The McCollough Effect and Facial Emotion Discrimination in Patients With Schizophrenia and Their Unaffected Relatives

Simon A. Surguladze1*, Eka D. Chkonia2, Archil R. Kezeli3, Maya O. Roinishvili3, Daniel Stahl1, and Anthony S. David1

1Department of Psychosis Studies, Institute of Psychiatry, King’s College London, PO Box 69, De Crespigny Park, London SE5 9AF, UK; 2Department of Psychiatry, Tbilisi State Medical University, Tbilisi, Republic of Georgia; 3Department of Behaviour and Cognitive Functions, Beritashvili Institute of Physiology, Tbilisi, Republic of Georgia

*To whom correspondence should be addressed; tel: +442078480363, fax: +44207848572; e-mail: simon.2.surguladze@kcl.ac.uk

Abnormalities in visual processing have been found consistently in schizophrenia patients, including deficits in early visual processing, perceptual organization, and facial emotion recognition. There is however no consensus as to whether these abnormalities represent heritable illness traits and what their contribution is to psychopathology. Fifty patients with schizophrenia, 61 of their first-degree healthy relatives, and 50 psychiatrically healthy volunteers were tested with regard to facial affect (FA) discrimination and susceptibility to develop the color-contingent illusion [the McCollough Effect (ME)]. Both patients and relatives demonstrated significantly lower accuracy in FA discrimination compared with controls. There was also a significant effect of familiality: Participants from the same families had more similar accuracy scores than those who belonged to different families. Experiments with the ME showed that schizophrenia patients required longer time to develop the illusion than relatives and controls, which indicated poor visual adaptation in schizophrenia. Relatives were marginally slower than controls. There was no significant association between the measures of FA discrimination accuracy and ME in any of the participant groups. Facial emotion discrimination was associated with the degree of interpersonal problems, as measured by the Schizotypal Personality Questionnaire in relatives and healthy volunteers, whereas the ME was associated with the perceptual-cognitive symptoms of schizotypy and positive symptoms of schizophrenia. Our results support the heritability of FA discrimination deficits as a trait and indicate visual adaptation abnormalities in schizophrenia, which are symptom related.

Key words: affect discrimination/McCollough effect/heritability/visual processing/schizophrenia

Introduction

Abnormalities in visual perception have been demonstrated in schizophrenia, eg, investigators have reported deficits in early visual processing,1 motion detection,2 form perception,3 object recognition,4 as well as higher-level processes, such as perceptual organization and integration.5 It has been postulated that most of these visual processing deficits are related to dysfunctional magnocellular pathways.6 Another visual function, relevant to schizophrenia, is the processing of facial emotional expressions, which is implicated in guiding interpersonal relationships as a part of a socioemotional processing stream.7 Facial emotion processing has been found consistently to be dysfunctional in schizophrenia.8-11 Neurocognitive12-15 and neuroimaging16 studies have shown that abnormal facial emotion processing contributed to impaired social functioning of patients. These deficits have been found in medication-free first-episode patients17 and are reported to be stable over the course of illness, being present in the acute state and in remission.18,19 A recent meta-analytical review20 showed a large effect size of the facial emotion recognition deficit in schizophrenia which was moderated by illness severity but not by medication status or illness duration. Moreover, there have been reports on the heritability of facial emotion discrimination,21-24 which together with the above studies indicate that these deficits represent potential biomarkers of the illness.25 The mechanism of impaired facial emotion recognition is not fully established; however, there is a strong evidence for involvement of impaired magnocellular pathway functioning.26

Visual processing has recently been studied with paradigms inducing optical illusions under varying stimulus contrast conditions.27 Schizophrenia patients demonstrate an increased susceptibility to some (eg, Muller-Lyer) but not other (eg, Hermann grid) illusions. The authors suggested that faulty perception in schizophrenia represented an upstream effect of deficient contrast sensitivity, which in itself is related to the magnocellular pathway. Experiments involving the development of
visual illusions have advantages over performance-based experiments: Being purely visual tasks, they require minimal involvement of language and memory processes that may be impaired in schizophrenia. Hence, the abnormalities may be taken to reflect pure deficits rather than results of generalized impairment.

In our study of schizophrenia patients and their first-degree relatives, we examined facial emotion recognition concurrently with susceptibility to the color-contingent illusion [the McCollough effect (ME)]. The ME involves perception of an illusory color in a black-white grid, developing after a series of adaptation trials where a subject has been exposed to horizontal patterns of one color alternating with vertical patterns of a different color (figures 1–3).

There is evidence in support of a purely local (primary visual cortex) basis of ME as well as indications of involvement of more complex integrational processes. Thus, previous studies have shown that ME reveals adaptive (compensation-correction) mechanisms of color constancy. According to Dodwell and Humphrey, the ME represents an error-correction mechanism (error correcting device) whose role is reducing discrepancies between the external environment and the internal representation, which may be mediated by top-down neural processes. Functional neuroimaging studies in healthy volunteers have also contributed to the understanding of the neurophysiology of the ME. One functional Magnetic Resonance Imaging (fMRI) study demonstrated activation of primary visual cortex during perception of illusory color. An fMRI study conducted by our group showed that the experience of the ME was associated with activation of left anterior fusiform gyrus, bilateral ventrolateral prefrontal cortex, and the left insula. We suggested that both early visual and “top-down” mechanisms were implicated in the processing of this illusory color.

Hence, the ME is a useful probe for investigating integrity of certain neural systems, which are subject to top-down integrative control processes and may be a valuable tool in research of visual processes implicated in schizophrenia. The ME largely depends on edge detection, sensitivity to which has been associated with the magnocellular pathway. It has been reported that abnormal functioning of the magnocellular pathway represents a vulnerability indicator for schizophrenia. On the other hand, as noted above, the ME also entails more global integrative perceptual processes that are assumed to be compromised in schizophrenia.

In our study, we contrasted 2 very different but well-characterized visual tasks: the facial affect (FA) discrimination accuracy task and experiments inducing ME in individuals with schizophrenia, first-degree relatives, and healthy controls.

Thus, the aims of our study were 2-fold:

First, to test the familiality of facial emotion discrimination deficits in a sample of patients and their first-degree relatives, and second, to introduce a new...
approach of testing visual adaptational processes—by employing a task inducing the ME. To this end, we set out to test—whether ME was affected by schizophrenia at all, and if so—would it be associated with FA discrimination task performance.

The study addressed the following questions:

1. Do patients with schizophrenia and their first-degree relatives differ from healthy controls in their ability to identify facial emotions, ie, is there a heritable component to facial emotion discrimination deficits in schizophrenia?
2. Is there a deficit in visual adaptation processes in schizophrenia—as measured by the ME paradigm, and if so, is this deficit heritable?
3. Is facial emotion discrimination correlated with the efficiency of visual adaptation processes?

We predicted that

(a) Patients would show poorer performance compared with healthy controls on FA discrimination tasks;
(b) First-degree relatives’ performance would be impaied on facial emotion discrimination, compared with healthy controls, but to a lesser degree than patients;
(c) Patients would show deficient performance in ME experiments, due to deficient magnocellular functioning or faulty integrative processes.

We did not have a specific prediction regarding the relationship between the ME and FA discrimination measures, leaving this issue open to exploration. However, if performance on the 2 was highly correlated, this might point to reliance on common neuroanatomical or cognitive processes. On the contrary, absence of such association between the ME and FA task performance would indicate that the ME reflects higher-order cognitive processes like adaptation.

Methods

Participants

The sample comprised 50 patients with schizophrenia (16 inpatients and 34 outpatients), 61 of their first-degree healthy relatives (35 siblings and 11 offspring and 15 parents), and 50 psychiatrically healthy controls. All participants were white Caucasians. The patients and their relatives were recruited at inpatient and outpatient services of M. Asatiani Institute of Psychiatry, Tbilisi, Republic of Georgia. The controls were recruited from the community within the catchment area of the above services via advertisements. The study was conducted in compliance with the Code of Ethical Principles for Medical Research Involving Human Subjects of the World Medical Association (Declaration of Helsinki) and approved by the local Research Ethics Committee. Subjects gave their informed consent and participated for a small fee. Clinical diagnosis of schizophrenia was confirmed using the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders (SCID-I for DSM-IV) and through available medical records.

Exclusion criteria for all 3 groups were history of drug or alcohol dependency or head injury accompanied with the loss of consciousness. Family history of psychosis was an exclusion criterion for the control group. We determined visual acuity by means of the Freiburg Visual Acuity Test. The inclusion criterion was a value of 0.8 in each eye (equivalent to 20/25 Snellen fraction). There was no statistically significant difference between the participant groups with regard to age and sex (table 1). The groups differed in years of education ($F(2,104) = 5.23, P = .007$), with controls and patient relatives spending more time in education compared with patients (respectively, $P = .026$ and $P = .004$). Healthy controls had no history of axis I psychiatric disorder, as determined by the screen interview of SCID-I.

All patients were taking antipsychotic medications: risperidone (7 patients), perazine dimalonate (taxil:an, 7 patients), clozapine (11 patients), zuclopenthixol (2 patients), haloperidol (12 patients), fluphenazine (4 patients), chlorprothixene (4 patients), and levomepromazine (3 patients). Drug doses were converted to chlorpromazine equivalents according to Agency for Healthcare Research and Quality recommendations. In addition to antipsychotic medication, 16 patients were taking anticholinergic biperiden, 3 patients—amitriptyline, 3 patients—carbamazepine.

Symptoms of schizophrenia were scored according to Scale for the Assessment of Negative Symptoms (SANS) and Scale for the Assessment of Positive Symptoms (SAPS). The investigator (E.D.C.) was trained on the SANS and SAPS to meet minimum intraclass correlation coefficients for each subscale of 0.70 ($P < .05$), based on agreements with the ratings of an Institute of Psychiatry expert diagnostician (S.A.S.). This was achieved for all subscales. Schizotypal personality features of the relatives and controls were assessed by the Schizotypal Personality Questionnaire-Brief (SPQ-B) version.

Experiments on FA Discrimination Accuracy

The computerized FA task used in this study was a slightly modified version that employed in our earlier study of depression and was based on the set of stimuli comprising black/white photographs of faces of white Caucasian individuals (Facial Expressions of Emotion: Stimuli and Tests). These photographs represent the morphed version of standard Ekman and Friesen’s instrument Pictures of Facial Affect. The faces were framed within an oval the size of which was manipulated to equate all images. The average visual angle of the faces subtended
Table 1. Demographic, Clinical, and Experimental Data

<table>
<thead>
<tr>
<th></th>
<th>Patients (n = 50)</th>
<th>Relatives (n = 61)</th>
<th>Controls (n = 50)</th>
<th>F</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>37.40 (1.3)</td>
<td>39.15 (2.0)</td>
<td>33.9 (1.8)</td>
<td>2.2</td>
<td>.114</td>
</tr>
<tr>
<td>Gender (female/male)</td>
<td>26/24</td>
<td>39/22</td>
<td>30/20</td>
<td>.444</td>
<td></td>
</tr>
<tr>
<td>Education, y</td>
<td>12.6 (0.4)</td>
<td>13.4 (0.4)</td>
<td>14.0 (0.4)</td>
<td>3.8</td>
<td>.025</td>
</tr>
<tr>
<td>Duration of illness, y</td>
<td>13.7 (1.3)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SANS</td>
<td>12.0 (0.8)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAPS</td>
<td>11.7 (4.6)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CPZ</td>
<td>527.6 (66.1)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>SPQ-B total</td>
<td>5.7 (3.9)</td>
<td>6.1 (4.0)</td>
<td>0.61</td>
<td>.54</td>
<td></td>
</tr>
<tr>
<td>SPQ-B cognitive-perceptual</td>
<td>2.2 (1.9)</td>
<td>1.9 (1.5)</td>
<td>0.79</td>
<td>.43</td>
<td></td>
</tr>
<tr>
<td>SPQ-B interpersonal</td>
<td>2.3 (1.7)</td>
<td>2.6 (1.8)</td>
<td>1.2</td>
<td>.23</td>
<td></td>
</tr>
<tr>
<td>SPQ-B disorganized</td>
<td>1.2 (1.3)</td>
<td>1.5 (1.5)</td>
<td>1.2</td>
<td>.25</td>
<td></td>
</tr>
</tbody>
</table>

Note: Br, response bias; CPZ, chlorpromazine equivalent; Pr, discrimination accuracy; SANS, Scale for Assessment of Negative Symptoms; SAPS, Scale for Assessment of Positive Symptoms; SPQ-B, Schizotypal Personality Questionnaire-Brief. Means (SDs) of demographic and clinical variables.

an area of 8.93° vertically by 4.77° horizontally, which was devised to be a visual angle equivalent to that of a social interaction held at approximately 1 m.

There were 2 experimental runs: One with photographs of faces expressing fear, disgust, and no emotion (neutral faces) and another with photographs of happy, sad, and neutral faces. We used 6 facial photographs per each emotion (3 male and 3 female) each of which had 3 different intensities of emotion (eg, 25%, 50%, and 100% fear). Each of the expressions was presented twice: for 500 ms and 2000 ms. Six emotionally neutral faces were presented 4 times each: 2 times for 500 ms and 2 times for 2000 ms. In order to avoid practice effects (which might develop if there was just 1 type of emotional expression vs. neutral), we mixed 2 different emotions within each experimental run. Thus, one experimental run (experiment 1) comprised 36 fearful faces, 36 disgusted faces, and 24 neutral faces and another (experiment 2) 36 happy faces, 36 sad faces, and 24 neutral faces. The order of experiments 1 and 2 was counterbalanced across the participants. Each face was presented individually, with an interstimulus interval of 1500 ms, during the first 500 ms of which a fixation cross was displayed.

The participants were instructed that they would see emotional (eg, either fearful or disgusted) or neutral faces in each run and were requested to use 1 of the 2 buttons of the gamepad to indicate whether the face they saw was emotional or neutral. The software recorded the button-press responses as “emotional” or “neutral” and the accompanying reaction times. The participants had practice trials before they started the experiments to ensure that they understood the task requirements. The facial stimuli in the practice trials were different from those used in the actual experiments. Although the task contained a variety of intensities of emotional expressions, presented at various durations, the patients did not find it particularly challenging because they were not required to explicitly label the expressions. We intentionally avoided the labeling instructions knowing that this may be problematic for people with schizophrenia. Thus, the task was easy to perform because it tested only the participants’ ability to distinguish emotional faces from the nonemotional ones.

Experiments With the ME

All subjects were pretested to ensure normal color vision. The experiments eliciting the ME started with the presentation of adaptation stimuli that comprised black-green vertical and black-red horizontal patterns with spatial frequency 0.6 c/grad. One adaptation trial lasted 10 s, which included presentation of 2 colored patterns, each appearing for 4 s, with an Inter-stimulus interval of 2 s (during which the participants viewed a black screen). Each trial was repeated 24 times, constituting a block, making the blocks of 4-min duration. After each block, the participants were shown the test stimuli for 60 s (figures 1–3). The test stimuli represented black-white horizontal and vertical patterns, and the participants were asked whether they saw any difference between the horizontal and vertical patterns. If the subjects reported experiencing the illusory color in the test stage (eg, they reported that the vertical patterns looked reddish and the horizontal ones—greenish), the experiment was discontinued. If they had not developed the illusion (ie, they correctly identified the black-white patterns as black-white), they were presented with the next block of adaptation stimuli. The maximal number of blocks per the whole experiment was 5, lasting 20 min in total. Thus, the subjects could have developed the ME after 1, 2, 3, 4, or 5 blocks ie, in 4, 8, 12, 16, and 20 min, respectively. All experiments were performed on a Siemens Fujitsu P796-1 monitor (31.0 cm × 23.3 cm and 1024 × 768 resolution). Refresh rate was 100 Hz, driven by a standard accelerated graphics card. Participants sat comfortably in a chair in front of the screen in a dimly illuminated room (about 0.5 lx) and were asked to maintain gaze on the screen although not to any specific point on it. The distance between the subject and the screen was approximately 60 cm.

Data Analysis

The FA task data were analyzed with regard to discrimination accuracy (Pr) and response bias (Br). Accuracy values would indicate an ability to discriminate accurately between emotional and neutral expressions.
Response bias scores would indicate a tendency to misidentify neutral faces as emotional.

The analytical procedure was as described by Corwin.51 In particular, discrimination accuracy, \( Pr = \frac{\text{number of hits} + 0.5/\text{number of targets} + 1}{\text{number of false alarms} + 0.5/\text{number of distractors} + 1} \). Response bias, \( Br = \frac{\text{number of false alarms} + 0.5/\text{number of distractors} + 1}{1 - Pr} \). We were interested in a general (rather than emotion-specific) ability to discriminate between any emotional expressions (e.g., fear, disgust, happy, sad) and neutral faces. For each of 2 experiments, we have computed the accuracy data for short and long duration (Pr short and Pr long, respectively) and the bias for short and long duration (Br short and Br long, respectively). The facial expression intensities (e.g., 25%, 50%, 100%) were not analyzed as separate variables because the investigation of the intensity effect was beyond the scope of this study.

The accuracy and bias data were then compared between the 3 groups: patients, relatives, and controls—using generalized estimating equations (GEE) analysis in order to control for the effect of nonindependency. GEE was chosen because it allows to model the mix of dependent (relatives) and independent data (control group) between subjects. An exchangeable correlation structure was used to fit the model. GEE provides unbiased estimates of the marginal effects, even if the assumed correlation structure is misspecified.52,53 To safeguard a possible misspecification against the variance/covariance matrix, robust Hubert White sandwich estimators were used to adjust SEs and hence CIs and \( P \) values.54 Type III analysis was used for statistical inference.

ME data were analyzed in terms of the induction time as a dependent variable (e.g., 4, 8, 12, 16, 20 min), again applying GEE to control for the effect of nonindependency, with pairwise post hoc comparisons between the 3 groups.

Subsequently, the analyses were performed exploring the possible associations between the visual processing abilities and the measures reflecting symptoms of schizophrenia (SAPS and SANS) or schizotypy (SPQ-B).

**Familiality Effects**

In order to obtain an appropriate estimate of familiality, a variance component analysis using random effects models was performed55 using patients and their healthy relatives data. Correlation between members from the same family was accounted for by including a random factor for families. This linear mixed model using restricted maximum likelihood estimation method provides an estimate of the between-family variability and the within-family variability. We therefore used the intrafamily correlation coefficient [Intra-class correlation (ICC)], which measures the percentage of the total residual variability due to family differences, as an index of familiality.

A likelihood ratio test was then used to assess the significance of the random (i.e., family) effect. These analyses were conducted in STATA 9.0 (Stata Corporation, College Station, Texas).

**Results**

**FA Task**

Two patients were unable to complete the FA task, and their data were excluded from the analysis. Response accuracy (Pr) data were entered into GEE analysis with experiments (Exp1 and Exp2) and duration (short and long) as within-subject variables and group (patients, relatives, and controls) as between-subject variable.

There was a significant main effect of group: Wald chi-square \( (df, 2) = 48.3; P < .001 \). Pairwise comparisons showed that the patients performed poorer in overall, regardless of the experiment or duration, compared with controls (\( P < .001 \)) and relatives (\( P < .001 \)). Importantly, relatives' performance was also poorer (\( P = .031 \)) than that of controls (table 1; figure 4).

GEE analysis was applied to response bias data with experiments (Exp1 and Exp2) and duration (short and long) as within-subject variables and group (patients, relatives, and controls) as between-subject variable. This analysis produced no significant main effects or interactions.

**McCollough Effect**

The ME did not develop in 9 patients (even with induction lasting 20 min) compared with 4 relatives and 3 controls. The between-group difference with regard to

![Fig. 4. Facial Affect Discrimination Accuracy in Patients With Schizophrenia, Their First-Degree Relatives and Healthy Participants. Mean Pr, mean discrimination accuracy. Error bars indicate SDs.](https://academic.oup.com/schizophreniabulletin/article-abstract/38/3/599/1865860/The-McCollough-Effect-and-Facial-Emotion/603)
abuse vs presence of ME was not significant: Wald chi-square (2) = 5.26; \( P = .07 \). Patient group tended to have larger number of individuals who did not develop ME compared with relatives (\( P = .07 \)) and controls (\( P = .06 \)). The clinical and FA data of the group of patients who developed the ME were compared with those who did not develop it. There was no significant difference between these groups of patients with regard to the scores of SANS (\( t = .18; \ P = .6 \)), SAPS (\( t = -.18; \ P = .86 \)), accuracy Pr (\( t = .66; \ P = .5 \)), or response bias Br (\( t = -1.2; \ P = .22 \)).

In further analyses, only the data of subjects who had developed the ME were used. GEE analysis was performed to compare the ME induction time between the groups, which demonstrated a significant effect of group: Wald chi-square (2) = 34.12, \( P < .001 \). Pairwise comparisons showed that patients needed significantly more time for ME induction compared with the relatives or healthy controls: \( P = .003 \) and \( P < .001 \), respectively. The induction time in relatives was longer than that in controls, although this difference did not reach statistical significance: \( P = .056 \). (Table 2; Figure 5).

**Associations With Symptoms and Hospitalization Status**

To explore possible associations with clinical measures (SANS, SAPS, and SPQ-B), we performed regression analyses with the measures where the groups demonstrated significant differences, ie, Pr and ME. Because the accuracy (Pr) scores showed the same pattern of results across both FA tasks, we produced a composite (mean) measure of accuracy: Pr total—based on means of both short and long presentation conditions across both experiments.

Linear regression analyses established that ME predicted the SAPS global score in schizophrenia patients (standardized beta = 0.35; \( P = .029 \)) and SPQ cognitive-perceptual deficits score in relatives (standardized beta = 0.34; \( P = .012 \)). Pr total predicted SPQ interpersonal deficits score in healthy controls (standardized beta = -0.43; \( P = .002 \)) and in relatives (standardized beta = -0.26; \( P = .045 \)). The Pearson’s correlations of Pr and ME with SANS global score were not significant: \( r = .14, \ P = .34 \) and \( r = .098, \ P = .55 \), respectively.

In none of the groups was there a significant association between Pr total and ME. In particular, in the patient group, Pearson’s correlations between Pr total and ME and Br total and ME were as follows: \( r = -.044, \ P = .79 \) and \( r = -.079, \ P = .64 \), respectively. In the relatives group, correlations between Pr total and ME and Br total and ME were \( r = .041, \ P = .76 \) and \( r = -.002, \ P = .99 \), respectively. In the control group, correlations between Pr total and ME and Br total and ME were \( r = -.175, \ P = .24 \) and \( r = -.14, \ P = .36 \), respectively.

To determine whether hospitalization status affected performance, we compared the ME and FA task results between the inpatient and outpatient subgroups. The contrasts show that there were no significant differences between the patients subgroups (ME performance: \( t(48) = 0.35; \ P = .73 \); Pr measure: \( t(46) = 0.85; \ P = .4 \)).

**Familiality Effects**

The random effects analysis was applied to the accuracy measure Pr total and demonstrated a familiality effect: The family scores accounted for a significant proportion of the variance of the accuracy variable: ICC rho = 25%, \( P = .03 \). In other words, the participants from the same families had stronger similarities in accuracy scores Pr total than those who belonged to different families. The ME measure did not show any familiality effect: ICC rho = 2%, \( P = .4 \).

**Medication Effect**

To explore any possible contribution of antipsychotic treatment on visual processing, we entered the accuracy, ME data, and chlorpromazine equivalent (CPZ) equivalent values into the correlational analysis. The only significant correlation was detected between the Pr (short) from Exp2 and CPZ: Spearman’s rho = -0.31, \( P = .04 \). This result did not survive the Bonferroni control for multiple comparisons.

### Table 2. Discrimination Accuracy and ME: Main Effects of Groups and Pairwise Contrasts

<table>
<thead>
<tr>
<th>Contrasts/Groups</th>
<th>Mean Difference</th>
<th>Standard Error</th>
<th>df</th>
<th>Significance</th>
<th>95% Wald CI</th>
<th>Lower</th>
<th>Upper</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pr</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patients</td>
<td>-0.1461</td>
<td>0.03172</td>
<td>1</td>
<td>&lt;.001</td>
<td>-0.2083</td>
<td>-0.0839</td>
<td></td>
</tr>
<tr>
<td>Relatives</td>
<td>-0.2051</td>
<td>0.02960</td>
<td>1</td>
<td>&lt;.001</td>
<td>-0.2631</td>
<td>-0.1471</td>
<td></td>
</tr>
<tr>
<td>Patients</td>
<td>-0.0590</td>
<td>0.02737</td>
<td>1</td>
<td>.031</td>
<td>-0.1126</td>
<td>-0.0053</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patients</td>
<td>2.74</td>
<td>0.930</td>
<td>1</td>
<td>.003</td>
<td>4.91</td>
<td>4.56</td>
<td></td>
</tr>
<tr>
<td>Relatives</td>
<td>4.22</td>
<td>0.726</td>
<td>1</td>
<td>&lt;.001</td>
<td>5.80</td>
<td>5.65</td>
<td></td>
</tr>
<tr>
<td>ME</td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patients</td>
<td>1.49</td>
<td>0.776</td>
<td>1</td>
<td>.056</td>
<td>2.04</td>
<td>3.01</td>
<td></td>
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<tr>
<td>Controls</td>
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</table>

Note: ME, McCollough effect; Pr, discrimination accuracy.
Discussion

We carried out 2 distinct visual processing tasks in patients with schizophrenia, their first-degree relatives and healthy controls. We have demonstrated a significant deficit in schizophrenia patients in discrimination accuracy of facial emotional expressions, which was not accompanied by response bias. The relatives' accuracy level was also significantly lower compared with healthy controls and, importantly, patients and relatives from the same families showed significantly stronger similarities in accuracy scores than those who belonged to different families.

Our finding of familiality effects in discrimination accuracy adds to the evidence of heritability of the FA discrimination deficit, which is in line with suggestions that this trait may serve as an intermediate phenotype for schizophrenia.25 We contribute to this knowledge by showing the relevance of this measure to the unaffected individuals—by demonstrating an association of facial emotion discrimination with an index of interpersonal problems in relatives and healthy volunteers (measured with the Schizotypal Personality Questionnaire).

Another set of experiments showed that the patients were abnormally slow in developing color-contingent illusions (ME) compared with healthy controls. This indicates deficient (inert) visual adaptation processes in schizophrenia.

One intriguing finding was that there appeared to be an association between the ME with the perceptual-cognitive symptoms of schizotypy and the positive symptoms of schizophrenia. We cannot resolve whether this association was determined by abnormal early vision or more complex integrational processes. Further studies are warranted that manipulate the contrast of the stimuli—to control contrast sensitivity-driven phenomena—similar to the approach described by Kantrowitz et al.27

Our moderate sample size did not allow us to examine the relationships between the ME and particular perceptual symptoms (eg, hallucinations) because these associations did not survive control for multiple comparisons.

We suggest therefore that our findings need replication and this study could serve as the first step in investigation of possible contribution of visual adaptation mechanisms to the symptoms of schizophrenia.

Limitations

Because all patients in our study were medicated, we had to determine whether the detected impairments were attributable to schizophrenia or to the effect of antipsychotic treatment. A recent meta-analysis of facial emotion processing studies demonstrated that patients on antipsychotic medication did not differ from those who were unmedicated.20 Another way of testing the possible medication effect is to examine the association between dose in chlorpromazine equivalents and the degree of impairment. We found no such association, which again was in agreement with the findings of the meta-analysis. Finally, the FA discrimination accuracy deficit was found in unaffected relatives, who were not taking any antipsychotic medication, which confirms that these deficits may be related to schizophrenia rather than medication.

The ME abnormality in the relatives, although showing the same pattern as in patients, was just marginally different from the healthy controls. There was no association of the ME and the medication dosage, which helps to rule out the effect of antipsychotic medication on ME. We acknowledge that studies on medication naïve patients are warranted to fully examine the ME in schizophrenia. The patients group was heterogenous in terms of hospitalization status, comprising both inpatients (16) and outpatients (34). The choice of the patients was determined by 2 main factors: their ability to participate in amnesia, which was consistent with evidence implicating early visual areas in development of ME rather than mnemonic processes. Some studies however indicate a more complex character of the ME by showing the relationship between ME and central cholinergic activity,56 hormonal state,57 or extraversion.58 Our results show that the ME measure was not associated with the ability to recognize facial emotions, which may indicate that the ME task did not tap on the same magnocellular pathway that is implicated in FA discrimination.

One intriguing finding was that there appeared to be an association between the ME with the perceptual-cognitive symptoms of schizotypy and the positive symptoms of schizophrenia. We cannot resolve whether this association was determined by abnormal early vision or more complex integrational processes. Further studies are warranted that manipulate the contrast of the stimuli—to control contrast sensitivity-driven phenomena—similar to the approach described by Kantrowitz et al.27

Our moderate sample size did not allow us to examine the relationships between the ME and particular perceptual symptoms (eg, hallucinations) because these associations did not survive control for multiple comparisons.

We suggest therefore that our findings need replication and this study could serve as the first step in investigation of possible contribution of visual adaptation mechanisms to the symptoms of schizophrenia.
the FA and ME tasks and the availability of the relatives who were willing to take part in the study. Although in our case this heterogeneity did not affect performance in FA and ME tasks, it may have been relevant for attentional deficits potentially associated with the inpatients' status. This should be taken into account in future studies.

The experiments on the ME were conducted without looking at the extinction (strength) of the effect. This was to keep the experiments as short as possible to avoid fatigue in schizophrenia patients. Thus, our findings on ME deficit should be considered as preliminary, pending further studies that address both aspects of the ME—adaptation duration and strength.

In conclusion, our results indicate deficient facial emotion processing in patients with schizophrenia, which is shared with first-degree relatives. Schizophrenia patients also showed problems with developing the color contingent after-effect (ME) that may reflect either abnormalities in early vision (low contrast sensitivity) or more complex adaptational processes. Both facial emotion discrimination and ME measures were independently associated with the symptoms of schizotypy and schizophrenia, respectively. Thus, concurrent investigation of ME and FA discrimination may open new avenues for uncovering underlying mechanisms of psychopathology in schizophrenia.

Supplementary Material

Supplementary material is available at http://schizophreniabulletin.oxfordjournals.org.

Funding

The Royal Society UK (2006/R2-IJP).

Acknowledgment

Conflict of interest: nothing to declare.

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