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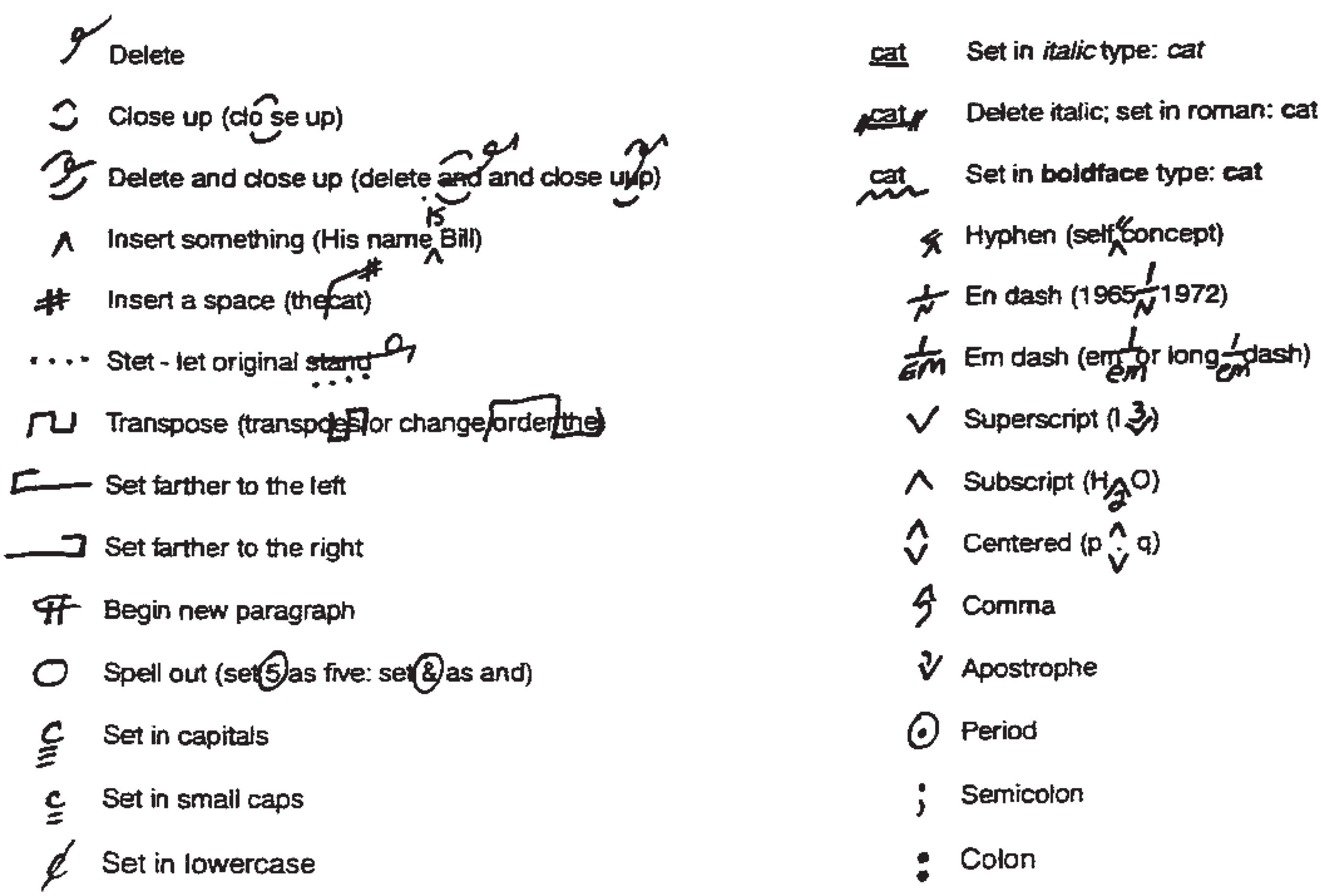

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Vernier Threshold in Patients With Schizophrenia and in Their Unaffected Siblings

Szabolcs Kéri
University of Szeged

Oguz Kelemen
Bács-Kiskun Country Hospital

György Benedek and Zoltán Janka
University of Szeged

The aim of this study was to investigate magnocellular (M) and parvocellular (P) visual functions in nonmedicated patients with schizophrenia and in their unaffected siblings. Possible abnormalities in cortical integration of retinal receptive fields were also addressed. Twenty-two nonmedicated patients with schizophrenia, their unaffected siblings, and 20 age- and IQ-matched healthy control subjects received 4 vernier acuity tasks (blue-on-yellow, frequency-doubling, achromatic low and high contrast conditions) in which they were asked to detect the spatial alignment of dots and gratings. Results revealed that the patients with schizophrenia and their unaffected siblings showed selective dysfunctions in the frequency-doubling and achromatic low contrast conditions, which were devoted to investigate M pathways. In the isoluminant blue-on-yellow and high contrast achromatic conditions, there were no significant differences between the experimental groups. These results suggest that the deficit of M pathway is an endophenotype of schizophrenia.

AQ: 1

Patients with schizophrenia show various dysfunctions in visual perceptual tests. Recently, visual dysfunctions have been explained in the framework of retino-geniculo-occipital magnocellular (M) and parvocellular (P) pathways and their cortical recipients (Schwartz, Tomlin, Evans, & Ross, 2001). These pathways originate in the ganglion cells of the retina and project to the primary visual cortex through the lateral geniculate nucleus. The M pathway is sensitive for low luminance contrast, low spatial frequency, and rapid temporal changes. In contrast, the P pathway prefers static stimuli with medium and high spatial frequency (fine details of objects) and colors (Schiller, Logothetis, & Charles, 1990; Van Essen & Gallant, 1994). Novel evidence suggests that there is a considerable overlap between the two pathways even in the primary visual cortex (Sawatari & Callaway, 1996; Vidyasagar, Kulikowski, Lipnicki, & Dreher, 2002). Therefore, the classic view (Goodale & Milner, 1992; Ungerleider & Mishkin, 1982; Van Essen & Gallant, 1994) that the dorsal occipito-parietal stream dominated by the M pathway exclusively participates in motion perception, detection of spatial location, and visuomotor functions and the ventral occipito-temporal stream dominated by the P pathway is selectively engaged in object recognition and color perception has several limitations (Farrer, Passingham, & Frith, 2002; Kourtzi, Bulthoff, Erb, & Grodd, 2002).

Regarding the physiological mechanism of visual impairments in schizophrenia, results are controversial. Most of the studies investigating the M and P pathways used visual backward masking and luminance contrast sensitivity measurements (Butler et al., 2003; Cadenhead, Serper, & Braff, 1998; Green, Nuechterlein, & Mintz, 1994; Merritt & Balogh, 1989; Schwartz, McGinn, & Winstead, 1987; Slaghuis, 1998; Slaghuis & Curran, 1999). However, the physiological mechanisms of these procedures are very complex, and hence the interpretation of results is not straightforward. In visual backward masking tasks, the subject must identify a briefly presented target, which is then followed by a second irrelevant stimulus, the mask. It has been suggested that an excessive transient M pathway response to the mask results in an increased interruption of the sustained P pathway response to the target (Butler et al., 2003; Green et al., 1994). However, studies using specific tasks with high and low spatial frequency gratings as masks have provided inconsistent results (Butler et al., 2003; Merritt & Balogh, 1989; Slaghuis & Curran, 1999). Schwartz et al. (1987) observed selective contrast sensitivity impairments for temporally modulated low spatial frequency gratings, which are believed to stimulate M pathways. In contrast, Slaghuis (1998) demonstrated both M and P pathway impairments in negative symptom schizophrenia. Finally, many patients with schizophrenia show intact contrast sensitivity for M pathway stimuli (Kéri, Antal, Szekeres, Benedek, & Janka, 2000).

Recently, two electrophysiological studies directly revealed a more prominent impairment of M pathways (Butler et al., 2001; Doniger, Foxe, Murray, Higgins, & Javitt, 2002). Butler et al. (2001) recorded steady-state evoked potentials over the occipital cortex of patients with schizophrenia and control subjects. They found lower responses for stimuli devoted to the stimulation of M pathways (low spatial frequency with low luminance contrast) in the patients compared with the control subjects. There was no significant difference for P pathway stimuli (high spatial frequency with isoluminant colors). During a perceptual closure task, Doniger et al. (2002) registered reduced P1 component over the dorsal occipito-parietal cortex, whereas the ventral N1 component was

AQ: 4

Szabolcs Kéri, Departments of Psychiatry and Physiology, University of Szeged, Szeged, Hungary; Oguz Kelemen, Bács-Kiskun Country Hospital, Kecskemét, Hungary; György Benedek, Department of Physiology, University of Szeged; Zoltán Janka, Department of Psychiatry, University of Szeged.

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spared. Doniger et al. concluded that the selective reduction of dorsal stream P1 is consistent with impaired M pathway functions in schizophrenia.

The human visual system has a remarkable capacity to process small spatial alignment such as horizontal displacement of dots or
F1 gratings (see Figure 1). The main feature of vernier effect is that it is related to the ability of the visual system to detect small spatial offsets. *Vernier threshold* is the minimal spatial offset (displacement) that can be detected. Poor vernier thresholds result in larger spatial displacement of stimuli (i.e., larger s arc). To achieve this, the visual cortex has to integrate information from many retinal receptive fields (Wilson, 1986). The input to this integrative cortical stage depends on stimulus parameters. It is well established that M pathway cells do not respond when stimuli and background exclusively differ with respect to their color but not with respect to their luminance (isoluminant condition; Hubel & Livingstone, 1990; Schiller & Colby, 1983). In addition, short-wavelength-sensitive cones in the retina, which are essential for the processing of blue–yellow color opponency, provide only 10% of the M cell input in the lateral geniculate nucleus (Chatterjee & Callaway, 2002). Therefore, the isoluminant blue-on-yellow vernier task was not expected to significantly stimulate the M pathway.

Lee, Wehrhahn, Westheimer, and Kremers (1995) investigated the role of M and P pathways in vernier functions. These authors demonstrated that at luminance contrasts of 20% and below, vernier acuity was exclusively mediated by the M pathway, whereas at higher contrast levels both pathways were stimulated. Therefore, we expected that in the low luminance contrast vernier task (5%), the M pathway would be dominantly stimulated, similarly to the frequency doubling condition in which dynamically modulated low spatial frequency gratings are used (McKendrick, Johnson, Anderson, & Fortune, 2002; Merigan & Maunsell, 1990).

There are at least two critical issues that remain to be determined. The first is the question of antipsychotic medication. It has been shown that drugs blocking dopamine receptors may induce profound visual dysfunctions (Harris, Calvert, Leendertz, & Phillipson, 1990; Kéri, Antal, Szekeres, Benedek, & Janka, 2002). In addition, first generation antipsychotics with high affinity to dopamine D2 receptors predominantly disrupt the detection of dynamic stimuli with low luminance contrast and low spatial frequency, which suggests detrimental effects on M pathway functions (Kéri et al., 2002; but see Butler et al., 2003). Second, it is unclear whether M or P pathway dysfunctions are endophenotypes of schizophrenia that can be detected in unaffected biological relatives. To elucidate these issues, we used vernier threshold tasks with well-established physiological bases in nonmedicated patients with schizophrenia and their unaffected siblings. We hypothesized that patients with schizophrenia and their siblings show more pronounced impairments in M pathway tests.

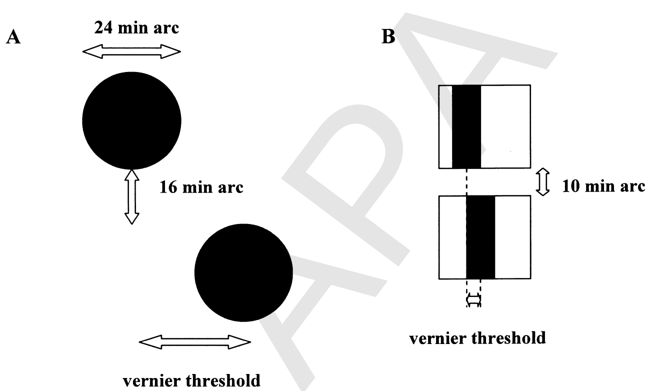

Figure 1. Vernier threshold tasks. A: Two dots were displaced on the monitor. In the achromatic two-dot condition, black dots were presented against a white background. The contrast between the dots and the background was 5% or 50%. In the blue-on-yellow condition, blue dots were presented against a yellow background. B: In the frequency-doubling condition, counterphase-modulated low spatial frequency (1 cycle/degree) gratings were used (only two vertically displaced dark bars are shown). The subject's task was to decide whether the lower dot/grating was to the left or to the right in relation to the upper dot/grating.

Method

Subjects

Twenty-two patients with schizophrenia (14 male and 8 female), their 22 unaffected siblings (10 male and 12 female), and 20 healthy control subjects (13 male and 7 female) participated in the study. The patients and their siblings were recruited from a larger sample follow-up at the psychosis prevention outpatient unit of the University of Szeged Hospital, Szeged, AQ: 2
Hungary. The diagnosis was based on the *DSM–IV* (*Diagnostic and Statistical Manual of Mental Disorders*, 4th ed.; American Psychiatric Association, 1994) criteria. All subjects received the International Neuropsychiatric Interview Plus (Sheehan et al., 1998) and the Wechsler IQ test (Wechsler, 1981). The siblings were free from Axis I or Axis II disorders. Clinical symptoms were determined with the Scale for the Assessment of Positive Symptoms (SAPS; Andreasen, 1984b) and Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1984a) instruments. The patients were unmedicated for at least 2 weeks before testing. History of neurological disorders, head injury, substance abuse, and electroconvulsive therapy were the exclusion criteria. All subjects had normal or corrected-to-normal visual acuity. The three experimental groups did not differ in age and IQ ($p > .20$, two-tailed t tests). The clinical and demographic data are shown in Table 1. After complete description of the study to the subjects, T1
written informed consent was obtained.

Stimuli and Procedure

The experimental procedure consisted of the following parts, which were administered to each subject: (a) blue-on-yellow vernier threshold, (b) achromatic vernier threshold (low and high contrast), (c) frequency-doubling vernier threshold, and (d) contrast threshold measurements for achromatic dots. The order of administration was counterbalanced across subjects. The luminance contrast threshold measurement always preceded the low contrast vernier threshold task.

We adopted the modified procedure of McKendrick et al. (2002), which was previously used in clinical psychophysics. Stimuli were presented on a gamma-corrected ViewSonic PF815 monitor (resolution: 800 × 600 pixel, refresh rate: 75 Hz; VSG graphic card, version 5.02, Cambridge Research System Ltd, Rochester, United Kingdom). The monitor was controlled by an IBM-compatible PC. The viewing distance was 9.5 m.

In the blue-on-yellow condition, two blue dots were presented against a yellow background. The luminance of the background was 80 cd/m^2, as determined by a Spectra Pritchard 1980A-CD photometer. The vertical distance between the dots was 16 min arc. The diameter of the dots was 24 min arc (see Figure 1). The stimulus chromacity was defined using a three-dimensional cone contrast space (Cole & Hine, 1992). There was no luminance contrast between the dots and background (isoluminant condition). To achieve isoluminant condition, we carried out a heterochromatic

Table 1
Clinical and Demographic Characteristics of the Experimental Groups

Characteristic	Schizophrenia patients (n = 22)		Siblings (n = 22)		Control subjects (n = 20)	
	M	*SD*	*M*	*SD*	*M*	*SD*
Age (years)	35.6	12.1	35.8	13.6	32.4	9.4
Wechsler's IQ	104.3	10.8	107.4	10.3	108.1	12.7
SAPS	8.7	4.7				
SANS	6.7	3.0				
Duration of illness (years)	10.4	7.4				

Note. SAPS = Scale for the Assessment of Positive Symptoms; SANS = Scale for the Assessment of Negative Symptoms.

flicker-photometry procedure (Omtzigt, Hendriks, & Kolk, 2002). A flickering disk (3° of visual angle) was presented against a dark background. The fore- and background color alternated at a rate of 10 Hz. The luminance level of the foreground was adjusted by the experimenter until the subject judged the flicker to be minimal. This luminance level was individually determined and then was used in the vernier experiment.

In the achromatic conditions, two black dots were presented against a white background in a similar manner to that in the blue-on-yellow condition. Luminance contrast was defined according to the Michelson formula (the absolute difference between the luminance of the stimulus and background divided by their sum). In the low contrast condition it was 5%, whereas in the high contrast condition it was 50%.

In the frequency-doubling condition, stimuli were two sinusoidal luminance-contrast gratings, vertically separated by 10 min arc (see Figure 1). The spatial frequency was 1 cycle per degree. *Spatial frequency* was defined as the number of cycles (a dark bar with minimal luminance and a white bar with maximal luminance) per 1° of visual angle. The Michelson contrast was 90%. Adjacent bars with minimal and maximal luminance changed places several time during a second (counterphase modulation). The grating was counterphase modulated at 25 Hz. The spatial phase of the upper grating was random. The lower grating was horizontally displaced from the upper grating. The maximum displacement was 0.5 cycle of the grating.

Vernier acuity thresholds were determined in each condition (blue-on-yellow, frequency doubling, achromatic low contrast, and achromatic high contrast) using a two-alternative forced-choice method. The location of the upper stimulus (dot or grating) was constant, whereas the lower stimulus was displaced either to the left or to the right of the upper stimulus (spatial offset). Subjects were asked to indicate the direction of spatial offset by pressing different keys on the computer keyboard (*1* for left, *9* for right). The initial displacement was 400 s arc. The displacement was decreased by 10% after three consecutive correct responses and was increased by 10% after one incorrect response. The staircase was terminated after four reversals. The final vernier threshold was the average value obtained from two separate staircase procedures.

Luminance contrast threshold was determined for the achromatic dots in each subject. For the measurement of contrast threshold, a yes/no staircase procedure was used that terminated after four reversals. Subjects were asked to press a button on the computer keyboard (*0*) if they detected the dots. The initial contrast was 70%. The contrast was decreased by 10% after three successive correct responses and was increased by 10% after a single incorrect response at a given contrast level. The final contrast threshold was the average value obtained from two separate staircase procedures (for further methodological details, see McKendrick et al., 2002).

Data Analysis

Vernier and luminance contrast threshold values were log-transformed. We assessed the normality of data distribution and the equality of variance with Kolmogorov–Smirnov and Levene's tests, respectively. The data did not deviate from normality or equality of variance. We used analysis of variance (ANOVA), in which group (patients with schizophrenia, siblings, and control subjects) was the between-subjects factor and vernier task type (blue-on-yellow, achromatic low and high contrast, and frequency doubling) was the within-subject factor. Huynd–Feldt corrections were used to adjust the univariate ANOVA results for violations of the compound symmetry assumption. Experimental groups' performances in M and P pathway tasks were compared through the use of F tests. We performed two-tailed t tests for post hoc comparisons, analysis of demographic data, and comparison of luminance contrast thresholds. For multiple comparisons, t tests were Bonferroni corrected. Spearman's correlation coefficients were calculated between vernier threshold values and SAPS and SANS scores. The level of significance was .05.

Results

Each subject was able to judge the direction of spatial offset at the maximal displacement level. One control subject and 2 patients with schizophrenia had luminance contrast thresholds higher than 5%. These subjects were excluded from the achromatic low contrast condition.

The ANOVA revealed main effects of group, $F(2, 58) = 5.60$, $p < .01$, and vernier task type, $F(3, 174) = 164.01$, $p < .01$. The two-way interaction was also significant, $F(6, 174) = 2.30$, $p < .05$. The effect of vernier task type and the interaction remained significant after Huynd–Feldt correction, $\varepsilon(2.4, 141.8) = 0.82$, $p < .01$, and, $\varepsilon(4.9, 141.8) = 0.82$, $p < .05$, respectively. Planned comparisons with F tests revealed that the patients with schizophrenia performed worse on the M pathway tests (frequency doubling and achromatic low contrast) but not on the P pathway test (blue-on-yellow) compared with the control subjects, $F(1, 58) = 9.91$, $p < .01$. Bonferroni-corrected t tests confirmed this result, blue-on-yellow: $t(40) = -0.97$, $p = .33$; frequency doubling: $t(40) = -3.68$, $p < .01$; achromatic low contrast: $t(37) = -4.52$, $p < .01$; achromatic high contrast: $t(40) = -1.26$, $p = .22$. Analyses with F and t tests indicated that the siblings were also selectively impaired in the two M pathway tests compared with the control subjects, $F(1, 58) = 4.44$, $p < .05$; blue-on-yellow: $t(40) = 0.72$, $p = .47$; frequency doubling: $t(40) = -2.81$, $p < .01$; achromatic low contrast: $t(39) = 2.58$, $p < .05$; achromatic high contrast: $t(40) = -0.90$, $p = .40$ (see Table 2). The F test did T2
not indicate significant task-specific differences between the patients and their siblings ($p > .20$). There were no significant differences between the three groups regarding luminance contrast threshold for achromatic dots (t test, $p > .30$; see Table 2). The male and female patients performed similarly (t test, $p > .50$). There was no significant correlation between vernier threshold values and SAPS and SANS scores ($p > .20$).

Discussion

The results of this study indicate that nonmedicated patients with schizophrenia and their siblings are selectively impaired in vernier tasks devoted to the testing of M pathways. Performances on the blue-on-yellow and achromatic high contrast tests were spared. We did not observe a generalized deficit in vernier tasks,

Table 2
Vernier and Luminance Contrast Threshold Values in the Experimental Groups

Threshold value	Schizophrenia patients (n = 22)		Siblings (n = 22)		Control subjects (n = 20)	
	M	*SD*	*M*	*SD*	*M*	*SD*
Frequency doubling (s arc; M pathway)	149.4**	78.3	106.8*	38.1	66.3	26.4
Low contrast (5%; s arc; M pathway)	113.1**	49.7	82.1*	24.1	55.6	18.5
Blue-on-yellow (s arc; P pathway)	94.3	33.5	77.8	30.3	70.2	28.9
High contrast (50%; s arc; M and P pathways)	37.2	22.0	33.3	17.1	21.4	16.9
Luminance contrast threshold (%)	3.1	1.9	2.4	1.4	2.8	1.3

Note. Asterisks indicate significant differences for log-transformed vernier threshold values compared with the controls (t tests, * $p < .05$ and ** $p < .005$). M = magnocellular; P = parvocellular.

which suggests that cortical integration of retinal receptive fields is effective in patients with schizophrenia if the input to this cortical integrative stage is not supplied by the M pathways (low contrast and frequency-doubling conditions). However, a deficit of cortical integration in a higher order representation of the motion and depth cues of the receptive field cannot be ruled out by these results.

Consistent with the present results are three previous studies also suggesting that the deficit of M pathway and dorsal stream is an endophenotype of schizophrenia (Chen, Nakayama, Levy, Matthysse, & Holzman, 1999; Green, Nuechterlein, & Breitmeyer, 1997; Kéri, Kelemen, Benedek, & Janka, 2001). Siblings of patients with schizophrenia show dysfunctions in backward masking tasks in which the location of the target must be detected, which is a putative dorsal stream function (Green et al., 1997; Kéri et al., 2001). Chen, Nakayama, et al. (1999) found impaired motion perception in siblings of patients with schizophrenia, also supporting the impairment of the dorsal stream. The main problem with these studies is that they are not able to distinguish between M pathway and dorsal stream processing, despite the fact that these are not entirely interchangeable, because of the overlap of M and P pathways in the primary visual cortex (Sawatari & Callaway, 1996; Vidyasagar et al., 2002). Two studies demonstrated that patients with schizophrenia show dorsal stream deficits even when M pathways may be spared (Chen, Palafox, et al., 1999; Kéri et al., 2000). In both studies, patients with schizophrenia showed normal contrast sensitivity for low spatial frequency gratings, a putative test for M pathway functions. Also notable is the intriguing finding that the same patients displayed impaired motion perception (Chen, Palafox, et al., 1999) and target location backward masking (Kéri et al., 2000), which suggests deficient cortical processing in the dorsal stream. Further studies are warranted to explore the possible differential role of M pathway and dorsal stream dysfunctions in schizophrenia.

Patients with schizophrenia regularly show more pronounced AQ: 3 deficits on tests of dorsal stream functions as compared with ventral stream tasks (Brenner, Wilt, Lysaker, Koyfman, & O'Donnell, 2003; O'Donnell et al., 1996; but see also Tek et al., 2002). A recent functional magnetic resonance imaging study found a decreased activation of the motion sensitive visual area (V5), a characteristic part of the dorsal stream, while there was no impairment in the ventral stream (Braus, Weber-Fahr, Tost, Ruf, & Henn, 2002). It is interesting that this study demonstrated functional abnormalities of the thalamus during visual stimulation, which may indicate a precortical origin of visual impairments. However, impaired top-down modulation may explain these results. Postmortem histological studies are warranted to investigate whether there is a primary impairment in the visual relay nuclei of the thalamus in schizophrenia.

Previous studies suggested that M pathway dysfunctions are related to the negative symptoms of schizophrenia (Butler et al., 2003; Slaghuis, 1998). We found no significant relationship between vernier thresholds and clinical symptoms, which may be the consequence of small sample size and restricted range of the variables. Longitudinal studies are necessary to elucidate this issue.

Testing nonmedicated patients allowed the exclusion of short-term confounding effects of antipsychotic medications, although the duration of the drug-free period was short in light of the sustained modification of cellular functions by antipsychotics (Corson, Nopoulos, Miller, Arndt, & Andreasen, 1999). Our findings are not due to task difficulty, because the most difficult vernier task was the blue-on-yellow condition, in which the control subjects showed the highest threshold. In this P pathway dominated condition, the patients with schizophrenia and their siblings had spared performances. Similarly, the patients and their siblings exhibited normal luminance contrast threshold. Because low luminance contrast processing is related to the M pathway, this finding may seem contradictory to the M pathway hypothesis of schizophrenia, which was supported by our vernier results. However, it is likely that the frequency-doubling and low contrast vernier tasks are more sensitive measures of M pathway functions than simple luminance contrast threshold detection for stationary objects, demonstrating even subtle visual impairments (McKendrick et al., 2002).

The impairment of the M pathway–dorsal system is not unique for schizophrenia. Patients with Williams's syndrome (Atkinson et al., 1997), autism (Spencer et al., 2000), and children with hemiplegia (Gunn et al., 2002) also show M pathway–dorsal stream impairments. In these cases, abnormal neurodevelopment may be implicated. Indeed, the growing literature shows that schizophrenia can be characterized by neurodevelopmental deficits and genetic traits (Lewis & Levitt, 2002), and the M pathway deficit may be specifically related to these factors. The M pathway undergoes late development (Benedek, Benedek, Kéri, & Janáky, 2003) and shows more plastic properties than the P pathway (Armstrong, Neville, Hillyard, & Mitchell, 2002). These findings suggest that the M pathway–dorsal stream impairment may be a marker of disorders characterized by neurodevelopmental anomalies with genetic or environmental origin. The similar psychophysical findings in the above mentioned disorders support the view that these clinical conditions may comprise a group of disorders with similar origins.

In conclusion, our data indicate that patients with schizophrenia and their unaffected siblings show impaired performances on vernier tasks devoted to test visual M pathways. This deficit is more pronounced compared with data from P pathway tests. Our results are in accordance with the increasing number of studies indicating more severe M pathway–dorsal stream dysfunctions in patients with schizophrenia and in their unaffected biological relatives (Braus et al., 2002; Brenner et al., 2003; Chen, Nakayama, et al., 1999; Chen, Palafox, et al., 1999; Green et al., 1997; Kéri et al., 2001; O'Donnell et al., 1996). The investigation of M pathway functions in larger samples is under way in our laboratory to elucidate whether it is a useful endophenotype for molecular genetic studies.

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