional connectivity among the OFC, the dorsal ACC, the precuneus, and the amygdala [103,104]. On the other hand, these patients present an increase of functional connectivity between the rostral ACC and the hippocampus, which are two important areas of the DMN, together with structural alterations in the white matter that correlate with symptom severity [105]. Taken together, these results could reflect the lack of prefrontal regulation over subcortical and cortical regions involved in social appraisal and emotional generation systems [106].

In order to participate in the rich human social life, it is necessary not only to perceive others, but also to understand them. Crucially, we attribute an inner mental world to social agents, and we infer their intentions, beliefs, and wishes through several sources. The ability to do so allows us to maintain social interactions. Several behavioral works show that MDD patients present ToM deficits [107–113]. These deficits can persist after depressive symptom remission and their intensity correlates with the risk of recurrence [114,115].

Currently, there is an interesting line of evidence that arises from studies dealing with the performance of MDD in social dilemmas. An important characteristic of social dilemmas is that there exist goals which tend to clash; hence, the analytical difficulty of social dilemmas increases. For example, the pursuit of self-interest can often be accomplished with the use of coercion or deception, yet such behaviors tend to have the effect of eroding social bonds [116]. In the Prisoner's Dilemma (see above), people with subclinical depression behave in such a way that they optimize their payoffs [117]. Moreover, other studies exploring the effects of mood in social dilemmas have shown that healthy participants tend to cooperate regardless of the social context, whereas depressed participants modulate their behavior in more rational ways [118,119]. Another example is a study that uses the Ultimatum Game. In this game, one player (the "proposer") makes an offer to another player (the "responder") regarding how to split an amount of money between them. The responder can either accept the offer, in which case the money is split as proposed, or reject it, in which case neither player receives any money. Patients with MDD playing as responders accept unfair offers, whereas healthy people tend to reject them [120]. These unfair offer acceptances would appear more "rational" from a standard economic standpoint (i.e., maximizing payoffs). Indeed, MDD patients made more money in the game. Notably, the acceptance rate correlated with cardiac vagal control, which is a peripheral measure of emotional regulation, notwithstanding that MDD patients report higher levels of disgust, anger and surprise upon receiving unfair offers.

The above finding indicates that depressive subjects use emotional regulation processes when making social interactive decisions that may, in fact, help them in managing emotional reactions and, in turn, lead to more acceptances [120]. The induction of sad moods in healthy people generates the opposite behavior, that is, more rejection of unfair offers [121], together with an increase of anterior insula activation and a decrease of ventral striatum activation. However the use of reappraisal, that is, evaluating an emotional situation as more positive, generates more unfair offer acceptances together with a modulation in both the dlPFC and the insular cortex [122]. Notably, other studies that evaluate more severe depressive patients show equal [123] or more [124] rejections of unfair offers. In addition, as proposers, MDD patients give more money than healthy people do [123].

This evidence suggests that MDD patients avoid social rejections, and yet the precise behavior seems to depend on mood symptom severity. Interestingly, studies on individuals who exhibit subthreshold depression (dysphoria) show that they perform better than non-dysphoric controls at ToM and other social cognitive tasks, such as detecting deception [125–128]. Thus, some authors suggest that the increasing activity in DMN, which contributes to task-relevant mental simulation and spontaneous cognition, could also account for the fact that depressed individuals perform better in sequential decision-making tasks and analytical thinking [129,130]. In this way, depression (at least at first) induces cognitive changes that enhance capacities for analysing and solving key social problems, suggesting a "social rumination function" that could facilitate behavioral adaptation in difficult social situations [129,131].

Interestingly, MDD patients show a hyperactivity of the DMN, which correlates with rumination and presents a very similar spatial pattern to that of the areas activated by ToM tasks. In fact, some authors indicate that this network is one of the general domain brain networks which is recruited by mentalizing processes [132].

Conclusions and implications for treatment

Human beings are intrinsically social and gregarious, and virtually all of their actions are directed toward others or produced in response to others [133]. However, more than a third of the world's population is affected by mental illnesses at some point in life, with social impairments being one of the most prominent and disabling features [134]. Social processes are also highly relevant for recovery. The essence of psychosocial therapy should thus be a social interaction structured to benefit patients [135].

Given the intimate association of depressive symptoms and social functioning in MDD, treatments must not only target core depressive symptoms, but also the significant impairments in cognitive and social functioning experienced by people with depression. Indeed, patients with MDD rate treatment outcomes, such as wellbeing, quality of life, and functioning, as more important than symptom relief [136]. Moreover, the primary goal of depression treatment is restoration of functioning to allow the person to flourish. Nevertheless, only few clinical trials in depression emphasize outcomes of cognition or social functionality [137]. As our review indicates, MDD patients present significant alterations in social cognitive skills that can impact deeply in their quality of life. Moreover, neurobiological evidence shows that these patients present abnormal engagements of the key brain systems implicated in social processes.

However, most of the studies dealing with social skills in MDD patients focus on individual mechanisms and observational perspectives. There only exist few studies that explore social skills using interactive mechanisms. Interactive experimental paradigms, like social games, have many advantages. One is that these paradigms give us the possibility to evaluate social decision making in ecological paradigms where the result of a player's decision depends on the decisions of the other player. Another advantage is that, in these games, normal people behave following both the rational construction of the game and the social/moral norm (like fairness, inequity aversion). Interestingly, these social norms vary according to cultural differences [138–140] and represent the standard behavior of the social group or community. Therefore, the behavior represents an indicator of social adaptation. Still another advantage is that we can evaluate how a specific population, like MDD patients, behaves. Importantly, since these behaviors can be measured and correlated with a certain biological activity, these paradigms could help us to identify biological markers of these behaviors and potential targets of medical intervention [141]. Additionally, the performance of the people in these games reflects their behavior in daily life [142].

Most of the evidences reviewed here, however, are still incipient and have limitations that are necessary to address in future research. For instance, most of the fMRI studies have small sample sizes and small effect size. Thus, it is still necessary to replicate the results in order to confirm the neurobiological mechanisms proposed. Another large limitation is that almost all of the findings are corelational in nature. In order to directly test the role of social cognition and the neural circuitry that is supporting it in depression, it is necessary both to experimentally manipulate these processes and systems and to measure the effect of these manipulations on symptoms and functioning of depressive patients. For example, using transcranial magnetic stimulation and direct current stimulation, studies have revealed the causal role of dlPFC in reputation formation[143] and social norm compliance [144]. In Table 1 we point out some research needs in this area. We believe that the use of interactive paradigms for studying social impairments in MDD is a powerful tool to identify the underlying cognitive and neurobiological alterations. The development of translational studies focused on social cognition in MDD can generate novel therapeutic approaches addressed not only to symptom reduction, but also to increasing functionality, social integration and the quality of life of these patients. Thus, it is possible to elaborate integral therapeutic interventions at biological, psychological and social levels with a solid scientific basis (see Figure 2). To achieve this, however, more research on neuroscience, psychological and clinical integration is required.

Table 1.

Social cognition in major depressive disorder: research needs	
1	Studying the biological bases of social skills in depressive subjects using interactive ecological paradigms
2	Studying the evolution of social skills in depressive subjects with different symptom severity (disphoric, depressive and recovery subjects)
3	Elaborating population norms for social cognition ⊠ age, gender, culture, ethnicity effects
4	Elaborating a standard battery to measure social cognition in MDD research trials
5	Carrying out large sample studies of social cognition in acute MDD versus controls and other psychiatric diseases
6	Studying the effect of impaired social cognition on disability in MDD
7	Incorporating social cognition outcomes in registration clinical trials
8	Developing novel social interventions targeting social cognitive skills

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