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New Findings for Face Processing Deficits in the Mental Disorder of Schizophrenia

Toshiaki Onitsuka[1], Tomoya Tsunoda[1,2] and Naotoshi Ohara[1]
[1]Department of Neuropsychiatry, Graduate School of Medical Sciences,
Kyushu University
[2]Department of Neuropsychiatry, Self-Defense Forces Sasebo Hospital
Japan

1. Introduction

Human faces may be one of the most familiar visual stimuli, and the most important stimulus for human social life. The highly evolved human ability to recognize faces represents an important component of species social communication, naturally selected to solve adaptive problems critical for survival, such as distinguishing friend from foe, familiar from unfamiliar, related from unrelated. Moreover, face recognition represents an evolutionarily significant element of nonverbal decoding that may be viewed as a neuropsychological building block for sociality that is a core ingredient of extraversion. Indeed, a critical component of the kind of effective nonverbal decoding seen in extraverts may presuppose a capacity to extract social and affective cues from faces. Face recognition may be qualitatively different from other memory recognition systems in terms of the underlying neurobiological structure and developmental trajectory. It has been suggested that a special brain processing network, present early in development, underlies face perception. For example, two-month-old infants recognize their mothers' faces among others (Morton & Johnson, 1991), showing that infants have the perceptual skill of eye gaze from birth (Farroni et al., 2002). These studies suggest that face recognition is formed in the first few months of life. Face recognition includes many processes (perception of the face configuration, retention, memorization, and verification of face images). There has been uniform agreement that the facial recognition processes involves multiple brain areas.

In the last several decades, methods of cognitive neuroscience have been applied fruitfully to the study the cognitive deficits in schizophrenia; however, the studies of the processes for facial recognition and recognition of facial expressions have received less attention. Recently, application of neuroscience techniques, including electroencephalography (EEG), magnetoencephalography (MEG), and structural and functional magnetic resonance imaging (MRI) to the study of face recognition has produced a new field of neuroscience. Therefore, abnormalities in the face recognition processes associated with psychiatric diseases have been revealed using these techniques.

This chapter first presents an overview of the recent findings from studies on the neuronal mechanisms of face recognition, based on the current theoretical models. Second, the findings on abnormalities of facial recognition in patients with schizophrenia will be reviewed.

2. Face-responsive brain regions

Research to investigate the direct neuronal activities of face recognition require invasive procedures, so most studies have been investigated by examining monkey brains. Hasselmo et al. (1989) reported that the superior temporal sulcus (STS) and the inferior temporal gyrus (ITG) in monkeys contain neurons which responded only to a picture of human or monkey faces. Neurons in the STS analyze mainly the changeable aspects of faces; the emotional expression (Perrett et al., 1984), eye gaze (Eifuku et al., 2004), and mimicry (Oram & Perrett, 1996). The ITG seems to be the site for storage of static features of faces (Hasselmo et al., 1989; Perrett et al., 1984, 1985). The amygdala is involved in emotional perception and regulation, and production of emotional feelings (Nakamura et al., 1992). In addition, several other brain regions are involved in face recognition. The region of the orbitofrontal cortex and the vicinity are activated by faces for longer durations and with lower activation compared to the temporal lobes (Thorpe et al., 1983; Rolls et al., 2006; Wilson et al., 1993). The section below presents an overview of each region related to face recognition.

2.1 Fusiform gyrus

A key brain region of the network for face recognition is the fusiform gyrus (FG) (Allison et al., 1999; Damasio et al., 1982; Kanwisher et al., 1997), which is located on the ventromedial surface of the temporal and occipital lobes (see Figure 1). The FG is bordered medially by the collateral sulcus and laterally by the occipitotemporal sulcus; both of the sulci are frequently interrupted with bifurcations, particularly in the anterior and posterior part of the FG (Naidich et al., 1987; Ono et al., 1990).
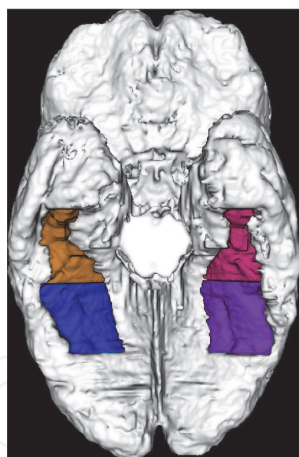

Fig. 1. Ventral view of a three-dimensional reconstruction of the FG.
The gray matter of the anterior fusiform gyrus is shown in red (left subject) and orange (right subject). The gray matter of the posterior fusiform gyrus is shown in purple (left subject) and blue (right subject).

Although both face and non-face object perception use the ventral pathway (Martin et al., 2001), EEG, MEG and fMRI studies have reported that faces are perceived, at least in part, by a separate processing stream in the ventral pathway (Allison et al., 1999; Kanwisher et al., 1997; Watanabe et al., 1999). Using fMRI, Kanwisher et al. (1997) reported that the middle portion of the FG responded predominantly to faces, leading them to call it the fusiform face area (FFA). Many studies have supported that the FFA is more active when stimuli have

been detected as faces. The most robust face-sensitive activation is consistently found on the lateral side of the right middle portion of the FG (Kanwisher et al., 1997). Additionally, the FFA responds to a wide variety of face stimuli; front and profile photographs of faces (Tong et al., 2000), line drawings of faces (Spiridon et al., 2002), animal faces (Chao et al., 1999b; Tong et al., 2000) and even faces of cartoon characters (Tong et al., 2000). Although the FFA shows the strongest increase in blood flow in response to faces, it also responds to non-face stimuli, such as houses (Aguirre et al., 1998; Ishai et al., 1999), chairs (Ishai et al., 1999), tools (Chao et al., 1999a), landscapes (Epstein et al., 1998) or nonsense stimuli (Clark et al., 1996; Epstein et al., 1998). Some fMRI studies suggest that the right FFA, also associated with face perception, can also be recruited in the processing of objects that subjects are highly familiar with, as in cases of expert knowledge for objects (Gauthier et al., 1999, 2001). Indeed, similar EEG responses to faces and, after repeated training, to other objects, have been reported (Tanaka et al., 2001; Rossion et al., 2002). This observation has led to the suggestion that functional and anatomical specialization for faces in healthy adults could simply reflect our extensive experience with human faces (Gauthier et al., 2001). An important assumption of the expertise hypothesis is that object expertise can be developed entirely as an adult, and the predictor of processing style is merely the amount of practice. This assumption has remained implicit in subsequent research (Gauthier et al., 1997; Grill-Spector et al., 2004; Rossion et al., 2002).

2.2 Superior temporal sulcus region

The STS is the sulcus separating the superior temporal gyrus (STG) from the middle temporal gyrus (MTG) in the temporal lobe of the brain (see Figure 2). The STS is frequently interrupted (the proportion of STS that have a single continuous sulcus is 36% for the right side and 28% for the left side), and the posterior part of the STS is extremely varied (Ono et al., 1990). For face processing by the STS, Kanwisher et al. (1997) reported that the superior and lateral regions of the right STS were significantly activated, as observed using fMRI, when the subjects viewed faces. Although the STS is generally involved in the perception of biological motion (Bonda et al., 1996; Decety et al., 1999; Grossman et al., 2000), for face recognition, the STS is also activated by mimics of faces. The STS responds to dynamic aspects of faces beyond gaze shifts (Calder et al., 2002), including lip movements (Puce et al., 1998) and emotion expression (Narumoto et al., 2001) as well as complex social cues such as trustworthiness (Winston et al., 2002). Moreover, it has been reported that the changeable aspects of faces are processed by a second face-specific processor located in the posterior part of the STS (Chao et al., 1999; Haxby et al., 1999; Hoffman & Haxby, 2000; Puce et al., 2007). For instance, Hooker et al. (2003) reported that STS activity accompanied extracting directional information from a gaze relative to directional information from an arrow and relative to eye motion without relevant directional information. In this study, subjects attempted to detect a particular directional cue provided either by gaze changes on an image of a face or by an arrow presented alone or by an arrow superimposed on the face. Another control condition was included in which the eyes moved without providing meaningful directional information. Moreover, Hoffman and Haxby (2000) reported that activation of the STS depended on the aspects of the face upon which attention was focused. In their results, the activity in the STS region was significantly higher when subjects were paying attention to the changeable aspects of the face than in the invariant condition. Such selectivity for gaze processing was observed only in the STS, but not in the FG.
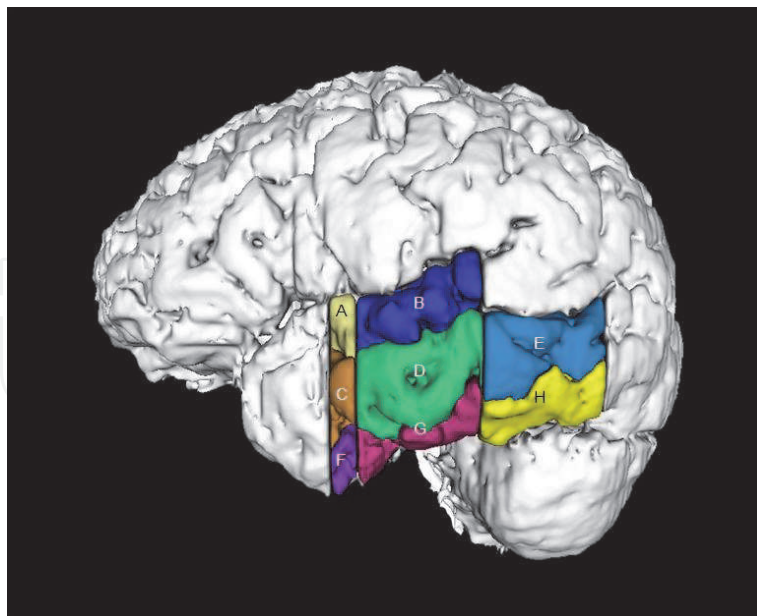
Fig. 2. STG, MTG, and ITG in the left hemisphere on a three-dimensional image.
A: anterior STG, B: posterior STG, C: anterior MTG, D: mid- MTG, E: posterior MTG, F: anterior ITG, G: mid- ITG, H: posterior ITG.

2.3 Inferior occipital gyrus (Occipital face area: OFA)

In addition to the FG and the STS, the lateral inferior occipital gyrus is one of the face-responsive regions (Adolphs, 2003; Halgren et al., 1999). The FG and the STS are responsible for preliminary face analysis according to the Haxby model (Haxby et al., 1999, 2000), and it is suggested that the OFA may glean more significant information from faces compared to both the FG and the STS (Haxby et al., 1999; Hoffman & Haxby, 2000). However, the OFA is activated by tasks in which the FFA is activated, thus suggesting that the OFA is also involved in processing the identity of faces (Hoffman & Haxby, 2000).

2.4 Other regions

Many research studies have shown that there are other regions engaged in face perception; the anterior portion of the middle temporal gyrus (aMTG) (see Figure 2), the orbitofrontal cortex (Nakamura et al., 2000; Sergent et al., 1992) and the right ventrolateral prefrontal cortex (Marinkovic et al., 2000; Vignal et al., 2000). The aMTG and the orbitofrontal cortex show both show a high activity when faces of famous or familiar persons are viewed. Studies with epilepsy patients also revealed the role of the right ventrolateral prefrontal cortex relevant to face processing.

The amygdala also participates in face recognition. The amygdala is an almond-shaped nucleus located deep within the medial temporal lobes of the brain in complex vertebrates. The amygdala has a primary role in the processing and memory of emotional reactions, and it is considered to be part of the limbic system. Actually, the recognition of emotions elicited a stronger response than neutral face detection. The activation occurs first in the STS (140–170 ms after the stimulus onset) and later in the right amygdala (around 220 ms after the stimulus onset) (Streit et al., 1999). Moreover, it has been reported that the amygdala plays a crucial role in processing fear (LaBar et al., 1998; Morris et al., 1998). Subsequent studies also showed activation of the amygdala in response to a happy face (Sheline et al., 2001) as well

as negative emotional faces like those expressing fear, sadness, anger (Blair et al., 1999) and disgust (Gorno-Tempini et al., 2001).

Additional regions in other parts of the brain also participate in face perception. For example, lip-reading elicits activity in regions associated with auditory processing of speech sounds (Calvert et al., 1997) and the perception of eye gaze direction elicits activity in parietal regions that are associated with spatial attention (Hoffman & Haxby, 2000).

3. Face recognition systems

Bruce and Young proposed a model of distributed face processing in humans (Bruce & Young, 1986). The essences of their model are that face perception differs qualitatively from the perception of other objects or words, and there may be several modules that independently process face information in human brain. Each face stimulates seven distinct types of mental processing; pictorial, structural, visually derived semantic, identity-specific semantic, name, expression and facial speech. According to this model, face perception processing consists of a four stage process; the first-stage being structural encoding to provide descriptions suitable for the analysis of facial speech, for the analysis of expression and for face recognition units; the second-stage being recognition of familiar faces, which involves a match between the products of structural encoding and previously stored structural codes describing the appearance of familiar faces; the third-stage is identification of specific semantic codes accessed from person identity nodes; and in the fourth-stage, name codes are retrieved.

Haxby et al. (2000) postulated a face processing model mediated by distributed neural systems in the human brain with core and extended systems. The core system is composed of regions in the visual cortex (inferior occipital cortex, FG, STS), which modulate the visual analysis of faces. In their model, the extended system of neural networks is as follows; intraparietal sulcus, auditory cortex, amygdala, insula, limbic system, and temporal pole.

Both models postulate multistage processing of face information and separate means of face analysis. Although these models do not take into account the attention effect, unconscious face processing is also important for face perception. With regard to this point, Bauer suggested a two-route model (Bauer, 1984), which was later developed by Ellis and Young (1990). Bauer reported that the skin conductance response was highly sensitive to the presentation of familiar faces in patients with prosopagnosia despite their inability to recognize familiar faces due to damage to the inferior temporal lobes (Bauer, 1984). Therefore, Bauer concluded that visual information processing remained partially separate from conscious face processing. The human brain has two neural visual pathways for the analysis of face information, both being directly connected with the limbic system. First, the ventral stream is responsible for overt face recognition. It starts at the visual association cortex and continues via the inferior temporal lobes to the amygdala and hypothalamus. The ventral stream, sometimes called the "what pathway", is associated with configuration recognition and object representation. Second, the dorsal stream is responsible for the covert analysis of facial stimuli. This pathway starts at the visual association cortex and comprises the superior temporal and inferior parietal lobes, the cingulate gyrus and the hypothalamus. The dorsal stream is sometimes called the "where pathway" or "how pathway". Bauer's model may account for the face recognition deficits accompanying some other disorders such as Capgras syndrome (Ellis & Young, 1990), unilateral spatial neglect (Vuilleumier, 2000) and the blind sight phenomenon (Morris et al., 1998).

4. Prosopagnosia

Neuropsychological studies have reported a number of brain-damaged patients who exhibited a disproportionate impairment for recognizing faces as compared to other objects. Especially, prosopagnosia has been crucial in the development of theories of face recognition. Prosopagnosia is one of the disorders of face perception, usually associated with bilateral or right unilateral occipito-temporal lesions (Damasio et al., 1982; Farah, 1991; Landis et al., 1988; Wada & Yamamoto, 2001). For example, Wada and Yamamoto (2001) reported a case of prosopagnosia caused by a hematoma limited to the right FG and to the lateral occipital region.

The word "prosopagnosia" derives from the Greek *prosopon* (face) and *gnosis* (knowledge), which was introduced by Bodamer (1947). Damasio et al. (2000) described that the deficit of prosopagnosia is not limited to human faces. The magnitude of the deficit of visual agnosia varies in prosopagnosia; a farmer cannot recognize his cows individually and a bird-watcher cannot identify different species of birds. According to their description (Damasio et al., 2000), patients with prosopagnosia can recognize a pencil, or an article of furniture, or a car, as, respectively, pencils, furniture, and cars; but they often cannot decide whether such an article belongs to them or not, or who the specific manufacturer of a given car is. Although dysfunction of the FG produces difficulties in identifying a face, patients with prosopagnosia can recognize facial expressions (Morris et al., 2001). The dissociation between recognizing a face and a facial expression may be associated with the tectopulvinar system, which processes facial expression perception. Thus, patients with prosopagnosia can recognize a face implicitly because of emotional responses to facial expressions even if they are not perceived consciously. This suggests that emotion also plays a significant role in face recognition, as a relatively independent module.

5. The face-related potentials

The evidence regarding face recognition comes from two very different methodologies, neuroimaging and neurophysiology. Face processing also can be assessed by metabolic activity, blood deoxygenation, and glucose uptake in the human brain (Haxby & Gobbini, 2002; Henson et al., 2003). Metabolic brain imaging techniques produce information about the regions that are related to the face recognition processes. Meanwhile, neurophysiological studies, including EEG and MEG, have revealed the individual regions associated with human face processing with a high temporal resolution.

Of particular note, numerous EEG studies, especially event-related potentials (ERP) studies have revealed the presence of face-sensitive potentials are absent or attenuated to non-face visual stimuli (Jeffreys, 1989). ERPs are measures of neurophysiological activity, and represents a non-invasive method used to clarify the time course of visual object categorization processes in the human brain. Artifact-free EEG segments time-locked to stimuli onsets have been averaged over many trials, revealing the timing and magnitude of consistent neural processing elicited by stimuli. Data from ERP studies have demonstrated the negative potential recorded at occipitotemporal leads, the N170, to be larger for faces than objects in humans (Bentin et al., 1996; Botzel et al., 1995; Rossion et al., 2000). For MEG, the magnetic counterpart of N170 (M170) to the face is also larger for faces than objects (e.g., Lu et al., 1991).

There is a face-sensitive potential with a maximum amplitude and minimum latency to faces than other objects at vertex electrodes, called vertex positive potentials (VPP) (Botzel & Grusser, 1989; Jeffreys, 1989). It has been reported that the VPP represents the positive counterpart of the N170 (Botzel et al., 1995).

Intracranial ERP recordings of epilepsy patients have also found face-sensitive N200, which were recorded from both hemispheres of the FG and the ITG (Allison et al., 1994, 1999). Several discrepancies have been reported between the scalp N170 and intracranial N200. For example, the intracranial face-N200 showed no hemispheric asymmetry, whereas right-greater-than-left scalp face-N170 amplitudes have been frequently demonstrated (Henson et al., 2003; Itier & Taylor, 2004). The scalp N170 generally corresponds to the intracranial N200, but the N170 may be an overlapping potential and, therefore, the N170 is not exactly the same as the N200.

6. Evaluation of face processing

As emotionally expressive stimuli, faces can bridge multiple levels of the emotional response. Faces can be classified as showing positive and negative emotions. In addition, faces also express categorical emotions. The universality of facial expressions that communicate anger, disgust, fear, happiness, sadness, and surprise is widely accepted (Ekman, 1994; Izard, 1994). Subjects tend to mistake fear and surprise, disgust and anger, and neutrality and sadness (Ekman, 1972; Gur et al., 2002). Most facial expression research is done with static, posed images (Edwards et al., 2002); however, naturally elicited expressions will change within milliseconds and there are differences in the way in which some subjects respond to posed vs. natural expressions (Davis & Gibson, 2000). This section will focus on the neuropsychological findings for processing of static images of emotional faces. It should also be noted that most studies used their own original emotional face recognition tests. For example, one research group has worked extensively with emotional face processing. The Penn Emotion Acuity Test (PEAT) (Kohler et al., 2000) consists of 10 happy, 10 sad, and 20 neutral expressions of Caucasian faces. The task requires the participant to indicate the emotion depicted on a 7-point Likert-type intensity scale (1 = very sad, 2 = moderately sad, 3 = somewhat sad, 4 = neutral, 5 = somewhat happy, 6 = moderately happy, 7 = very happy). The Emotion Intensity Differentiation task (Emodiff) presents two faces of the same individual showing the same emotion (happy or sad), requiring the participant to select the more intense expression. A total of 40 face pairs (20 happy, 20 sad) are used in this study (Silver et al., 2002). However, various tests are used to assess the recognition of emotional faces in reading the following sections.

Although threatening stimuli, including faces, are detected more rapidly in visual search tasks, studies that emphasize identification of expressions, rather than detection of differences, commonly find that happy faces elicit faster response times than other expressions (Leppanen & Hietanen, 2004). Happy faces are also widely found to be identified more accurately (e.g., Gur et al., 2002). Both of these effects could be due to structural aspects of the expressions as visual stimuli, rather than the affective meaning itself. Happy expressions are of a very different structural conformation and rely more on visual analysis of the mouth area and less on the eye areas than negative expressions (sadness, fear, anger; [Ekman & Friesen, 1978; Smith et al., 2003]).

7. Behavioral studies for face recognition in schizophrenia

Recently, interest in the neural physiology of emotional processing in schizophrenic patients has undergone a resurgence. Impaired emotional functioning is fundamental to schizophrenia, and negative symptoms, including flat affect, are debilitating and resistant to intervention. These emotional impairments include deficits in how emotional meaning is assigned to incoming sense data, how emotions are felt and elicited by life circumstances (e.g., inappropriate affect, persecutory delusions), and how emotions are encoded and transmitted during communication (e.g., symptoms of flat or restricted affect).

In particular, the ability to identify and interpret facial expressions is imperative for effective non-vocal social communication. Individuals with schizophrenia have an impaired ability to both recognize faces and both discriminate and respond to emotional facial expressions, which are activities critical to social functioning (Addington & Addington, 1998; Edwards et al., 2002; Kerr & Neale, 1993; Mandal et al., 1998; Wolwer et al., 1996). However, to simplify, the findings of neuropsychological tests for emotional face recognition will be the focus of this section.

It has been repeatedly reported that patients with schizophrenia have deficits in the recognition of emotional faces. For example, Kohler et al. (2000) performed PEAT and the age recognition task with facial images for 35 patients with schizophrenia and 45 normal controls. The patients with schizophrenia performed worse than the controls on both the PEAT and age recognition task without differential deficits. In patients with schizophrenia, a poor performance on PEAT correlated with the severity of negative and positive symptoms, with no significant correlation for age recognition. Hooker and Park (2002) tested 20 chronic, medicated schizophrenia patients and 27 normal control participants in a battery of face recognition and affect recognition tasks. They demonstrated that schizophrenia patients were less accurate than normal control participants on face recognition, facial affect recognition and vocal affect recognition tasks, but among schizophrenia patients, only affect recognition performance was related to social functioning.

It was also reported that schizophrenia patients with persecutory delusions were slower than normal controls for identifying angry faces (Green & Phillips, 2004). Patients also misattribute disgust and fear to neutral faces at a higher error rate than healthy controls (Kohler et al., 2003). These behavioral deficits in the recognition of emotional faces are stable over at least a few months (4 weeks [Gaebel & Wolwer, 1992]; 4 and 12 weeks [Wolwer et al., 1996]; 12 weeks [Addington & Addington, 1998]) and appear to be unrelated to either the symptom severity or medication status (Edwards et al., 2002). Differences among schizophrenia subtypes have also been reported. Paranoid-subtype patients are more accurate than non-paranoid-subtype patients at identifying negative facial expressions (Davis & Gibson, 2000; Kline et al., 1992; Lewis & Garver, 1995; Phillips et al., 1999). Although the reaction time is slower, paranoid-subtype patients may be more accurate judges of facial affect than normal control subjects. For example, Davis and Gibson (2000) reported an enhanced perception in paranoid-subtype patients relative to normal controls for negative facial expressions. However, the precise nature of emotional face recognition deficits in patients with schizophrenia remains unclear.

8. Neuroanatomical abnormalities of the fusiform gyrus in schizophrenic patients

MRI studies have provided useful findings regarding the subtle structural brain abnormalities in patients with schizophrenia (Shenton et al., 2001). It should be noted that only positive MRI findings on FG have been included in this section. Paillère-Martinot et al. (2001) investigated the gray and white matter volumes in early onset schizophrenic patients using voxel-based morphometry. They reported significant gray matter reductions in the medial frontal gyri, left insula, left parahippocampus and left FG in patients. Lee et al. (2002) manually measured the FG volume and reported bilateral FG reduction, which was specific to first-episode schizophrenia compared with first-episode manic psychosis. Moreover, Onitsuka et al. (2003) reported an association between FG reduction and delayed facial recognition memory deficits in male patients with chronic schizophrenia. Premkumar et al. (2008) demonstrated that a longer duration of illness was associated with a smaller volume of left FG grey matter. In addition, the authors suggested that the right middle frontal cortex is particularly vulnerable to the long-term effects of schizophrenia, whereas the dorsomedial prefrontal cortex, FG and cerebellum are affected by both a long duration of illness and aging. In the study by Bangalore et al. (2009), the duration of untreated illness was found to be inversely correlated with gray matter changes in the left FG. Recently, Goghari et al. (in press) reported the left FG to decrease by 11% in patients and 7% in their biological relatives compared with normal controls.

The FG abnormalities represent a social communication disturbance of the disease, expressed symptomatically as asociality and social anhedonia, and neuropsychologically due to poor facial memory, and this three-variable relationship was uninfluenced by medication. Negative symptoms emphasize a loss of interest, motivation, and pleasure in social interaction which may reflect a risk factor that is present long before the onset of the disease and may mediate the relationship between the FG volume and lower scores on neuropsychological tests of facial recognition memory (Onitsuka et al., 2003) as well as lower levels of extraversion (Onitsuka et al., 2005), all of which may constitute the well-known disease-related disturbance of social communication.

9. Neuroimaging findings for neutral/emotional face recognition deficits in schizophrenia

Patients with schizophrenia obviously have deficits in the neural substrate of visual face processing. As noted before, a number of structural and functional studies have also reported abnormalities in the FG. A meta-analysis of voxel-based MRI studies showed decreased volume of the left FG in around a quarter of studies, whereas only 5% or fewer showed a reduction in the right FG (Honea et al., 2005). Functionally, a deficit of FG activation relative to control subjects is also evident in functional MRI studies of matching facial identity and emotion (Quintana et al., 2003). Moreover, structural MRI has also revealed decreased STG gray matter volumes (Wright et al., 2000); thus other brain areas related face recognition may be affected in patients with schizophrenia.

In humans, various brain regions have been associated with emotion processing, as described before. Volume reductions of several brain regions involved in emotional face recognition have been reported, including the amygdala, prefrontal and orbitofrontal regions (Shenton et al., 2001; Wright et al., 2000). The amygdala may be structurally and

functionally abnormal in patients with schizophrenia. Brain morphometric studies have indicated that there is a 6% bilateral amygdala volume reduction on average in patients with schizophrenia compared to healthy subjects (Wright et al., 2000). The responses of the amygdala to facial emotion appear to be abnormal in schizophrenic patients. For example, several studies have reported blood flow hypoactivation of the amygdala elicited by fearful faces compared with the activities to neutral faces in patients with schizophrenia (Aleman & Kahn, 2005). On the other hand, recent studies have shown hyperactivation of the medial temporal lobe, especially the amygdala, to neutral faces in schizophrenic patients (Holt et al., 2006; Surguladze et al., 2006). Differences in the methodology and required tasks may account for some of the disparate results.

Functional neuroimaging studies have suggested several brain regional deficits in patients with schizophrenia, including a lack of FG activation relative to healthy subjects in facial affect and individual identification matching tasks (Quintana et al., 2003). Phillips et al. (1999) found differences between paranoid and non-paranoid schizophrenia patients using a gender discrimination task to compare expressions of fear, anger, and disgust to a mildly happy/neutral baseline in an fMRI study. Both groups showed decreased activation and accuracy at emotion identification relative to controls, and decreased amygdala activity to fearful faces. Paranoid patients showed increased visual cortex activity to fearful faces than non-paranoid patients, and abnormal activity in the inferior frontal cortex, anterior cingulate cortex, and visual cortex to angry expressions. Non-paranoid patients showed no activation of the insula to disgust, and some abnormal activation of the amygdala to faces showing disgust. There was also a trend for paranoid patients to be more accurate than non-paranoid patients at classifying negative emotions. In contrast to the decreased activation of visual and emotional processing areas in schizophrenic patients, functional neuroimaging studies have found *increased* responses in the amygdala and hippocampus to happy, neutral, and fearful expressions using non-face visual stimuli as the baseline of brain activity (Holt et al., 2006; Kosaka et al., 2002).

In summary, the results of a large number of neuroimaging studies suggest that facial emotion recognition in schizophrenic patients is impaired, especially for negative emotions. This impairment may be related to structural and functional abnormalities of the amygdala in patients with schizophrenia.

10. Face-sensitive visual evoked potentials in schizophrenic patients

In this section, the evoked potentials or ERPs elicited by neutral or emotional face images of schizophrenia will be reviewed for each component.

10.1 P1 (P100)

The P1 is a positive potential at the occipital area elicited at around 100 ms after the stimulus onset and generated within the extrastriate cortex (Pourtois et al., 2005). This potential is associated with the global processing of visual perception. The "global processing" is the initial stage of face categorization (Liu et al., 2002). Herrmann et al. (2004) reported a normal occipital P1 amplitude to images of neutral faces and buildings, with larger P1 amplitudes to faces than to buildings. Similarly, some studies reported normal P1 during processing of faces or facial affect in schizophrenic patients (Doniger et al., 2002; Schechter et al., 2005). On the other hand, some studies revealed P1 deficits during the processing of

faces (Haenschel et al., 2008; Yeap et al., 2006); thus, the differences in P1 during face processing in schizophrenic patients remain to be elucidated.

The mechanism of the P1 evoked during processing of emotion identification remains unclear, and the effects of facial emotional expressions on the P1 are still being debated even for healthy subjects. The difficulties of required tasks may account for the P1 during emotion recognition (Di Russo et al., 1999; Hillyard et al., 1998). Furthermore, the P1 is very sensitive to the properties of the stimulus such as contrast, spatial frequency, and luminance (Tobimatsu et al., 1993; Rebai et al., 1998). Pourtois et al. (2005) reported that the P1 was significantly enhanced by low-spatially-filtered fearful faces in healthy subjects. However, several investigators reported no significant effects of facial expressions on P1 amplitudes in either normal controls or schizophrenics (Campanella et al., 2006; Johnston et al., 2005; Turetsky et al., 2007). Obayashi et al. (2009) reported no significant effects of the facial expressions for normal controls or patients with schizophrenia, regardless of spatially-filtered face images, thus suggesting that recognition of facial emotional expressions is higher-level visual processing, and that lower-level visual perception as reflected by the P1 cannot account for this process. Further studies will be necessary to reveal the mechanism responsible for the P1 component for face processing.

10.2 N170

The N170 is a negative potential recorded over occipitotemporal areas at around 170 ms, and is considered to function as an index of the structural encoding of faces (Bentin & Deouell, 2000), and the extraction of configural information (Goffaux et al., 2003; Rossion et al., 1999). The N170 is also associated with the local processing of identifying individual faces (Liu et al., 2002).

As noted before, the N170 is larger for faces than for objects in healthy subjects. However, Onitsuka et al. (2006) reported that male chronic schizophrenia patients showed specific N170 reduction to faces compared to normal controls (see Figure 3). Additionally, in this study, patients with schizophrenia, but not normal controls, showed a significant association between right posterior FG reduction and N170 reduction to faces but not to other objects at right posterior temporal electrodes. It has been repeatedly reported that patients with schizophrenia showed reduced N170 amplitudes to neutral and emotional faces (Caharel et al., 2007; Campanella et al., 2006; Lee et al., 2010; Lynn & Salisbury, 2008; Obayashi et al., 2009; Turetsky et al., 2007).

However, the effects of facial emotional expressions on N170 still remain controversial. For example, Batty and Taylor (2003) found that the N170 amplitude to fearful faces was larger than the N170 to neutral faces in healthy subjects. On the other hand, it was reported that N170 was unaffected by any emotional expressions in healthy subjects (Eimer, 2000; Eimer & Holmes, 2002). The authors documented that the N170 reflects the process of identification and recognition of faces, but not facial emotional expressions. Lynn and Salisbury (2008) demonstrated that healthy subjects showed bilateral differences in N170 amplitude among facial expressions, while schizophrenic patients failed to show this modulation. However, Obayashi et al. (2009) reported no significant effects of facial expressions for normal controls or schizophrenics, regardless of spatially-filtered face images. They also suggested possible dysfunction of the magnocellular and parvocellular pathways, which may underlie the deficits associated with facial recognition in schizophrenic patients. Therefore, electrophysiological studies have indicated that the N170

amplitude reduction of schizophrenia during face and facial affect processing may reflect deficient processing of facial structures and facial structure encoding.
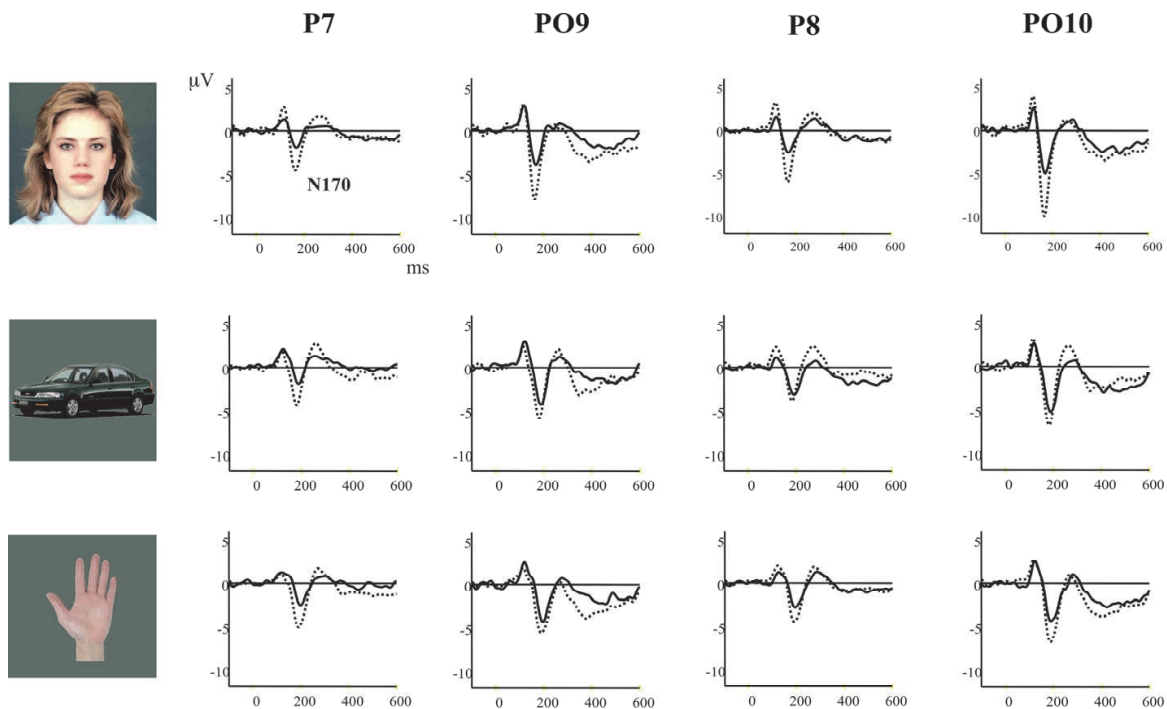

Fig. 3. The group average N170 waveforms at P7/P8 (typical N170 recording sites) and PO9/PO10 (more ventral sites with the largest effect).
The waveforms of patients with schizophrenia (n = 20) are shown in solid lines and those of normal controls (n = 16) are shown in dashed lines.

With regard to clinical correlations in patients with schizophrenia, a few studies have reported an association between the N170 and the clinical features. One study found significant associations between N170 and social dysfunction in patients with schizophrenia. Obayashi et al. (2009) reported correlations between reduced N170 amplitude in response to faces and lower Global Assessment of Functioning (GAF) scores in patients with schizophrenia. The N170 amplitude to faces could therefore function as a neurophysiological index of social functioning in schizophrenia. Further studies of social functioning of patients with schizophrenia using face processing paradigms would be of value to schizophrenia research. An fMRI study of healthy subjects suggested that the right FFA can be activated by highly trained object categories (Gauthier et al., 1999) (e.g., by birds in expert bird watchers). Moreover, Gauthier and Nelson (2001) speculate that the functional and anatomical specialization for faces in normal adults could simply be a result of our experience with human faces. As reviewed in this article, a number of findings suggest that anatomical/functional abnormalities underlie facial processing problems in patients with schizophrenia. Whether such abnormalities in schizophrenia are secondary to the limited experience of patients and/or are based on abnormalities in the neural substrate of face perception, it seems reasonable to speculate that abnormalities in face processing are related to the disinterest in social contact and social isolation observed in schizophrenic patients. In summary, a number of face-ERP studies have reported N170 amplitude reduction specific to faces, thus indicating that the neuronal populations involved in face perception may be

specifically reduced in patients with schizophrenia. Moreover, schizophrenia may be characterized by deficits in the modulation of the N170 responses to different face stimuli.

10.3 N250

Beyond the N170 potential, face-sensitive potentials also exist, but only a few studies have examined the N250 potential in patients with schizophrenia. The N250 is an affect-related negative potential that occurs approximately 250 ms after the stimulus onset at fronto-central electrode sites, and is sensitive to the emotional content of a face and familiar faces (Streit et al., 1999, 2001b; Tanaka et al., 2006).

In an MEG study, Streit et al. (2001a) found decreased amplitude in the 150-250 ms potential at left inferior parietal cortex, and in the 250-350 ms potential at the left inferior prefrontal cortex. In an ERP study, Streit et al. (2001b) reported decreased ERPs at frontal sites in schizophrenia patients compared to normal controls between 180-250 ms. These studies may indicate deficits in decoding of emotional information in patients with schizophrenia beyond the N170 potential.

However, the findings of previous studies tend to be mixed with regard to the N250 in patients with schizophrenia. For instance, other researchers have suggested that these deficits are secondary flow-on effects of broader deficits in the structural encoding of faces (Johnston et al., 2005; Turetsky et al., 2007), and reported abnormally small N170 amplitudes, but normal N250 responses. This finding suggested impaired facial feature encoding but unaffected emotion decoding (Johnston et al., 2005). Further research will therefore be needed to clarify this issue.

11. Conclusion

In this chapter, we have reviewed recent research on the neural mechanisms of facial recognition. Face perception may be one of the most familiar visual stimuli and the most important stimulus with regard to social interactions. Studies in monkeys showed face-selective neurons in the monkey brain. Human research to clarify the neuronal systems of face perception were also performed in patients, including those with prosopagnosia, and contributed significant information that helped in the elucidation of the human face processing system. Two models of human face processing, the Bruce and Young model and the Haxby model have so far been proposed. In addition, Bauer's model that has two neural visual pathways (the ventral stream and the dorsal stream) account for attentional face processing. The results from the study of human face processing show that there are face perception-related regions, including the FFA, STS, OFA, aMTG, the orbitofrontal cortex and the right ventrolateral prefrontal cortex, and the amygdala.

For face recognition deficits of patients with schizophrenia, this chapter reviewed findings of behavioral, structural and functional studies. Neuropsychological findings for face processing have indicated deficits in the formation and retention of memory for face configuration information in patients with schizophrenia. Brain morphometric studies have indicated that abnormalities of the FG may be associated with the pathophysiology of schizophrenia. Neurophysiologically, schizophrenia may be characterized by deficits in modulating the N170 in response to different face stimuli.

Although the respective roles of the different brain regions involved in face processing have emerged, both neuroimaging and neurophysiology studies, including FRP studies, have

positively contributed to elucidating the neural network responsible for face processing in the human brain.

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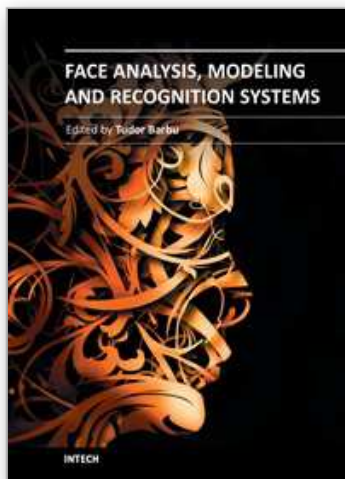
Face Analysis, Modeling and Recognition Systems
Edited by Dr. Tudor Barbu

The purpose of this book, entitled Face Analysis, Modeling and Recognition Systems is to provide a concise and comprehensive coverage of artificial face recognition domain across four major areas of interest: biometrics, robotics, image databases and cognitive models. Our book aims to provide the reader with current state-of-the-art in these domains. The book is composed of 12 chapters which are grouped in four sections. The chapters in this book describe numerous novel face analysis techniques and approach many unsolved issues. The authors who contributed to this book work as professors and researchers at important institutions across the globe, and are recognized experts in the scientific fields approached here. The topics in this book cover a wide range of issues related to face analysis and here are offered many solutions to open issues. We anticipate that this book will be of special interest to researchers and academics interested in computer vision, biometrics, image processing, pattern recognition and medical diagnosis.

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