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Review

Colour vision defects in schizophrenia spectrum disorders: A systematic review

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ABSTRACT

This systematic review synthesized the existing literature to summarize colour vision disturbances experienced by patients with schizophrenia. A comprehensive literature search compliant with PRISMA-2020 was conducted in Medline and Embase from inception to February 28, 2023. Studies were included if they: (1) included people diagnosed with schizophrenia, (2) investigated colour vision, (3) had a comparator with or without schizophrenia. Study quality appraisal was performed using the NIH Study Quality Assessment Tool. Seven studies of fair quality with 695 patients were included, of whom, 46.5% (n = 323) patients were diagnosed with a schizophrenia-spectrum disorder. Compared to healthy controls, patients with schizophrenia either made more mistakes in discriminating between colours, or were delayed in recognizing colours. One study found that Positive and Negative Syndrome Scale for Schizophrenia (PANSS) scores correlated weakly with error scores related to colour vision impairments. The most common shortcomings were lack of sample size justification (k = 7, 100%), and lack of blinding (k = 7, 100%). Our review indicates early evidence of colour vision deficits among patients with schizophrenia, and an unclear relationship between severity of schizophrenia with colour vision deficits. Possible mechanisms may include alterations in retinal dopamine transmission or schizophrenia-related cognitive deficits interacting with colour vision outcomes. Future studies may benefit from large registry analyses of patients with various schizophrenia spectrum disorders, analyzing ocular parameters (e.g., OCT), collecting

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data on cognitive impairment, and pursuing multivariate analyses to elucidate mechanisms for schizophrenia-related colour vision changes.

1. Introduction

Visual impairments are commonly seen among patients with schizophrenia spectrum disorders (Butler et al., 2001; Fernandes et al., 2019a). In fact, among a survey of patients with schizophrenia, up to 52% of participants reported visual distortions before seeking formal treatment for their condition (Phillipson and Harris, 1985). Most commonly, contrast perception, forward and backward masking, and colouration, among other visual deficits, are dysregulated in schizophrenia (Butler et al., 2001; Green et al., 2011; Kiss et al., 2010). Pathophysiologically, there are various hypotheses implicating critical structures in the visual pathway including the retina, optic nerve, lateral geniculate nuclei, and primary visual cortex. Particularly, visual signal alterations are thought to be influenced by fluctuating dopamine, acetylcholine, GABA, and NMDA neurotransmitter levels, which cause aberrations in cortical functioning and subsequent reality distortions in the form of visual symptoms (Adámek et al., 2022; Fornaro et al., 2011, 2014). Previously, retinal dopamine levels were associated with colour vision deficits among patients with schizophrenia, which was further supported by the influence of antipsychotic type on colour vision symptoms (Fernandes et al., 2019b).

The relationship between visual deficits and psychotic disorders are suspected to be bidirectional with strong correlation with underlying genetic predispositions. (Ramsay et al., 2020; Schubert et al., 2005) Vision has diagnostic and prognostic value among patients with schizophrenia, and there is an association with quality of life with vision in these populations including elevated risk of suicide and poor real-life functioning (Granö et al., 2015; Silverstein and Rosen, 2015; McCleery et al., 2020). To date, most studies investigating visual deficits among patients with schizophrenia have primarily focused on visual deficits in isolation and focused on discerning the pathophysiologic basis of such deficits. The disease burden of primary psychotic disorders with comorbid visual deficits imposes further challenges in daily functioning including exacerbating visual hallucinations and social isolation (Granö et al., 2015; Silverstein and Rosen, 2015; McCleery et al., 2020).

Despite the growing attention, there are no systematic reviews, to our knowledge, synthesizing visual colour discrimination outcomes in this population. Discerning the bidirectional relationship between vision deficits and schizophrenia spectrum disorders may provide greater prognostic insight to identify at-risk populations and inform screening protocols. Thus, this systematic review aims to synthesize the existing literature to summarize colour vision disturbances experienced by patients with schizophrenia, and generate hypothesis-generating trends with respect to predictors and outcomes.

2. Methods

The protocol was uploaded to OSF *a priori* at https://osf.io/8dja4/. Of note, this manuscript shares the same protocol with another pertaining to bipolar disorder and vision. We chose to dedicate a separate manuscript due to volume of information, and for clarity purposes.

2.1. Search strategy and inclusion criteria

MEDLINE, and EMBASE were searched for studies that investigated the bidirectional relationship between schizophrenia and colour vision deficits. A manual search of the Cochrane trial register and ClinicalTrials.gov was also performed. A librarian (RS) was involved to optimize the search strategy. The search key is available in Table A1. The search terms were also entered into Google Scholar and PsychINFO, and a hand search was performed to ensure that relevant articles were not missed.

Studies were included if they met the following criteria: (1) included people diagnosed with schizophrenia (based on DSM, ICD, or clinical diagnosis), (2) investigated colour vision (as measured by Ishihara plates or any other tools to measure colour vision), (3) had a comparator with or without schizophrenia. No restrictions were applied in terms of age or language. The primary outcome was colour vision, as measured by Ishihara plates, and secondary outcomes included colour discrimination, as measured by the Dichotomous-15 (D-15) Color Blindness Test or Cambridge Colour Test. Of note, we excluded studies with the Stroop test given their particular focus on attention as opposed to colour vision.

2.2. Study screening

The studies were imported into COVIDENCE, through which study screening was conducted. The PRISMA-2020 guidelines were employed from title to full-text screening stages, and the process was performed in duplicate by independent reviewers (AG, JT, NF, VD, KL, IL, SW, SL) for this study, after a calibration exercise (Page et al., 2021). Discrepancies were discussed and resolved with consensus between both reviewers, and a third adjudicator. The references of included studies were also screened using the same systematic approach to capture any additional relevant articles.

2.3. Data extraction

Reviewers (AG, JT, NF, VD, KL, IL, SW, SL) independently extracted relevant data from the included articles and recorded the data onto a Microsoft Excel spreadsheet designed *a priori*. Demographic data was also collected, including age, sex, gender, ethnicity, study country, sample size, and year of publication. Information regarding clinical presentation (i.e., symptoms, diagnostic criteria), and history of physical or mental comorbidities was also gathered. Discrepancies were discussed and resolved with consensus between both reviewers, and a third adjudicator.

2.4. Quality assessment/risk of bias

Quality assessment was independently conducted by seven investigators (NF, KL, VD, IL, SW, IYZM, SL) using the NIH Study Quality Assessment Tool. Conflicts in the quality assessment were discussed to reach a consensus. The following scores were used for cohort (prospective or retrospective) and cross-sectional studies: 0–5 (poor), 6–10 (fair), 11–14 (good). While the following scores were used for case-series: 0–3 (poor), 4–6 (fair), and 7–9 (good).

2.5. Statistical analysis

Heterogeneity among the included studies' outcomes, and follow up times precluded our ability to perform a meta-analysis. Therefore, all studies were narratively synthesized.

3. Results

3.1. Study and participant characteristics

Fig. 1 illustrates the screening process. We identified 2957 studies. After removing 551 duplicates, 2406 studies were screened. One hundred thirty five full texts were reviewed and 128 were excluded. Seven studies, published between 2002 and 2022, met inclusion criteria.

Table 1 summarizes individual study information and patient

characteristics. Studies most commonly originated from Europe (k = 2, 28.6%). Five studies (71.4%) were of cross-sectional study design, one study (14.3%) was prospective cohort study design, and one study (14.3%) was case-control study design. Six studies (85.7%) had general population as controls, and one study (14.3%) had healthy controls.

Altogether, 695 patients were included, with mean age 38.1 (SD = 7.4) years and at least 41.3% (n = 287) of the sample were female. Approximately, 46.5% (n = 323) patients were diagnosed with a schizophrenia-spectrum disorder, with the remainder of patients being the general population (39.1%, n = 272), siblings (12.4%, n = 86), or healthy controls (2.0%, n = 14). All studies used either DSM-IV or DSM-V criteria.

3.2. Colour vision

Table 2 summarizes individual study results as meta-analysis was not possible.

Three studies assessed colour vision through D-15 tests. Generally, patients with schizophrenia demonstrated greater total error scores for D-15 tests, and total PANSS was weakly correlated with D-15 scores. Particularly, Shuwairi et al. (2002) assessed 16 patients with schizophrenia and 14 healthy controls, among whom no differences in colour-axis-specific errors were observed in both Farnsworth and Lanthony D-15 tests (Shuwairi et al., 2002). However, patients with schizophrenia made greater random errors on both the Fanrsworth-Musell 100 Hue test (5.25 errors v. 1.5 errors, $p < 0.05$) and Lanthony New Colour Test (2.5 errors v. 0.3 errors, $p < 0.05$). Duan et al. (2022) assessed 61 patients with first-episode schizophrenia and 52 healthy controls, among who patients with schizophrenia had significantly higher Farnsworth-Munsell D-15 total error scores (19.3 v. 12.9, $p = 0.000$); however, their scores clinically corresponded to slightly colour blind/colour amblyopia (Duan et al., 2022). Variance in D-15 scores were weakly correlated with total PANSS score (r = 0.266, $p = 0.039$). Finally, Dahdouh et al. (2020) assessed 50 patients with predominantly chronic schizophrenia and severe psychosis, and 50 healthy controls (Dahdouh et al., 2020). Patients with schizophrenia had significantly higher Farnsworth-Munsell D-15 total error scores (30.3 v. 12.4, $p < 0.001$) corresponding to moderate colour blindness, and total PANSS weakly explained variance in error scores ($r = 0.310$, $p = 0.017$).

Fernandes and colleagues (2019) assessed 66 patients with schizophrenia and 64 healthy controls (Fernandes et al., 2019b). Across all 3 colour axes, compared to healthy controls, patients with schizophrenia had significantly greater chromatic discrimination scores on both trivector and ellipses tests (effect sizes not reported) corresponding to greater colour vision impairment. When comparing patients on typical versus atypical antipsychotics, there were non-significant differences in colour discrimination abilities on the Deutan (red-green) axis ($p = 0.089$). Finally, brief psychiatric rating scale scores weakly correlated with chromatic discrimination across all colour axes in both trivector and ellipse colour discrimination tests.

Keri and colleagues (2005) assessed 72 patients with schizophrenia and 60 healthy controls (Kéri et al., 2005). They observed moderate differences (Cohen's $d = 0.49$, $p = 0.01$) in patients with schizophrenia with more time spent per arc due to greater displacement in Vernier threshold tests – corresponding to greater colour vision impairment (98.3 s v. 86.0 s).

Kogata and Iidaka (2021) compared 16 patients with chronic schizophrenia to 15 healthy controls, among whom identical colour search accuracy rates were seen (98% v. 98%) (Kogata and Iidaka, 2021). However, on average, patients with schizophrenia had a slower response time (1235 ms v. 531 ms) and were significantly slower to respond in right visual fields as demonstrated by mean laterally index

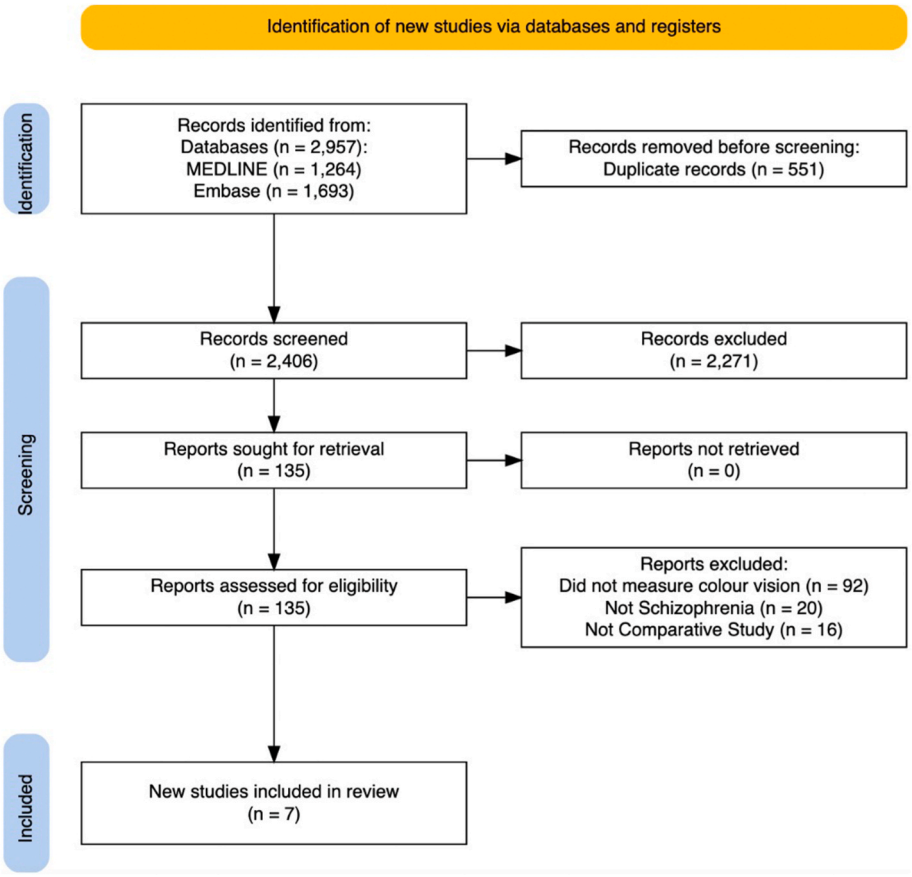

Fig. 1. Prisma diagram.

(1.4 v. 1.3, $z = 2.22$, $p = 0.03$).

Finally, Breboin and colleagues (2015) compared 50 patients with schizophrenia and 45 health controls (Brébion et al., 2015). Schizophrenia was significantly associated with reduced accuracy in recognizing both coloured and black-and-white pictures (effect size not reported, $p < 0.01$).

3.3. Quality assessment

All studies (k = 7, 100%) were of fair quality. The most common shortcomings were lack of sample size justification (k = 7, 100%), and lack of blinding (k = 7, 100%).

4. Discussion

This systematic review narratively synthesized 7 observational studies investigating colour vision outcomes among patients with schizophrenia. Studies generally included patients with schizophrenia of milder severity, as measured by the PANSS or BPRS. Our review's most notable findings were that patients with schizophrenia generally had non-specific colour blindness, and that severity of schizophrenia may correlate with severity of colour blindness symptoms.

Across the majority of included studies, patients with schizophrenia demonstrated non-specific colour vision deficits compared to healthy controls. Particularly, compared to healthy controls, patients with schizophrenia either made more mistakes in discriminating between colours, or were delayed in recognizing colours. In keeping with studies investigating the dopamine-inducing effects of antidepressants, such changes may be attributed to altered dopaminergic transmission (Fornaro et al., 2011, 2014; Djamgoz et al., 1997). While Lee et al. (2013) observed consistently reduced retinal nerve fibre layer (RNFL) and macular thickness among patients with schizophrenia, there is sparse literature implicating RNFL thickness with colour vision in patients with other diseases (i.e., multiple sclerosis, optic neuritis) (Lee et al., 2013; Barbur and Wong, 2022; Yekta et al., 2022; Bellato et al., 2023; Perna et al., 2023). Rather, as suggested by Polo and colleagues' (2016) survey of Parkinson's Disease patients in which RNFL thickness correlated with increased D-15 scores, structural and function colour vision changes in schizophrenia may be from alterations in dopamine (Polo et al., 2016). Likewise, among Büttner and colleagues' (1994) cohort of patients with Parkinson's Disease, those who were given L-dopa experienced significant reductions in colour vision error scores, further supporting the relationship between dopamine and colour vision (Büttner et al., 1994). However, in previous preclinical models, dopamine-related colour vision loss was thought to preferentially affect blue-sensitive cones, which may contradict our review's non-specific colour vision findings and suggest a multifactorial mechanism (Djamgoz et al., 1997). This may further be supported by Shuwairi et al. (2002) and Kogata and Iidaka (2021) finding no interaction between colour vision outcomes and antipsychotic use, and Fernandes and colleagues (2018) not observing a differential impact of second generation antipsychotics, with less hypodopaminergic effects, on tritan (blue-yellow) colour vision (Fernandes et al., 2019b; Shuwairi et al., 2002). Of note, these studies were of either case-control or cross-sectional design; thus, it is difficult to truly establish the temporality requirement for causation between schizophrenia or antipsychotic use and colour vision. Ultimately, without standardized documentation of retinal characteristics (e.g., optical coherence tomography) or functional magnetic resonance imaging (fMRI) studies, it is difficult to elucidate the specific mechanism.

The severity of schizophrenia may correlate with the extent of colour blindness, albeit weakly. For instance, where both Duan et al. (2022) and Dahdouh et al. (2020) assessed colour vision via the Farnsworth D-15 test, the latter study observed higher total error scores which may correlate with its higher baseline proportion of patients with severe PANSS-measured schizophrenia; however, neither study individually established a strong correlation between schizophrenia severity and colour blindness (Duan et al., 2022; Dahdouh et al., 2020). Given previous findings of dopamine receptor expression correlating with severity

Table 2
Summary of individual study results.

Study	Study Results
Shuwairi et al. (2002)	● Patients with schizophrenia made greater random errors on both the Fanrsworth-Musell 100 Hue test (5.25 errors v. 1.5 errors, p < 0.05) and Lanthony New Colour Test (2.5 errors v. 0.3 errors, p < 0.05).
Duan et al. (2022)	● Patients with schizophrenia had significantly higher Farnsworth-Munsell D-15 total error scores (19.3 v. 12.9, p = 0.000). ● Variance in D-15 scores were weakly explained by total PANSS score (r = 0.266, p = 0.039). ● No correlation between iris characteristics and F-15 scores.
Fernandes et al. (2019a,b)	● Patients with schizophrenia had higher chromatic discrimination thresholds than healthy controls (p < 0.001). ● Patients with schizophrenia had higher length's thresholds (p < 0.001), differences between the angle (p < 0.001), lower eccentricities values (p < 0.001), and larger area of the ellipses than controls for all 3 ellipses (p < 0.001). ● When comparing patients on typical versus atypical antipsychotics, there were non-significant differences in colour discrimination abilities on the Deutan (red-green) axis (p = 0.089)
Kéri et al. (2005)	● Patients with schizophrenia had greater colour vision impairment according to Vernier colour threshold test (98.3s v. 86.0s, $p = 0.01$).
Kogata and Iidaka (2021)	● Patients with schizophrenia had a slower reaction time when the target emerged from the right visual field than controls (1235ms v. 531ms, p-value NR) ● Patients with schizophrenia had a greater categorical perception effect for stimuli in the left visual field compared to the right visual field (mean laterally index, 1.4 v. 1.3, $z = 2.22$, $p = 0.03$).
Brébion et al. (2015)	● Schizophrenia was significantly associated with reduced accuracy in recognizing both coloured and black-and-white pictures (effect size not reported, p < 0.01).
Dahdouh et al. (2020)	● Total Farnsworth-Munsell D-15 error scores were higher in patients with schizophrenia than healthy controls (30.32 ± 7.42 v. 12.39 ± 1.0, $p < 0.001$). ● Total PANSS weakly explained variance in error scores (r = 0.310, p = 0.017).

Table 1
Study characteristics.

Study	Country	Design	Sample Size	Mean Age, years	% Female	Study Quality
Shuwairi et al. (2002)	USA	Cross Sectional	30 (16 schizophrenia, 14 healthy)	44.4	0.0	8/14 (Fair)
Duan et al. (2022)	China	Prospective Cohort	113 (61 schizophrenia, 52 general population)	29.0	64.6	10/14 (Fair)
Fernandes et al. (2019a,b)	Netherlands	Cross Sectional	108 (58 schizophrenia, 50 general population)	35.8	44.4	9/14 (Fair)
Kéri et al. (2005)	Hungary	Cross Sectional	218 (72 schizophrenia, 86 siblings, 60 general population)	NR	37.6	9/14 (Fair)
Kogata and Iidaka (2021)	Japan	Cross Sectional	31 (16 schizophrenia, 15 general population)	48.8	34.4	9/14 (Fair)
Brébion et al. (2015)	Spain	Cross Sectional	95 (50 schizophrenia, 45 general population)	46.2	36.0	9/14 (Fair)
Dahdouh et al. (2020)	Lebanon	Case-Control	100 (50 schizophrenia, 50 general population)	NR	36.0	9/14 (Fair)

Abbreviations: Not Reported (NR).

of schizophrenia, this finding may allude to dopamine-mediated colour vision changes (Duan et al., 2022; Cui et al., 2015). Notably, as our only prospective cohort study, Duan et al. (2022) observing reduced schizophrenia severity and total error scores among patients post-antipsychotic treatment contrasted with the lack of an impact of antipsychotics seen by the majority of non-cohort studies in this review (Duan et al., 2022; Cui et al., 2015). One possibility may be their inclusion of a sample of patients with mild MoCA-measured cognitive impairments. Oomen et al. (2023) previously observed that among patients with first-episode psychosis, severity of cognitive impairments were associated with poor prognosis of any schizophrenia-related symptoms (Oomen et al., 2023). In our review, as Kogata and Iikada (2021) attributed colour vision deficits to poor semantic processing from schizophrenia as observed by slower reaction times and lateralized effects, perhaps cognitive impairments may similarly interact with colour vision outcomes (Kogata and Iidaka, 2021). This is further supported by statistical correlation between MoCA scores and D-15 total error scores in Duan and colleagues' (2022) study, along with improvements in MoCA, PANSS, and D-15 scores after antipsychotic treatment (Duan et al., 2022). However, as Duan et al. (2022) only performed univariate analyses as opposed to multivariate regressions or ANCOVA, it is difficult to comment on any confounding or effect modification between cognitive impairment, schizophrenia severity, and colour vision.

Our systematic review's strengths are its rigorous methodology including a broad search strategy encompassing multiple databases and a manual hand search, and a duplicate systematic review approach to reduce reviewer bias. Studies were generally of fair quality on NIH assessment suggesting minimal influence of individual study biases on our results. Finally, our study is the first of its kind to clinically contextualize existing studies on colour vision and schizophrenia.

The most significant limitations of this review were that the majority of studies were of cross-sectional design which ultimately limited our ability to establish temporality between schizophrenia severity, antipsychotic use, cognitive impairment and colour vision outcomes. The limited documentation of data including baseline schizophrenia severity and cognitive impairment limits, and heterogeneity in outcome reporting for colour vision precluded meta-analysis and limited the strength or generalizability of our conclusions. Finally, as we search only 2 databases known to primarily have observational studies, our search strategy may be biased towards interventional studies. Future studies should establish large registries with various forms of schizophrenia spectrum disorders, have larger sample sizes, cohort designs, ocular parameters (e.g., OCT), and pursue multivariate analyses to elucidate mechanisms for schizophrenia-related colour vision changes.

5. Conclusion

Our review indicates early evidence of colour vision deficits among patients with schizophrenia, and an unclear relationship between severity of schizophrenia with colour vision deficits. Possible mechanisms may include alterations in retinal dopamine transmission or schizophrenia-related cognitive deficits interacting with colour vision outcomes. However, our preliminary results are limited primarily by studies of cross-sectional design and low sample sizes. Future studies may benefit from large registry analyses of patients with various forms and severities of schizophrenia spectrum disorders, analyzing ocular parameters (e.g., OCT), collecting data on cognitive impairment, and pursuing multivariate analyses to elucidate mechanisms for schizophrenia-related colour vision changes.

Collaboration

MS conceived the study, which was led by JT and AG. KL, VD, IL, and SW completed level I and II screening. IYZM and SL extracted the data and conducted quality appraisal under the supervision of JT. JT, AG, and MS drafted the work, and AG conducted the analyses. RS curated the search strategy. AZ, AS, and SC made substantial contributions to the interpretation of the results. NF made substantial contributions to the discussion. All authors critically revised the manuscript, approved the final version for publication, and agreed to be accountable for all the aspects of the work. JT and AG had full access to all the data in the study and can take responsibility for the integrity of the data and the accuracy of the data analysis.

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.nsa.2024.104046.

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