HOW SOCIAL COGNITION DEFICITS AFFECT PSYCHOPATHOLOGY – A NEUROSCIENTIFIC APPROACH

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Abstract

Humans are substantially a social species. Effective mental treatment cannot be obtained without addressing social behavior. Social cognition refers to the mental processes underlying social interactions, which allow individuals to make sense of the other peoples' behavior, to decipher emotions on their faces, and to draw conclusions about their intentions. The core domains of this multifaceted concept are theory of mind, social cue perception, attributional style and emotion perception/processing.

The amygdala, orbital frontal cortex and temporal cortex areas are typically activated during the processing of information within social-emotional context. The aforementioned brain areas are recognized as the major components of the so-called "social brain" - specialized for the social interactions in humans.

Adequate perceiving and processing of the social information is essential for an effective social functioning, which becomes obvious when it goes awry. Various psychiatric disorders are characterized by social cognitive deficits, among which schizophrenias, depression-anxiety and autism spectrum disorders were most broadly studied to date. Growing evidence suggest that these deficits underlie poor functional outcomes in patients with mental health impairments and have an important role in the initiation and maintenance of the disorders' symptoms.

One of the most important goals of social neuroscience research is to provide a treatment intervention that will improve patients' social cognitive skills and the functional outcome.

All together, the present review aims to provide a contemporary overview of the concept of social cognition, to outline its relation to psychopathology, and to discuss the implications for clinical practice and treatment.

Key words: social cognition, psychopathology, treatment implications

Sažetak

Čovek je društveno biće. Za postizanje i očuvanje mentalnog zdravlja neophodno je imati očuvanu socijalnu kogniciju. Socijalna kognicija obuhvata skup mentalnih procesa koji čine osnovu socijalnih interakcija, a koji omogućavaju razumevanje ponašanja drugih ljudi, shvatanje značaja izraza njihovih lica, i procenu namera osoba iz okruženja. Ključne domene ovog složenog koncepta predstavljaju: teorija uma, opažanje socijalnih znakova, kauzalna atribucija, opažanje i procesuiranje emocija. Obradu informacija socijalno-emocionalnog konteksta karakteriše aktivacija amigdala, orbitofrontalnog i temporalnog korteksa, glavnih regiona takozvanog „društvenog mozga“, specijalizovanog za socijalne interakcije svojstvene ljudima.

Značaj ispravnog opažanja i tumačenja socijalnih znakova, funkcija neophodnih za uspešnu socijalnu adaptaciju, dolazi do izražaja kada su ove sposobnosti narušene. Brojne psihijatrijske poremećaje prati deficit socijalne kognicije, a među njima ispadi su najpodrobnije ispitivani u grupi shizofrenija, depresivnom poremećaju, autizmu, itd. Zanimljivo je da sve više naučnih dokaza ukazuje da ovi ispadi imaju važnu ulogu u nastanku i održavanju određenih simptoma psihijatrijskih bolesti, kao i da predstavljaju glavni uzrok narušene opšte funkcionalnosti pacijenata.

Jedan od najvažnijih ciljeva istraživanja u oblasti socijalnih neuronauka je obezbeđivanje terapijskih intervencija koje bi poboljšale socijalne veštine pacijenata, a time i njihovu funkcionalnost. U aktuelnom pregledu biće razmotreni specifični terapijski modaliteti čija je primena u dosadašnjoj literaturi najbolje proučena: trening socijalnih veština, psihofarmakološke intervencije, neinvazivne tehnike stimulacije mozga.

Cilj ovog pregleda je prikaz savremenih saznanja o konceptu socijalne kognicije i njegovoj povezanosti sa psihopatologijom, kao i razmatranje terapijskih implikacija od značaja za kliničku praksu.

Ključne reči: socijalna kognicija, psihopatologija, terapijske implikacije

Social interactions depend on the exchange of signals such as speech, facial expressions, eye gaze, body posture and the movements. Social cognition represents a set of skills needed for the understanding of these interactions and successful social functioning. It refers to the mental processes underlying social interactions, which allow us to draw inferences about other people's beliefs, intentions and behaviors [1]. Simply put, social cognition research deals with questions on how we make sense of the behavior of others, in which ways we decipher emotions on their faces, and how we draw conclusions about other people's intentions. In everyday life, an individual encounters numerous social cues from the faces of others, their voices and gestures. Adequate perceiving and processing of those social information and subsequent appropriate responses are needed for effective social communication and functioning. In this review we aimed to provide a contemporary overview of the concept of social cognition, to outline its relation to psychopathology, and

to discuss the implications for clinical practice and treatment.

Recent research in the field of social neuroscience has begun to shed light on the underlying biological processes of social cognition and behavior and to identify the roles of specific neural structures/circuits. The so called "social brain network", specialized for social interactions in humans, refers to the brain areas typically activated during processing information within social-emotional context [2, 3]. The current literature has highlighted amygdala, orbital frontal cortex and temporal cortex areas as the major components of "social brain" [4, 5]. Temporal cortex participates in perceiving of the social relevant stimuli (i.e. social perception), whereas the amygdala, cingulate gyrus, somatosensory and orbitofrontal cortices have a joint role in linking the aforementioned social perception to emotions, motivation and neurocognition (i.e. social cognition) [6] (see **Figure 1**).

Figure 1. Component processes of social cognition and their neural correlates

The domains of social cognition

Social cognition is a multifaceted construct comprised of several sub-domains. Among them, the most broadly studied in the current literature are the following four core domains of social cognition:

Theory of mind (ToM) - represents the ability to accurately infer the mental states of others and to understand their behavior in a social environment ("to take someone's viewpoint") [7]. In other words, ToM is largely automatic process that allows us to make predictions

about other people's actions, based on their beliefs, intentions, emotions and desires. It develops fully only in humans, probably as an adaptive response to their increasingly complex social organization [8]. The activation of medial prefrontal cortex and adjacent paracingulate cortex has been consistently associated with this social cognitive domain [9]. The example of a prototypical assessment task for ToM is The Reading the Mind in the Eyes Task [10], where respondents are required to draw a conclusion about the mental state of others only by looking at a picture of their eyes (see **picture 1**).

Picture 1. The Reading the Mind in the Eyes Task

Social cue perception- represents the ability to make inference about certain social situation. That is to understand and appraise social roles, rules and contexts, using given verbal and non-verbal social cues. Through experience, we learn various facts about social situations. The temporal poles are brain regions responsible for applying this general knowledge about social situations to the situation that currently confronts us, through specifying the kinds of thoughts and feelings most likely to occur in a particular context (i.e. previously observed or felt in similar situations in the past) (3). The Profile of Nonverbal Sensitivity task [11], a measure of individual differences in the ability to decode nonverbal social signs, is an example of a prototypical assessment task for this domain. Therein, participants are asked to name the situations that would prompt the social cue observed on a video-taped scenes containing different facial expressions, voice intonations, bodily gestures (for example- “expressing gratitude”, “asking forgiveness”, “expressing jealous anger”, etc.).

Attributional style – refers to the characteristic ways in which individuals explain the causation of events. It represents individual’s tendency to attribute the cause of events to the self, others or the environment. Healthy individuals usually display a so called “self-serving” bias,

which means that they preferentially attribute negative events to the external causes and positive events to themselves [12]. Fronto-temporo-parietal network has been recognized as a neural correlate of causal attribution in social situations [13]. The Internal, Personal and Situational Attributions Questionnaire (IPSAQ) [14] represents prototypical task designed for assessing the causal locus. Therein, respondents are asked to categorize the causes of positive and negative social situations as being either internal (i.e. caused by themselves), personal (i.e. caused by other people), or situational (i.e. caused by certain circumstances).

Emotion perception and processing – refers to the ability to correctly identify other people’s emotions (primarily from their facial expressions, but also through vocal prosody), to discriminate between different emotions, understand their meaning, and to adequately manage emotions and emotional reactions [15]. This domain is fundamental for deciphering others’ affective states and social signs. Previous research indicated that amygdala has a crucial role in attaching emotional values to faces, thus enabling us to recognize their expressions (3). Typical assessment of this domain includes the tasks measuring participants’ ability to correctly recognize universal emotions (i.e. happiness, sadness, fear, anger, neutral) represented by facial expressions or emotional prosody in speech. The Degraded Facial Affect Recognition Task [16] is an prototypical example, where the photographs presenting emotional facial expressions has been blurred in order to enhance the contribution of interpretation. (see **Picture 2**).

Picture 2. The Degraded Facial Affect Recognition Task

Social cognition deficits in psychiatric disorders

Although social interactions require a set of sophisticated skills, for most people they are routine and most social signals are processed automatically, without

awareness. The necessity of possessing such skills, crucial for successful social functioning, becomes obvious only when they are altered or insufficient. Various psychiatric disorders are characterized by impaired social cognition and social functioning. There is growing evidence that social cognitive deficits underlie poor functional outcomes in psychiatric patients and have an important role in the initiation and maintenance of the disorder [17, 18]. Hereby, we will focus on the three disorders where most of the research has been done to date: schizophrenia, depression-anxiety and autism spectrum disorders.

Schizophrenias – current literature highlights the critical role of social cognitive deficits in functional disability in psychotic disorders and their potential role in the formation and maintenance of certain psychotic symptoms. For example, the tendency to blame other people rather than circumstances or situational factors for negative events (i.e. personalizing attributional bias), is well established in paranoid patients [19]. It has been also hypothesized that emotion recognition (ER) deficits, through negative misperceiving of social-emotional cues, participate in the occurrence of paranoid elaborations [20] and social withdrawal/isolation [21], that often accompany schizophrenia. Some other psychotic symptoms, such as thought/language disorganization and delusions of control/persecution, could be understood in light of a disturbed capacity of patients to relate their own intentions to behavior and to monitor others' intentions (i.e. ToM deficits) [8]. Altogether, schizophrenia is associated with a broad range of social cognitive deficits, among which the ER has been most widely explored to date. Deficits in ER have been recognized as a hallmark of schizophrenia [22]. Altered recognition of negatively valence emotions (i.e. fear, anger) is evident early in the course of schizophrenia, years before the onset of full-blown psychosis [23], and it is believed to further generalize across the emotional valences following the illness progression [24]. Since it has been shown that some of the healthy siblings of patients with schizophrenia also exhibit similar but subtle deficits, it has been proposed that ER alterations might represent the indicators of vulnerability to schizophrenia-spectrum disorders [25].

Depression-anxiety spectrum disorders – According to cognitive theories, biased information processing (i.e. the tendency to interpret ambiguous information in a threatening way) is common among depression-anxiety spectrum disorders and contributes to the risk of their development. The normal self-serving bias is altered in depressed individuals, who tend to attribute positive events to the external causes and negative events to the internal ones. Thus, vulnerability to depression-anxiety spectrum disorders might arise from a bias to blame oneself for a failure in a global way and subsequent decreased

self-worth, hopelessness and depressed mood [26]. Impaired facial ER for many of the basic emotions (i.e. happiness, anger, fear, disgust, sadness) has also been repeatedly reported for this group of patients [27]. Moreover, the recent study conducted by our research group reported altered recognition of happy facial expression even in depression-vulnerable healthy individuals [28]. It seems that biased processing of positive emotions increases the susceptibility to negative affective states and therefore it should be a target for specific preventive strategies.

Autism spectrum disorders (ASD) – are developmental disorders characterized by profound difficulties in understanding other people's thoughts and emotions, and severely impaired social communication and functioning [29]. Affected individuals fail to form normal peer relationships, to engage in reciprocal social behavior and to adequately respond to nonverbal social cues (such as emotional facial expressions). ASD are frequently associated with clinically prominent deficits in ER (across multiple expressions), ToM and social cue perception, even in cases with normal or high IQ and other cognitive abilities [30]. In other words, social cognition may be selectively impaired in those individuals, leaving the other cognitive functions intact [8]. It has been argued that the development of social cognitive skills primarily depends on the amygdala-fusiform system, and that deficits in this network have a crucial role in ASD development [31]. The neuroimaging studies supported the notion of dysfunction in the specific social cognitive neural substrate in ASD by showing decreased activation in some of the major components of "social brain" (i.e. medial prefrontal cortex, amygdala, fusiform gyrus) in individuals with ASD while performing social cognitive tasks [32, 33, 34]. Therefore, the search for the neurobiological underpinnings and causes of ASD should probably focus on the social cognitive deficits.

Treatment implications

One of the most important goals of the social neuroscience research is to provide a treatment interventions that will ameliorate patients' social cognitive skills and improve their functional outcome. Each social cognitive process requires its own specific therapeutic intervention. Structured behavioral training programs targeting facial ER and ToM deficits have been well validated in schizophrenia and showed positive effects [35]. Social cognitive training programs for individuals with ASD are also under development. Although it has been shown that ToM skills can be taught to individuals with ASD, the evidences that these skills could be maintained over time or generalized to other setting/new context are still lacking [36]. Thus, further research in this domain and

continuous refinement of the intervention strategies are still needed.

Psychopharmacological interventions could also be beneficial. Oxytocin (OXT) is a neuropeptide known to strongly modulate amygdala function and to promote pro-social behavior [37]. Decreased peripheral OXT levels have been reported in patients with schizophrenia [38], ASD [39] and depression [40]. Therefore, OXT has lately been widely examined as a promising novel treatment approach, particularly in synergistic combination with psychotherapy, for an adjunctive treatment of various psychiatric disorders characterized by social dysfunction [41].

It is also possible that noninvasive brain stimulation techniques (such as transcranial magnetic stimulation and transcranial direct current stimulation) could be used in future to enhance social cognition through stimulating particular social processing neural systems in a targeted manner [42,43]. Nonetheless, there are many concerns that need to be addressed before, such as safety concerns and potential unintended long-term consequences.

Humans are essentially a social species. The treatment in psychiatry cannot be obtained without addressing social behavior. The clarification of these issues is an essential step toward developing an intervention that is capable of minimizing social cognition deficits. We hope that our review contributed to a better understanding of the social cognition concept, its underlying neural mechanisms, and importance for psychiatric clinical practice.

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