

Running head: FACIAL EMOTION PROCESSING IN SCHIZOPHRENIA

## Facial Emotion Processing in Paranoid and Non-Paranoid Schizophrenia

Sophie Jacobsson

Bachelor Degree in Cognitive Neuroscience C, 15hp

Spring 2010

School of Humanities and Informatics

The University of Skövde, Sweden

**Facial Emotion Processing in Paranoid and Non-Paranoid Schizophrenia**

Submitted by Sophie Jacobsson to the University of Skövde as a final year project towards the degree of B.Sc. in the School of Humanities and Informatics. The project has been supervised by Katja Valli.

**2010-06-07**

I hereby certify that all material in this final year project which is not my own work has been identified and that no work is included for which a degree has already been conferred on me.

Signature: \_\_\_\_\_

### Abstract

The aim of this essay is to investigate how paranoid and non-paranoid schizophrenic patients differ in the processing of emotional facial expressions from healthy individuals, and how this could lead to deficits in the area of social cognition. Researchers have conducted many behavioral and neuroimaging studies on facial emotion processing and emotion recognition in schizophrenia. Several studies have shown that individuals with schizophrenia have deficits in recognizing and processing emotional facial stimuli. It is known that patients with different subtypes of schizophrenia also show differences in facial emotion processing. It has also been shown that patients with schizophrenia uses different strategies in the processing of emotional faces compared to healthy individuals.

Key words: Schizophrenia, Paranoid Schizophrenia, Facial processing, Emotion, Social cognition

Table of contents

Abstract

Introduction .....5

1. Schizophrenia .....6

2. Neural Correlates of Facial Emotion Processing .....8

    3.1. Facial Emotion Processing in Schizophrenia .....12

    3.2. Facial Emotion Processing in Paranoid and Non-Paranoid Schizophrenia .....14

        3.2.1. Processing of Anger .....14

        3.2.2. Processing of Fear .....15

        3.2.3. Processing of Disgust .....17

        3.2.4. Processing of Trustworthiness .....17

    3.3. Cognitive Strategies in Facial Emotion Processing .....19

    3.4. The Role of Facial Emotion Processing in Social Cognition .....22

4. Discussion .....23

5. Limitations .....26

6. Future Research .....28

7. Conclusions .....28

References .....30

## 1. Introduction

The aim of this essay is to conduct a theoretical investigation of the deficit in facial emotion processing in two different subtypes of schizophrenia: Individuals with paranoid schizophrenia and individuals with non-paranoid schizophrenia and what effect it could have on social cognition. According to several studies (Doop & Park 2009; Gur et al., 2007; Michalopoulou et al., 2008) that have been reviewed, schizophrenic patients have difficulties in facial emotion processing and this may also have an effect on social cognition (Marwick & Hall, 2008). There are several approaches to study social cognition and studies in processing of facial expressions could be one way to come near the schizophrenics underlying deficits in social cognition which can give more understanding behind their apparent difficulties in daily life. It is important to study the underlying cognitive deficits because it could help in right diagnosing of different subtypes in schizophrenia, leading to right treatment and a possible better prognosis. Understanding of the neural basis of cognitive deficits in schizophrenia could also be a potential therapeutic tool for cognitive therapies (Marwick & Hall, 2008) as social cognition and interaction training (Roberts & Penn, 2008).

Recognizing of facial expressions is importance in social interaction, facial expressions has a communicative function to observers of them. Our facial expressions and recognition of them evolved as we became engaged in a more complex nonverbal communication. The perception and production of facial expressions are cognitive processes and specific cortical and sub cortical areas are responsible for these processes (Erickson & Schulkin, 2003). According to Erickson and Schulkin (2003) emotions and facial expressions are social and communicative tools, which serves as an interpreting function of other peoples' intentions and

goals. Humans and other animals use facial expressions to communicate information to other members in the species. Facial responses are regulated and modified through interactions with others. The ability to express and interpret facial expressions leads to increased fitness and survivability in an evolutionary perspective (Erickson & Schulkin, 2003). If schizophrenics individuals have deficits in facial emotion processing it means that they misinterpret the feelings of other people and this leads to deficits in understanding of other people and bad coping of social interactions. Schizophrenics deficit in facial emotion processing make it more understandable to see why they have severe difficulties of interactions with other people.

In this review of facial emotion processing in paranoid and non-paranoid schizophrenia, a presentation of schizophrenia will be given and a description of the differences between paranoid and non-paranoid schizophrenia. Then, a review of the neural correlates of general face processing in healthy individuals, studies that have been conducted on facial emotion processing in schizophrenic patients and differences in facial emotion processing in paranoid and non-paranoid will be presented. This essay also mentions the possible strategies for processing of emotional face expressions and how deficits in face emotion processing in schizophrenia have an influence on social cognition. The last section is devoted to a discussion about the results of the conducted studies with schizophrenic patients, the importance of perception and recognition of emotional facial expressions and suggestions for further studies.

## 2. Schizophrenia

According to *the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)*, schizophrenia is a common, chronic neuropsychiatric condition with symptoms including abnormal mental features, in other words, positive symptoms such as

hallucinations and delusions e.g. believing that one has been abducted by aliens and symptoms of absence in normal mental features, negative symptoms, including deficits in affect and social skills (American Psychiatric Association, 2000). The disorder is characterized by marked social and occupational dysfunction, and also difficulties in interpreting the emotions and intentions of other people (Marwick & Hall, 2008). Schizophrenia appears to have a strong genetic hereditary and it can be related to abnormalities in the structure and chemistry of the brain. Social factors such as stress could also be linked to the cause of schizophrenia. Medication is the most common part of the treatment in schizophrenia and it can reduce the psychotic symptoms. Schizophrenic patients could become needing of assistance in their daily life, with therapy they can improve social and occupational skills (American Psychiatric Association, 2000).

Schizophrenia can be divided into several different subtypes and it is known that patients diagnosed with different subtypes have differences in facial processing (Marwick & Hall, 2008). There are five subtypes in schizophrenia according to DSM-IV: The paranoid type (295.3), disorganized type (295.1), catatonic type (295.2), undifferentiated type (295.9) and the residual type (295.6). The paranoid type is suffering from delusions or frequent auditory hallucinations, this is the only subgroup that has paranoid symptoms and since this subtype have an additional of mental features (hallucinations and delusions) this means that they have positive symptoms and no reduced effect on negative symptoms such as inappropriate or flat affect etc. The disorganized type has affect that is flat or inappropriate, this means that this type have an absence of mental features since their affect have a negatively effect. This type is also disorganized in speech or behavior. The catatonic type has symptoms such as motor immobility, hyperactivity, mutism or negativism and peculiar behavior etc. The undifferentiated type meets only the basic criteria for schizophrenia, but no criteria for paranoid, disorganized or catatonic types. The residual type has one time met the criteria for catatonic, disorganized, paranoid or

undifferentiated schizophrenia but has no longer any symptoms from these subtypes. These patients have either negative symptoms such as reduced affect or characteristic symptoms of schizophrenia such as hallucination, delusions, disorganized speech or behavior etc. It is important to remember that all of these subtypes fulfill the basic criteria for schizophrenia but they are different in relating to each other (American Psychiatric Association, 1994).

In order to measure different subtypes in schizophrenia the most often used measure is the Marder Scale for paranoid and non-paranoid schizophrenia. Some studies classified schizophrenics as either paranoid (Paranoid type, 295.3) or non-paranoid (Catatonic, 295.2, Undifferentiated, 295.9, Disorganized type, 295.3 and Residual type, 295.6) and places all schizophrenics without paranoid symptoms into one group: the non-paranoid group in other words, so in this non-paranoid type one can find different schizophrenics subtypes, this means that there are an overrepresentation of non-paranoid schizophrenics. (Zalewski, Johnson-Selfridge, Ohriner, Zarrella & Seltzer, 2008).

### 3. The neural correlates of facial emotion processing

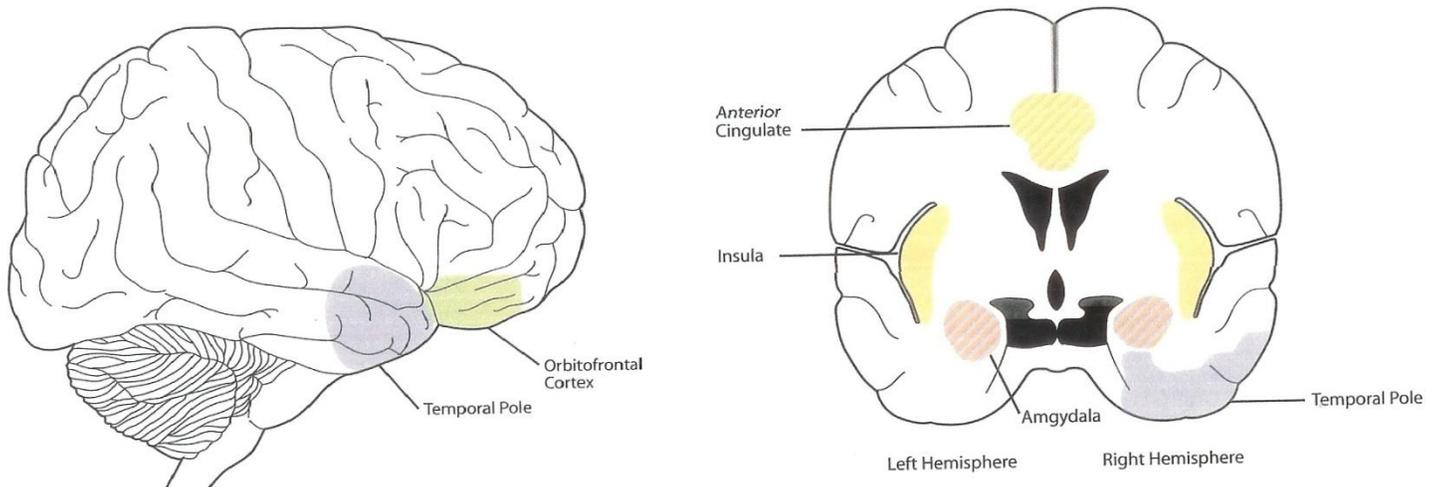
Face perception is essential to our social interactions (Gazzaniga, Ivry & Mangun, 2009b). The human face is the most common and most important visual stimuli we process. Faces are something that we process daily, and when we see someone's face we can easily judge how the person is feeling (Haxby, Hoffman & Gobbini., 2000; Itier & Batty, 2009). Based on other individuals' facial expressions we tend to interact with them differently, depending on our perception of the expression (Itier & Batty, 2009).

It can be difficult to define what an emotion is but Smith and Kosslyn (2007, p. 535) define emotion as "a relatively brief episode of synchronized responses (which can include

bodily responses, facial expression, and subjective evaluation) that indicates the evaluation of an internal or external event as significant". It seems that face expressions represent an inner feeling about the responses to environmental events or internal content.

When a social target has been identified, the next step is to determine if the target is willing to interact and if it is approachable. This type of social information arises from changeable aspects of the face, such as the eyes and the mouth. Changes in eye and mouth show facial expressions and are indicators of different emotions (Pinkham, Penn, Perkins & Lieberman, 2003). The amygdala is responsible for social and emotional stimuli (Adolphs, 2001), since amygdala is responsible for social and emotional stimuli, it is common that it activates during any kinds of facial expressions when one perceives a face (Gazzaniga et al., 2009b). The responsible cortical areas in general facial emotion processing is regions of the occipital and temporal cortices such as Fusiform Gyrus (FG) (Haxby et al., 2000), a region also called the Fusiform Face Area (FFA) (Gazzaniga et al., 2009b), and Superior Temporal Sulcus (STS) (Haxby et al., 2000).

Different emotions, in turn, seem to have different responsible brain areas (see Table 1.). For example, in James Blair's (1999) functional Magnetic Resonance Imaging (fMRI) study, the orbitofrontal cortex (OFC) was active when the participants viewed angry, but not sad faces. The activity of amygdala and right temporal pole is associated with sad faces while anterior insula and anterior cingulate cortex activity is linked to faces of disgust (Gazzaniga et al., 2009b). Gazzaniga et al., (2009b) suggest that the limbic system in the brain is responsible for emotion processing, and Adolphs (2001) suggests that several cortical regions in the temporal lobe activates when one is perceiving a social stimuli: the amygdala, right somatosensory cortices, orbitofrontal cortices and cingulate cortices (see figure 1 for activated brain areas).



**Fig. 1.** Brain pictures adopted directly from Gazzaniga et al., (2009), p. 384.

**Table 1**  
Emotions and associated brain areas

Emotion	Associated Brain Area
Fear, untrustworthiness	Amygdala
Trustworthiness, happiness	Orbitofrontal cortex
Sadness	Temporal Pole
Disgust	Anterior Insula, Anterior Cingulate Cortex

Structures involved in social cognition include sensory and association neocortex for social perceptual processing (e.g. superior temporal sulcus and fusiform gyrus). Also a network consisting of amygdala, prefrontal cortex, cingulate cortex, and right somatosensory-related cortices are activated in conjunction with social behavior (Adolphs, 2001). Somatosensory related cortices includes Brodmann areas 1, 2 and 3 and it contains of a somatotopic

representation of the body and it is also called the sensory homunculus (Gazzaniga et al., 2009b). Right somatosensory cortices are important in recognizing the feelings of other people. We recognize an individual's inner emotional state by having internally somatosensory representations that tells us how an individual feels when one is perceiving a facial expression. (Adolphs, Damasio, Tranel, Cooper & Damasio, 2000)

Winston, Strange, Doherty and Dolan (2002) performed an fMRI study on the perception of trustworthiness of faces. Subjects viewed faces and rated them in trustworthiness. Happy faces were rated as significantly more trustworthy than neutral faces, whereas angry and sad faces were rated as significantly less trustworthy. Significant activation bilaterally in amygdala was found in the contrast of untrustworthy to trustworthy faces. The more untrustworthy the subjects rated faces, the greater was the Blood Oxygenation Level Dependent (BOLD) response evoked in the amygdala. In contrast, orbitofrontal cortex was activated during processing of trustworthy faces. Gazzaniga et al. (2009b) suggested that the amygdala should be activated during any kind of facial expression but in this study (Winston et al., 2002) the amygdala was significant more activated during processing of untrustworthy faces such as angry and fearful faces, this could mean that negative facial expressions, such as untrustworthy faces, evoke a greater activation in amygdala than neutral or positive expressions.

In sum, the following areas are actively involved in face processing in healthy individuals: FG and STS are involved in general face processing. Processing of emotional faces activates the limbic system in the brain, but specifically the amygdala (Gazzaniga et al., 2009b). Other important brain areas that activate when one is perceives social stimuli, such as emotional faces are: right somatosensory cortices, orbitofrontal cortices and cingulate cortices. Social cognition network includes sensory and association neocortex (e.g. STS and FG), and a network

consisting of amygdala, prefrontal cortex, cingulate cortex, and right somatosensory related cortices.

### 3. 1. Facial emotion processing in schizophrenia

Patients with schizophrenia have deficits in recognition and identification of emotional expressions in faces. It is still unclear whether these deficits result from abnormal processing of emotional faces or an impaired ability to process complex visual stimuli such as faces (Doop & Park, 2009). Doop and Park (2009) found a correlation between worse social functioning and facial emotion matching errors in a study with patients with schizophrenia and a healthy control group. Patients with schizophrenia showed general deficits in processing of emotional facial expressions which is associated with reduced social functioning. In a study by Radua (2010) they found that the neural response to eyes was similar in patients with schizophrenia and healthy individuals. On the other hand, they detected a lower activation in amygdala and hippocampus region in schizophrenic patients.

Gur et al. (2007) conducted an event-related fMRI study and they found that patients with schizophrenia showed reduced limbic activation compared to healthy controls in a facial emotion identification task. In controls, the analysis showed that greater amygdala activation was associated with correct identifications of threat-related (anger and fear) expressions. Analysis showed significant activation for the emotion identification task in a network of regions which included amygdala, hippocampus, thalamus, FG and frontal and visual association cortex in healthy controls and schizophrenic patients, however, several regions showed significantly greater activation in controls. In controls there were significantly different activation levels in amygdala and other limbic regions related to anger and fear compared to neutral stimuli. Controls showed greater activation for correct responses to angry faces in inferior

frontal and orbitofrontal regions, in contrast patients with schizophrenia showed less activation for correct identification of threat-related expressions of anger and fear. The difference between patients and controls was significant for anger in FG and amygdala, it was also significant for fear in all regions.

Michalopoulou et al. (2008) used fearful stimuli in an fMRI study. The participants viewed grey-scaled images showing facial emotional expressions of fear, sadness and neutral faces. In healthy subjects, the processing of fearful faces showed a significantly greater activation within the amygdala, right-sided parahippocampal gyrus, FG, superior temporal gyrus, middle occipital and temporal gyri and right insula than the processing of neutral faces. Processing of facial fear led to a significantly greater activation of amygdala in healthy subjects compared to schizophrenic patients, similar to what was found by Gur et al. (2007). Healthy subjects also had greater activation within the right FG and superior temporal gyrus in response to fearful faces compared to neutral faces, compatible with the results of the study by Gur et al. (2007). Participants with schizophrenia showed significantly reduced activation within these regions during facial fear processing compared to healthy controls. These results may reflect a deficit in the visual processing of facial fear in people with schizophrenia.

The above-mentioned studies show that patients with schizophrenia have reduced limbic activation in processing of emotional expressions compared to healthy individuals. Correct identifications of threat-related expressions, such as anger and fear, were linked to greater activation of the amygdala and other limbic regions in healthy controls compared to neutral stimuli. In contrast, patients with schizophrenia had reduced activation for correct identifications of threat-related expressions. The difference between patients and controls was significant for anger in FG and amygdala and for fear in all regions (amygdala, hippocampus, thalamus, FG,

frontal and visual association cortex). The conclusion of this is that patients not only have deficits in emotional processing of faces, they also show a deficit in the processing of faces in general, since FG activation is lower in schizophrenic patients during processing of emotional expressions and FG is involved in general face processing (Gazzaniga et al., 2009b).

### 3.2. Facial emotion processing in paranoid and non-paranoid schizophrenia

Individuals with non-paranoid schizophrenia as opposed to individuals diagnosed with paranoid schizophrenia have normal levels of brain activation during passive viewing of emotional facial expressions (Williams et al., 2004). This explains why individuals with non-paranoid schizophrenia rate faces differently from individuals with paranoid schizophrenia but similarly to healthy individuals (Pinkham et al., 2008).

One extensive study of several kinds of different facial expressions was made by Phillips et al. (1999). They compared the neural responses to facial expressions in paranoid, non-paranoid schizophrenic patients and healthy subjects with fMRI scanning. Subjects viewed black and white facial expressions of fear, anger and disgust, and also expressions of mild happiness. All patients were less accurate in identifying emotional expressions and they also showed less activation to these expressions than healthy controls.

In the following section I will present different studies investigating different emotional expressions such as threat related emotions, such as anger and fear, and also non-threatening face expressions such as disgust and trustworthy faces in paranoid and non-paranoid schizophrenia.

#### 3.2.1. Processing of anger

In the study by Phillips et al. (1999), in healthy individuals the inferior frontal gyrus, anterior cingulate gyrus, putamen, superior temporal gyrus, hippocampus, cerebellum and FG were activated in response to angry expressions. In healthy individuals, activation in response to neutral expressions could be seen in the cerebellum, superior temporal gyrus, anterior cingulate gyrus, medial frontal gyrus and caudate nucleus (deactivation of angry expression). Activation in response to neutral expressions in the paranoid schizophrenics was shown in the inferior frontal gyrus and anterior cingulate gyrus and a little activation in response to angry expressions. Non-paranoid schizophrenics showed activation in the posterior cingulate gyrus, inferior temporal gyrus, cerebellum and thalamus in response to angry expressions, and little activation to neutral expressions.

These results show a reduced neural response in both groups of schizophrenic patients, but especially in paranoid schizophrenics, in regions activated by angry expressions compared to healthy individuals. Healthy individuals showed significantly greater activation in response to angry expressions compared to the schizophrenic patients in the inferior frontal gyrus, putamen and cerebellum, and also significantly greater activation to neutral expressions in the superior temporal gyrus, medial frontal gyrus and anterior cingulate gyrus. Non-paranoid schizophrenics showed significantly greater activation in the cerebellum, thalamus and inferior temporal gyrus to expressions of anger compared to paranoid schizophrenics. No brain regions were activated significantly more in the paranoid schizophrenics.

### 3.2.2. Processing of fear

A study by Phillips et al. (1999) showed that in healthy controls, fearful expressions activated the limbic system and visual cortical regions: hippocampus, amygdala, the

middle temporal gyrus, the superior temporal gyrus, putamen, cerebellum and the inferior frontal gyrus. Activation in response to neutral expressions could be seen in superior and middle temporal gyri. In all schizophrenic patients, fearful expressions activated the postcentral gyrus and inferior frontal gyrus and activation in response to neutral expressions was demonstrated in visual areas: the middle temporal gyrus and fusiform gyrus. Paranoid schizophrenics showed activation in the fusiform gyrus, cerebellum, precentral gyrus and lingual gyrus in response to fearful expressions, but not in the amygdala. Non-paranoid schizophrenics showed activation in the middle and superior temporal gyri, also in non-paranoid schizophrenics there was no activation in the amygdala in response to fearful expressions. Healthy individuals showed significantly greater activation compared with the schizophrenic patients in response to fearful expressions in the superior temporal gyrus, amygdala and putamen. No brain regions were activated significantly more in the schizophrenic patients compared to healthy individuals.

Williams et al. (2004) found, when participants were viewing fearful and neutral facial expressions, that emotion recognition was significantly impaired in the schizophrenia patients compared to healthy controls. There were no brain regions that showed greater activity in schizophrenic patients than in healthy subjects. Regarding differences between paranoid and non-paranoid schizophrenia, paranoid patients had greater impairment for recognition of fear. Paranoid patients had also significant reduction in the lateral prefrontal cortex compared to non-paranoid schizophrenics.

In the same study (Williams et al., 2004) processing of fearful faces in healthy subjects activated limbic, prefrontal and visual brain regions. Schizophrenic patients showed significantly reduced activity in the right amygdala, related central gray region, both medial and lateral prefrontal cortices and bilaterally in fusiform gyrus in comparison with healthy subjects.

Also an additional reduction in the visual region could be observed in both subtypes of schizophrenia. Patients with paranoid schizophrenia showed significantly reduced activity, compared to healthy subjects, in the right amygdala, central gray area, both medial and lateral prefrontal cortices and bilateral fusiform gyri. Paranoid patients showed a reduction in the lateral prefrontal cortex relative to non-paranoid patients, and together with reductions in amygdala, it leads to impairment in integration and processing of threat-related signals in this schizophrenic subtype.

### 3.2.3. Processing of disgust

In the study by Phillips et al. (1999), the processing of disgust activated the insula and striatum in healthy individuals. In schizophrenic patients the insula was also activated in response to facial expressions of disgust. Non-paranoid schizophrenics showed significantly greater activation in response to expressions of disgust in the superior temporal gyrus, thalamus, postcentral gyrus and amygdala compared with paranoid schizophrenics (Phillips et al., 1999).

### 3.2.4. Processing of trustworthiness

Pinkham, Hopfinger, Pelphrey, Piven and Penn (2008) used both paranoid individuals with schizophrenia and another group with schizophrenia but without any paranoid tendencies. All groups in the study showed significant activation of the social cognitive network: amygdala, fusiform face area (FFA), STS and ventrolateral prefrontal cortex (VLPFC) while they were judging the trustworthiness of faces. Subjects with paranoid schizophrenia showed significantly reduced neural activation in the right amygdala, FFA and left VLPFC compared to controls and in the left VLPFC as compared to non-paranoid schizophrenia individuals during the task. Non-paranoid schizophrenia group also showed significantly less activation in the FFA

compared to healthy control, but in contrast to the other group, non paranoid individuals showed similar levels of activation to controls in all the other brain regions. The results demonstrated that the patients with paranoid schizophrenia have significant reductions in neural activation during tasks involving judgment of complex social stimuli and therefore they have also deficits in complex social cognition.

Phillips et al. (1999) suggests that the absence of amygdala activation in schizophrenic patients and their impairment in emotion recognition and emotion processing may be linked to reduced neural responses in limbic structures. In schizophrenic patients, visual cortical regions and regions important for visual processing (fusiform, lingual and temporal gyri) did not fail to activate. Therefore, the reduced neural response in schizophrenic patients was not a reflection of the schizophrenics' avoidance of the facial stimuli since the visual regions were activated. Phillips et al. 1999 suggest that the impaired neural response in the amygdala, insula, frontal regions and to a lesser extent, visual cortical regions, during emotional stimuli, may be a result of specific deficits in the attention and processing of emotional visual information.

The conclusion that can be drawn based on these studies is that non-paranoid individuals have similar levels of activation in the processing of emotional stimuli as healthy individuals when compared to paranoids. Paranoid individuals had lower levels of activation in response to angry expressions compared to healthy controls. Non-paranoids had greater activation than paranoid individuals in processing of angry expressions. Both patient groups had reduced activation in response to angry expressions compared to healthy individuals. In the matter of the expressions of fear, paranoids showed activation in FG but not in the amygdala, and non-paranoids had no amygdala activation in response to expressions of fear. Healthy individuals showed a greater activation in amygdala in response to this kind of stimuli and they also showed

an activation of limbic, prefrontal and visual regions in the brain, whereas schizophrenics showed reduced activity in the amygdala, prefrontal cortices and bilateral FG, compared to healthy individuals. In processing of trustworthy faces, paranoids showed a reduced neural activation in the right amygdala, FFA and left VLPFC compared to controls and in left VLPFC compared to non-paranoids. Non-paranoids had also a reduced activation in FFA compared to healthy individuals but they had similar levels of activation in all the other brain regions (amygdala, STS and VLPFC).

### 3.3. Cognitive strategies in facial emotion processing

Matching emotional faces can be done with different strategies. One is to process the facial expression holistically, in other words, to use a configuration-based strategy which enables an intuitive and automatic processing of the emotion on the face. Another strategy is to match common features individually and independently by a feature-based strategy. The schizophrenic patients use the second one of these strategies, according to Fakra, Salgado-Pineda, Delaveau, Hariri and Blin (2008), because in these patients' brain the regions that are involved in emotional face processing, such as bilateral amygdala, putamen and inferior frontal cortex (Williams et al., 2004), fail to activate.

Schizophrenic patients have reduced activation in left FG. FG is a region that is highly associated with face processing (Haxby et al., 2000) and schizophrenic individuals usually adopt a feature-based strategy when they process emotional faces. The schizophrenic patients had longer reaction times in the matching condition compared to healthy subjects in the study by Fakra et al. (2008). The increased reaction times represent the use of a more time-consuming cortical pathway compared to automatic or intuitive emotional face processing (Fakra et al.,

2008).

Fakra et al. (2008) investigated the neural bases of different cognitive strategies for emotional face processing in schizophrenia. The task was to perform facial affect identification and matching of emotional faces during fMRI scanning. Fourteen schizophrenic patients and fourteen matched controls participated in the study. The results were that both groups had similar networks engaged in facial affect processing, but in the schizophrenic patients, regions of the limbic system, especially the amygdala which is a part of automatic processing in emotions (Gazzaniga et al., 2009b), failed to activate in response to stimuli. Instead, neocortical areas such as inferior parietal cortex (IPC), left middle temporal cortex and right precuneus were activated, which are normally not engaged in emotional judging of stimuli.

The results by Fakra et al. (2008) showed a greater bilateral BOLD response in amygdala in healthy subjects compared to schizophrenic patients, but in contrast, the schizophrenic patients showed an increased response in neocortical network. Psychophysiological interactions (PPI) revealed a functional connectivity between prefrontal regions and the left amygdala in healthy controls, but the PPI couldn't reveal such interactions in schizophrenic patients. Compared to controls the patients showed decreased activation of regions involved in face processing (FG) and increased activation in brain regions associated with feature analysis (inferior parietal cortex, left middle temporal lobe and right precuneus). These findings suggest that schizophrenic patients adopt a different kind of cognitive approach when identifying facial expressions. The results indicate that patients may use a feature-based rather than configuration-based processing, and this means that schizophrenic patients may be using a compensatory strategy due to limbic dysfunction.

Fakra et al. (2008) investigated the neural activity during two different types of emotional face processing, the matching condition which is based on automatic and intuitive processing, and the labeling condition which is dependent on higher cognitive processes, such as judgment and interpretation of emotional facial expressions. They found activation of both cortical and sub-cortical structures, such as amygdala, anterior and middle cingulate as well as parahippocampal gyri, putamen, thalamic pulvinar nuclei and right middle temporal gyrus. These areas are involved in automatic processing of emotional information. During the labeling condition they observed activation of cortical and sub-cortical regions, such as medial prefrontal regions and hippocampus nuclei, which are instead involved in the cognitive processing of emotional stimuli. Both labeling and matching conditions activated inferior occipital and FG, which are involved in face processing (Gazzaniga et al., 2009b). The subjects with schizophrenia had similar activation patterns for both matching and labeling conditions as healthy subjects. But during the matching condition the patients had significantly lower activation in several limbic structures, especially in the amygdala. The patients with schizophrenia did not show any correlation between activity in amygdala and other brain regions, and this may be so because the patients may be employing a different cognitive strategy when they are faced with emotional tasks, rather than to process it automatically or intuitively.

Neuroimaging studies of schizophrenic patients performing emotion facial tasks have shown dysfunction of the limbic system, especially in the amygdala and the frontal areas compared to healthy individuals. Neuroimaging methods demonstrate a failure to activate amygdala in schizophrenic individuals (Fakra et al., 2008). Fakra et al. (2008) suggest, based on other studies, that this lack of limbic activation is compensated by an over activation of the PFC in schizophrenic individuals. This could be a possible answer to why amygdala is under activated

in schizophrenic patients, because they use another strategy to process emotional faces. Instead of processing faces emotionally they rather process them cognitively. Neuroimaging studies have pointed out that the amygdala is involved in processing of emotional facial expressions, particularly negative emotions such as fear and anger (Phillips et al., 1999). Activation of the amygdala is only apparent when subjects perform automatic or intuitive processing of emotional stimuli (Fakra et al., 2008). Interestingly, this activation is reduced when subjects are asked to label the emotion or to attend to non-emotional characteristics of the stimuli. Neuroimaging studies have also revealed that regions of the neocortex, such as the prefrontal cortex, are involved in the cognitive appraisal of processing emotional stimuli and the inhibition of emotional reactions. These findings suggest that different cortical regions are involved when identifying facial affect depending on the strategy (intuitive or cognitive) (Fakra et al., 2008).

#### 3.4. The role of facial emotion processing in social cognition

It is important to recognize expressions in each face because of the ability to adapt to the social environment. Schizophrenics often perform poorly in social interactions and they also tend to misinterpret social cues (Phillips et al., 1999). In the studies that have been presented in this review, one can see that schizophrenics clearly have deficits in processing of emotional stimuli and that it has an effect on social cognition.

Social cognition is defined as: "social cognition is the processing of any information which culminates in the accurate perception of the dispositions and intentions of other individuals" (Brothers, 1990, p. 28). The cortex surrounding the STS along with the FG is associated with social cognition, and these regions are involved in visual perception of socially relevant stimuli (Adolphs, 2001). As schizophrenics have deficits in social cognition, this may

have an influence on their social functioning (Michalopoulou et al., 2008), which could be linked to schizophrenics' deficits in facial emotion processing, according to Doop and Park (2009).

In a review by Marwick and Hall (2008), they discuss the relation between face processing and social cognition in schizophrenia. Marwick and Hall (2008) suggest that a deficit in facial identification may lead to social difficulties. Several brain areas are involved in social cognition: the STS, amygdala and prefrontal cortex. During social judgment of trustworthy faces, schizophrenics show abnormalities in blood flow, specifically to amygdala (Pinkham et al., 2008). There is a link between brain regions that are abnormal in schizophrenia and social judgment from facial expressions, and this could mean that schizophrenic patients have deficits in several components which are involved in social cognition and this leads to difficulties in processing of emotional stimuli (Marwick & Hall, 2008).

#### 4. Discussion

Patients with schizophrenia have significant impairments in social cognition and social functioning (Pinkham et al., 2008). Research suggests that their social dysfunction has a cause in the neural systems that underlie social cognition. Pinkham et al. (2008) speculates that it is possible that individuals with paranoid schizophrenia are not applying appropriate emotional significance to facial stimuli when they are asked to make a complex social judgment, and this may be an explanation for their social dysfunction. Schizophrenic patients have greater impairment for perception of negative emotional stimuli compared to positive facial affect, especially perception of fear (Phillips et al., 1999; Williams et al., 2005).

Neuroimaging studies of facial emotion processing shows that individuals with non-paranoid schizophrenia are better at facial emotion perception than individuals with paranoid

schizophrenia. Phillips et al. (1999) discuss the underlying causes to paranoid schizophrenics' difficulties in identifying of threat-related emotions, anger and fear. First, schizophrenics identify a threat when there is none, but the amygdala has decreased activation during threat-related stimuli, such as anger and fear, and this is true in both paranoid and non-paranoid individuals. Second, schizophrenics avoid the threat, but then the visual regions and FFA shouldn't be activated, but they are so, this is true according to the study by Phillips et al. (1999). On the other hand, studies by Michalopoulou et al. (2008) and Williams et al. (2005), comes to different conclusions regarding activation of the visual regions in Schizophrenic patients. Visual regions have a reduced activation during processing of fear in the study by Michalopoulou et al. (2008) and Williams et al. (2005) in both paranoid and non-paranoid schizophrenia, compared to healthy individuals. These contradictions could be explained by the year of the carrying out studies since the studies by Michalopoulou et al. (2008) and Williams et al. (2005) is more recent compared to the study by Phillips et al. (1999). This conveys that the result of the more recent studies is more reliable than the study by Phillips et al. (1999). The third suggestion from Phillips et al. (1999) of underlying causes of Schizophrenics deficits in identifying of threat-related emotions is that schizophrenics have deficits in labeling the correct emotion. Explanation number three that schizophrenics have deficits in labeling emotions correctly seems to be right together with explanation two, according to all the reviewed studies here. It also seems that paranoids especially have troubles with processing of emotional face stimuli. The amygdala has been linked to recognition of negative emotion perception, especially threat detection. Individuals with schizophrenia have abnormal amygdala activation (Breiter et al., 1996) and several functional imaging studies have shown that these individuals are less accurate in identifying emotions and they also show reduced amygdala activation to fearful expressions (Michalopoulou et al., 2005), The amygdala is an important area for social cognition (Pinkham, 2003) and since schizophrenic

individuals have abnormal amygdala activation, they also have deficits in social cognition.

The social environment is complex, and it is hard to predict other people's behavior. Facial expressions are a cue that informs us how one self should behave (Adolphs, 2001). Social cognition has been shaped by evolution. Early human social groups were smaller (Adolphs, 2001), and we needed to evolve our social cognition when the social groups were becoming bigger, in order to understand the complex emotional facial expressions which we developed. In many situations, individuals must decide whether one should approach someone or avoid the person, trust or distrust the person (Winston et al., 2002). Thus, Facial emotion recognition may have an evolutionary basis. Some emotional facial expressions might serve as reliable signals of threat (Radua et al., 2010). Lesion studies have demonstrated that subjects with bilateral damage in the amygdala judge people to look more trustworthy than normal subjects do, the amygdala makes it possible for us to discover possible threats (Adolphs, 2001). When we meet other people we tend to look on their faces more than their bodies, as the facial expressions provide the most cues regarding affective states. Facial expression is one example of emotional behavior, important in both social cognition and basic survival (Erickson & Schulkin, 2003).

Impaired recognition of threat related expressions (anger and fear) may result from dysfunction of emotional face processing and this could lead to misinterpretations of expressions as being threatening. Schizophrenics have abnormalities in the social network in the brain. Schizophrenics also show delayed interpretation of correct identification of emotional expressions, accurate and fast responses to threat stimuli is critical for basic survival (Green & Phillips, 2004). Emotions are important to correctly recognize because they prepare our behavior to the right action, e.g., running when you face a dangerous encounter. People interpret facial expressions and based on that they decide how to react (Gazzaniga, Heatherton & Halpern,

2009a). It can mean a lot of difficulties if schizophrenics' don't have the ability to match the right expression to the right emotion, as they end up with difficulties in interpreting other people's emotions and predicting others behavior. The conclusion could be that schizophrenic individuals, especially paranoid schizophrenics, would not have the best survivability based on their deficits in interpreting emotional expressions.

## 5. Limitations

It can be somewhat difficult to study emotions and emotional face processing systematically (Gazzaniga et al., 2009b). It is also problematic to come up with an absolute definition of what an emotion is. Some studies have few participants, such as the study by Phillips et al. (2004), this could be an explanation to why this study comes to different conclusions than the studies by Pinkham et al. (2008) and Williams et al. (2004), regarding visual processing of faces. It is problematic to divide schizophrenia into paranoid and non-paranoid schizophrenia, since the "subtype" non-paranoid schizophrenia contains all other subtypes in schizophrenia except the one with paranoid Schizophrenia (American Psychiatric Association, 2000), this means that there could be a high variety in the non-paranoid subtype regarding processing of emotional faces. There is more problems in dividing schizophrenic patients into a paranoid or a non-paranoid subtype, one individual can be characterized with paranoid symptoms, while in earlier life the same individual could be fitted in the non-paranoid subtype, this would results in that non-paranoid subtypes has longer mean duration of illness (Zalewski et al., 2008). Studies conducted with schizophrenic patients are also problematic, because the patients are often on medication, and this may influence the results. Antipsychotic medication could affect limbic regions and also some regions of neocortex, but, on the other hand, the literature suggests that antipsychotic medication has little influence on activation patterns

(Phillips et al., 2003); therefore medication cannot be used as treatment option to alleviate symptoms related to social functioning.

There are also some limitations with this review. It only takes brainimaging studies in account, excluding behavioral, electrophysiological and neuroanatomical studies due to several reasons. First: not many studies have investigating the differences in paranoid and non-paranoid schizophrenia, and the most studies that have been conducted have been used brainimaging methods. Second: Since not many studies have been conducted with paranoid and non-paranoid schizophrenia with other methods, this review only take the brainimaging in account when the deficits in facial emotion processing in schizophrenia is reviewed. Otherwise, it is hard to compare the studies between healthy, schizophrenia in general and paranoid and non-paranoid schizophrenia, if this review take other kinds of methods in account and using of behavioral, electrophysiological and neuroanatomical results when schizophrenia is reviewed, it is complicating to compare these results with only brainimaging studies of the differences between paranoid and non-paranoid schizophrenia regarding facial emotion processing. Third: If It had been conducted more research regarding the differences between paranoid and non-paranoid Schizophrenia it would, anyway, be too extensiveness to review behavioral, electrophysiological and neuroanatomical studies. These are the reasons to why this review only takes brainimaging in account when investigating the differences between paranoid and non-paranoid schizophrenia, this should be take into consideration when making conclusions of the studies above since other methods could possibly be revealing different results than results of neuroimaging studies.

## 6. Future research

More research should be done with different using of methods such as behavioral,

electrophysiological and neuroanatomical studies investigating the differences between paranoid and non-paranoid schizophrenia and the deficits in facial emotion processing. Researchers in the field should investigate other kinds of treatment beyond medication. In Roberts and Penn's (2009) study they investigate potential training in social cognition and interaction in schizophrenic patients. Social cognition and interaction (SCIT) is a 20-week group treatment which is designed to improve social functioning in schizophrenia by improved social cognition. The treatment contains of several phases: Emotions, which handles dysfunction of emotion perception, and integration in where the participants take the learned skills into their own lives. In the study by Roberts & Penn (2008) they used an emotion perception task as a social cognitive measure, and in the future, we need to investigate not only the schizophrenic patients, but also the different kind of subtypes in this disorder, regarding facial emotion processing. Processing of facial emotion recognition could be done by different approaches, for example, by viewing facial expressions and then matching a similar expression, or identify a facial expression (Roberts & Penn (2008). The next step could be to use standardized movies of social interactions as a measure of processing of emotional faces, and investigate how these stimuli could be linked to social cognition through tests in social functioning. Based on this, further training of social cognition and interaction should be developed, such as SCIT.

## 7. Conclusions

This review comes to several conclusions based on the research in facial emotion processing of schizophrenia: Individuals with schizophrenia have deficits in recognition and processing facial emotion stimuli compared to healthy individuals. Schizophrenic patients show greater impairment for perception of negative emotions compared to positive emotions, with the greatest impairment for the perception of fear. Research has shown that individuals with non-

paranoid schizophrenia are better at recognizing fear and disgust but not anger than paranoid individuals. Amygdala is one brain area which is involved in the processing of emotional expressions of faces. The amygdala shows a greater activation in negative facial expressions, especially fear, than in neutral facial expressions, and it has also been linked to threat detection. Individuals with schizophrenia are not only less accurate in identifying emotional stimuli, they also show decreased amygdala activation to fearful expressions, this makes it harder for these patients to recognize threat-related stimuli. Schizophrenic patients may have difficulties in recognizing fear due to deficits in the visual network (Williams et al., 2004), and it could be an explanation to why they troubles with facial emotion processing. Deficits in processing of emotional stimuli are likely to lead to deficits in social cognition.

## References

- Adolphs, R., Damasio, H., Tranel, D., Cooper, G., Damasio, A. R. (2000). A role for somatosensory cortices in the visual recognition of emotion as revealed by three-dimensional lesion mapping. *The Journal of Neuroscience*, 20(7), 2683–2690.
- Adolphs, R. (2001). The neurobiology of social cognition. *Current Opinion of Neurobiology*, 11(2), 231-239.
- American Psychiatric Association. (2000). *Diagnostic and statistic manual of mental disorders* (4th ed.). Washington DC: American Psychiatric Press.
- Blair, R. J. R., Morris, J. S., Frith, C. D., Perret, D. I and Dolan R. J. (1999). Dissociable neural responses to facial expressions of sadness and anger. *Brain*, 122, 883, 893.
- Breiter, H. C., Etcoff., N. L., Whalen, P. J., Kennedy, W. A., Rauch, S. L., Buckner, R. L., Strauss, M. M., Hyman, S. E., & Rosen, B. R. (1996). Response and habituation of the human amygdala during visual processing of facial expression. *Neuron*, 17, 875-887.
- Brothers, L. The social brain: a project for integrating primate behavior and neurophysiology in a new domain. *Concepts Neurosci*, 1990, 1, 27–51.
- Couture, S. M., Penn, D. L, & Roberts, D. L. (2006). The functional significance of social cognition in schizophrenia: A review. *Schizophrenia Bulletin*, 32(S1), S44-S63.

- Doop, M. K & Park, S. (2009). Facial expression and face orientation processing in schizophrenia. *Psychiatry Research*, 170(2-3), 103-107.
- Erickson, K., & Skulking, J. (2003). Facial expressions of emotion: A cognitive neuroscience perspective. *Brain and Cognition*, 52(1), 52-60.
- Fakra, E., Salgado-Pineda, P., Delaveau, P., Hariri, A. R., & Blin, O. (2008). Neural bases of different cognitive strategies for facial affect in schizophrenia. *Schizophrenia Research*, 100(1-3), 191-205.
- Gazzaniga, M. S., Heatherton, T. F., Halpern, D. F. (2009a). *Psychological science*. Norton: London.
- Gazzaniga, M. S., Ivry, R. B., & Mangun, G. R. (2009b). *Cognitive neuroscience: The biology of the mind*. Norton: New York.
- Green, M. J. & Phillips, M. L. (2004). Social threat perception and the evolution of paranoia. *Neuroscience and Biobehavioral Reviews*. 28, 333-342.
- Gur, R. E., Loughhead, J., Kohler, C. G., Elliot, M. A., Lesko, K., Ruparel, K., Wolf, D. H., Bilker, W. B., & Gur, R. C. (2007). Limbic activation associated with misidentification of fearful faces and flat affect in schizophrenia. *Archives of General Psychiatry*, 64(12), 1356-1366.
- Haxby, J. V., Hoffman, E. A., & Gobbini, M. I. (2000). The distributed human neural system for face perception. *Trends in Cognitive Sciences*, 4(6), 223-232.

Hooker, C. & Park, S. (2002). Emotion processing and its relationship to social functioning in Schizophrenia patients. *Psychiatry research*, 112(1), 41-50.

Itier, R. J., & Batty, M. (2009). Neural bases of eye and gaze processing: The core of social cognition. *Neuroscience and Biobehavioral Reviews*, 33(6), 843-863.

Marwick, K., & Hall, J. (2008). Social cognition in schizophrenia: a review of face processing. *British Medical Bulletin*, 88, 43-58.

Michalopoulou, P. G., Surguladze, S., Morley L. A., Giampietro, V. P., Murray, R. M., & Shergill, S., S. (2008). Facial fear processing and psychotic symptoms in schizophrenia: functional magnetic resonance imaging study. *The British Journal of Psychiatry*, 192, 191-196.

Phillips, M. L., Williams, L., Senior, C., Bullmore, B. T., Brammer, M. J., Andrew, C., Williams S. C, David A. S. (1999). A differential neural response to threatening and non-threatening negative facial expressions in paranoid and non-paranoid schizophrenics. *Psychiatry Research*, 92, 11–31.

Pinkham, A.E., Hopfinger, J.B., Pelphrey, K.A.; Piven, J.; Penn, D.L. (2008). Neural bases for impaired social cognition in schizophrenia and autism spectrum disorders. *Schizophrenia Research*, 99(1-3), 164-175.

- Pinkham, A. E., Penn, D. L., Perkins, D. O., & Lieberman, J. (2003). Implications for the neural basis of social cognition for the study of schizophrenia. *The American Journal of Psychiatry*, 160 (5), 815-824.
- Radua, J., Phillips, M. L., Russell, T., Lawrence, N., Marshall, N., Kalidindi, S., El-Hage, W., McDonald, C., Giampietro, V., Brammer, M. J., David, A. S., Surguladze, S. A. (2010). Neural response to specific components of fearful faces in healthy and schizophrenic adults. *Neuroimage*, 49(1), 939-946.
- Reske, M., Habel, U., Kellermann, T., Backes, V., Shah, N. J., Wilmsdorff, M. V., Gaebel, W., Zilles, & K., Schneider, F. (2009). Differential brain activation during facial emotion discrimination in first-episode schizophrenia. *Journal of Psychiatric Research*, 43, 592-599.
- Roberts, D. L., Penn, D. L. (2009). Social cognition and interaction training (SCIT) for outpatients with schizophrenia: A preliminary study. *Psychiatry Research*, 166, 141-147.
- Smith, E. E. & Kosslyn, S. M. (2007). *Cognitive Psychology: Mind and Brain*. Pearson: New Jersey.
- Williams, L. M., Das, P., Harris, A. W. F., Liddell, B. B., Brammer, M. J., Olivieri, G., Skerrett, D., Phillips, M. L., David, A. S., Peduto, A., & Gordon, E. (2004). Dysregulation of arousal and amygdala-prefrontal systems in paranoid schizophrenia. *American*

*Journal of Psychiatry*, 161(3), 480-490.

Winston, J. A., Strange, B. A., Doherty, J. O., & Dolan, R. J. (2002). Automatic and intentional brain responses during evaluation of trustworthiness of faces. *Nature Neuroscience*, 5(3), 277-283.

Zalewski, C., Johnson-Selfridge, M. T., Ohriner, S., Zarrella, K., Seltzer, J. C. (2008). A review of neuropsychological differences between paranoid and nonparanoid schizophrenia. *Schizophrenia Bulletin*, 24(1), 127-145.