



REVIEW

Facial emotion processing in schizophrenia: a review of behavioural and neural correlates

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Abstract

Schizophrenia is one of the most severe psychiatric conditions, often associated with deficits in social cognition. Social cognition deficits are predictors of functionality in patients and involve theory of mind, attributional style, social perception, and emotional processing. In particular, facial emotion processing (an important domain of emotional processing) seems to be particularly related to cognitive and social functioning, and to positive and negative symptoms. Patients with schizophrenia have difficulties in processing emotional faces; however, those impairments are still far from fully understood. In this review, we addressed the behavioural and neural correlates of facial emotion processing in schizophrenia. Despite studies showing impairments in both positive and negative faces, the most consistent findings involved negative faces. Moreover, patients with schizophrenia showed abnormalities in the social brain neural circuit during facial emotion processing. While some studies described hypoactivation of brain areas related to emotional processing, such as the amygdala, others reported hyperactivation, leading to a high number of inconsistencies. The findings are limited by the experimental designs used, and the clinical and demographic characteristics of patients. Despite such variable findings, there has been growing interest in developing psychosocial interventions focused directly on social cognitive impairments in schizophrenia, with potential impact on patient's ability to perceive emotional faces. We provide a critical perspective on current evidence and suggest new pathways of research. The understanding of the mechanisms underlying facial emotion processing in schizophrenia could enhance functionality and quality of life by providing innovative approaches to the interpersonal difficulties patients frequently experience.

Keywords: Schizophrenia, Facial Emotion Processing, Social Cognition.

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Introduction

Schizophrenia (SZ) is one of the most debilitating psychiatric disorders, often associated with impairments in social cognition [1,2]. Social cognition is described as the ability to understand thoughts, feelings, and intentions of others and of the self in the context of social interactions [3,4]. Impairment in social cognition is associated with poor functioning in patients with SZ [5,6]; and according to a recent meta-analysis [7], it appears to be more strongly related to community functioning (a direct indicator of everyday functioning) than neurocognition (e.g., processing speed, attention, learning and working memory, problem solving). Furthermore, social cognition is frequently a mediator of the relationship between functional outcomes and neurocognition [8]. Together with neurocognition, impairments in social cognition are observed in prodromal and early phases of psychosis - as well as in unaffected family members - remaining largely unaffected by pharmacological treatment [9–16]. Thus, social cognition has been identified as a main area of research in SZ through the exploration of interpersonal difficulties experienced by patients and its daily life consequences.

The National Institute of Mental Health (NIMH) [17] consensus statement recognized theory of mind, attributional style, social perception, and emotional perception or process as the main domains of social cognition in SZ. Later, four general social cognitive processes have been proposed: experience sharing, mentalizing, experiencing and emotion regulation, and perception of social cues [3]. Of greater relevance to this review, perception of social cues allows appropriate responses to others' facial expressions, voices and body movements, facilitating social interactions [3]. In SZ, studies on social cue perception have focused mainly on facial emotion processing. This domain also includes emotional perception, usually assessed by tasks requiring the identification or discrimination of emotional faces.

In SZ, research has showed overall deficits in facial emotion processing and abnormal neurological responses to emotional stimuli [1,18,19]. The comprehension of those deficits will provide insight on the mechanisms underlying some of the SZ symptomatology. For example, Addington et al. [13] found that facial emotion recognition partially mediates the relationship between cognitive and social functioning. Hall et al. [20] reported that patients experiencing positive symptoms were impaired in recognising basic facial emotions when compared to a subgroup of patients without positive symptoms (scores of <4 on PANSS (positive symptoms items)). Furthermore, several emotional biases have been associated with positive and negative symptoms [21–24].

This narrative review aims to describe the behavioural and neural correlates of facial emotion processing in SZ, by providing a more comprehensive understanding of the maladaptive behaviour of patients with SZ in social context. We divided it in two main sections; the first one provides

an overview focused on behavioural studies, and the later reviews neurobiological data and the mechanisms underlying facial emotional process in SZ. Lastly, intervention techniques, future directions and concluding remarks are presented.

Behavioural correlates of facial emotion processing

As previously stated, several studies have identified impairments in facial emotion processing in patients with SZ; however, findings differ according to the emotion presented. Strauss et al. [25] reported that deficit syndrome patients, in whom negative symptomatology is predominant, had more severe impairments in identifying happy faces rather than negative (disgust, angry, fearful, sad) and neutral ones. Derntl et al. [26] examined the effects of emotional faces (angry and neutral) on inhibition processes using an emotional stop signal task; patients performed significantly worse than controls in neutral trials, supporting the hypothesis that neutral face processing is impaired. Park et al. [27] investigated the interaction between sustained attention and emotion by presenting emotional faces (happy, neutral, and sad) as background during a continuous performance test. Results revealed a steeper sensitivity decline over time when relevant stimuli were displayed conjointly with a happy face (as compared to a sad one), suggesting that happiness expressions decreased patients' performance. The authors concluded that an impaired recognition of happy faces withdrew processing resources from the attentional task.

Although research has described impairments in the processing of positive facial expressions, such as happiness [28,29], the most consistent findings involve negative ones [30–32]. Kohler et al. [33] found higher error rates for the identification of sad faces than happy ones in SZ. Zhu et al. [34] investigated eye movements during facial emotion recognition in SZ. Overall, the authors reported a significant higher time and effort processing negative faces, suggesting a valence-specific deficit. Recently, Jang et al. [35] assessed facial emotion processing over time using an eye-tracker. The authors concluded that, despite relatively preserved early attentional capture by emotion, patients with SZ withdrew their attention from faces later when one of the two faces presented in conjunction displayed negative emotions (sad and angry). Jan et al. suggested that the diminished processing of negative faces in later phases may relate to affective vulnerabilities in SZ.

Healthy individuals tend to process potential threatening stimuli, namely angry faces, more quickly and effectively, even without conscious awareness of the stimuli [36,37]. This advantage prepares the body to deal or to react rapidly to imminent threats, enhancing the chances of survival. In SZ, however, studies investigating this phenomenon often report contradictory findings. Recently, Grave et al. [38] explored the interference of facial expressions in psychotic

disorders using an letter-discrimination task. Participants were asked to discriminate a target-letter among distractor-letters and to ignore an adjoining facial expression (happy, angry and neutral). Grave et al. found that patients with psychotic disorders were more prone to interference by happy faces than neutral and angry ones. The results suggested a weaker detection of potential social threat, leading to a lower impact of task-irrelevant angry faces during the attentional task. The perceived threat from the social environment is a central feature of SZ, related to maladaptive appraisals of somehow anomalous experiences [39]. Therefore, patients may display a phenomenon of sensitisation to threat, leading to a weaker processing of angry faces [38]. For instance, in a study using morphed facial expressions, Huang et al. [40] found that patients with SZ categorize intermediate angry faces as happy ones. Nevertheless, once the emotional signal became evident, patients' perception changed more quickly (in comparison to controls). Huang et al. highlighted that patients may decrease the valence of threatening facial expressions in order to regulate affect. Using a facial emotional attribution test, Premkumar et al. [41] showed that psychotic patients were less accurate than controls in recognizing fearful and angry faces (compared to happy and neutral), revealing more fear-as-anger misattributions. Moreover, the propensity for such bias was associated with a longer duration of illness [41]. Another possible explanation for an overall impairment in negative facial expressions is that happy faces are easier to categorise due to highly salient features (e.g. smile) [42]. However, the robust happy advantage found in a large number of studies with healthy individuals seems to reflect its emotional valence and not low-level visual features [43,44].

Several factors related to the experimental design may contribute to the inconsistency of findings across studies in SZ, namely the characteristics of the facial stimuli (e.g., intensity, original database, gender, colour of the picture) and the emotion categories (e.g., angry, neutral, fearful, sad and happy) [19]. In fact, social cues provided by the human face differ strikingly depending on the emotion expressed (even among negative emotions). For example, angry faces usually signal imminent aggression, while fearful faces are related to potential threat in the environment [45]. Another possible explanation for the inconsistent findings may be that the paradigms vary significantly across studies. For instance, task instructions can be divided into two categories: explicit and implicit [46]. Explicit tasks aim at the emotional content of stimuli and usually involve identification, recognition or discrimination of emotional faces; a meta-analysis on this subject stated that patients' performance did not differ significantly between explicit tasks [1]. On the other hand, implicit task instructions are not directed toward the emotional content of stimuli and aim to investigate how emotional stimuli interfere with a specific task [46]. In SZ, when comparing both implicit and explicit processing, implicit emotion processing seems less affected [46]. These

results suggest that emotional faces are correctly perceived by patients with SZ at non-awareness level or at early processing but may be biased at higher top-down processing levels [47–49]. Nevertheless, a study using event-related potentials found impaired bottom-up processing rather than top-down dysfunction for facial emotion recognition in SZ [50]. Investigation on the neural activity of facial emotion processing might help in clarifying the processes substratal to facial emotion processing and its deficits.

Neural correlates of facial emotion processing

A preponderance of social information is gathered by observing facial expressions. Interpretation of such information requires the interaction of specific brain areas, such as the amygdala (for emotional perception), insula and basal ganglia (for emotional recognition), and prefrontal cortex and temporal lobe (for more complex social judgments) [4]. In SZ, abnormalities in social brain neural circuitry during facial emotion processing have been widely reported; however, results are somehow conflicting [51–53]. For example, some studies reveal under-recruitment of the amygdala [51,52], particularly in the processing of fearful faces [54], while others have reported intact activity [55] or even hyperactivity [56,57].

A meta-analysis demonstrated that impairments in facial emotion processing in SZ are related to an under-recruitment of the amygdala, accompanied by hypoactivation of segments of the so-called 'social brain system', including not only the bilateral fusiform and right superior frontal gyri, but also right lentiform nucleus [58]. A meta-analysis conducted by Anticevic et al. [59] found that the bilateral amygdala had a significantly lower activation in patients with SZ in response to aversive emotions. Gur et al. [51] reported a significant decrease of activation in the left amygdala, together with bilateral hippocampus activation, during the performance of an emotional valence discrimination task by individuals with SZ. A study by Dowd et al. [18] revealed that the brain activity during emotional experience was largely intact in SZ; however, the ventral striatum and left putamen showed reduced activation to positive stimuli (words, images, and faces). Choudhary et al. [60] observed hypoactivation of the fusiform gyrus and bilateral dorsolateral prefrontal cortex in first-episode patients. Using functional near-infrared spectroscopy, Watanuki et al. [61] reported lower activation of the right precentral and inferior frontal areas in SZ during an emotional face task.

Contrariwise, several studies have revealed limbic hyperactivation in patients with SZ exposed to emotional faces [54]. Habel et al. [62] reported hyperactivation of the right precentral gyrus, extending to the right inferior frontal gyrus. A meta-analysis revealed that, during facial emotion perception, a larger signal was found in the cuneus, left parietal lobule, right precentral gyrus, and left temporal lobe; areas which are not commonly activated in emotional tasks (with the exception of the left temporal lobe) [53]. The au-

thors speculated that patients might recruit non-emotional regions as a compensatory process. Of greater relevance, the results highlighted that patients with SZ do not simply exhibit reduced activation when processing emotional stimuli. Also, Li et al. [58] reported that patients with SZ, but not controls, activated the left insula, which plays an important role in emotion regulation and in processing unpleasant stimuli [58].

The magnocellular pathway is usually involved in the processing of emotional signals, and is more sensitive to low-visual-acuity information (low-spatial-frequency: LSF) [63]. This pathway supports the rapid transformation of evolutionary relevant stimuli to subcortical regions, allowing a more automatic processing [64]. In opposition, the parvocellular pathway, usually involved in the processing of fine-grained details, is more sensitive to high-visual-acuity (high-spatial-frequency: HSF) [63]. Techniques to elicit unconscious responses to faces by subliminal presentation have been recently used in SZ. For example, Kim et al. [50] measured the event-related potentials during the presentation of spatial frequency modulated face stimuli (neutral versus fearful). The authors postulated that impairments in emotion processing may be rather associated with reduced activity in the LSF-dependent, automatic visual pathway. Using an event-related potential paradigm, Komlósi et al. [65] reported increased brain responses to fearful faces at a later processing stage (compared to neutral faces), which could reflect hyperresponsivity to fearful stimuli.

It is important to notice that the use of different emotion categories and experimental designs, as mentioned above, might influence findings, leading to reported differences in neural activity across studies.

Psychosocial interventions

Evidence-based psychosocial interventions, such as social skills training, cognitive behavioural therapy and cognitive remediation, may influence social cognition in SZ; however, their main target does not directly involve social cognition and, in particular, facial emotion processing. Also, there seems to be little impact of pharmacological treatment on social cognition in SZ [10,66].

There has been growing interest in developing psychosocial interventions focusing on social cognitive impairment in SZ. Available programs differ on key characteristics, namely duration training, group size, and social cognition domains involved [67]. For example, Metacognitive Training (MCT) is a group intervention focused on cognitive biases (e.g. jumping to conclusions) underlying delusions [68]. The authors combined group MCT with an individual approach and found a reduction of psychotic symptoms and cognitive biases (in comparison to cognitive remediation program).

Regarding facial emotion processing, several research groups have been developing and testing intervention programs that specifically target this social cognition domain.

Frommann et al. [69] developed the Training of Affect Recognition (TAR), a 12-session program administered to pairs of patients at a time. TAR focus on facial emotion perception and comprises three parts: a) identification and discrimination of specific facial features associated with basic emotions; b) increasingly holistic processing mode, considering first-impressions, non-verbal information and different emotion intensities; and c) integration of mimicked expressions into the social, behavioural, and situational context. Inpatients with SZ who received this intervention had significant improvement in facial emotion perception, in comparison with patients in a time-matched neurocognitive remediation program or treatment-as-usual (TAU) [70].

Penn et al. [71] developed the Social Cognition and Interaction Training (SCIT) program, a manualized group intervention aimed at improving social cognition and social functioning. SCIT involves three phases: a) emotion training, addressing emotion perception impairments; b) figuring out situations, focusing on attributional biases and theory of mind; and c) integration, in which patients apply the learned skills to interpersonal problems in their daily lives. Roberts et al. [72] compared the efficacy of SCIT plus TAU with TAU alone. The authors found improvements related to the SCIT program, in particular in emotion perception (but not attributional bias and theory of mind) and social skills.

In order to combine the successful elements of the SCIT and TAR, Horan et al. [73] developed the Social Cognitive Skills Training (SCST), a 12-session integrative social cognitive intervention designed to address the four social cognition domains. SCST was associated with significant improvement in facial emotion perception. Latter, Horan et al. [74] assessed the efficacy and treatment-outcome of 24-session SCST. The program led to greater improvement over time in emotional processing (including facial emotion perception and emotional management) compared to computerized neurocognitive remediation, standard illness management skills training, and treatment combining elements of SCST and neurocognitive remediation [74].

Rocha et al. [75] developed a Metacognitive and Social Cognition Training (MSCT) program designed to remediate deficits (emotion recognition, theory of mind and social perception) and to correct biases in social cognition (e.g. jumping to conclusions, self-serving bias, bias against disconfirmatory evidence, need for closure, and liberal acceptance). MSCT is based on social cognition programs [71–73] and metacognitive training for SZ [68]. The authors reported improvement in several social cognition measures (theory of mind, social perception, emotion recognition) after the application of the program, thus providing evidence for the efficacy of combining remediation and debiasing approaches. In addition, patients assigned to the MSCT program had significantly greater improvement in psychosocial functioning and general schizophrenia-related symptoms, when compared to a TAU group [68].

Innovative third-wave psychological interventions for

psychosis—contextual acceptance, mindfulness or compassion-based therapies—have also shown promising results in group contexts, with positive effects on variables such as social functioning [76–78].

In aggregate, there is evidence of a positive effect of social cognitive training, mainly in facial emotion recognition and theory of mind domains [67]. Recently, computerized online social cognitive games and virtual reality have been used, revealing high patient satisfaction [79]. Lastly, the use of adjunctive therapies, such as oxytocin, might boost the effects of psychosocial interventions on social cognition [79].

Future directions

Despite growing attention to facial emotion processing in SZ during the past two decades, its mechanisms and impact on patients' daily functioning are still far from fully understood. Future studies ought to focus on the neural mechanisms underlying the conscious detection of emotional faces. In particular, the pathways involved in the access to visual awareness by social-threat stimuli in SZ are still unclear. Since psychotic disorders are related to a predominantly sense of threat and danger, the detection of social threats in SZ should imprint the patients' social functioning.

Besides neural responses, other physiological correlates could be assessed. For example, heart-rate variability (HRV) is linked to emotional processing in healthy individuals [80]. In SZ, reduced HRV has been found, suggesting a constant perception of threat that leads to the maintenance of an overall elevated sympathetic arousal and decrease parasympathetic activity [81]. In keeping with these results, paranoid patients are more likely to attribute threat to ambiguous stimuli [81]. To our knowledge, no studies have investigated HRV in patients with SZ when exposed to emotional faces. Moreover, the role of oxytocin on the processing of emotional faces is of growing interest [83]. Averbek et al. [84] found that the administration of oxytocin improved the ability of patients with SZ to recognize emotional faces, and a systematic review about the conjoint use of oxytocin in interventions directed to social cognition reported favourable results [79].

Concluding remarks

In order to analyse facial emotion processing in SZ, we have reviewed several behavioural and neural studies. Impairment in facial emotion processing has been widely reported, affecting patients' functioning and quality of life. Studies with behavioural tasks have shown deficits concerning happy faces. In opposition, the most consistent findings involve negative emotions (sadness, anger and fear). Regarding neural responses, there is evidence of abnormal activation of areas related to the processing of emotional faces, namely the amygdala; however, such results are still inconsistent. Findings are limited by the

heterogeneity in clinical (e.g., phase of illness, subtype, pharmacological treatment) and demographic characteristics of patients (e.g. age, gender). Furthermore, studies differ considerably in paradigms and experimental design (e.g. emotion categories, response format, stimulus complexity, control tasks, and behavioural, neural and physiological measures). Notwithstanding these inconsistent findings, interventions focused on emotion perception tend to improve patients' ability to correctly process facial expressions.

Investigating the mechanisms involved in facial emotion processing in SZ and the behavioural, neural and physiological responses to emotional faces could enhance daily functioning and quality of life by providing innovative approaches to the interpersonal difficulties.

Abbreviations

HRV: Heart-rate variability; HSF: High-spatial-frequency; LSF: Low-spatial-frequency; MCT: Metacognitive Training; MSCT: Metacognitive and Social Cognition Training; NIMH: National Institute of Mental Health; SCIT: Social Cognition and Interaction Training; SCST: Social Cognitive Skills Training; SZ: Schizophrenia; TAR: Training of Affect Recognition; TAU: Treatment-as-usual.

Competing interests

The authors declare no conflict of interest.

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