



Processing emotional expression and facial identity in schizophrenia

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Abstract

Previous studies showed that schizophrenic patients have a deficit in facial information processing. The purpose of the present study was to test the abilities of patients with schizophrenia and normal controls in emotion and identity matching when these two dimensions were varied orthogonally. Subjects (20 schizophrenic patients and 20 controls) had to report if two faces had the same emotion or belonged to the same person. When the task concerned one type of information (i.e. emotion or identity), the other one was either constant (same person or same emotion) or changed (different person or different emotion). Schizophrenic patients performed worse than controls for both kinds of facial information. Their deficit was more important when the secondary factor was changed. In particular, they performed at chance level when they had to match one emotion expressed by two distinct persons. Finally, correlation analysis indicated that performance/deficit in identity and emotion matching co-varied and that in such tasks performance is negatively correlated with the severity of negative symptoms in patients. Schizophrenic patients present a generalised deficit for accessing facial information. A facial emotion and an identity-processing deficit are related to negative symptoms. Implications for face-recognition models are discussed.

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1. Introduction

Patients with schizophrenia show abnormal performance on facial emotion recognition and identity

matching (Cutting, 1981; Salem et al., 1996). These deficits, which affect the ability of patients to interpret others' intentions or desires, might play a role in the social disorders associated with schizophrenia.

Abnormal performance on facial emotion recognition tasks have been reported in schizophrenia (Cutting, 1981; Habel et al., 2000; Salem et al., 1996). The neural correlates of facial emotional expression are less active during emotion perception

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among patients with schizophrenia (Gur et al., 2002; Phillips et al., 1999), but performance improves after training in affect recognition (Frommann et al., 2003). The deficit could be more important for fear and sadness (Edwards et al., 2001). Some reports also indicated that this deficit extended to all facial information processing (Kerr and Neale, 1993; Archer et al., 1994; Salem et al., 1996; Franck et al., 2002; Hooker and Park, 2002), but some patients with schizophrenia seem to have greater difficulty in processing emotion-related information than other kinds of facial information (Gooding et al., 2001; Gooding and Tallent, 2002; Hooker and Park, 2002). Finally, the impairment is not restricted to facial affect but might also extend in the recognition of affective prosody (Edwards et al., 2001).

Studies of individual cases of schizophrenia have not revealed any consistent pattern of deficit: patients typically exhibit quite specific deficits, but not necessarily for the same facial information, e.g., in face recognition, emotion recognition or identity matching (Archer et al., 1994; Evangeli and Broks, 2000). Archer et al. (1994) interpreted these observations in accordance with Bruce and Young's (1986) model of face recognition. Bruce and Young (1986) proposed that there are separate functional components for the processing of facial emotion, for the processing of unfamiliar faces, and for familiar face recognition. These components are believed to be independent. This assumption is supported by experimental studies in healthy participants (Bruce, 1986; Campbell et al., 1996), electrophysiological recordings in monkeys (Hasselmo et al., 1989) and functional imaging studies (Phillips et al., 1998; Sergent et al., 1994). Neuropsychological studies (Parry et al., 1991; Humphreys et al., 1993; Schweich and Bruyer, 1993) showed that these processes could be selectively damaged (for a model of facial information processes and their neuro-anatomical correlates, see Haxby et al., 2000, 2002).

Nevertheless, some recent studies suggest that facial emotion processing and face recognition may interact (Baudouin et al., 2000a,b; Dolan et al., 1996; Schweinberger and Soukup, 1998; Tiberghien et al., 2003). For example, healthy participants were not able to pay attention to emotion when identity was varied (Schweinberger and Soukup, 1998). The experiment of Baudouin et al. (2002) extends this observation to patients suffering from schizophrenia. They observed

that schizophrenic participants could not selectively attend to facial emotion regardless of the identity displayed. The ability/deficit in classifying faces according to emotion was significantly correlated with the ability/deficit in classifying faces according to identity. Similarly, Young et al. (1996) studied a patient with a partial bilateral amygdectomy who was poor at recognizing facial emotions. She was not impaired on face-recognition and identity-matching tasks, except in the special case where she had to recognize the same person with two distinct facial emotions. Under those circumstances, she tended to perceive two different persons; the deficit in facial emotion recognition led to a deficit in identity processing.

Thus, it appears that the variation of one kind of facial information may interfere with the processing of another. Moreover, other studies reported a positive correlation in performance for emotion and identity tasks, for right brain-damaged patients (Weddell, 1989), for controls, and for lobotomized participants (Braun et al., 1994).

These observations do not favor the independence hypothesis. By contrast, they indicate that identity and emotion processing are interrelated. To reconcile these two opposing views, one may suggest that a deficit in the processing of one kind of facial information is concomitant with some decrease of the ability to process the other kind, which nevertheless does not reach an "impaired" level. This hypothesis would explain both observations of correlation and dissociation cases.

Finally, these observations suggest that the ability to respond selectively either to facial identity or emotion without interference from the other (e.g., facial expression) does not result from implementing independent processes, but rather that these processes interact and may reciprocally interfere. Thus, attention to one kind of facial information would require the intervention of active dissociation processes that allow selective responses to each kind of facial information. These attentional processes would need to be used to disregard information that is not relevant for the task.

In previous studies, each type of information was generally tested individually, i.e., participants did not need to rule out the variations of irrelevant facial information to complete the task—for example, the emotion was not varied when the task involved

identity judgments (Archer et al., 1994). The purpose of the present study was to test the abilities of patients with schizophrenia and of controls in emotion and identity matching when these two dimensions were varied orthogonally. Exactly the same photographs were used in two delayed matching tasks (identity matching and emotion matching). Pairs of faces, belonging to the same person or not, and expressing the same emotion or not, were presented to subjects during two tasks. The instruction was the only difference between the two tasks (“is the person the same?” or “is the emotion the same?”). Contrary to previous studies, the emotion was varied when the instruction was about identity, and reciprocally. Thus, participants had to disregard the irrelevant information.

The independence hypothesis would predict that when the task concerned one kind of information, variation in the other would not interfere with performance. Conversely, the interactive hypothesis, which we favor, would predict that variation of identity/emotion might interfere in the matching of emotion/identity. This interference would be greater for patients with schizophrenia because of their attentional problems. The relation between a deficit in facial information processing and the severity of schizophrenic symptoms, evaluated with the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1983) and the Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1984), was also investigated.

2. Methods

2.1. Participants

Twenty patients with schizophrenia (5 females, 15 males; mean age: 38.15 years, range 21–60) and 20 healthy control participants (7 females, 13 males; mean age: 36.7, range 21–55) volunteered to participate in the study. All the subjects provided written consent after a complete description of the study. All patients (Table 1) were hospitalized in the Vinatier psychiatric hospital in Lyon (France). They were assessed using the Mini International Neuropsychiatric Interview (MINI) (Lecrubier et al., 1997) to determine the presence of schizophrenia according to DSM-IV criteria (American Psychiatric Association, 1994) and to exclude concomitant neurological or psychiatric

Table 1

Characteristics of patients with schizophrenia and control subjects; means (SD)

	Schizophrenic patients (n=20)	Normal controls (n=20)
Age (years)	38.15 (9.5)	36.7 (13.4)
Gender		
Male	15	13
Female	5	7
Education level	10.4 (2.7)	11.9 (1.9)
Illness duration ^a	12.6	–
Medication ^b	16.3 (7.24)	–
Type ^c		
Paranoid	8	–
Disorganized	1	–
Undifferentiated	7	–
Residual	4	–
SAPS score ^d	29 (16.1; range 17–88)	–
SANS score ^e	40.9 (23.2; range 10–74)	–

^a Time elapsed since onset of psychotic symptoms (years).

^b Haloperidol equivalence per diem (mg).

^c Schizophrenia subtypes (DSM-IV).

^d Maximum score: 170.

^e Maximum score: 125.

disorders. Exclusion criteria included visual difficulty, history of neurological illness or trauma, alcohol or drug dependence according to DSM-IV criteria, and age older than 65 years. All patients were receiving antipsychotic medication (principally olanzapine and risperidone) and were clinically stable at the time of testing (mean illness duration: 12.6, SD: 9.9; mean total SANS score: 40.9, SD: 23.2; mean total SAPS score: 29, SD: 16.1). Neuropsychological assessment (Mattis and Stroop) revealed no evidence of attentional deficit (mean: 36.4, SD: 0.8), initiation deficit (mean: 33.3, SD: 5.3), construction deficit (mean: 6, SD: 0), concepts deficit (mean: 36.6, SD: 3.5), and memory deficit (mean: 23.6, SD: 2.3) (mean total: 135.9, SD: 10.1). None of controls reported evidence of neurological diseases or psychiatric problems. The patients with schizophrenia and the controls did not differ significantly in sex ($\chi^2=1.07$) and age ($t=0.54$).

2.2. Material

Color photographs of five persons (5 clean-shaven males, with no facial particularity like scars or glasses and with approximately the same hairstyle; mean age: 26 years, range 24–29) expressing five different emotions (happiness, fear, sadness, anger



Fig. 1. Example of face with five expressions used in the experiments.

and disgust) were used in the experiment (Fig. 1). Eleven judges had previously rated the emotion of each portrait. They were students or laboratory and university staff members, unfamiliar with the models, and blind to the aim of the study. They had to indicate for each photograph to which of six categories they belonged (happy, sad, disgusted, angry, fearful, neutral, and others). The minimal inter-rater agreement was 55% for each photograph (range: 55–100%), significantly above chance level (chance level=14%, t -test: $t(10)=2.56$, $P<0.05$). Photographs for which another emotion was selected by more than three raters were not used. All information about background and body were eliminated. The faces were 12 cm high and 7.5 cm wide when presented on a computer monitor.

2.3. Procedure and design

Each trial began with the presentation of a fixation point for 1 s. After a blank screen that lasted 1 s, a first

face appeared on the left of the screen for 1 s. After a second blank screen lasting 1 s, a second face appeared on the right of the screen and remained on it until the participant gave an answer. These two faces belonged either to the same person or to two distinct persons. Similarly, both faces expressed either the same emotion or two distinct emotions. All the participants were shown each face and each emotion but only some subset of face/emotion combinations. Indeed, it was possible to associate faces according to the identity in 10 pairs (i.e., persons A/B, A/C, A/D, A/E, B/C, B/D, B/E, C/D, C/E and D/E), and it was also possible to associate faces according to the emotion in 10 pairs (i.e., happy/sad, happy/fearful, happy/angry, happy/disgusted, sad/fearful, sad/angry, sad/disgusted, fearful/angry, fearful/disgusted and angry/disgusted). For each participant, two people (e.g., persons A/B) were associated with two particular emotions (e.g., happy/sad). The four photographs (i.e., person A happy and sad, person B happy and sad) were presented in pairs in all possible orders and position (16 possibilities; see Table 2). The other pairs were associated with the other possible pairs of emotions. That made a total of 160 trials (10 pairs * 16 trials per pair). The association between pairs of people and pairs of emotions was alternated across participants.

The experiment took place in two sessions. During one session, the participants were required to classify faces according to their identity and, in the other, they were required to classify faces according to their emotion. In each of these two sessions, participants sat in front of a monitor, at an approximate distance of 70 cm, with the index finger of each hand on a response key. They were instructed to press one key when the

Table 2

The 16 possible combinations of two faces (person A and person B) and two emotions (happy and sad)

	Same person		Different persons	
	First face	Second face	First face	Second face
Same emotion	PA happy	PA happy	PA happy	PB happy
	PA sad	PA sad	PA sad	PB sad
	PB happy	PB happy	PB happy	PA happy
	PB sad	PB sad	PB sad	PA sad
Different emotions	PA happy	PA sad	PA happy	PB sad
	PA sad	PA happy	PA sad	PB happy
	PB happy	PB sad	PB happy	PA sad
	PB sad	PB happy	PB sad	PA happy

two faces belonged to the same person or expressed the same emotion and the other key when it was not the case. Exactly the same photographs were presented in both sessions. Participants were instructed to respond as accurately and as quickly as possible. The position of the keys (right/left) and the order of the two sessions were alternated between participants. For patients with schizophrenia, the two sessions took place on two different days and, for controls, on one day.

2.4. Data analysis

The factors were group (patients with schizophrenia vs. controls), matching dimension (identity vs. emotion), response (same vs. different) and change of the second dimension (change vs. constant). The first factor was manipulated between subjects; the three other factors were manipulated within subjects. Table 2 presents mean percentages correct and latencies. As shown in Table 2, the differences in standard deviations between controls and patients with schizophrenia were considerable. Therefore, we performed a logarithmic transformation before analyzing the data. We performed a $2 \times 2 \times 2 \times 2$ analysis of variance (ANOVA) (group \times matching dimension \times response \times change) on percentage accuracy and latencies for correct responses.

We also calculated the Spearman correlation coefficients between performances for identity and emotion as well as between SANS/SAPS total scores and subscores on the one hand and performances on

the other. A non-parametric test was used because we did not postulate a linear link between these variables.

3. Results

3.1. Percentage accuracy and latencies for correct responses

The factor group was significant for percentage and latencies: patients with schizophrenia performed significantly less accurately (82.2% vs. 91.8%; $F_{1, 38} = 15.64$, $P < 0.001$) and more slowly than controls (1317 vs. 949 ms; $F_{1, 38} = 14.99$, $P < 0.001$). The factor of matching dimension was also significant for both measures: participants performed significantly less accurately and more slowly in the emotion-matching task than in the identity-matching task (81.3% vs. 92.6%; $F_{1, 38} = 38.23$, $P < 0.0001$; 1246 vs. 1020 ms: $F_{1, 38} = 29.75$, $P < 0.0001$). The factor response was significant for both measures: participants performed better and faster when they had to say “different” rather than “same” (89.5% vs. 84.5%; $F_{1, 38} = 5.55$, $P < 0.05$; 1112 vs. 1154 ms; $F_{1, 38} = 4.78$, $P < 0.05$). The factor change was also significant for percentage and latencies: performances were more accurate and faster when the second dimension did not change (92.7% vs. 81.2%; $F_{1, 38} = 28.64$, $P < 0.0001$; 1054 vs. 1212 ms: $F_{1, 38} = 122.08$, $P < 0.0001$) (Table 3).

The following interactions were significant for both percentage and latencies: matching dimension *

Table 3
Mean percentages and latencies (ms) for correct emotion and identity matching of schizophrenic patients and controls

Task	Emotion				Identity			
	Same		Different		Same		Different	
Response	No	Yes	No	Yes	No	Yes	No	Yes
<i>Percentage (%)</i> :								
Schizophrenic patients	92.4	48.4	81.4	80.1	90.6	80.5	91.9	92.0
(SD)	(13.1)	(27.6)	(21.1)	(18.8)	(8.6)	(25.7)	(10.0)	(11.8)
Controls	98.1	71.5	93.9	84.8	98.3	96.1	95.3	96.4
(SD)	(2.3)	(12.3)	(8.5)	(10.0)	(2.3)	(3.8)	(5.6)	(3.8)
<i>Latencies (ms)</i> :								
Schizophrenic patients	1138	1643	1426	1426	1121	1242	1312	1225
(SD)	(255)	(708)	(366)	(394)	(442)	(548)	(895)	(582)
Controls	833	1283	1041	1176	747	886	811	816
(SD)	(218)	(298)	(235)	(321)	(204)	(248)	(219)	(227)

change (respectively, $F_{1, 38}=43.49$, $P<0.0001$ for percentage; $F_{1, 38}=46.36$, $P<0.0001$ for latencies), response * change (respectively, $F_{1, 38}=14.83$, $P<0.0001$ for percentage and $F_{1, 38}=96.18$, $P<0.0001$ for latencies) and matching dimension * response * change (respectively, $F_{1, 38}=9.96$, $P<0.001$ for percentage; $F_{1, 38}=17.80$, $P<0.001$ for latencies). This last three-way interaction indicated that the interaction of response * change was significant when the matching dimension was on emotion ($F_{1, 38}=31.58$, $P<0.0001$ for percentage; $F_{1, 38}=75.54$, $P<0.0001$ for latencies). When the matching dimension was on identity, the interaction response * change was significant for latencies ($F_{1, 38}=29.41$, $P<0.0001$) but only tended to be significant for percentage ($F_{1, 38}=4.02$, $P<0.06$).

When the matching dimension was on emotion, changing identity had a significant detrimental effect when the response was “same” for both accuracy and latencies (59.9% vs. 95.3%: $F_{1, 38}=28.89$, $P<0.0001$; 1463 vs. 986 ms: $F_{1, 38}=115.76$, $P<0.0001$). When the response was “different”, changing identity had a significant detrimental effect for latencies (1301 vs. 1234 ms: $F_{1, 38}=5.10$, $P<0.05$) but not for accuracy (82.4% vs. 87.6%: $F_{1, 38}=1.00$, NS). Thus, changing identity in emotion matching interfered with accuracy and latencies when the two faces expressed the same emotion (“same” response) and interfered with latencies when the two faces did not have the same emotion (“different” response).

When matching dimension was on identity, changing emotion had a significant detrimental effect on latencies when the response was “same” (1064 vs. 934 ms: $F_{1, 38}=69.21$, $P<0.0001$) but had no effect when the response was “different” ($F_{1, 38}=0.53$, NS).

Elsewhere, changing identity in emotion matching had a more detrimental effect than changing emotion in identity matching when the response was “same” (difference of 35.4% vs. 6.1% and 477 vs. 130 ms).

The interaction group * matching dimension was significant for percentage ($F_{1, 38}=6.10$, $P<0.05$). It indicated that both controls and patients with schizophrenia were more accurate in identity matching than in emotion matching (96.5% vs. 87.1% for controls: $F_{1, 38}=6.89$, $P<0.01$; 88.8% vs. 75.6% for patients with schizophrenia: $F_{1, 38}=37.44$, $P<0.0001$), but the difference between the two tasks was stronger for patients with schizophrenia (13.1% vs. 9.4%). The

performances for patients with schizophrenia were nevertheless worse than for controls both for identity ($F_{1, 38}=9.60$, $P<0.01$) and emotion ($F_{1, 38}=13.80$, $P<0.001$). The interaction between group * change was significant for percentage ($F_{1, 38}=6.05$, $P<0.05$), but it was modulated by the interaction of group * response * change ($F_{1, 38}=6.06$, $P<0.05$). It indicated that the interaction of response * change was significant for controls ($F_{1, 19}=36.57$, $P<0.0001$) and for patients with schizophrenia ($F_{1, 19}=10.10$, $P<0.01$). For controls, changing the second dimension had a significant detrimental effect either when the response was “same” (98.2% vs. 83.8%: $F_{1, 19}=73.19$, $P<0.0001$) or “different” (94.6% vs. 90.6%: $F_{1, 19}=17.43$, $P<0.001$), but this detrimental effect was stronger when the response was “same” (14.4% vs. 4.0%). For patients with schizophrenia, changing the second dimension had a significant detrimental effect when the response was “same” (91.5% vs. 64.4%: $F_{1, 19}=13.47$, $P<0.01$) but had no effect when the response was “different” (86.6% vs. 86.1%: $F_{1, 19}=0.01$). Moreover, when the response was “same”, the detrimental effect of changing the second dimension was stronger for patients with schizophrenia than for controls (27.1% vs. 14.4%).

Thus, to summarize these results, performance of all participants was impaired by the change of the second dimension. Nevertheless, performance was more dramatically impaired for patients with schizophrenia than for controls. Table 2 further indicates that for patients with schizophrenia, performance was at chance level when they had to match the same emotions in different faces (correct responses: 48.4% vs. 71.5% for controls).

The global interaction (group * matching dimension * response * change) was not significant either for percentage ($F_{1, 38}=2.85$, NS) or latencies ($F_{1, 38}=0.45$, NS).

3.2. Correlation analysis

We calculated Spearman correlation coefficients between performance for identity matching and emotion matching (see Fig. 2). For accuracy, there was a significant positive correlation between the two tasks both for accuracy ($\rho=0.53$, $P<0.001$) and latencies ($\rho=0.72$, $P<0.0001$). Thus, the more/less accurate and fast participants on identity matching

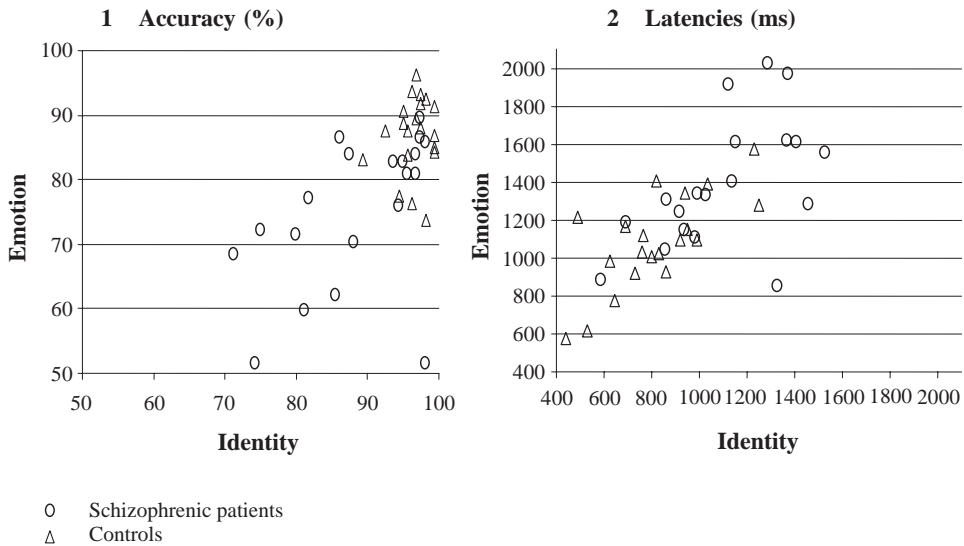


Fig. 2. Correlation between percentage of correct responses and latencies in identity and emotion matching for schizophrenic patients and controls.

also tended to be the more/less accurate and fast participants on emotion matching. When we considered patients with schizophrenia alone, the correlation was also significant for both accuracy ($\rho=0.53$, $P<0.05$) and latencies ($\rho=0.61$, $P<0.01$). Then, when a patient had a deficit in one task, he tended also to have a proportional deficit in the other task. When only controls were considered, the correlation was always significant for latencies ($\rho=0.62$, $P<0.01$) but not for accuracy ($\rho=0.14$, $P>0.50$).

For patients with schizophrenia, we also calculated Spearman correlation coefficients between SANS or SAPS total scores and subscores and performances for identity or emotion matching. Performances were not correlated with total SAPS scores, but the delusions subscore was correlated with both emotion matching ($\rho=0.46$, $P<0.04$) and emotion matching when identity changed ($\rho=0.68$, $P<0.01$). The SANS score was negatively correlated with identity matching ($\rho=-0.48$, $P<0.05$) and tended to be negatively correlated with emotion matching ($\rho=-0.41$, $P<0.08$). Moreover for emotion matching, the SANS score was negatively correlated with emotion matching when identity changed ($\rho=-0.54$, $P<0.02$). Three SANS subscores were correlated with emotion matching when identity changed (affective flattening: $\rho=-0.48$, $P<0.03$;

avolition–apathy: $\rho=-0.49$, $P<0.03$; inattentiveness: $\rho=-0.53$, $P<0.02$). Thus, the deficit in matching one emotion expressed by two distinct persons observed for patients with schizophrenia co-varied with the severity of negative symptoms (see Fig. 3). No other correlation was significant.

Fig. 3 also indicates that four patients performed far below chance levels (from 0% to 12.5% correct) in emotion matching from different identities. Two of them also performed below chance when matching the

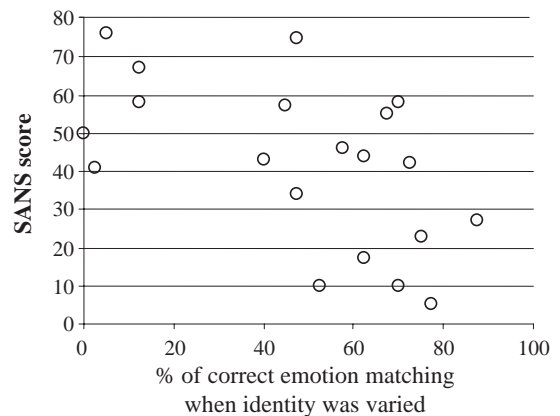


Fig. 3. Correlation between SANS score and percentage of correct responses in emotion matching (“same” response) when identity was varied for schizophrenic patients.

same identity from a different emotion (both 12.5%). All these patients performed above chance levels in other conditions. They evidently mistook a change of identity (or emotion) for a change of emotion (or identity). This finding parallels the observation reported by Young et al. (1996) with an amygdalectomy patient.

4. Discussion

The results of the present study indicate that patients with schizophrenia were impaired in processing facial emotion and identity in a delayed matching task. In addition, this study shows that this impairment was greater when the second facial dimension manipulated in the experiment varied. In other words, patients with schizophrenia were impaired in the processing of facial information such as identity and emotion, but they also exhibited impairment in selectively responding to each of them. Variations of identity in emotion matching even gave rise to performances that were at chance level. Performances in emotion and identity matching were significantly and positively correlated; a deficit for one kind of facial information was associated with a deficit for the other. Finally, the severity of the negative symptoms, especially affective flattening, avolition–apathy and inattentiveness, co-varied with deficits in facial processing; notably the higher these scores were, the greater the interference of identity on emotion matching. Moreover the SAPS delusions subscore was positively correlated with both emotion matching and emotion matching when identity changed.

In the current study we showed that although schizophrenic patients were impaired in correctly identifying the identity and emotion of face stimuli, this impairment was greater for emotional stimuli. Such a result indicates that even though schizophrenic patients have a general difficulty in processing facial information, the difficulty is particularly acute for facial emotion. This is consistent with previous studies which have shown that such patients have greater difficulty processing emotion compared with other kinds of facial information, e.g., gender or age (Gooding et al., 2001; Gooding and Tallent, 2002). Such a difference might result from active symptoms, the acute phase of schizophrenia, resulting in the

greatest impairment in emotion perception (Penn et al., 2000).

The present experiment furthermore indicates that patients with schizophrenia have problems in selectively attending to one facial dimension—either identity or emotion—while ignoring variation of the second. Some of them may even mistake a difference of identity for a difference in emotion or a difference of emotion for a difference in identity. This last observation is similar to the case of an amygdalectomy patient who said that she saw two different persons when she was presented with the face of one person expressing two different emotions (Young et al., 1996). Both observations indicate that the ability to respond selectively to identity or emotion may be impaired after either a cerebral damage or severe psychiatric disorders. Consequently, it appears that some cognitive processes are implied in the dissociation of both kinds of facial information, and that these processes can be altered in schizophrenia. This hypothesis is strengthened by studies that have shown some kinds of interference between facial emotion and identity for healthy participants (Schweinberger and Soukup, 1998; Schweinberger et al., 1999; Baudouin et al., 2000a, b). Consequently, the ability to respond specifically to one kind of facial information (e.g., identity) without interference from the other kinds (e.g., facial expression) does not result from implementing independent processes but, rather, these processes interact and may reciprocally interfere. Thus, selective attention to one kind of facial information needs the intervention of an active dissociation process that allows selective responses to each kind of facial information. The association between negative symptoms and the difficulty in dissociating identity and emotion in our studies suggests that the dissociation processes are affected by such symptoms. Correlations between negative symptoms and some disabilities in facial emotion processing have already been reported (Silver et al., 2002). The fact that affective flattening was correlated with performance in our task leads us to speculate that perception and expression of emotions are closely linked. The correlation with the avolition–apathy subscore suggests that patients impaired in recognizing others' expressions are not able to interact with other people in social activities. In contrast, the attentional impairment, as measured by the SANS

inattentiveness subscore, could result in an overall decrease in the patients' performance. Further investigations are required on the role of negative symptoms in facial emotion processing to determine whether dissociation processes are affected by such symptoms. The positive correlation between the patients' performance in our tasks and their delusions subscore suggests that the more severe the delusions are, the better the patients are at recognizing others' emotions. This is consistent with the notion that in delusional patients top-down processes cause them to be hyperattentive to others' expressions.

The precise nature and location of these dissociation processes are unclear, but some suggestions can be advanced. First, many studies have implicated the amygdala in the processing of facial emotion (e.g., Breiter et al., 1996; Streit et al., 1999). Young et al. (1996) reported that damage to this region may cause deficits in matching identity, and that these processes can be altered in schizophrenia. This region was also proposed as a critical structure in the cognitive dysfunction in schizophrenia (see Evangeli and Broks, 2000; Edwards et al., 2001), and some studies have indicated abnormalities in schizophrenia with regard to amygdala activation during affect recognition (e.g., Schneider et al., 1998; Phillips et al., 1999; Gur et al., 2002). Indeed, some of the patients we described here exhibited the same kind of deficit as seen in the amygdectomy patient reported by Young et al. (1996). Thus, one may suspect that the amygdala plays a role in the ability to dissociate facial emotion and identity.

Elsewhere, an increasing number of authors working on face recognition question the role of the frontal and prefrontal cortex in face recognition. These regions were reported as being activated in tasks that imply facial information processing (Allison et al., 1999; Guillaume and Tiberghien, 2001; Marinkovic et al., 2000; Sergent et al., 1994). Damage to these areas may cause deficits in face recognition (Rapcsak et al., 1996) while stimulation gives rise to hallucinations of faces (Vignal et al., 2000). Guillaume and Tiberghien (2001) observed that the N200 amplitude was increased in an episodic memory task when the context (and, more particularly, the emotional expression) was varied between encoding and recognition stage. Grady et al. (2000) observed that the activity of the frontal lobe increased when face quality was

degraded and they explained this result by the allocation of supplementary cognitive resources to analyze the stimulus (see also Kanwisher and Moscovitch, 2000). Many authors have suggested that the frontal lobe is part of the system for face perception, and that its interaction with the fusiform gyrus is a critical element of face processing (Marinkovic et al., 2000; Vignal et al., 2000). Elsewhere, cognitive dysfunctions in schizophrenia have been linked to a decrease of activity in the frontal lobe and an abnormal coordination of activity between the frontal and temporal lobes (for a review, see Mitchell et al., 2001). One may then suggest that the ability to dissociate facial emotion and identity may result from the coordination of the frontal and temporal lobes. The temporal lobe analyzes facial stimuli while the frontal lobe modulates temporal lobe activities according to the characteristics of the stimulus and the intention, expectancy and task. Some aspects of the cognitive dysfunctions in schizophrenia could result from an impaired coordination of these structures. This hypothesis needs to be tested in further research.

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