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Performance during Face Processing Differentiates Schizophrenia Patients with Delusional Misidentifications

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Key Words
Delusional misidentification syndrome · Schizophrenia · Face recognition · Performance · Capgras · Fregoli

Abstract
Background: Delusional misidentification syndrome (DMS) is of considerable interest, but rarely diagnosed clinically. It is supposed to occur relatively frequently in schizophrenia, and to be related to the pathophysiology of face processing. Two antagonistic forms of DMS are the hypoidentification (Capgras) and hyperidentification (Fregoli) syndromes. We aimed to highlight differences between these subtypes using a face recognition memory task. ***Methods:*** Twenty schizophrenia patients (10 with DMS) and 21 healthy controls memorized the images of unknown neutral faces (targets). After a 10-min interval, accuracy and reaction times were recorded during a recognition task consisting of targets (newly learned faces), as well as familiar and unfamiliar face images. The 10 DMS patients could be further subdivided into 6 patients with Fregoli syndrome and 4 with Capgras syndrome. ***Results:*** Patients with DMS had longer reaction times than controls or patients without DMS ($p < 0.001$). Fregoli patients had longer reaction times ($p < 0.001$) and lower discrimination accuracy than Capgras patients ($p = 0.019$). These results were independent of other clinical variables. ***Conclusions:*** Face recognition differs between clinically identified subgroups of schizophrenia and between types of DMS. The results indicate independent pathophysiological mechanisms for Capgras (hypoidentification) and Fregoli (hyperidentification) syndromes in schizophrenia.

Introduction

Facial processing is critical for communication between humans, and there are indications of severe impairments in schizophrenia [1–3]. Several studies have focused on face identity and face emotion recognition in schizophrenia. Poor accuracy in face identity recognition has been reported in comparison to healthy controls [3–5] as well as to psychiatric controls [1, 6]. The impairment is more pronounced in patients with a longer duration of illness [7], suggesting progression over the course of the disease. Furthermore, the deficit is likely to have a genetic component, since non-affected relatives of schizophrenia patients were found to perform better than patients, but worse than healthy controls, in face memory tests in terms of accuracy [8] and speed [4]. Heritability,

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however, has also been suggested for eye movements [9, 10], which are part of the visual system involved in face processing [11] and have repeatedly been shown to be altered in schizophrenia.

The morphological recognition of a face elicits an affective response, and both types of information are supposed to be necessary to decide whether someone is familiar or not. In the case of correctly decoded facial information but lack of an emotional response, one perceives a familiar face as alien. This is a possible explanation of delusional misidentification syndromes (DMS) [12–14], such as Capgras syndrome [15], where a familiar person is perceived as being alien, or Fregoli syndrome, in which an unknown person is taken for a familiar one [16]. Besides that, there are at least 2 other misidentification syndromes related to persons: intermetamorphosis, where people change both physical and mental identities, and subjective doubles, when another person is transformed into the patient (patient considers the other as double of him/herself). The literature refers to a variety of other misidentification syndromes, e.g. reduplicative paramnesia (a place exits simultaneously in 2 locations) or foreign reduplicative paramnesia (a familiar place exists in another location), which are related to places [for reviews, see 17 and 18]. Misidentifications have also been reported for limbs, time and objects [19]. The delusion of inanimate doubles (transformed objects) occurs along with the Capgras delusion in approximately 7% of DMS cases [20]. The prevalence of DMS is reported to be 3–4% in a psychiatric population, with most cases occurring in schizophrenia [21, 22].

Because of the severe disturbance in the recognition of persons, which results in delusional beliefs about identity, DMS have been suggested to represent a target for research on face processing pathophysiology in schizophrenia in general [13, 23]. Presumably, schizophrenia patients with DMS would be more disturbed in face processing than patients without DMS. Psychophysiological differences between the 2 groups were reported in an auditory P300 study that demonstrated differences between schizophrenia patients with DMS and without DMS in latencies and amplitudes in frontal and central areas [24]. Edelstyn et al. [25] reported increased total reaction times in a case series of 5 DMS patients and 3 patients without DMS but with schizophrenia, as well as problems with famous face recognition in the DMS patients. However, another group failed to find differences between schizophrenia patients with and without DMS in neuropsychological assessments, including the Benton facial recognition task [26].

Homogeneity in performance between schizophrenia patients with and without DMS could also stem from heterogeneity within the group with DMS. In fact, the previously mentioned study included patients with Capgras, Fregoli, intermetamorphosis and mixed types into the group with DMS [26]. Capgras patients were shown to exhibit poor performance in facial recognition memory tests, tests on facial emotion recognition and recognition of familiar faces. However, Capgras patients correctly rejected unfamiliar faces [14, 27]. Capgras syndrome is considered to be a syndrome of hypoidentification [28–30]. In contrast, a case of Fregoli delusion following damage of the right anterior fusiform gyrus and atrophy of the parahippocampal gyrus was demonstrated to have intact recognition of familiar faces [31]. Indeed, Fregoli syndrome can be seen as a syndrome of hyperidentification, in which feelings of familiarity are attached to unfamiliar subjects [28–30, 32]. In fact, Fregoli patients seem to rely mostly on the feeling of familiarity in face recognition tasks [33]. Both psychopathology and performance in face processing are very likely to be different, even contradictious, between delusions of the Capgras and Fregoli type. Hence, a heterogeneous group of the various DMS types could lack marked differences when compared with patients without DMS.

Cognitive models of face processing describe the process starting from the structural encoding of facial information. If the face seen is a familiar one, it will contact its representation in the face recognition unit (FRU). In the model of Bruce and Young [34], the following steps are placed in a sequential way: the FRU activates the person identity node (PIN), which contains biographic and semantic information. The final step is the name retrieval, which is activated by the PIN since names are stored independently from other information. Ellis and Young [35] then adapted this model, introducing a 2-route model with an affective component for familiar face recognition. However, Breen et al. [36] criticized the Ellis and Young model, and argued that face recognition is subserved only by the ventral visual-limbic pathway. Instead, they suggested that a second pathway leads from the FRU to the affective response. If the person viewed is very familiar, the affective response will be strong. The affective response will interact with the PIN, which will lead to the name retrieval. Breen et al. [36] argue that Capgras patients have a disruption in the affective response to familiar stimuli, while the FRU is still intact. Later, Ellis and Lewis [13] presented another modified face recognition model that incorporated the FRU, PIN and affective response from the Breen model, but, in contrast, the PIN

Table 1. Participants' demographics

	Controls	DMS positive	DMS negative	χ^2, F or t value	p
n	21	10	10		
Gender					
Men	12	6	8	1.567	0.488
Women	9	4	2		
Age, years	26.10 ± 4.17	27.85 ± 6.33	29.35 ± 7.89	1.129	0.334
Chlorpromazine equivalents		435.50 ± 526.56	483.29 ± 333.53	–1.055	0.308
PANSS total		62.40 ± 17.16	59.40 ± 14.90	1.335	0.202
PANSS positive		14.10 ± 5.09	13.90 ± 3.84	0.100	0.922
PANSS negative		19.00 ± 5.87	16.70 ± 5.85	1.926	0.073
Duration of illness, years		4.40 ± 3.83	6.40 ± 6.13	–0.875	0.393
Episodes, n		3.10 ± 3.25	5.90 ± 7.33	–1.105	0.284

Data presented as means ± SD.

information and the affective response are separately passed on to an integrative device that compares conclusions from both types of incoming information. According to Ellis and Lewis [13], Capgras delusion results from a disruption in the affective response, which will lead to opposing information content in the integrative device, and will therefore result in a delusional attribution (i.e. the familiar person must have been replaced by an imposter). Finally, Ellis [37] suggested disturbances within the PIN to explain Fregoli delusions.

In order to identify alterations in face processing of schizophrenia patients, we investigated schizophrenia patients with and without DMS in comparison to controls by applying a modified recognition memory task that focused on structural facial information. Therefore, our task consisted of neutral faces only. In a study phase, subjects learned unfamiliar faces. Newly learned and highly familiar (famous) faces were chosen to investigate: (1) recognition memory (newly learned faces), and (2) recognition of very familiar stimuli (famous faces). During the test phase, subjects were also confronted with unfamiliar foils, to control for false alarms.

We hypothesized that schizophrenia patients with DMS would display more pronounced deficits in the recognition of face images when compared with patients without DMS or controls. We expected, upon subdividing the group of DMS patients, to see a differential pattern of performance deficits: Fregoli patients being more likely to have increased false alarm rates due to hyperidentification and reduced discrimination accuracy; Capgras patients, with hypoidentification, were expected to display an increased hit rate for familiar faces, a reduced false alarm rate, as well as a negative response bias. We hypothesized that both DMS types would have decreased recognition memory for the newly learned faces.

Methods

Participants

Twenty patients with schizophrenia according to DSM-IV criteria and 21 healthy controls matched for gender and age were investigated as part of a larger study on the neuropsychiatric features of schizophrenia. All patients were inpatients of the University Hospital of Psychiatry Bern at the time of study participation. Age, chlorpromazine equivalents and PANSS scores [38] are given in table 1. All study participants were free of medical disorders (other than schizophrenia in the study patients), as determined by clinical examination, structured interview and review of their medical history. Because the subjects were recruited for a larger project, every person had 1.5-T T_1-weighted structural MRI. None of the participants had any detectable structural intracranial pathology. Diagnoses were given by board-certified psychiatrists (H.H., W.S. and T.J.M.) after extensive exploration and review of the case history. Inclusion criteria were: (1) diagnosis of schizophrenia or schizoaffective disorder according to DSM-IV; (2) age 18–45 years. Exclusion criteria were: (1) substance abuse or dependence (other than nicotine); (2) history of electroconvulsive treatment; (3) history of head trauma or neurological disorders. One trained investigator (H.H.) conducted all PANSS ratings. Chlorpromazine equivalents were calculated according to the literature [39, 40]. Schizophrenia patients were screened for the presence of a DMS using the Bern Assessment of Delusional Misidentification [41], a structured interview which has been validated in schizophrenia [42]. Briefly, patients were asked to indicate whether they had experienced delusional misidentifications of subjects, and, if so, the type (Capgras or Fregoli), frequency, familiarity of the misidentified person, stability of the delusion as

Table 2. Diagnostic characteristics of schizophrenia patients

Patient No.	DSM-IV diagnosis (schizophrenic type)	Type of DMS	Objects of misidentification	Frequency
1	paranoid	Fregoli	unknown persons	daily
2	paranoid	Fregoli	unknown persons	weekly
3	paranoid	Fregoli	unknown persons	monthly
4	paranoid	Fregoli	unknown persons	monthly
5	paranoid	Fregoli	acquaintances	daily
6	disorganized	Fregoli	unknown persons and animals	monthly
7	paranoid	Capgras	parents and girlfriend	daily
8	paranoid	Capgras	family members	weekly
9	paranoid	Capgras	mother	monthly
10	paranoid	Capgras	close friends	monthly
11	paranoid	none		
12	paranoid	none		
13	paranoid	none		
14	paranoid	none		
15	catatonic	none		
16	disorganized	none		
17	disorganized	none		
18	disorganized	none		
19	schizoaffective	none		
20	schizoaffective	none		

well as the behavioral consequences (e.g. fear or aggression) were obtained. The groups of schizophrenia patients were characterized by the presence (DMS+) or absence of a DMS (DMS–). Only patients in whom the syndrome could clearly be diagnosed or ruled out were included. The characteristics of the DMS and DSM-IV diagnoses are provided in table 2. The average duration of education showed no differences between DMS+ (11.5 ± 3.5 years) and DMS– (12.0 ± 3.3 years; $t = -0.327$; $p = 0.747$) patients.

The investigation was conducted in accordance with the Declaration of Helsinki and approved by the local ethics committee. Before entering the study, all subjects provided written informed consent.

Procedures

A slightly modified facial recognition memory task was applied, which consisted of a study phase, when subjects were presented with the target stimuli, and a recognition phase, when target stimuli and familiar distracters were discriminated from novel distracters.

During the study phase, participants were presented with 30 images of unfamiliar faces with neutral affect (15 men and 15 women, all adults of different ages). The faces were displayed as black and white photographs with equal luminescence. Images were taken from the same source as in earlier studies [5, 43], which is a collection of portraits of Swedish people that have been edited and cut in order to reduce the image to the face only, leaving very little background or hair to the image. Face images appeared in random order, each displayed for 2 s with a blank screen interval of 2 s. Subjects were asked to memorize the faces to be able to recognize them later when they would be presented with these faces as well as with unfamiliar and familiar (i.e. famous) faces.

The recognition phase followed after an interval of 10 min. During the recognition phase, subjects were presented with 30 faces, each displayed for 2 s. Ten of the faces were from the previously memorized set (newly learned targets), 10 were familiar (famous) persons, and 10 were completely unfamiliar to the participants. All images were balanced for gender among the face categories. Familiar faces with neutral affect were obtained from various sources and digitally changed so that they were equal in luminescence, black and white features, and size. The familiar faces contained people of high celebrity status, including movie and sport stars, musicians, scientists and politicians. The familiar faces were prominent dead or living persons that frequently appeared in the Swiss media at the time of the study (2004).

Faces were presented in random order with a blank screen interval. Subjects were instructed that during this task they would be confronted with faces shown during the study phase as well as with faces of celebrities (both of which were considered to be familiar) in contrast with completely unfamiliar faces. Study participants were asked to indicate whether the image displayed was familiar or unfamiliar by pressing 1 of 2 mouse buttons as quickly as possible (left button for 'familiar' and right button for 'unfamiliar'). Reaction times and responses were recorded.

Performance Measures

Correct identification of a newly learned target face was defined as a 'hit', as was the correct identification of a familiar distracter, whilst the false recognition of an unfamiliar face as familiar was termed 'false alarm'. Due to our study design, we had 2

Table 3. Recognition memory performance measures between control and schizophrenia groups

	Controls	DMS+	DMS–	Test	
				F	p
Hit rate					
Overall	12.00 ± 4.48	7.40 ± 3.78	8.30 ± 5.42	2.334	0.111
Newly learned	6.62 ± 3.12	4.60 ± 3.27	3.30 ± 3.83	1.399	0.260
Familiar	5.38 ± 3.02	2.80 ± 2.30	5.00 ± 2.16	2.389	0.105
False alarm rate					
Overall	1.33 ± 1.53	2.30 ± 2.79	3.00 ± 3.59	2.294	0.115
				χ^2	p
Discrimination accuracy (A′)					
Overall	0.59 ± 0.07	0.66 ± 0.75	0.53 ± 0.26	0.868	0.648
Newly learned	0.78 ± 0.62	0.78 ± 0.80	0.98 ± 1.03	0.281	0.869
Familiar	0.54 ± 0.18	0.97 ± 1.30	0.98 ± 1.65	0.750	0.687
Response bias (B′′)					
Overall	0.24 ± 0.28	0.20 ± 0.33	–0.05 ± 0.44	3.787	0.151
Newly learned	0.07 ± 0.33	0.14 ± 0.34	–0.08 ± 0.18	3.347	0.188
Familiar	0.28 ± 0.33	0.20 ± 0.39	–0.18 ± 0.46	7.440	0.024[1]

[1] DMS+ > DMS– p = 0.039, controls > DMS– p = 0.003.

types of hit (newly learned and familiar) and 1 type of false alarm. Reaction times were recorded for each stimulus that had been responded to.

Data Analysis

In the first step, all performance data were analyzed and compared between controls, DMS+ and DMS– patients. In the second step, we compared the performance of the DMS subtypes (Fregoli and Capgras) with DMS– schizophrenia patients. Reaction times were compared between DMS subtypes, patients without DMS and controls.

The composition of the proportional hit rate (overall, newly learned and familiar faces) and the false alarm rate was transformed using the arcsine square root formula to satisfy normality assumptions for parametric tests. Using ANOVA, hit rates and false alarm rates were compared between groups. Post hoc analyses were computed using the Bonferroni t method of correction.

Hit rate and false alarm rate for each participant were converted into the nonparametric signal detection indices of discrimination accuracy A′ and response bias B′′ [44]. Discrimination accuracy A′ is defined as the ability to discriminate targets from distracters. High values represent strong discrimination ability. Response bias B′′ refers to the probability of accepting a stimulus as a target when uncertain. Equal probability of 'familiar' and 'unfamiliar' responses in an uncertain state will lead to a B′′ value of 0. Positive values (0–1) indicate more liberal responses, and negative values (–1 to 0) more conservative responses. Conservative response bias is defined as higher probability of choosing 'unfamiliar' when uncertain. These nonparametric measures (A′ and B′′) were analyzed as dependent variables using the Kruskal-Wallis H test. Pairwise comparisons were computed using Mann-Whitney U tests.

Reaction times for all stimuli were compared using univariate analysis with the factors group, response (correct or incorrect) and stimulus type (newly learned, familiar, or unfamiliar faces), as well as their interaction. Demographic variables were compared between groups using one-way ANOVAs or χ^2 tests where appropriate. Finally, performance measures for the schizophrenia patients were correlated with PANSS positive and negative subscores as well as the PANSS total scores. Likewise, performance measures were entered in an ANCOVA, with PANSS subscores and total scores as covariates. Statistical analyses were performed using SPSS® 15.0.

Results

Missed Stimuli

The proportion of missed stimuli (i.e. stimuli with no response) was not different between groups for the total amount of stimuli, as well as for familiar targets and unfamiliar distracters. However, the proportion of missed newly learned targets differed significantly [F(2, 41) = 3.852, p = 0.030] with DMS– patients having a higher proportion of missed targets than controls (post hoc Bonferroni t test: p = 0.026).

Table 4. Recognition memory performance measures between DMS types and patients without DMS

	Fregoli	Capgras	DMS–	Test	
				F	p
Hit rate					
Overall	8.17 ± 3.54	6.25 ± 4.35	8.30 ± 5.42	2.191	0.142
Newly learned	4.67 ± 2.73	4.50 ± 4.43	3.30 ± 3.83	0.008	0.992
Familiar	3.50 ± 2.17	1.75 ± 2.36	5.00 ± 2.16	3.783	0.044*
False alarm rate					
Overall	2.33 ± 2.25	2.25 ± 3.86	3.00 ± 3.59	0.340	0.717
				χ^2	p
Discrimination accuracy (A′)					
Overall	0.43 ± 0.21	0.99 ± 1.17	0.53 ± 0.26	2.216	0.344
Newly learned	0.41 ± 0.28	1.33 ± 1.06	0.98 ± 1.03	5.073	0.037**
Familiar	0.47 ± 0.33	1.73 ± 1.90	0.98 ± 1.65	0.490	0.794
Response bias (B′′)					
Overall	0.18 ± 0.30	0.24 ± 0.41	–0.05 ± 0.44	1.799	0.424
Newly learned	0.18 ± 0.27	0.08 ± 0.46	–0.08 ± 0.18	3.492	0.179
Familiar	0.28 ± 0.35	0.08 ± 0.46	–0.18 ± 0.46	3.389	0.188

* p = 0.041, post hoc Bonferroni test (Capgras < DMS–); ** p = 0.019, Mann-Whitney U test (Fregoli < Capgras).

Accuracy

Measures of accuracy between controls and schizophrenia groups are given in table 3. Neither hit rates and false alarm rates nor the nonparametric signal detection index of A′ (discrimination accuracy) differed significantly between groups. However, for the response bias B′′ to familiar faces, DMS– patients showed negative values in contrast to DMS+ and controls. Thus, DMS– patients display a higher probability of responding 'don't know' in an uncertain state. In contrast, DMS+ and controls had positive values for B′′, and are therefore more likely to respond 'know' when uncertain.

DMS+ patients were subdivided according to the type of misidentification and compared in terms of accuracy with schizophrenia patients without DMS (table 4). Hit rates differed only for familiar targets: Capgras patients had a lower proportion of hits than patients without DMS (p = 0.041). In terms of discrimination accuracy, Fregoli patients had lower accuracy than Capgras patients when discriminating newly learned targets from distracters (p = 0.019). Response bias did not differ significantly between these groups.

Reaction Time

Univariate analysis of the reaction time in the whole sample for all images revealed a strong effect for group [$F(2, 1{,}108) = 21.199$, $p < 0.001$], response (i.e. correct or false) [$F(1, 1{,}108) = 10.085$, $p = 0.002$] as well as for the group × stimulus × response interaction [$F(4, 1{,}108) = 3.843$, $p = 0.004$]. Post hoc Bonferroni tests demonstrated that schizophrenia patients with DMS had longer reaction times than healthy controls and schizophrenia patients without DMS (both $p < 0.001$).

When we divided the group of patients with DMS into the specific DMS type, univariate analysis of the reaction time for all images also revealed a strong effect for group [$F(3, 1{,}108) = 25.903$, $p < 0.001$], response [$F(1, 1{,}108) = 10.085$, $p = 0.002$], a stimulus × group interaction [$F(6, 1{,}108) = 2.197$, $p = 0.041$], as well as a stimulus × response × group interaction [$F(6, 1{,}108) = 3.866$, $p = 0.001$]. Post hoc Bonferroni tests demonstrated that patients with Fregoli syndrome (i.e. hyperidentification) had longer reaction times than patients with Capgras syndrome, patients without DMS or controls (each $p < 0.001$; fig. 1).

Impact of Clinical Features

None of the parameters tested correlated significantly with the PANSS positive, negative or total scores or the

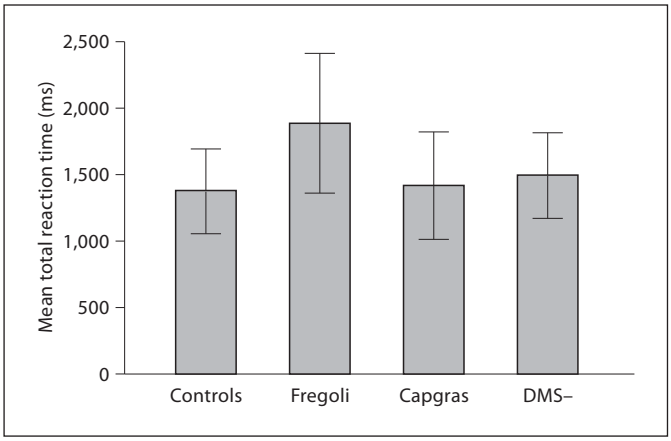

Fig. 1. Bars display mean reaction times for all stimuli (with error bars indicating 1 SD). Post hoc Bonferroni tests revealed that the Fregoli group had longer reaction times than all other groups ($p < 0.001$).

chlorpromazine equivalents; nor did the test performance significantly co-vary with PANSS positive, negative or total scores.

Discussion

In the present study, we were able to demonstrate differences in the performance of schizophrenia patients with and without DMS during a challenging face identity recognition task. Patients with DMS had longer reaction times compared to controls and patients without DMS. The patients without DMS showed an increased number of missing responses to the target faces (newly learned) and a negative response bias for familiar faces. Besides that, no significant differences in accuracy were elicited. These limited findings parallel previous reports of few or no differences between patients with and without DMS in face recognition, memory performance or neuropsychological tests [24–26]. As in our schizophrenia patients without DMS, negative response bias has recently been reported for schizophrenia patients in a visual object memory task [45].

However, when we subdivided DMS patients into Capgras and Fregoli syndromes, a different pattern became evident. It is only the Fregoli type that differs in reaction time, with longer reaction times than Capgras patients, patients without DMS or controls. Along with this finding, Fregoli patients also had a reduced discrimination accuracy for the newly learned target faces as compared to Capgras patients. Capgras patients in turn had a lower hit rate for familiar faces as compared to schizophrenia patients without DMS. However, response bias did not differ between groups. None of the parameters correlated or co-varied with PANSS subscores, which gives rise to the notion that performance in our task was not related to psychopathological phenomena other than delusional misidentifications.

Fregoli patients were expected to have an overall low discrimination accuracy and an increased false alarm rate, due to hyperidentification [32]. In our study, however, the false alarm rate was not different between schizophrenia groups. This could be due to the rather difficult task that led to false alarms in every schizophrenia group. In contrast, discrimination accuracy varied across groups with Fregoli patients displaying the lowest. Still, because of considerable variance in the data, the difference became significant only in newly learned target faces. Others have reported reduced visual memory, intrusions and confabulations in brain-damaged patients with Fregoli syndrome [29]. In fact, the Fregoli syndrome patient B.C., reported in the study of Edelstyn et al. [33], even had higher discrimination accuracy than the psychotic controls; however, all the recognition of targets relied on feelings of familiarity rather than conscious recollection. This is different to our findings. Still, the severity of DMS may vary between subjects.

The reduced discrimination accuracy and increased reaction times found in this study generally support the idea of a hyperidentification syndrome in Fregoli patients [29, 30, 32]. However, if this was entirely true and the PINs were generally disturbed as suggested by Ellis [37], the response bias had to be highly positive, because in any case of doubt hyperidentifiers will claim to know the face rather than reject it. In our study, the response bias was only slightly positive and close to that of the controls. Again, the deficit in Fregoli patients results in hyperidentification, and it might be a deficit in error monitoring. Indeed, brain-damaged patients with Fregoli syndrome have been reported to frequently suffer from right frontal damage [30].

In Capgras patients with hypoidentifications, we expected negative response biases, reduced false alarm rates and increased hit rates for familiar faces. As mentioned above, false alarm rates were not different between groups. Capgras patients had reaction times comparable to those of controls. Discrimination accuracy for newly learned faces among schizophrenia patients was highest in Capgras patients, indicating that they showed superior performance in discriminating the newly learned target

from distracters. However, hit rates for familiar faces were lower in Capgras patients as compared to schizophrenia patients without DMS. Our findings are somewhat close to those of Young et al. [27], who reported severe problems in identifying familiar faces in 2 Capgras patients. Their patients differed in their recognition memory performance (1 was within the limits of the control group). Both patients suffered from organic psychosyndrome, which may account for the difference compared to our schizophrenia sample. In contrast to our findings, Breen et al. [46] reported 1 patient with Capgras syndrome in whom visual processing of familiar and unfamiliar faces was intact, but was unable to identify facial expressions. All published studies used different face recognition tasks, differences in the results might therefore also stem from the different tasks.

Our findings within the group of schizophrenia patients fit to the well-established facial memory disturbance in schizophrenia in general [3, 4, 8, 47]. However, the finding is in contrast to the results of Lykouras et al. [26] who reported no differences between patients with and without DMS in the performance in a variety of neuropsychological tasks, including the Benton visual retention task.

Coltheart et al. [48] suggested 2 factors for monothematic delusions, such as Capgras syndrome. The first is the abnormality that results in a delusional belief. The second abnormality forces the person to stick to the delusion against all other opposing evidence and represents a failure in updating cognitive hypotheses. This study aimed to investigate the abnormality that leads to the delusional belief.

The important findings of our study are the various differences between Capgras and Fregoli-type DMS in schizophrenia patients in a modified face recognition memory task. Others have reported neurophysiological differences between schizophrenia patients with and without DMS, i.e. in the auditory P300 [24]. In addition, we demonstrated that the group of patients with DMS is not only heterogeneous in psychopathological symptoms (hypoidentification vs. hyperidentification), but also in the performance of face recognition tasks (reaction time and accuracy). In fact, we suggest a different pathophysiological mechanism in face processing between the 2 groups of DMS. The mechanism, however, still remains unclear. Some authors favour the view that the DMS stem from neuropathological lesions, as Fregoli and Capgras syndromes have been demonstrated in various case reports of patients with different forms of brain damage, mostly in the right hemisphere [17]. In addition, the mechanism for Fregoli and Capgras delusions cannot be one that exclusively leads to either misidentification, since there are reports of patients with both types of DMS [18]. From lesion case studies with similarity in neuropsychiatric tests between Capgras and Fregoli patients, it has been concluded that both DMS types might stem from right frontal damage and the direction of attribution as hypo- or hyperfamiliar depends on motivational aspects [30]. However, brain-damaged patients are fairly different from patients with schizophrenia. In our study, we consider schizophrenia to be the underlying brain pathology, because none of the subjects investigated had any neuroanatomical pathology in the MRI scan. However, the previously mentioned differences in facial recognition challenge the idea of a common underlying pathology for both DMS types in schizophrenia.

The present investigation has some limitations, however. We did not control for attention or working memory impairments in other domains. In addition, the findings concerning the type of DMS were somewhat post hoc results. We did not have the chance to recruit 2 adequately sized and matched patient samples of schizophrenia patients with DMS according to the misidentification types of Capgras [15] or Fregoli [16]. The prevalence of the syndromes, however, limits the number of subjects available.

Furthermore, the study did not aim to investigate the impact of emotional faces, which could be relevant in DMS as well. Nonetheless, we focused on the processing of neutral affect faces in this first step. Our study was not designed to allocate the disturbance in face processing to a failure during encoding or recognition. An optimal study design had to use familiar faces of each subject's environment in contrast to completely unfamiliar faces, but then stimulus material would lack standardization across the study participants – besides the tremendous effort needed to collect individualized stimulation images.

Our task was also quite challenging for the healthy control subjects. This could have contributed to the small effect in comparison to the patients. Some authors consider the dysfunction in face processing in schizophrenia to be related to general neurocognitive impairments or to the influences of drug treatment [1, 49], while others have failed to reproduce these findings [3]. Impaired error monitoring has been suggested to contribute to the face recognition deficit in schizophrenia [50]; however, our task was not designed to control for this possible impact. Psychopathology, as already mentioned, had no impact on the results.

In conclusion, our study showed that face processing impairments in schizophrenia are different according to the clinical presence or absence of a DMS as well as to the type of misidentification. Further, it indicated that different types of delusional misidentifications, i.e. false-positive and false-negative misidentifications may rely on different pathophysiological mechanisms. Future research will help to disentangle the components of face processing and their specific meaning for schizophrenia symptoms in general and, in particular, for distinct types of DMS.

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References

1 Addington J, Addington D: Facial affect recognition and information processing in schizophrenia and bipolar disorder. Schizophr Res 1998;32:171–181.
2 Hall J, Harris JM, Sprengelmeyer R, Sprengelmeyer A, Young AW, Santos IM, Johnstone EC, Lawrie SM: Social cognition and face processing in schizophrenia. Br J Psychiatry 2004;185:169–170.
3 Sachs G, Steger-Wuchse D, Kryspin-Exner I, Gur RC, Katschnig H: Facial recognition deficits and cognition in schizophrenia. Schizophr Res 2004;68:27–35.
4 Gur RE, Nimgaonkar VL, Almasy L, Calkins ME, Ragland JD, Pogue-Geile MF, Kanes S, Blangero J, Gur RC: Neurocognitive endophenotypes in a multiplex multigenerational family study of schizophrenia. Am J Psychiatry 2007;164:813–819.
5 Walther S, Federspiel A, Horn H, Bianchi P, Wiest R, Wirth M, Strik W, Muller TJ: Encoding deficit during face processing within the right fusiform face area in schizophrenia. Psychiatry Res 2009;172:184–191.
6 Archer J, Hay DC, Young AW: Face processing in psychiatric conditions. Br J Clin Psychol 1992;31(part 1):45–61.
7 Kucharska-Pietura K, David AS, Masiak M, Phillips ML: Perception of facial and vocal affect by people with schizophrenia in early and late stages of illness. Br J Psychiatry 2005;187:523–528.
8 Calkins ME, Gur RC, Ragland JD, Gur RE: Face recognition memory deficits and visual object memory performance in patients with schizophrenia and their relatives. Am J Psychiatry 2005;162:1963–1966.
9 Hong LE, Mitchell BD, Avila MT, Adami H, McMahon RP, Thaker GK: Familial aggregation of eye-tracking endophenotypes in families of schizophrenic patients. Arch Gen Psychiatry 2006;63:259–264.
10 Turetsky BI, Calkins ME, Light GA, Olincy A, Radant AD, Swerdlow NR: Neurophysiological endophenotypes of schizophrenia: the viability of selected candidate measures. Schizophr Bull 2007;33:69–94.
11 Haxby JV, Hoffman EA, Gobbini MI: Human neural systems for face recognition and social communication. Biol Psychiatry 2002;51:59–67.
12 Ellis HD, de Pauw KW, Christodoulou GN, Papageorgiou L, Milne AB, Joseph AB: Responses to facial and non-facial stimuli presented tachistoscopically in either or both visual fields by patients with the Capgras delusion and paranoid schizophrenics. J Neurol Neurosurg Psychiatry 1993;56:215–219.
13 Ellis HD, Lewis MB: Capgras delusion: a window on face recognition. Trends Cogn Sci 2001;5:149–156.
14 Ellis HD, Young AW, Quayle AH, De Pauw KW: Reduced autonomic responses to faces in Capgras delusion. Proc Biol Sci 1997;264:1085–1092.
15 Capgras J, Reboul-Lachaux J: L'illusion des sosies dans un delire systematise chronique. Bull Soc Clin Med Ment 1923;11:6–16.
16 Courbon P, Fail G: Illusion de fregoli. Bull Soc Clin Med Ment 1927;15:121–124.
17 Devinsky O: Delusional misidentifications and duplications: right brain lesions, left brain delusions. Neurology 2009;72:80–87.
18 Christodoulou GN, Margariti M, Kontaxakis VP, Christodoulou NG: The delusional misidentification syndromes: strange, fascinating, and instructive. Curr Psychiatry Rep 2009;11:185–189.
19 de Pauw KW: Delusional misidentification: a plea for an agreed terminology and classification. Psychopathology 1994;27:123–129.
20 Anderson DN, Williams E: The delusion of inanimate doubles. Psychopathology 1994;27:220–225.
21 Joseph AB: Observations on the epidemiology of the delusional misidentification syndromes in the Boston metropolitan area: April 1983 – June 1984. Psychopathology 1994;27:150–153.
22 Kirov G, Jones P, Lewis SW: Prevalence of delusional misidentification syndromes. Psychopathology 1994;27:148–149.
23 Phillips ML, David AS: Facial processing in schizophrenia and delusional misidentification: cognitive neuropsychiatric approaches. Schizophr Res 1995;17:109–114.
24 Papageorgiou C, Lykouras L, Alevizos B, Ventouras E, Mourtzouchou P, Uzunoglu N, Christodoulou GN, Rabavilas A: Psychophysiological differences in schizophrenics with and without delusional misidentification syndromes: a P300 study. Prog Neuropsychopharmacol Biol Psychiatry 2005;29:593–601.
25 Edelstyn NM, Oyebode F, Booker E, Humphreys GW: Facial processing and the delusional misidentification syndromes. Cogn Neuropsychiatry 1998;3:299–314.
26 Lykouras L, Typaldou M, Mourtzouchou P, Oulis P, Koutsaftis C, Dokianaki F, Michalopoulou PG, Havaki-Kontaxaki M, Christodoulou C: Neuropsychological relationships in paranoid schizophrenia with and without delusional misidentification syndromes: a comparative study. Prog Neuropsychopharmacol Biol Psychiatry 2008;32:1445–1448.
27 Young AW, Reid I, Wright S, Hellawell DJ: Face-processing impairments and the Capgras delusion. Br J Psychiatry 1993;162:695–698.
28 Christodoulou GN: The syndrome of Capgras. Br J Psychiatry 1977;130:556–564.
29 Feinberg TE, Eaton LA, Roane DM, Giacino JT: Multiple Fregoli delusions after traumatic brain injury. Cortex 1999;35:373–387.
30 Feinberg TE, Keenan JP: Where in the brain is the self? Conscious Cogn 2005;14:661–678.
31 Hudson AJ, Grace GM: Misidentification syndromes related to face specific area in the fusiform gyrus. J Neurol Neurosurg Psychiatry 2000;69:645–648.
32 Christodoulou GN: Delusional hyper-identifications of the Fregoli type: organic pathogenetic contributors. Acta Psychiatr Scand 1976;54:305–314.
33 Edelstyn NM, Drakeford J, Oyebode F, Findlay C: Investigation of conscious recollection, false recognition and delusional misidentification in patients with schizophrenia. Psychopathology 2003;36:312–319.
34 Bruce V, Young A: Understanding face recognition. Br J Psychol 1986;77(part 3):305–327.

35 Ellis HD, Young AW: Accounting for delusional misidentifications. Br J Psychiatry 1990;157:239–248.
36 Breen N, Caine D, Coltheart M: Models of face recognition and delusional misidentification: a critical review. Cogn Neuropsychol 2000;17:55–71.
37 Ellis HD: Delusions: a suitable case for imaging? Int J Psychophysiol 2007;63:146–151.
38 Kay SR, Fiszbein A, Opler LA: The positive and negative syndrome scale (PANSS) for schizophrenia. Schizophr Bull 1987;13:261–276.
39 Rey MJ, Schulz P, Costa C, Dick P, Tissot R: Guidelines for the dosage of neuroleptics. I. Chlorpromazine equivalents of orally administered neuroleptics. Int Clin Psychopharmacol 1989;4:95–104.
40 Woods SW: Chlorpromazine equivalent doses for the newer atypical antipsychotics. J Clin Psychiatry 2003;64:663–667.
41 Steffen T, Müller TJ: Merkmale der Personenverkennungen bei schizophrenen Patienten: Eine explorative Studie. Bern, University of Bern, 2006.
42 Eichenberger B, Müller TJ: Entwicklung und Testung eines computergesteuerten Stimulationsparadigmas zur Erfassung der Verhaltensparameter von schizophrenen Patienten mit Personenverkennung, schizophrenen Patienten ohne Personenverkennung und gesunden Probanden. Bern, University of Bern, 2004.
43 Lehmann C, Mueller T, Federspiel A, Hubl D, Schroth G, Huber O, Strik W, Dierks T: Dissociation between overt and unconscious face processing in fusiform face area. Neuroimage 2004;21:75–83.
44 Snodgrass JG, Corwin J: Pragmatics of measuring recognition memory: applications to dementia and amnesia. J Exp Psychol Gen 1988;117:34–50.
45 Luck D, Montoya A, Menear M, Achim AM, Lal S, Lepage M: Selective pair recognition memory impairment with no response bias in schizophrenia. Psychiatry Res 2009;169:39–42.
46 Breen N, Caine D, Coltheart M: The role of affect and reasoning in a patient with a delusion of misidentification. Cogn Neuropsychiatry 2002;7:113–137.
47 Yoo SS, Choi BG, Juh RH, Park JM, Pae CU, Kim JJ, Lee SJ, Lee C, Paik IH, Lee CU: Working memory processing of facial images in schizophrenia: fMRI investigation. Int J Neurosci 2005;115:351–366.
48 Coltheart M, Langdon R, McKay R: Schizophrenia and monothematic delusions. Schizophr Bull 2007;33:642–647.
49 Whittaker JF, Deakin JF, Tomenson B: Face processing in schizophrenia: defining the deficit. Psychol Med 2001;31:499–507.
50 Silver H, Goodman C, Bilker W, Gur RC, Isakov V, Knoll G, Feldman P: Impaired error monitoring contributes to face recognition deficit in schizophrenia patients. Schizophr Res 2006;85:151–161.