ISSN 0362-1197, Human Physiology, 2024, Vol. 50, No. 6, pp. 569–575. © Pleiades Publishing, Inc., 2024.
Russian Text © The Author(s), 2025, published in Fiziologiya Cheloveka, 2025, Vol. 51, No. 1, pp. 3–12.

Visual Contrast Sensitivity in Schizophrenia and Schizotypal Disorder

I. I. Shoshina[a], *, A. V. Kosikova[a], A. S. Fedorova[a], V. A. Smolyaninova[a], A. A. Moritz[a], O. V. Limankin[b], A. A. Torgovtsev[b], A. V. Hanko[b], S. I. Lyapunov[c], and T. P. Fernandes[d]

[a] *St. Petersburg State University, St. Petersburg, Russia*
[b] *Kashchenko Psychiatric Hospital No. 1, St. Petersburg, Russia*
[c] *Prokhorov General Physics Institute, Russian Academy of Sciences, Moscow, Russia*
[d] *Federal University of Paraiba (UFPB), Department of Psychology, Joao Pessoa, Brazil*
**e-mail: shoshinaii@mail.ru*

Received September 16, 2024; revised October 7, 2024; accepted October 8, 2024

Abstract—Schizophrenia is characterized by a wide range of symptoms that also manifest themselves in other disorders, which served as the basis for the emergence of ideas about schizophrenia spectrum disorders. Studies indicate inconsistency of data on the characteristics of visual contrast sensitivity in schizophrenia and schizotypal disorder, which is part of the structure of schizophrenia spectrum disorders. The study involved 30 patients diagnosed with paranoid schizophrenia, 18 patients with schizotypal disorder, and 30 people without psychopathology and neurological diseases. Contrast sensitivity was recorded when presenting Gabor elements with a spatial frequency from 0.4 to 10 cycles/deg, using the adaptive staircase procedure. Contrast sensitivity in both the schizophrenia group and the schizotypal disorder group was lower in the area of high spatial frequencies, compared to the conditionally healthy control. Thus, the identified disorders are common to both schizophrenia and schizotypal disorder. The obtained data are considered as evidence of a special nature of the discordance in the interaction of the magnocellular and parvocellular channels of the visual system with a shift towards the dominance of the magnocellular system.

Keywords: contrast sensitivity, schizophrenia, schizotypal disorder, magnocellular system, parvocellular system, dorsal and ventral cortical pathway

DOI: 10.1134/S0362119724701159

INTRODUCTION

Schizophrenia is characterized by a wide range of symptoms that also appear in other disorders, which served as the basis for the emergence of ideas about schizophrenia spectrum disorders. Schizotypal disorder is a diagnostic phenotype within the schizophrenia spectrum with genetic, biological, and behavioral characteristics common to schizophrenia [1]. The most significant features for the clinical diagnosis of schizotypal disorder are cognitive-perceptual and disorganizational symptoms [2]. Experimental study of visual perception processes is important for clarifying the mechanisms of cognitive-perceptual disorders in both schizophrenia and schizotypal disorder.

Visual perception disorders occur in more than 60% of patients with schizophrenia and are represented by visual hallucinations, distortion of the perception of spatio-temporal characteristics of visual stimuli [3–9], decreased integration of contours [10, 11], perception of movement [12], and color [13]. There is very little data on visual perception disorders in other disorders that have symptoms similar to schizophrenia. J. Simon et al. [14], report high-level impairments of visual perception (visual gnosis) in patients with schizotypal disorder. The authors obtained data on changes at all stages of the formation of a complex image and its subsequent comparison with images from memory. While B.F. O'Donnell et al. [15], demonstrated no impairment of visual processing in the early stages of perception, even in the presence of pronounced schizotypal symptoms.

Although visual processing and attention deficits account for a significant portion of the disability associated with schizophrenia [16], the source of these impairments remains unclear. The results of experimental studies indicate the importance of coordinated interaction of two large-scale neural networks, the magno- and parvocellular systems, to ensure the integrity of visual perception, which is impaired in schizophrenia [3, 9, 17–20]. Magnocellular neurons of the lateral geniculate nucleus (LGN) of the thalamus at the cortical level give rise to the dorsal, and the parvocellular, to the ventral flow of information from the caudal to the prefrontal zones of the cerebral cor-

Table 1. Antipsychotic therapy outcomes in patients with schizophrenia and schizotypal disorder

Therapy		Schizophrenia	Schizotypal disorder
Antipsychotics	1st generation	5	0
	2nd generation	22	13
	3rd generation	10	5
Availability of enhancer	No	30	9
	Trihexyphenidyl	5	0
	Biperiden	2	1
Chlorpromazine equivalent		480(186)	412(195)

tex. Neurons of these systems are distinguished by their sensitivity to contrast, which is often used in studies of their functional activity. Contrast is the ratio of brightness between the light and dark phases of a pattern. Contrast sensitivity, recorded in different ranges of spatial frequencies, is a value inverse to the threshold contrast, i.e., the minimum distinguishable value of the difference in brightness. Spatial frequency is measured as the number of changes from light to dark (sinusoidal cycles) per angular degree (ang. deg) of the visual field. Patterns containing up to 0.5 cycles/deg have a low spatial frequencyand those from 7 cycles/deg have a high spatial frequency. Large cells of the magnocellular layers of the LGN of the thalamus are specific to the perception of low spatial frequencies, high frequencies are specific to small cells of the parvocellular layers [21, 22].

According to the results of most studies, a decrease in the activity of the magnocellular system is observed in schizophrenia [3, 23], while other authors, on the contrary, report an increase in its activity [17, 19, 24]; some studies report hypofunction of the parvocellular system [25], or a decrease in the function of both systems [3, 21, 26–28]. Most of these studies are based on data on the contrast sensitivity of the visual system. There are very few data regarding contrast sensitivity in individuals with schizotypal disorder. B.W. Kent et al. [29], in their study used only one spatial frequency of 0.5 cycles/deg and several options of temporal modulation: 0, 1, 5, 10, and 15 Hz. The authors demonstrated a decrease in contrast sensitivity in schizotypal disorder when presented with a stationary sinusoidal grating and a grating with a temporal frequency of 15 Hz. Thus, the results of the study indicate a decrease in the activity of the magnocellular system, while in this case only indirect conclusions can be made about the activity of the parvocellular system.

Objective—To investigate the characteristics of visual contrast sensitivity in schizophrenia and schizotypal disorder.

METHODS AND MATERIALS

The study involved 30 people without a history of neurological and psychiatric diseases (18 women, 31.0 ± 10.4 years); 37 patients (19 women, 35.6 ± 10.6 years) with a diagnosis of paranoid schizophrenia (*F*20 according to ICD-10), and 18 patients (10 women, 22.6 ± 3.4 years) with a diagnosis of schizotypal disorder (*F*21 according to ICD-10).

According to the protocol, the criteria for inclusion of patients in the study were:

— Age from 18 to 45 years.

— Diagnosis of paranoid schizophrenia (F20 according to ICD-10), as well as schizotypal disorder (F21 according to ICD-10).

— Vision is normal or corrected with glasses or lenses.

The exclusion criteria for patients were as follows:

— Ophthalmological diseases that are not corrected by glasses or lenses and lead to a decrease in visual functions.

— Organic lesions of the central nervous system (CNS).

— The presence of severe acute and chronic somatic diseases requiring the use of constant additional pharmacological therapy.

— Alcohol or drug addiction.

— For women: pregnancy or lactation period.

Patients were examined in a subacute state 1–2 weeks after admission to a hospital for treatment. All patients received antipsychotic therapy (Table 1).

For participants in the control group, the inclusion criteria for the study were: age 18–45 years, no history of neurological or psychiatric pathologies, and normal vision or vision corrected to normal with lenses or glasses. The exclusion criteria corresponded to those in the patient group. The day before the study, participants were asked not to drink alcohol, and on the day of the study, not to drink caffeine, and to refrain from smoking.

Contrast sensitivity was determined using the adaptive staircase procedure [30, 31]. During binocular presentation, the threshold contrast was recorded using a computer program that allows test images to be generated on a monitor of any type without prior calibration. The program was developed under the supervision of S.I. Lyapunov (Prokhorov General Physics

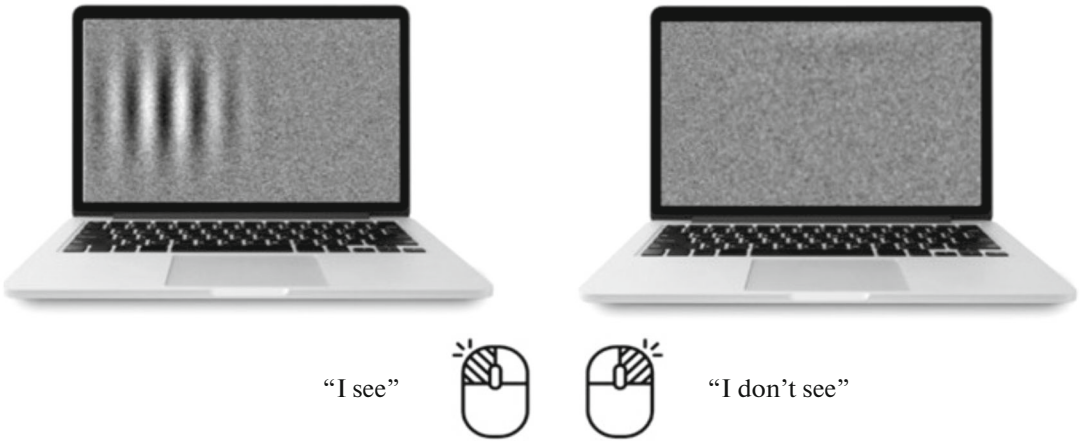

Fig. 1. Demonstration of the procedure for registering contrast sensitivity.

Institute, Russian Academy of Sciences, Moscow). The stimuli used were Gabor elements—gratings with a brightness distribution that obeys a sinusoidal law, with a decrease in the amplitude of the sinusoid from the center to the edges of the stimulus according to the Gaussian law [32]. Stimuli with spatial frequencies of 0.4; 1.0; 3.0; and 10.0 cycles/deg were presented on the screen of an HP Pavilion Aero 13-be0822nw (61R48EA) AMD Ryzen 7 (size 13.3″) against a mask in the form of additive white noise with a refresh rate 60 Hz. The distance from the subject to the monitor screen was 53 cm. Stimuli were displayed to the left or right of the center of the screen, starting with low spatial frequencies. When analyzing the data, the frequency of 0.4 cycles/deg was considered to be a low spatial frequency range, the frequency of 1.0 and 3.0 cycles/deg was considered to be a medium spatial frequency range, and the frequency of 10.0 cycles/deg was considered to be a high spatial frequency range. The subject's task was to press the left mouse button when he saw a stimulus, and the right mouse button when the stimulus was absent (Fig. 1).

The head position of the study participant was maintained immobile using a standard forehead-chin rest.

Statistical processing was carried out using IBM SPSS Statistics 26. As a statistical method of data analysis, nonparametric criteria for two or more unrelated samples were chosen: Mann–Whitney *U*-criterion and Kruskal–Wallis *H*-test; the choice is related to the fact that the Kolmagorov–Smirnov test did not reveal a normal distribution at all frequencies, and also due to the unequal dispersions of the compared groups [33]. In this case, the arguments of the functions were two numerical vectors (two samples). The groups were analyzed in pairs: healthy norm–schizotypal disorder, healthy norm–paranoid schizophrenia, paranoid schizophrenia–schizotypal disorder. Bonferroni correction for multiple comparisons was used. Correlation analysis was performed using Spearman's correlation coefficient.

RESEARCH RESULTS

Clinical picture. Results of the assessment of the clinical picture of the condition of the study participants suffering from schizophrenia and schizotypal disorder, using *PANSS* are presented in Table 2. Significant differences between patient groups were found only for the total scale of positive symptoms.

Table 2. Clinical characteristics of groups of patients suffering from schizophrenia and schizotypal disorder

Clinical characteristics	Schizophrenia	Schizotypal disorder	*p-value* of Kruskal–Wallis
Duration of the disease	10.9(8.9)	7.0(3.4)	0.172
SAS	2.2(1.5)	2.6(2.2)	0.595
PANSS positive scale	14.3(3.9)	10.2(2.5)	0.006*
PANSS negative scale	19.8(5.8)	15.9(5.4)	0.174
PANSS General Psychopathology Scale	32.7(6.9)	32.4(4.6)	0.638
PANSS total score	66.8(13.7)	58.5(9.1)	0.188
Chlorpromazine equivalent	480(186)	412(195)	0.631

* $p < 0.01$.

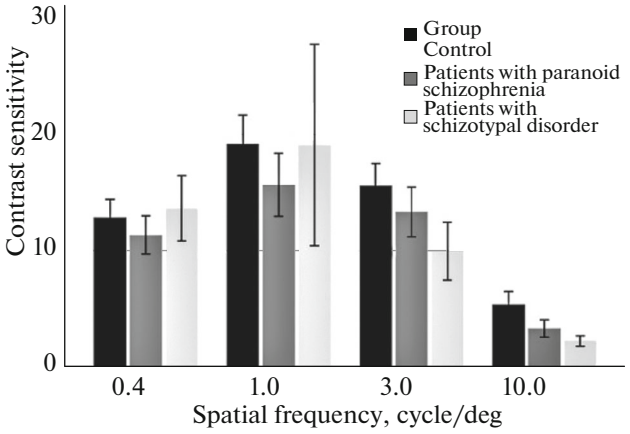

Fig. 2. Contrast sensitivity in different spatial frequency ranges in schizophrenia, schizotypal disorder and in a healthy control group.

Contrast sensitivity. The average values of contrast sensitivity (mean ± standard deviation) in the group of patients with paranoid schizophrenia when presented with Gabor elements containing a low spatial frequency of 0.4 cycles/deg were 11.4 ± 4.4, average frequencies of 1.0 cycles/deg were 15.7 ± 7.2 and 3.0 cycles/deg were 13.3 ± 5.7, and high spatial frequency of 10 cycles/deg was 3.3 ± 2.0 (Fig. 2).

In the group of patients with schizotypal disorder, the indicators were, respectively, 13.6 ± 4.34, 19.1 ± 9.9, 9.9 ± 3.4, and 2.2 ± 0.6; in the healthy control group, 12.4 ± 4.1, 19.2 ± 6.6, 15.6 ± 5.1, and 5.4 ± 3.0, respectively.

Results of statistical analysis using the Kruskal–Wallis criterion indicates a decrease, compared with the participants of the control group, in contrast sensitivity in patients with paranoid schizophrenia when perceiving stimuli with a spatial frequency of 10 cycles/deg ($Z = -2.772$, $p = 0.017$), in patients with schizotypal disorder when perceiving stimuli with a spatial frequency of 3 cycles/deg ($Z = -0.001$, $p = 0.004$) and 10 cycles/deg ($Z = -3.878$, $p = 0.000$). No significant differences were found between the contrast sensitivity indices of the groups of patients with paranoid schizophrenia and schizotypal disorder at all spatial frequencies.

Correlation analysis of clinical indicators of *PANSS* patients with schizophrenia showed a significant correlation of scores on the general psychopathology scale with contrast sensitivity in the range of average spatial frequencies (3 cycles/deg, $r = 0.37$, $p = 0.02$). Whereas in the group of patients with schizotypal disorder, a significant correlation was found for scores *PANSS* scales of negative symptoms with contrast sensitivity, also in the range of medium spatial frequencies ($r = 0.67$, $p = 0.04$). Thus, for these groups of subjects, a correlation was found between clinical indicators and contrast sensitivity in the range of average spatial frequencies, the perception of which is provided by neurons of both the magno- and parvocellular systems.

No correlations were found between the contrast sensitivity indices in the studied spatial frequency ranges and the chlorpromazine equivalent value or the Parkinsonism severity scale scores in any of the patient groups. However, this does not remove the limitation of the present study related to the possible influence of antipsychotic therapy on the magnitude of contrast sensitivity.

DISCUSSION OF RESULTS

The results of the present study indicate changes in contrast sensitivity of the visual system in both schizophrenia and schizotypal disorder, compared with healthy controls. In both cases, differences are observed in the perception of high spatial frequencies, to which parvocellular neurons are more specific [21, 22]. The decrease in the activity of this system, compared to the magnocellular system, the sensitivity of which does not differ from the indicators of healthy controls, indicates a special nature of the discordance of these neural systems in patients with similar symptoms. The obtained data are considered by us as confirmation of the hypothesis about the dominance of the magnocellular system of information perception over the complementary parvocellular system in schizophrenia and schizotypal disorder. That is, the results of the present study indicate a special nature of the interaction of the magno- and parvosystems in schizophrenia and schizotypal disorder.

The importance of the coordinated work of these complementary systems is determined by the fact that their interaction at different levels of information processing ensures the integrity of perception. The discrepancy in the work of the magno- and parvocellular systems also manifests itself in the perception of average spatial frequencies, in the processing of which they mutually participate [21]. Misalignment in their work can be considered as one of the factors increasing the level of internal noise in the visual system in psychopathology [21, 34]. In the group of patients with schizotypal disorder, the dispersion of the contrast sensitivity index in the range of average spatial frequencies is much higher than in the group of patients with schizophrenia. It is possible that the reason is the smaller number of subjects in this group.

Several studies of contrast sensitivity in individuals with different durations of schizophrenia have shown increased activity of the magnocellular system, in particular in patients with a first psychotic episode who have not received long-term antipsychotic therapy [17, 19, 24, 35, 36]. In this case, the activity of the parvocellular system remained consistent with healthy controls or was reduced. Thus, in these cases, a different nature of the mismatch in the interaction of the

magno- and parvocellular neural systems was observed. The present study included patients in a subacute condition. In this regard, we expected to see higher values of contrast sensitivity to stimuli containing low spatial frequencies, to which neurons of the magnocellular system are more specific.

The absence of such differences, compared with healthy controls, in both groups of patients may be due to the absence in our sample of patients with a first episode who did not receive drug treatment [24, 35]. Accordingly, the influence of antipsychotic therapy that patients received over many years, including at the time of participation in the study, on the contrast sensitivity indices cannot be ruled out.

Contrast perception, which begins at the level of the retina, is associated with the level of its dopamine activity [37]. Dopamine improves visual contrast detection by mediating lateral inhibition via D_2 receptors [38]. In research J.P. Harris et al. [39], in eight patients with schizophrenia, contrast thresholds were measured during the presentation of stationary gratings with spatial frequencies of 0.5, 2.0, and 8.0 cycles/deg first before and then 2 to 3 days after a therapeutic injection of a typical depot neuroleptic (stelazine, haloperidol). The drug increased contrast sensitivity at low and decreased it at medium and high spatial frequencies. The results of our early studies have shown that patients with schizophrenia treated with atypical antipsychotics demonstrated a more pronounced decrease in contrast sensitivity in the low spatial frequency region than patients treated with typical antipsychotics [18]. Then, as in the study by Y. Chen et al. [35], patients receiving atypical antipsychotics showed unimpaired visual contrast detection (for the perception of a grating with a spatial frequency of 0.5 cycles/deg and a temporal modulation of 5 Hz), while those receiving typical neuroleptics showed higher contrast detection thresholds.

Most of the patients in the present study were receiving second-generation antipsychotic therapy. The therapeutic efficacy of drugs in this group is determined by the coefficient reflecting the ratio of 5-HT2a affinity to D_2-affinity. That is, such drugs have an effect on both dopamine and serotonin receptors and are dopamine–serotonin antagonists (sertindole, olanzapine, aripiprazole, etc.). Blockade of 5-HT2a receptors in mesocortical structures indirectly promotes an increase in dopamine content in the prefrontal cortex of the brain, which may determine the procognitive effect of second-generation antipsychotics. Third-generation drugs, which some of our patients also received, are partial dopamine receptor blockers. In any case, all antipsychotic drugs used to treat schizophrenia spectrum disorders affect dopamine receptors, reducing the level of dopamine in the mesocortical structures.

It can be assumed that patients with schizotypal disorder are characterized by less subcortical dopaminergic hyperreactivity than patients with schizophrenia [1, 40]. Indeed, in our group of participants with schizotypal disorder, positive clinical symptoms were significantly less pronounced than in the group of patients with schizophrenia. However, in terms of *PANSS,* the patients did not differ on the general psychopathology scale. This may be why we did not observe differences in contrast sensitivity of the visual system in patients with schizophrenia and schizotypal disorder. No such differences were found in contrast sensitivity and B.F. O'Donnell et al. [15].

Several studies have reported that patients with schizotypal personality disorder exhibit more specific visual processing deficits, which are evident when performing tasks involving working memory [2, 15, 41, 42]. L.J. Siever and K.L. Davis [1] reported preservation of prefrontal cortex volume in individuals with schizotypal disorder and reduced dopamine activity in the striatum, compared with chronic schizophrenia patients. These factors explain the results of A.A. Chepeliuk and M.G. Vinogradova on the presence of compensatory strategies in individuals with schizotypal disorder that increase the efficiency of task performance to a normative level [2]. In the present study, we failed to detect differences in performance on an early-level contrast detection task between patients with schizophrenia and schizotypal disorder.

Also, significant correlations of scores on the scales were established for both groups of patients. *PANSS* and contrast sensitivity in the range of medium spatial frequencies. Direct correlation dependences indicate that the increase in scales is associated with an increase in contrast sensitivity in the range of medium spatial frequencies. Processing of average spatial frequencies is carried out by neurons of both the magnocellular and parvocellular systems. The obtained data can be considered as evidence of the importance of the interaction of these systems and that the change in state will be reflected, first of all, in contrast sensitivity in the range of medium spatial frequencies. Confirmation or refutation of such a justification can be obtained as a result of the analysis of data from longitudinal studies currently being conducted.

CONCLUSIONS

Participants in the study with schizophrenia and schizotypal disorder demonstrated reduced contrast sensitivity in the high spatial frequency range compared to healthy controls. The obtained data are considered as evidence of the peculiar nature of the discordance in the interaction of the magnocellular and parvocellular channels of the visual system with a shift towards the dominance of the magnocellular system.

It can be assumed that disturbances in low-level sensory processes can affect high-level cognitive processes and lead to the manifestation of psychotic symptoms characteristic of schizophrenia [34, 43, 44].

FUNDING

The work was carried out with financial support from the Russian Science Foundation (grant No. 24-25-00494).

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

All studies were conducted in accordance with the principles of biomedical ethics set out in the 1964 Helsinki Declaration and its subsequent amendments. They were also approved by the local bioethics committee of the National Medical Research Center of Psychiatry and Neurology named after V.M. Bekhterev (St. Petersburg), protocol no. EK-I-120/19 dated October 24, 2019.

Each research participant or their legal representative (parent) gave voluntary written informed consent after receiving an explanation of the potential risks and benefits, as well as the nature of the upcoming study and the right to withdraw from the study at any stage without explanation.

CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

REFERENCES

1. Siever, L.J. and Davis, K.L., The pathophysiology of schizophrenia disorders: perspectives from the spectrum, *Am. J. Psychiatry*, 2004, vol. 161, no. 3, p. 398.
2. Chepeliuk, A.A. and Vinogradova, M.G., The performance of visual perceptual tasks in patients with schizotypal personality disorder, *Psychol. Russ.: State Art.*, 2021, vol. 14, no. 2, p. 42. https://doi.org/10.11621/pir2021.0204
3. Zemon, V., Herrera, S., Gordon, J., et al., Contrast sensitivity deficits in schizophrenia: a psychophysical investigation, *Eur. J. Neurosci.*, 2021, vol. 53, no. 4, p. 1155.
4. Jurišiü, D., Cûavar, I., Sesar, A., et al., New insights into schizophrenia: a look at the eye and related structures, *Psychiatr. Danubina*, 2020, vol. 32, no. 1, p. 60.
5. Tumova, M.A., Shoshina, I.I., Stanovaya, V.V., et al., Cognitive functioning and visual system characteristics in schizophrenia: a cross-sectional study, *Psikhiatriya*, 2023, vol. 21, no. 3, p. 36.
6. Silverstein, S.M., Keane, B.P., Demmin, D.L., and Fradkin, S.I., Visual impairments in schizophrenia: their significance and unrealized clinical potential, *Psychiatr. Danubina*, 2020, vol. 32, no. 1, p. 72.
7. Shoshina, I.I., Mukhitova, Yui.V., Tregubenko, I.A., et al., Contrast sensitivity of the visual system and cognitive functions in schizophrenia and depression, *Hum. Physiol.*, 2021, vol. 47, no. 5, p. 516. https://doi.org/10.1134/S0362119721050121
8. Fernandes, T.P., Shoshina, I.I., Oliveira, M.E.C., et al., Correlates of clinical variables on early-stage visual processing in schizophrenia and bipolar disorder, *J. Psychiatr. Res.*, 2022, vol. 149, p. 323.
9. Kosikova, A.V., Shoshina, I.I., Lyapunov, S.I., et al., Characteristics of visual contrast sensitivity and ocular microtremor in schizophrenia, *Psikhiatriya*, 2024, vol. 22, no. 1, p. 58.
10. Keane, B.P., Paterno, D., Kastner, S., et al., Intact illusory contour formation but equivalently impaired visual shape completion in first- and later-episode schizophrenia, *J. Abnorm. Psychol.*, 2019, vol. 128, no. 1, p. 57.
11. Keane, B.P., Erlikhman, G., Serody, M., and Silverstein, S.M., Spatial frequency contributions to contour integration deficits in schizophrenia, *Schizophr. Res.*, 2022, vol. 240, p. 78.
12. Martínez, A., Gaspar, P.A., Hillyard, S.A., et al., Impaired motion processing in schizophrenia and the attenuated psychosis syndrome: etiological and clinical implications, *Am. J. Psychiatry*, 2018, vol. 175, no. 15, p. 1243.
13. Fernandes, T.M.P., Silverstein, S.M., Butler, P.D., et al., Color vision impairments in schizophrenia and the role of antipsychotic medication type, *Schizophr. Res.*, 2019, vol. 204, p. 162.
14. Simon, Yu.A., Bizyuk, A.P., Isaeva, E.R., et al., Specific perception (visual gnosis) and cognition in patients with schizotypal disorder, *Klin. Spets. Psikhol.*, 2018, vol. 7, no. 2, p. 97.
15. O'Donnell, B.F., Bismark, A., Hetrick, W.P., et al., Early stage vision in schizophrenia and schizotypal personality disorder, *Schizophr. Res.*, 2006, vol. 86, nos. 1–3, p. 89.
16. Arkin, S.C., Ruiz-Betancourt, D., Jamerson, E.C., et al., Deficits and compensation: attentional control cortical networks in schizophrenia, *NeuroImage Clin.*, 2020, vol. 27, p. 102348.
17. Shoshina, I.I., Shelepin, Yu.E., Vershinina, E.A., and Novikova, K.O., The spatial-frequency characteristics of the visual system in schizophrenia, *Hum. Physiol.*, 2015, vol. 41, no. 3, p. 251. https://doi.org/10.1134/S0362119715030159
18. Shoshina, I.I., Shelepin, Yu.E., and Semenova, N.B., Frequency-contrast sensitivity of visual stimulus perception in patients with schizophrenia treated with atypical and typical antipsychotics, *Hum. Physiol.*, 2014, vol. 40, no. 1, p. 35. https://doi.org/10.1134/S0362119714010150
19. Isaeva, E.R., Tregubenko, I.A., Mukhitova, Yu.V., and Shoshina, I.I., Functional states of the magnocellular and parvocellular neural systems and cognitive impairments in schizophrenia at different stages of the disease, *Ross. Psychol. Zh.*, 2021, vol. 18, no. 1, p. 74.
20. Ifrah, C., Herrera, S.N., Silverstein, S.M., et al., The relationship between clinical and psychophysical assessments of visual perceptual disturbances in individuals at clinical high risk for psychosis: a preliminary study, *Brain Sci.*, 2024, vol. 14, no. 8, p. 819.
21. Shoshina, I.I. and Shelepin, Yu.E., *Mekhanizmy global'nogo i lokal'nogo analiza zritel'noi informatsii pri shizofrinii* (Mechanisms of Global and Local Analysis of Visual Information in People with Schizophrenia), St. Petersburg: VVM, 2016.
22. Edwards, M., Goodhew, S.C., and Badcock, D.R., Using perceptual tasks to selectively measure magnocellu-

lar and parvocellular performance: Rationale 97 and a user's guide, *Psychon. Bull. Rev.*, 2021, vol. 28, no. 4, p. 1029.

23. Calderone, D.J., Hoptman, M.J., Martínez, A., et al., Contributions of low and high spatial frequency processing to impaired object recognition circuitry in schizophrenia, *Cereb. Cortex*, 2013, vol. 23, no. 8, p. 1849.
24. Kiss, I., Fábián, Á., Benedek, G., and Kéri, S., When doors of perception open: visual contrast sensitivity in never-medicated, first-episode schizophrenia, *J. Abnorm. Psychol.*, 2010, vol. 119, no. 3, p. 586.
25. Muraveva, S.V., Pronina, M.V., Moiseenko, G.A., et al., Analysis of visual cognitive impairments in schizophrenia at the early stages of the disease and their correction by interactive virtual environment, *Hum. Physiol.*, 2017, vol. 43, no. 6, p. 625. https://doi.org/10.1134/S0362119717060056
26. Almeida, N.L., Fernandes, T.P., Lima, E.H., et al., Combined influence of illness duration and medication type on visual sensitivity in schizophrenia, *Braz. J. Psychiatry*, 2019, vol. 42, no. 1, p. 27.
27. Shoshina, I.I., Shelepin, Yu.E., Vershinina, E.A., and Novikova, K.O., Functional features of the magnocellular and parvocellular systems in schizophrenia, *Psikhol. Psikhofiziol.*, 2014, vol. 7, no. 4, p. 77.
28. Shoshina, I.I., Shelepin, Yu.E., Vershinina, E.A., and Novikova, K.O., The spatial-frequency characteristics of the visual system in schizophrenia, *Hum. Physiol.*, 2015, vol. 41, no. 3, p. 251.
29. Kent, B.W., Weinstein, Z.A., Passarelli, V., et al., Deficient visual sensitivity in schizotypal personality disorder, *Schizophr. Res.*, 2011, vol. 127, nos. 1–3, p. 144.
30. Nadler, M.P., Miller, D., and Nadler, D.J., *Glare and Contrast Sensitivity for Clinicians*, New York: Springer-Verlag, 1990.
31. Yu, Q., Zhang, P., Qiu, J., and Fang, F., Perceptual learning of contrast detection in the human lateral geniculate nucleus, *Curr. Biol.*, 2016, vol. 26, no. 23, p. 3176.
32. Belozerov, A.E., Theoretical evaluation of three-band stimuli as optotypes for measuring visual acuity in comparison with Gabor elements, *Sens. Syst.*, 2013, vol. 27, no. 2, p. 108.
33. Kupriyanov, I.V., Calculation of the effect size when using nonparametric tests for comparing samples in psychological research using the R software, *Inzh. Vestn. Dona*, 2016, no. 2, p. 38. http://www.ivdon.ru/ru/magazine/archive/n2y2016/3610.
34. Shoshina, I.I., Mukhitova, Yu.V., Tregubenko, I.A., et al., Contrast sensitivity of the visual system and cognitive functions in schizophrenia and depression, *Hum. Physiol.*, 2021, vol. 47, no. 5, p. 516. https://doi.org/10.1134/S0362119721050121
35. Chen, Y., Levy, D., Sheremata, S., et al., Effects of typical, atypical, and no antipsychotic drugs on visual contrast detection in schizophrenia, *Am. J. Psychiatry*, 2003, vol. 160, p. 1795.
36. Kéri, S. and Benedek, G., Visual contrast sensitivity alterations in inferred magnocellular pathways and anomalous perceptual experiences in people at high risk for psychosis, *Visual Neurosci.*, 2007, vol. 24, p. 183.
37. Masson, G., Mestre, D., and Blin, O., Dopaminergic modulation of visual sensitivity in man, *Fundam. Clin. Pharmacol.*, 1993, vol. 7, no. 8, p. 449.
38. Djamgoz, M.B., Hankins, M.W., Hirano, J., and Archer, S.N., Neurobiology of retinal dopamine in relation to degenerative states of the tissue, *Vision Res.*, 1997, vol. 37, no. 24, p. 3509.
39. Harris, J.P., Calvert, J.E., Leendertz, J.A., and Phillipson, O.T., The influence of dopamine on spatial vision, *Eye*, 1990, vol. 4, part 6, p. 806.
40. Abi-Dargham, A., Kegeles, L.S., Zea-Ponce, Y., et al., Striatal amphetamine-induced dopamine release in patients with schizotypal personality disorder studied with single photon emission computed tomography and iodobenzamide, *Biol. Psychiatry*, 2004, vol. 55, no. 10, p. 1001.
41. Farmer, C.M., O'Donnell, B.F., Niznikiewicz, M.A., et al., Visual perception and working memory in schizotypal personality disorder, *Am. J. Psychiatry*, 2000, vol. 157, no. 5, p. 781.
42. Maróthi, R. and Kéri, S., Enhanced mental imagery and intact perceptual organization in schizotypal personality disorder, *Psychiatry Res.*, 2018, vol. 259, p. 433.
43. Javitt, D.C., When doors of perception close: bottom-up models of disrupted cognition in schizophrenia, *Annu. Rev. Clin. Psychol.*, 2009, vol. 5, p. 249.
44. Dias, E.C., Butler, P.D., Hoptman, M.J., and Javitt, D.C., Early sensory contributions to contextual encoding deficits in schizophrenia, *Arch. Gen. Psychiatry*, 2011, vol. 68, no. 7, p. 654.