



# Recognition memory for faces in schizophrenia patients and their first-degree relatives<sup>☆</sup>

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## Abstract

It has consistently been shown that schizophrenia patients are impaired in recognition memory for faces. However, studies have not examined the specificity of this deficit relative to other cognitive functions nor the relationship between this deficit and particular schizophrenia symptoms. In addition, no studies have examined recognition memory for faces in unaffected biological relatives of schizophrenia patients who likely share some of the genetic diathesis for this disorder without presenting the potential confounds of mentally ill study samples. The Faces subtests from the Wechsler Memory Scale—Third Edition were used to evaluate recognition memory for faces in 39 schizophrenia patients, 33 of their first-degree relatives and 56 normal controls. Both schizophrenia patients and their relatives were impaired, relative to control participants, in recognition memory for faces after partialing out group differences in spatial attention or verbal memory. Further, recognition memory for faces was associated with positive symptoms in the schizophrenia group and schizotypal personality traits in the relative group. These findings may have important implications for reducing etiological heterogeneity among schizophrenia populations, identifying disorder susceptibility among their relatives and furthering understanding of disorder etiology.

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## 1. Introduction

Ever since it was observed clinically that individuals, despite being otherwise cognitively intact, could fail to recognize familiar faces (i.e. prosopagnosia) there has been interest in face recognition as a discrete mnemonic ability. Evolutionarily, it would make sense for face memory to be functionally and neuroanatomically distinct from other memory abilities, as the discrimination between familiar or unfamiliar, friend or enemy, related or unrelated is critical for survival. Therefore, there may have been selective pressure that led to the development of unique neural mechanisms for face memory [11]. Support for neural “hardwiring” of face analysis and recognition comes from the cross-cultural recognition of typical facial expressions (e.g. [12]), as well as the early devel-

opmental capacity of infants to recognize familiar faces [20].

Three lines of evidence have contributed to the identification of discrete neural circuits subserving face recognition ability: case studies of humans with focal brain lesions, neuroimaging research with neurologically intact individuals and single-unit recordings with nonhuman primates. In brain damaged adults, a double dissociation has been identified whereby patients may demonstrate impaired face recognition but intact object recognition (e.g. [13]) or impaired object recognition but intact face recognition (e.g. [35]), indicating separate processing centers for faces and objects. An early debate emanating from neuropsychological studies surrounded the hemispheric laterality of face recognition ability. Both autopsy results and CT-scan studies of individuals with prosopagnosia now suggest that the disability can be produced by a right hemisphere lesion alone [3]. Reviews of the neuropsychological literature are consistent with a right hemisphere advantage for face recognition [44]. Clinical observation supports the finding that patients with posterior right hemispheric disease show a disability in recognition of *unfamiliar* faces [3], suggesting that failure to recognize

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familiar faces and unfamiliar faces share overlapping neural substrates. Taken together, neuropsychological findings suggest that patients with damage to the posterior right hemisphere, most commonly the ventral occipitotemporal cortex, demonstrate selective loss of the ability to recognize faces that appears to be irrespective of face familiarity.

Findings from studies of brain damaged individuals have been supplemented with functional neuroimaging research that allows for greater spatial localization of face recognition ability. Consistent with case studies of individuals with brain lesions, neuroimaging studies have revealed a predominantly right-sided pattern of activation for memory for unfamiliar faces [27,28,32]. More specifically, regions of the fusiform gyrus, located within the ventral occipitotemporal cortex, have been found to be more active during face than object recognition (e.g. [25,40,41,49]). For example, regions of the fusiform gyrus show greater activation during the viewing of whole faces than of scrambled faces [40], letterstrings [41] or textures [41]. In an effort to test alternative interpretations of neuroimaging findings suggestive of neural specificity for faces, Kanwisher et al. [25] conducted a systematic set of experiments. These investigators found a region in the right fusiform gyrus that was more active during face viewing than object viewing. In addition, this area responded more strongly to intact two-tone faces than scrambled versions of the same faces, photographs of faces than houses, and photographs of human faces than hands, thus ruling out alternative interpretations of this activation pattern that hinge on luminance differences, exemplars of the same category or presence of any body part [25]. In addition to activation in the fusiform gyrus, half of the subjects in these experiments also showed activation for faces, compared to objects, in the region of the middle temporal gyrus/superior temporal sulcus [25].

Most of the studies reviewed thus far demonstrate involvement of the fusiform gyrus in face-processing tasks independent of mnemonic events such as encoding and retrieval. In a study examining activation patterns for face and word recognition memory, Kim et al. [28] found face recognition to be lateralized to the right hemisphere and located in an area including the right lingual and fusiform gyri. Similarly, Simons et al. [48] found recognition memory for faces, in comparison to objects, led to activation of the anterior temporal lobes (more extensively on the right), a region of the right parahippocampal gyrus, the right posterior fusiform gyrus and the anterior prefrontal cortex. This finding of frontal involvement in face recognition ability has been reported by other researchers [27,28,32,33]. Neuroimaging studies of face recognition memory also suggest that areas of activation are virtually the same for familiar or unfamiliar faces [28].

Single-unit recordings in nonhuman primates allow for even greater spatial resolution than neuroimaging studies for identifying neural substrates involved with face processing and memory. Rolls [45] reported on a series of studies of single cell recordings in the rhesus macaque that indicated

populations of neurons in the inferior temporal lobe that are specialized to respond to faces, suggesting involvement of posterior visual association cortex. Subsequently, other research groups have located cells in the temporal cortex in the macaque brain that are selectively responsive to faces, including the lateral and ventral surfaces of the inferior temporal cortex and the upper bank, lower bank and fundus of the superior temporal sulcus [37]. These investigators also proposed that properties of some of these cells suggest a role in the recognition of familiar individuals [37].

While various methodological factors make it difficult to make direct comparisons among findings from neuropsychological studies of brain injured adults, neuroimaging studies with neurologically intact individuals and single-unit recording studies in nonhuman primates, all three areas of research converge in identifying a specific neural circuit associated with face recognition. This evidence suggests that the right inferior occipitotemporal area, believed to be an association area involved in the transmission of visual information to mesial-temporal areas involved with memory, is critical to the recognition of faces. Secondary areas (e.g. frontal) likely support this ability.

Neuropsychological deficits in schizophrenia, including memory dysfunction, may provide important clues to underlying pathophysiology. Schizophrenia is a severe, persistent form of psychopathology that is associated with brain abnormalities. Neuropsychological tests have been used both to elucidate cognitive deficits that may be secondary to these abnormalities, as well as to suggest hypotheses about corresponding sites of neuropathology. Schizophrenia patients have been found to be impaired on a range of neuropsychological tasks (e.g. [8,16,36]), with some investigators suggesting the breadth of deficits reflects pathology in most brain areas. A recent investigation of first-episode schizophrenia patients, using a comprehensive neuropsychological battery, found a large generalized deficit in schizophrenia patients with superimposed deficits in memory and executive functions [5]. This finding was interpreted as support for dysfunction in an integrated frontal-temporal system in schizophrenia.

It has consistently been shown that patients with schizophrenia, when presented with faces portraying different emotions, have greater difficulty identifying the displayed emotion than normal controls (e.g. [14]). This finding has led investigators to examine component processes of emotion identification, including the ways in which schizophrenia patients scan faces, match facial expressions with emotion labels and recognize faces they have seen previously. Subsequently, multiple studies have found that schizophrenia patients are impaired in recognition memory for faces (e.g. [19,22]). However, it is unclear whether this represents a specific deficit in face memory or a more generalized deficit in memory function. For example, Hellewell et al. [22] reported that schizophrenia patients were impaired on the Warrington Recognition Memory Test for Faces; however, these patients also demonstrated impair-

ment on recognition memory tests for words and patterns (black-and-white wallpaper designs). Further, studies have not examined the relationship between deficits in recognition memory for faces and symptomatology in schizophrenia patients. Relationships among these deficits and certain schizophrenia symptoms, for example positive or negative symptoms, may allow for subgrouping of schizophrenia patients that provide insight to disorder etiology.

Reports of cognitive deficits in schizophrenia are open to some criticism because multiple factors associated with chronic mental illness (e.g. active psychotic symptoms, medication effects or lower education), as well as basic neuropathology, could influence performance and are difficult to disentangle. If cognitive deficits reflect an underlying genetic diathesis for schizophrenia, it is beneficial to study first-degree relatives because they share, on average, some of the genetic diathesis for schizophrenia without presenting the aforementioned complications. The investigation of first-degree relatives may also elucidate indicators of the genetic predisposition for schizophrenia and assist in the selection of family members for use in genetic linkage studies. Studies of the first-degree relatives of schizophrenia patients have indicated many neuropsychological deficits similar to those seen in their affected relatives [31]. However, we are unaware of any studies that have examined face memory in the biological relatives of schizophrenia patients.

Increased rates of schizotypal traits have been documented in the first-degree biological relatives of schizophrenia patients since the turn of the century (e.g. [30], originally published in 1909). It may be that relatives with such traits exhibit face recognition deficits similar to schizophrenia patients, contributing to the odd interpersonal behavior associated with schizotypy. Poreh et al. [39] found that students who scored higher on the Chapman schizotypy scales (Perceptual Aberration (PER), Magical Ideation (MAG) and Schizotypal Personality (STA) scales) made significantly more errors on both a facial affect recognition task and a face recognition task. Further analyses revealed that group differences in affect recognition disappeared after controlling for face recognition, suggesting that the effect was being driven by poor face recognition, rather than deficient emotion identification.

The current study sought to replicate the finding of impaired recognition memory for faces among schizophrenia patients using a common clinical measure of face memory that has not been reported in the research literature, the Faces subtest from the Wechsler Memory Scale—Third Edition (WMS-III) [51]. Provided that these patients proved to be impaired on this task, we aimed to investigate the task specificity of this deficit, as well as its relationship to schizophrenia symptoms and severity. Further, we sought to determine whether first-degree relatives show similar deficits to their affected relatives on the WMS-III Faces and to investigate the relationship between this deficit and different dimensions of schizotypy. We hypothesized that both schizophrenia patients and their relatives would be impaired on this WMS-III

task, relative to control participants. In the relative sample, we also predicted that face recognition ability would be negatively related to the presence of schizotypal traits, based on the findings in a schizotypal community sample. In addition, if these neuropsychological and personality measures are tapping the same diathesis, they should identify the same relatives (i.e. those with the greatest genetic vulnerability). The investigation of the relationship between face recognition ability and symptomatology in the schizophrenia group was largely exploratory in nature as the research literature did not provide enough evidence to warrant specific predictions.

## 2. Method

### 2.1. Participants

Thirty-nine schizophrenia inpatients were recruited from acute-care units of a regional metropolitan hospital. All patients met Diagnostic and Statistical Manual for Mental Disorders—Fourth Edition (DSM-IV) [2] criteria for schizophrenia, based on diagnostic interviewing using the Structured Clinical Interview for DSM-IV (SCID-IV) [15] and chart review. The patients were between the ages of 18 and 65, spoke English fluently, were not currently abusing alcohol or drugs, had not recently undergone ECT treatment and had no history of neurological disease, systemic disease known to involve CNS functioning, clinically significant head injury or mental retardation. Medical history relevant to these inclusion criteria was determined by review of medical records, supplemented by a semi-structured health history screening interview administered to the patient, and consultation with treating medical staff. Substance use status was determined by the SCID. In addition, patients were required to have a negative urine toxicology screen upon hospital admission or a minimum of 14 days inpatient stay between a positive urine toxicology screen date and the testing date.

Fifty-six normal control participants were recruited from the community via advertisement posters placed at regional hospital clinics and community vocational/technical schools, as well as through announcements at local churches. Control participants were excluded for the same general and medical criteria as the patients, as determined by health history interview. They were additionally excluded if they had any lifetime diagnoses of major affective, psychotic or substance dependence disorder, as determined by the SCID-IV, and if they or a first-degree biological relative had ever sought mental health treatment.

Thirty-three first-degree biological relatives from 18 families of schizophrenia patients, were recruited through written correspondence followed by phone contact. Relatives for the current investigation were excluded for the same general and medical criteria as the schizophrenia patients, as determined by health history interview. They were also interviewed

using the SCID-IV. Analyses were conducted both with a subgroup of relatives that met the rigorous diagnostic inclusion criteria established for the normal control participants and with a larger group that only excluded individuals with lifetime diagnoses of a psychotic disorder, current major affective disorder or current substance use disorder (i.e. included relatives who met lifetime, but not current, diagnoses of major affective disorder or substance dependence disorder).

To ensure that participants could see the stimuli presented in the study, the visual acuity of all participants was assessed by standard Snellen examination. Where necessary, participants wore corrective lenses during neuropsychological testing, and all participants with acuity at or worse than 20/100 were required to wear lenses that corrected to 20/30 or better.

## 2.2. Procedures

For all participants, diagnostic information was obtained from interviews using the SCID-IV (Modules A–E) and chart reviews. To confirm diagnostic assignments, a consensus team composed of advanced doctoral level students and clinical psychologists reviewed SCID, chart data and, when necessary, audio recordings of interviews. A reliability study performed on a group composed of 50 patients with diagnoses of schizophrenia, schizoaffective and psychotic mood disorders yielded a high level of inter-rater reliability between the diagnosis offered by the consensus team and the original interviewer ( $\kappa = 0.84$ ). All diagnostic and clinical ratings were made blind to performance on neuropsychological tasks.

A subset of schizophrenia patients ( $N = 31$ ) was interviewed using the Positive and Negative Syndrome Scale (PANSS) [26], a measure added to the assessment battery after the study had begun. These questions, which evaluate the presence of psychopathology in the preceding month, were asked during diagnostic interviewing with the SCID-IV. The PANSS consists of 30 items that are rated on a seven-point scale, from symptom not present at all to symptom present to an extreme degree. The questions comprise three scales: negative symptoms (7 items), positive symptoms (7 items) and general psychopathology (16 items).

Control participants and relatives were administered the Schizotypal Personality Questionnaire (SPQ) [42], which is a self-report measure based on DSM-III-R [1] criteria for schizotypal personality disorder (SPD). Schizophrenia patients were not administered the SPQ because schizotypal personality features are generally viewed as subsyndromal to schizophrenia as exemplified by DSM-IV specifically precluding the diagnosis of SPD in individuals currently diagnosed with schizophrenia. The SPQ assesses the nine major criterial features of SPD in DSM-III-R that contribute to three factors, as revealed by confirmatory factor analysis: cognitive-perceptual, social-interpersonal and disorganization [43]. The SPQ was modified to include 15

items from the L (lie) Scale and 30 items from the K (defensiveness) Scale of the Minnesota Multiphasic Personality Interview—Second Edition (MMPI-2) [6] in order to assess under-reporting of symptoms and response bias. Seven items from the Jackson Infrequency Scale were used to detect random responding [24]. These items were interspersed among the 74 SPQ items to form a 126 item true/false instrument. When responding to the items, participants were asked to refrain from considering episodes when they were under the influence of drugs or alcohol and from periods when they were just falling asleep or awakening.

All subjects were administered the Faces subtest from the WMS-III as part of a larger study investigating psychophysiological, neuropsychological and behavioral indices of risk for schizophrenia. The Faces subtest was added to the neuropsychological battery of the ongoing study in order to further characterize temporal lobe dysfunction in schizophrenia. In Faces I, the examinee is presented with a series of 24 photographs of neutral faces, one at a time, and is asked to remember each one. Immediately following this presentation, the examinee is presented with a second series of 48 faces and asked to indicate whether the face is one just previously seen or not; all target faces appear in the second series. The examinee is then instructed to try to remember the original faces for later testing. Thirty minutes after first presentation, during which time the examinee is engaged in unrelated neuropsychological tasks, the Faces II subtest is administered. In Faces II, the examinee is presented with a third series of 48 faces, the 24 target faces and a second set of 24 distractor faces, and is asked to indicate which faces were in the original set.

In order to investigate the task specificity of deficits in memory for faces, the performance of all participants on the Forward Spatial Span task from the WMS-III [51] and the Rey Auditory Verbal Learning Test (RAVLT) was considered. The Forward Spatial Span task includes a Spatial Span Board consisting of 10 blue cubes fastened to a white plastic board. The examiner taps specified sequences of blocks, of random location, at a rate of one per second. The examinee is required to repeat back the block taps in the same order. The number of blocks increases by one until the examinee consecutively fails two trials of the same length. The RAVLT is a measure of verbal list learning and memory. The examiner verbally presents five trials of the same list of 15 unrelated words. The examinee is required to freely recall as many words as possible following each of the five presentations, as well as after a distractor list and a 60 min delay. A delayed recognition component of the RAVLT was also administered. For this task, 50 words are presented orally and the examinee indicates, by responding yes or no, whether each word was from the original list of 15 words.

In order to obtain an estimate of general cognitive functioning, all participants were administered the Block Design subtest, which requires the examinee to replicate models or pictures of designs with blocks, and the Information subtest, which requires the examinee to respond orally to a series

of questions assessing general knowledge. Both of these subtests are from the Wechsler Adult Intelligence Scale—Revised (WAIS-R) [50]. All participants gave written informed consent and were compensated for their participation.

### 3. Results

#### 3.1. Demographics

Table 1 presents the means and standard deviations for all relevant demographic, neuropsychological and clinical variables. Effect sizes ( $d$ ) were calculated using the sample size-weighted pooled within-group standard deviation to provide estimates of group differences independent of sample size [23]. A  $\chi^2$ -analysis, comparing the proportion of males and females in each group, indicated that the groups were balanced for gender ( $\chi^2 = 3.74$ , d.f. = 2,  $P = 0.15$ ). Face recognition (Faces I and II, separately) by gender  $t$ -tests were computed within each group separately and none of these gender comparisons was significant. Gender did not correlate with performance on either Faces I or II in any of the groups ( $-0.04 < r < 0.26$ ,  $0.12 < P < 0.83$ ). Given that gender ratios did not differ between groups and gender was not related to performance on WMS-III Faces, it was not considered as a factor in the remaining analyses. The groups differed significantly in age ( $F = 8.56$ , d.f. = 2, 125,  $P < 0.001$ ), with post-hoc tests<sup>1</sup> indicating that the relatives were significantly older than the control participants and patients, who did not differ from each other. Education level differed significantly among groups ( $F = 21.19$ , d.f. = 2, 125,  $P < 0.001$ ), with post-hoc tests indicating that control participants had more years of education than the relatives and patients who did not differ from each other. IQ was prorated using scores on the Block Design and Information subtests according to the procedure developed by Tellegen and Briggs as cited in [47]. Four patients, but no relatives or controls, were excluded for having an IQ < 70, a defining feature of mental retardation. There was a significant difference among groups in IQ ( $F = 18.15$ , d.f. = 2, 125,  $P < 0.001$ ), with post-hoc tests indicating that schizophrenia patients had a lower IQ than relatives who had a lower IQ than controls.

#### 3.2. Face recognition

##### 3.2.1. Preliminary analyses

Visual inspection of the Faces record forms suggests that all participants put forth adequate effort with none of the participants utilizing a pure “yea” or “nay” saying response style. Fig. 1 shows the mean group differences in Faces I and II performance, as measured by the number of faces out of 48 that the examinee correctly recognized. A repeated measures analysis of variance (ANOVA), with Faces (I and

II) as the within subject factor and group (schizophrenia, relative and control) as the between subjects factor, failed to reveal a significant interaction between faces performance and group membership ( $F = 0.55$ , d.f. = 2,  $P = 0.58$ ) or a main effect for Faces task ( $F = 2.75$ , d.f. = 1,  $P = 0.10$ ). A main effect for group membership was revealed ( $F = 18.31$ , d.f. = 2,  $P < 0.001$ ). Appropriate follow-up one way ANOVAs and post-hoc tests were performed.<sup>2</sup>

**3.2.1.1. Faces I.** There was a significant difference among groups ( $F = 16.91$ , d.f. = 2, 125,  $P < 0.001$ ), with post-hoc tests indicating that schizophrenia patients recognized fewer faces than relatives who recognized fewer faces than controls on Faces I. When analyses were repeated, applying the rigorous diagnostic inclusion criteria established with the control participants to the relative sample (eight relatives were omitted from analysis), the group differences on Faces I remained statistically significant. In order to show that the significant differences between groups in face recognition were not attributable to group differences in age, education, or IQ, separate analyses of covariance (ANCOVAs) were computed using age, education and IQ as covariates.<sup>3</sup> The group differences on Faces I remained statistically significant.

**3.2.1.2. Faces II.** There was a significant difference among groups on Faces II ( $F = 14.37$ , d.f. = 2, 125,  $P < 0.001$ ), with post-hoc tests indicating that schizophrenia patients recognized fewer faces than relatives who recognized fewer faces than controls. An ANCOVA using age as a covariate indicated that group differences between relatives and controls on Faces II disappeared after controlling for age ( $P = 0.17$ ), suggesting that this difference on Faces II may be attributable to group differences in age. After adjusting for education in an ANCOVA, group differences between relatives and controls on Faces II also became nonsignificant ( $P = 0.08$ ). IQ was not a significant covariate and group findings remained significant after

<sup>2</sup> Given that relatives were selected for their genetic relationship to schizophrenia probands and some relatives came from the same families, some of the observations included in statistical analyses were not independent. In order to correct for violating the statistical assumption of independence, the degrees of freedom used to derive  $P$ -values in all  $t$ -tests, ANOVAs, ANCOVAs and post-hoc comparisons were adjusted by replacing the number of individuals with the number of families. When this conservative statistical procedure was used, all of the previously significant analyses remained significant ( $P < 0.05$ ).

<sup>3</sup> There is an on-going debate concerning the appropriateness of statistically adjusting for IQ differences between schizophrenia patients and control groups using ANCOVA [34]. The main criticism involves the possibility that adjusting for IQ may remove meaningful variance, resulting in either attenuation of real group differences or creation of spurious group differences on the dependent variable of interest. In this study, we found group differences on Faces I, both before and after adjusting for IQ, precluding the possibility that this group difference represents a statistical artifact. Further, the differential deficit criticism at the heart of this argument does not apply to well relative groups who lack a generalized cognitive deficit.

<sup>1</sup> All post-hoc tests used Fisher's least significant difference (LSD),  $P < 0.05$ .

Table 1  
Demographic, neuropsychological and clinical information by group

Variable	Schizophrenia ( <i>N</i> = 39 <sup>a</sup> )	Relative ( <i>N</i> = 33 <sup>a</sup> )	Control ( <i>N</i> = 56)	Effect size	
				S v. C	R v. C
<b>Demographics</b>					
Gender ratio (%male)	60.0	51.5	39.3	–	–
Age (years)	37.8 ± 8.2	43.9 ± 10.8	33.9 ± 12.9	0.35	0.82
Education (years)	13.1 ± 1.8	13.9 ± 2.0	15.9 ± 2.5	–1.25	–0.86
<b>Neuropsychological test scores</b>					
Faces I (number of faces correctly recognized)	34.6 ± 4.9	37.3 ± 5.1	40.2 ± 4.1	–1.26	–0.65
Faces II (number of faces correctly recognized)	35.4 ± 5.2	38.0 ± 3.9	40.3 ± 4.1	–1.07	–0.57
Faces percent retention	102.3 ± 11.8	102.9 ± 10.4	100.5 ± 8.0	0.18	0.27
Spatial span forward (number of blocks)	5.5 ± 0.9	6.2 ± 1.1	6.1 ± 0.9	–0.68	0.07
RAVLT: average number of words recalled on trials 1–5	7.5 ± 2.1 <sup>b</sup>	9.9 ± 1.8	11.3 ± 1.7	–2.02	–0.79
RAVLT: number of words recalled after 60s delay	6.6 ± 3.4 <sup>c</sup>	9.3 ± 3.3	–	–1.96	–1.01
RAVLT: number of words recognized after 60s delay	12.2 ± 2.7 <sup>c</sup>	13.5 ± 1.6 <sup>d</sup>	14.4 ± 0.9	–1.21	–0.74
Prorated IQ	93.8 ± 13.4	102.8 ± 12.5	111.5 ± 15.4	–1.21	–0.60
<b>Clinical scales</b>					
PANSS ( <i>N</i> = 31)	–	–	–	–	–
General	32.3 ± 6.8	–	–	–	–
Positive	21.2 ± 5.9	–	–	–	–
Negative	14.2 ± 4.3	–	–	–	–
SCID GAF ( <i>N</i> = 36)	27.2 ± 12.2	–	–	–	–

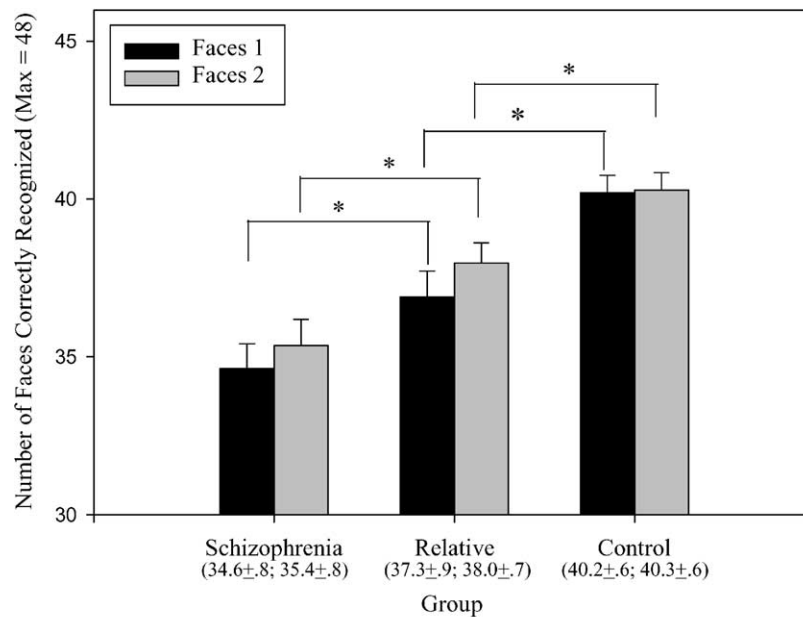
Data are presented as means plus or minus the S.D., unless otherwise indicated. Dashes indicate the scale was not employed with the participant group. S v. C: effect size of mean difference between schizophrenia patients and controls; R v. C: effect size of mean difference between relatives and controls; RAVLT: Rey Auditory Verbal Learning Test; PANSS: Positive and Negative Syndrome Scale; SCID GAF: Structured Clinical Interview for DSM-IV, Global Assessment of Functioning.

<sup>a</sup> Sample size unless otherwise noted.

<sup>b</sup> *N* = 38.

<sup>c</sup> *N* = 37.

<sup>d</sup> *N* = 32.



\* Brackets depict significant differences between groups as determined by Least Significant Difference (LSD) post hoc analyses ( $p < .05$ )

Fig. 1. Faces I and II performance by group (means and S.E.) for schizophrenia patients, relatives and controls as a function of the number of faces correctly recognized out of 48. Groups differ significantly on both Faces I and II using these raw scores, with schizophrenia patients recognizing fewer faces than relatives who recognized fewer faces than controls.

adjusting for IQ in an ANCOVA. All groups performed better on Faces II than I as indicated by the mean percent retention (i.e. number of faces correctly recalled on Faces II as a percentage of faces correctly recalled on Faces I), with no significant difference between groups ( $F = 0.68$ ,  $d.f. = 2, 125$ ,  $P = 0.51$ ). This unpredicted finding will be addressed in the discussion section.

### 3.3. Face recognition and psychopathology

In order to investigate the relationship between face recognition and specific schizophrenia symptomatology, correlations among the Faces subtests and the PANSS scales were examined. Within the schizophrenia group, there were no significant correlations between Faces I or II and the negative or general PANSS scales. However, the correlation between Faces I and the positive PANSS scale suggested a trend ( $r = -0.32$ ,  $P = 0.079$ ), with higher level of positive symptoms associated with poorer face recognition. The correlation between Faces II and the positive PANSS scale was in the same direction and significant ( $r = -0.36$ ,  $P < 0.05$ ). Within the schizophrenia group, neither performance on Faces I nor Faces II was significantly correlated with the Global Assessment of Functioning (GAF) Scale from the SCID-IV ( $0.00 < r < 0.02$ ,  $0.93 < P < 0.99$ ) nor the general PANSS scale ( $-0.23 < r < -0.21$ ,  $0.22 < P < 0.26$ ), suggesting that performance on these tasks was not merely an index of impairment in functioning or severity of psychopathology, respectively.

Our research team has previously shown using a larger sample, partially overlapping with this one, that there is a significant difference between relatives and control participants in schizotypal symptoms as measured by the SPQ [7]. For all but one control, for whom data had to be excluded due to failure to follow instructions (the subject responded both true and false to multiple items), SPQ data were available. The  $t$ -tests failed to reveal significant differences between relatives and controls on any of the validity scales (i.e. MMPI-2 L and K scales, Jackson Infrequency Scale). Given that the groups did not differ in response biases or random responding, all available SPQ scores were considered. Fig. 2 shows the correlation between Faces I and the total SPQ score in the relative group. There was a significant correlation between face recognition on Faces I and total SPQ score ( $r = -0.48$ ,  $P = 0.005$ ), the cognitive-perceptual factor ( $r = -0.41$ ,  $P = 0.017$ ), the social-interpersonal factor ( $r = -0.40$ ,  $P = 0.02$ ) and the disorganization factor ( $r = -0.47$ ,  $P = 0.005$ ). Raine [42] has reported that 55% of individuals in their community sample who scored in the top 10% of the SPQ met criteria for DSM-III-R schizotypal personality disorder as determined by the SCID. Moreover, it has been suggested that individuals with high SPQ scores may evidence cognitive deficits similar to those observed in schizophrenia patients (i.e. attentional processes [21]). Thus, in order to further investigate the relationship between SPQ score and Faces I performance, the relative group was

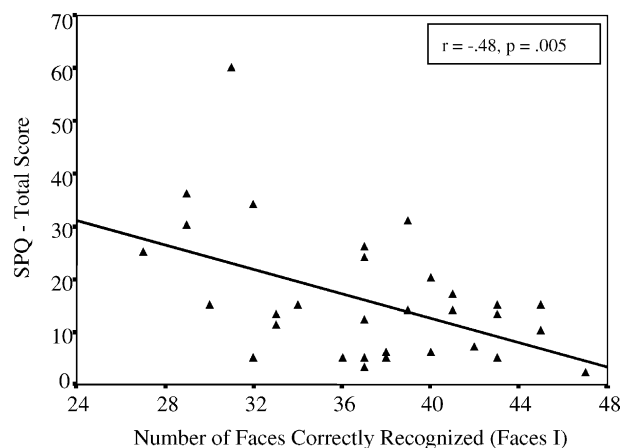


Fig. 2. Schizotypal personality questionnaire (SPQ) total score and Faces I (scatter plot with fitted line) for relatives. The correlation between SPQ total score and number of faces recognized on Faces I is significant, with higher scores on the SPQ associated with fewer recognized faces.

divided into two groups: relatives ( $N = 8$ ) whose scores fell within the highest 10% of control group scores (i.e. relatives endorsing the most SPQ items) and relatives ( $N = 25$ ) whose scores fell below this threshold. A  $t$ -test between the two groups revealed that the relative group endorsing more SPQ symptoms had significantly poorer face recognition on Faces I ( $t = 3.47$ ,  $d.f. = 31$ ,  $P < 0.002$ , Cohen's  $d = -1.41$ ). There were no significant correlations between face recognition on Faces II and total SPQ score or any of its component factors. Within the control group, there were no significant correlations between face recognition on Faces I or II and total SPQ score or any of its component factors.

### 3.4. Specificity of face recognition deficit

All groups were administered the Forward Spatial Span task from the WMS-III as a measure of simple visual attention. There was a significant difference among groups on the Forward Spatial Span task ( $F = 6.14$ ,  $d.f. = 2, 125$ ,  $P < 0.003$ ), with post-hoc tests indicating that schizophrenia patients had a shorter forward spatial span than relatives and controls who did not differ from each other. All groups were administered the RAVLT. The mean number of words recalled on the first five trials, as well as the number of words recalled and recognized after a 60 min delay were investigated as measures of verbal memory. There were significant differences between groups on five trial mean recall ( $F = 47.01$ ,  $d.f. = 2, 124$ ,  $P < 0.001$ ), delayed recall ( $F = 39.54$ ,  $d.f. = 2, 123$ ,  $P < 0.001$ ) and delayed recognition ( $F = 17.42$ ,  $d.f. = 2, 122$ ,  $P < 0.001$ ), with post-hoc tests indicating that schizophrenia patients identified fewer words than relatives who identified fewer words than controls, for all three variables. In order to show that the significant differences between groups in face recognition were not attributable to group differences in nonspecific aspects of spatial attention or to differences

in verbal memory, separate ANCOVAs were computed using forward spatial span and all three RAVLT variables as covariates. All group differences on Faces I remained statistically significant. Group differences on Faces II were statistically significant when adjusting for forward spatial span or RAVLT delayed recognition but were reduced to a trend when controlling for RAVLT five trial mean recall or delayed recall ( $P < 0.092$  and  $P < 0.097$ , respectively).<sup>4</sup>

#### 4. Discussion

Compared to control participants, schizophrenia patients were impaired in both immediate and delayed recognition memory for faces. These findings persisted when group differences in age, education and IQ were partialled out. While findings of impaired recognition memory for faces in schizophrenia patients represent a replication of previous findings, this is the first report in the research literature using WMS-III Faces, a common clinical neuropsychological measure that is readily available and easily administered.

To our knowledge, this is also the first report that demonstrated that unaffected first-degree biological relatives of schizophrenia patients were impaired in recognition memory for faces. Compared to control participants, these relatives were impaired in immediate recognition memory for faces and group differences were upheld after partialling out the effects of age, education and IQ.

However, group differences between relatives and controls on the delayed recognition task were reduced to a trend after partialling out the effects of age or education. Although this latter finding is not in keeping with our predictions, it is not completely unexpected given some of the WMS-III face's task characteristics. Whereas people typically do worse on delayed memory tasks in comparison to immediate memory tasks, presumably indicating the decay of memory traces over time, here all three groups performed as well or

better on the delayed recognition task. The WMS-III manual suggests similar findings in their normative sample (e.g. age-normed scores can decrease for individuals recognizing the same number of faces on Faces I and II, indicating that some individuals in the normative sample improved performance on the delayed task). While we do not have the data available to determine why this phenomenon occurs with this task, two possibilities seem likely. First, it could be that the recognition format of the task, which requires a second exposure to target stimuli during the immediate recognition task, facilitates memory consolidation and/or retrieval for the delayed recognition task. Alternatively, it may be that the set of distractor faces used in the delayed task is easier to discern as novel than the distractors in the immediate task. Support for the first hypothesis comes from a study by Grusser et al. [20] who demonstrated that a second exposure to stimuli during testing negated the decrease in performance associated with the delay period for stimuli presented only one time. In either case, it is difficult to interpret the failure to find significant group differences between relatives and controls on the delayed recognition task as this task did not perform as expected (i.e. with individuals demonstrating a decay in recognition memory over time).

Investigation of the relationship between recognition memory for faces and psychopathology revealed important associations. In the schizophrenia sample, there was a significant correlation between delayed recognition memory for faces and the positive scale of the PANSS, and a trend in the same direction for immediate recognition memory for faces. Bearing in mind that patients were tested during an acute episode, this correlation indicates that greater positive schizophrenia symptomatology was associated with poorer face memory performance. The significant correlation of face memory with positive symptoms, in the absence of a significant correlation with negative symptoms, is theoretically explicable [10,52]. Positive symptoms are more traditionally associated with impaired temporal lobe function than are negative symptoms, as demonstrated by the presence of hallucinations in individuals with temporal lobe epilepsy (e.g. [4]), and temporal lobe functioning is intimately involved in memory function in general, as well as face memory. Therefore, performance on tasks assessing recognition memory for faces may hold promise as a tool for creating neuropsychologically meaningful subgroups among individuals diagnosed with schizophrenia (e.g. those patients with greater temporal lobe involvement).

Among the relative sample, there were substantial correlations between self-reported schizotypal personality characteristics and recognition memory for faces. Total SPQ scores, as well as scores on all three of the factor scores, correlated negatively with immediate recognition memory for faces. This suggests that schizotypal characteristics in general, rather than specific trait subtypes, correlate with a face recognition deficit. Although additional evidence is clearly required to confirm a causal relationship (e.g. [29]), it is possible that neuropathology compromising the

<sup>4</sup> Faces I ANCOVA findings: Forward Spatial Span as a covariate (corrected model— $F = 11.61$ , d.f. = 3,  $P < 0.001$ ; covariate— $F = 1.01$ , d.f. = 1,  $P < 0.32$ ; group— $F = 17.31$ , d.f. = 2,  $P < 0.001$ ). RAVLT five trial mean as a covariate (corrected model— $F = 12.99$ , d.f. = 3,  $P < 0.001$ ; covariate— $F = 5.96$ , d.f. = 1,  $P < 0.02$ , group— $F = 4.03$ , d.f. = 2,  $P < 0.02$ ). RAVLT delayed recall as a covariate (corrected model— $F = 13.29$ , d.f. = 3,  $P < 0.001$ ; covariate— $F = 8.29$ , d.f. = 1,  $P < 0.005$ ; group— $F = 3.36$ , d.f. = 2,  $P < 0.038$ ). RAVLT delayed recognition as a covariate (corrected model— $F = 9.95$ , d.f. = 3,  $P < 0.001$ ; covariate— $F = 0.23$ , d.f. = 1,  $P < 0.63$ ; group— $F = 10.56$ , d.f. = 2,  $P < 0.001$ ). Faces II ANCOVA findings: Forward Spatial Span as a covariate (corrected model— $F = 9.74$ , d.f. = 3,  $P < 0.001$ ; covariate— $F = 0.58$ , d.f. = 1,  $P < 0.45$ ; group— $F = 14.41$ , d.f. = 2,  $P < 0.001$ ). RAVLT five trial mean as a covariate (corrected model— $F = 12.37$ , d.f. = 3,  $P < 0.001$ ; covariate— $F = 8.27$ , d.f. = 1,  $P < 0.005$ , group— $F = 2.44$ , d.f. = 2,  $P < 0.092$ ). RAVLT delayed recall as a covariate (corrected model— $F = 11.62$ , d.f. = 3,  $P < 0.001$ ; covariate— $F = 8.49$ , d.f. = 1,  $P < 0.004$ ; group— $F = 2.38$ , d.f. = 2,  $P < 0.097$ ). RAVLT delayed recognition as a covariate (corrected model— $F = 8.36$ , d.f. = 3,  $P < 0.001$ ; covariate— $F = 0.36$ , d.f. = 1,  $P < 0.55$ ; group— $F = 8.46$ , d.f. = 2,  $P < 0.001$ ).

ability to process and remember face stimuli may lead to mis-attributions about other people's intentions, causing individuals to act in suspicious or otherwise deviant ways. All these behaviors are hallmarks of schizotypy. A similar correlation of deficits has been observed in children with nonverbal learning disabilities (NVLD) who often show deficits not only in visuospatial abilities and nonverbal memory, but also disturbances in interpersonal skills as exemplified by poor social perception, judgment and interactive skills [18]. These children often have difficulty maintaining friendships, are withdrawn and may be described as odd or even bizarre [18]. It is believed that some of the social difficulties in these children result from deficits in processing nonverbal cues, including facial expressions (as reviewed in [46]). This syndrome in NVLD children, similar to deficits in face recognition, is thought to represent primarily right posterior brain dysfunction as evidenced by left-sided neurological signs and right parieto-temporal EEG spikes [18].

This report demonstrated that there is some task specificity associated with the recognition memory for faces deficit. Poor performance on this task is greater than would be predicted from measures of psychopathology, general attention deficits or global memory problems. Within the schizophrenia group, performance on neither of the WMS Faces tasks was correlated with GAF scores or the general scale from the PANSS. In addition, by statistically partialing out performance on the WMS-III Forward Spatial Span task and RAVLT immediate recall, delayed recall and delayed recognition, we were able to demonstrate that deficits in recognition memory for faces persisted. Thus, even though schizophrenia patients, as expected, demonstrated deficits in general attention and verbal memory, and relatives demonstrated deficits in verbal memory, there was also, superimposed on these group differences, a specific face memory impairment.

These findings have important implications. First, our finding that unaffected relatives of schizophrenia patients demonstrate impairment in recognition memory for faces, and the persistence of this effect after partialing out the effect of education, indicates this deficit cannot be attributed to factors associated with chronic mental illness, such as distractibility or lack of motivation due to active psychotic symptoms, to medication effects or to lower education levels. Impaired recognition memory for faces appears to be associated with the genetic diathesis for schizophrenia and may be a valuable indicator of disorder susceptibility. Second, recognition memory for faces is associated with specific symptomatology. In schizophrenia patients, there is a correlation between face memory and positive symptoms. As suggested earlier, with this finding it may be possible to create meaningful neuropsychological subgroups of schizophrenia patients, subgroups that may help eliminate some of the etiological heterogeneity among those diagnosed with schizophrenia via traditional means. By decreasing heterogeneity, we may be able to identify study samples that provide greater etiological insight. In addition, in the

relative group, there was a strong negative association between schizotypal characteristics and recognition memory for faces suggesting that these traits together may form a multivariate endophenotype. By identifying unaffected relatives of schizophrenia patients with this multivariate endophenotype, we may be able to locate relatives with the greatest schizophrenia vulnerability as well as family members that may be most useful for genetic linkage studies. Finally, this report may provide greater insight into the neuropathology associated with the schizophrenia diathesis. There has been extensive research into the neural localization of recognition memory for faces. Impaired recognition memory for faces among schizophrenia patients and their first-degree relatives suggests right occipitotemporal and/or frontal lobe involvement, adding to consistent, emerging evidence from diverse lines of research including neuropsychological and neuroimaging studies, as well as post-mortem histological findings (for review, see [9]). At the visual processing level, schizophrenia patients have been reported to evidence aberrations in visual scanning of faces, characterized by restricted fixations and disproportionate attention paid to nonfeature areas of the face (e.g. [17,38,53]), suggesting that face recognition deficits in schizophrenia patients (and their relatives) may be reflective of a basic problem in complex object processing [53].

There are a couple limitations to our findings worth noting. We did not use a nonverbal memory task with stimuli other than faces to compare to performance on the faces tasks. Given this limitation, we are unable to determine whether relatives have a specific deficit in memory for faces or a more generalized deficit in nonverbal memory. However, literature reviews of family-studies of schizophrenia have failed to reveal nonverbal memory deficits among first-degree relatives (e.g. [31]). A second potential limitation involves the somewhat limited symptom variability among the schizophrenia sample, with individuals exhibiting a greater preponderance of positive than negative symptoms. For instance, although 97% had two or more positive symptoms, only 10% had two or more negative symptoms. While this finding is not unexpected among psychiatric inpatients recently experiencing acute psychotic episodes, it may have reduced our ability to investigate relationships between recognition memory for faces and schizophrenia symptomatology. A schizophrenia sample with greater symptom variability might have been more sensitive to these relationships (i.e. the correlation between positive symptoms and face memory).

Given these limitations to the current study, future work is needed to strengthen confidence in some of our conclusions. Future studies are needed that use a delayed face recognition measure that shows the expected decrease in recall over time in order to investigate the temporal nature of the face memory deficit, neuropsychological batteries that include nonverbal memory tasks with nonface stimuli to further investigate the specificity of face memory deficits and a schizophrenia sample with greater symptom diversity to investigate face

memory relationships with symptomatology. Replication of our findings in studies employing these additional measures holds promise for helping to create meaningful groups among schizophrenia patients, identify relatives with the greatest susceptibility for schizophrenia and increase our understanding of the etiology of schizophrenia.

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