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Original Article

Effect of Chemical Exposure on Colour Vision

Sharmeen Shahid[1], Umar Siddique[1], Shoaib Rizwan[1], Mariyam Atta[1], Maria Naz[1], Ayesha Ghani[1]

[1] University of Lahore, Lahore, Pakistan

*Corresponding author: Sharmeen Shahid, Sharmeenshahid99@gmail.com

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ABSTRACT

Background: Occupational exposure to organic solvents and industrial chemicals may impair retinal or neuro-ophthalmic function, producing acquired colour vision defects that can remain undetected in workplace populations. Objective: This study aimed to evaluate the association between occupational chemical exposure and colour vision defects among industrial workers. Methods: A cross-sectional observational study was conducted at Pulcra Chemicals Pvt. Limited and Rudolf Chemicals Pakistan from July to November 2025. Eighty-two participants aged 20–60 years were included and divided equally into exposed and non-exposed groups. Participants with congenital colour blindness, ocular disease, systemic disease affecting vision, or medication use known to alter colour perception were excluded. Ishihara plates were used to screen for congenital red–green defects, and the Farnsworth D-15 test was used to assess acquired dyschromatopsia. Data were analyzed using SPSS, and associations were tested using chi-square and Fisher's exact tests. Results: Abnormal colour vision was identified in 33 of 41 exposed workers (80.5%) compared with 4 of 41 non-exposed participants (9.8%). The association between exposure status and D-15 findings was statistically significant, $\chi^2 = 42.866$, $p < 0.001$, with a large effect size. Tritan-type defects were the most frequent abnormalities among exposed workers. Conclusion: Occupational chemical exposure was strongly associated with acquired colour vision impairment, particularly blue–yellow defects. Routine colour vision screening and improved workplace safety measures are recommended. Keywords: Colour Vision Deficiency; Occupational Exposure; Organic Solvents; Farnsworth D-15 Test; Tritanopia; Industrial Workers.

INTRODUCTION

Colour vision is a specialized component of visual function that enables discrimination of chromatic differences through coordinated activity of retinal cone photoreceptors and post-retinal neural processing. Normal trichromatic vision depends mainly on short-, medium-, and long-wavelength cone systems, which allow perception across the blue, green, and red portions of the visible spectrum. Although inherited red–green colour vision deficiency is common and well described, acquired colour vision impairment is clinically distinct because it may develop later in life due to ocular disease, neurological dysfunction, systemic illness, medication use, nutritional deficiency, or environmental toxic exposure (1–6).

Occupational chemical exposure is an important but often under-recognized cause of acquired dyschromatopsia. Industrial workers may be exposed to organic solvents, heavy metals, pesticides, and other neurotoxic substances capable of affecting the retina, optic nerve, or visual pathways. Organic solvents such as toluene, xylene, styrene, propylene-based compounds, and carbon disulfide have been associated with impaired colour discrimination, particularly blue–yellow defects, because short-wavelength visual pathways appear vulnerable to toxic and metabolic injury. Such defects may remain unnoticed by workers until they interfere with visual performance, workplace safety, or routine occupational tasks, making screening clinically important in high-risk environments (7–13).

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Colour vision deficiency has public health relevance because visual discrimination is essential for daily functioning, occupational accuracy, hazard recognition, and quality of life. Population-based data show that inherited colour vision defects vary by sex and ethnicity, while acquired defects are influenced by age, ocular health, systemic disease, medication exposure, and environmental hazards. In Pakistan, where industrial safety monitoring and routine occupational vision screening may be inconsistent, workers exposed to chemical solvents may be at increased risk of undetected visual dysfunction. However, local evidence remains limited regarding the pattern and frequency of acquired colour vision abnormalities among industrial workers exposed to organic solvents and related chemicals (14–16).

Previous occupational studies have shown that workers exposed to solvents, pesticides, and heavy metals may demonstrate poorer performance on colour discrimination tests, including Farnsworth-based assessments. Blue–yellow impairment has been repeatedly described as an early indicator of solvent-related neurotoxicity, and colour vision testing has been proposed as a practical method for occupational surveillance. Despite this evidence, many available studies differ in exposure type, testing method, occupational setting, and participant characteristics, limiting direct generalization to Pakistani industrial settings. There is therefore a need for local data using standardized screening tools to determine whether chemical exposure is associated with acquired colour vision defects among workers in chemical-related industries (17–21).

This study was conducted to evaluate the association between occupational chemical exposure and colour vision defects among industrial workers using Ishihara plates to exclude congenital red–green colour vision deficiency and the Farnsworth D-15 test to assess acquired dyschromatopsia. The study hypothesis was that workers exposed to industrial chemicals would have a higher frequency of acquired colour vision abnormalities, particularly tritan-type defects, compared with non-exposed workers from similar sociodemographic backgrounds.

MATERIALS AND METHODS

This cross-sectional observational study was conducted at Pulcra Chemicals Pvt. Limited and Rudolf Chemicals Pakistan from July 2025 to November 2025. The study was designed to assess the association between occupational chemical exposure and acquired colour vision abnormalities among industrial workers. A cross-sectional design was selected because it allowed simultaneous assessment of exposure status and colour vision outcomes in the workplace population during the defined study period. The manuscript source reports 82 participants distributed equally into exposed and non-exposed groups, with 41 participants in each group; this sample size has therefore been used consistently in the revised section to resolve the earlier inconsistency between 80 and 82 participants.

Participants were recruited from industrial and non-exposed occupational groups after eligibility screening. The exposed group included workers aged 20–60 years who had worked for at least one year in environments involving occupational contact with chemicals or organic solvents, including manufacturing, painting, printing, agriculture-related chemical handling, and chemical processing work. The non-exposed group included individuals from similar sociodemographic backgrounds without routine occupational chemical exposure. Participants with known congenital colour blindness, pre-existing ocular disease such as glaucoma, cataract, or macular degeneration, systemic diseases known to affect vision such as diabetes mellitus or hypertension, and current or previous use of medications known to alter colour perception, including chloroquine or ethambutol, were excluded to reduce outcome misclassification and confounding.

Exposure status was operationally defined according to occupational history and workplace category. Participants were classified as exposed if they had a minimum of one year of work in a chemical-related industrial setting with reported exposure to solvents or industrial chemicals. Information was collected on duration of occupational exposure in years and type of chemical exposure, including commonly reported agents such as toluene/xylene, propylene/styrene, and carbon disulfide. Total years of exposure were grouped into 1–8 years, 9–16 years, and 17–24 years to describe exposure duration patterns and permit comparison across exposure categories. The main outcome variable was colour vision status assessed by the Farnsworth D-15 test and categorized as normal colour vision or abnormal colour vision, with defect pattern recorded where applicable, including tritanopia, deuteranopia, red–green defects, and mixed patterns.

Data collection was performed using a structured assessment process that included demographic information, occupational history, exposure duration, type of chemical exposure, and colour vision testing. Ishihara plates were used initially to screen for congenital red–green colour vision defects so that inherited colour vision abnormalities could be excluded or differentiated from acquired impairment. The Farnsworth D-15 test was then used to assess acquired dyschromatopsia and classify colour discrimination defects. Testing was performed under appropriate visual conditions using the same assessment protocol for exposed and non-exposed participants to minimize measurement bias. Participants were assessed using standardized instructions, and test findings were recorded immediately after assessment to preserve data accuracy.

Potential sources of bias were addressed through predefined eligibility criteria, use of the same colour vision assessment tools across both groups, and exclusion of participants with ocular, systemic, congenital, or medication-related causes of colour vision impairment. Selection bias was reduced by recruiting both exposed and non-exposed participants from comparable occupational and sociodemographic contexts. Confounding was addressed at the design stage through exclusion criteria and at the analysis stage by comparing colour vision outcomes between exposure groups. Age, duration of work, and exposure duration were considered clinically relevant variables because they may influence acquired visual dysfunction and occupational cumulative exposure.

Data were entered and analyzed using SPSS. Descriptive statistics were calculated for demographic and occupational variables, including frequencies and percentages for categorical variables such as age group, sex, exposure category, total years of exposure, and colour vision status. The chi-square test was used to examine the association between chemical exposure status and D-15 colour vision findings. Fisher's exact test was considered appropriate where expected cell counts were small because several colour vision defect categories had low frequencies. Statistical significance was set at $p < 0.05$. The primary comparison was the distribution of normal and abnormal colour vision findings between exposed and non-exposed participants, while secondary descriptive comparisons included defect patterns and exposure-duration categories.

Ethical approval was obtained from the relevant institutional review body before data collection, with approval number UOL/IRB/25/006/0008. Participants were included after informed consent, and confidentiality of participant information was maintained during data collection, analysis, and reporting. Data integrity was supported through standardized eligibility screening, uniform application of Ishihara and Farnsworth D-15 testing procedures, direct recording of findings, and use of statistical software for analysis.

RESULTS

A total of 82 participants were included, with equal distribution between the exposed group and non-exposed group, each comprising 41 participants. Most participants were aged 30–40 years, representing 37 participants (45.1%), followed by 23 participants (28.0%) aged 41–50 years and 19 participants (23.2%) aged 21–30 years. Only 3 participants (3.7%) were in the 51–60-year age category. The study population was predominantly male, with 78 male participants (95.1%) and 4 female participants (4.9%), reflecting the male-dominated nature of the industrial occupational setting.

Table 1. Demographic Characteristics of Participants

Variable	Category	Frequency (n)	Percentage (%)
Age group	21–30 years	19	23.2
	30–40 years	37	45.1
	41–50 years	23	28.0
	51–60 years	3	3.7
	Total	82	100.0
Sex	Male	78	95.1
	Female	4	4.9
	Total	82	100.0

Among participants with recorded occupational exposure duration, the largest proportion had 1–8 years of exposure, accounting for 51 participants (62.2%). A further 25 participants (30.5%) had 9–16 years of exposure, while 6 participants (7.3%) reported 17–24 years of exposure. This indicates that nearly two-thirds of the sample had relatively shorter cumulative exposure duration, although clinically relevant colour vision impairment was still observed in the exposed group.

Table 2. Distribution of Total Years of Occupational Chemical Exposure

Total Years of Exposure	Frequency (n)	Percentage (%)
1–8 years	51	62.2
9–16 years	25	30.5
17–24 years	6	7.3
Total	82	100.0

The Farnsworth D-15 test showed a marked difference in colour vision findings between exposed and non-exposed participants. Among exposed workers, only 8 of 41 participants (19.5%) had normal colour vision, whereas 33 participants (80.5%) demonstrated some form of colour vision abnormality. In contrast, 37 of 41 non-exposed participants (90.2%) had normal colour vision, while only 4 participants (9.8%) showed abnormal findings. The most frequent defect among exposed workers was binocular tritanopia, observed in 10 participants (24.4%), followed by L-tritanopia in 7 participants (17.1%) and R-tritanopia in 5 participants (12.2%). The association between exposure group and D-15 test category was statistically significant, $\chi^2 = 42.866$, df = 8, $p < 0.001$, with a large effect size by Cramer's V = 0.723.

Table 3. Farnsworth D-15 Colour Vision Findings by Exposure Group

D-15 Test Finding	Exposed n (%)	Non-Exposed n (%)	Total n (%)	p-value	Effect Size
Binocular tritanopia	10 (24.4)	1 (2.4)	11 (13.4)		
L-tritanopia	7 (17.1)	1 (2.4)	8 (9.8)		
Normal colour vision	8 (19.5)	37 (90.2)	45 (54.9)		
Binocular deuteranopia	2 (4.9)	0 (0.0)	2 (2.4)		
L-deuteranopia	2 (4.9)	1 (2.4)	3 (3.7)		
Binocular red–green defect	3 (7.3)	1 (2.4)	4 (4.9)		
R-deuteranopia with L-tritanopia	1 (2.4)	0 (0.0)	1 (1.2)		
R-deuteranopia	3 (7.3)	0 (0.0)	3 (3.7)		
R-tritanopia	5 (12.2)	0 (0.0)	5 (6.1)		
Total	41 (100.0)	41 (100.0)	82 (100.0)	<0.001	Cramer's V = 0.723

When D-15 findings were collapsed into normal versus abnormal colour vision, abnormal colour vision was present in 33 exposed participants (80.5%) compared with 4 non-exposed participants (9.8%). Exposed workers had approximately 38 times higher odds of abnormal colour vision than non-exposed participants, with an odds ratio of 38.16 and 95% confidence interval of 10.52–138.21. The relative risk was 8.25, indicating that abnormal colour vision was more than eight times as frequent among exposed workers compared with non-exposed participants. This dichotomized analysis confirms a strong and clinically meaningful association between occupational chemical exposure and acquired colour vision impairment.

Table 4. Association Between Chemical Exposure and Abnormal Colour Vision

Group	Normal Colour Vision n (%)	Abnormal Colour Vision n (%)	Risk of Abnormal Colour Vision (%)	Odds Ratio (95% CI)	Relative Risk (95% CI)	p-value
Exposed	8 (19.5)	33 (80.5)	80.5	38.16 (10.52–138.21)	8.25 (3.22–21.18)	<0.001
Non-exposed	37 (90.2)	4 (9.8)	9.8	Reference	Reference	

The overall chi-square analysis demonstrated a statistically significant relationship between exposure status and D-15 colour vision category. Pearson's chi-square value was 42.866 with 8 degrees of freedom and $p < 0.001$. The likelihood ratio test similarly supported a significant association, with a value of 50.507 and $p < 0.001$. Fisher's exact test was also significant, supporting the robustness of the association despite sparse counts in some colour vision defect categories. The linear-by-linear association was significant as well, $\chi^2 = 5.343$, $p = 0.021$.

Table 5. Chi-Square Test for Association Between Exposure Status and D-15 Findings

Test	Value	df	p-value
Pearson chi-square	42.866	8	<0.001
Likelihood ratio	50.507	8	<0.001
Fisher's exact test	43.134	—	<0.001
Linear-by-linear association	5.343	1	0.021
Number of valid cases	82	—	—

Overall, the findings show a strong association between occupational chemical exposure and abnormal colour vision. The dominant defect pattern among exposed workers involved tritan-type abnormalities, particularly binocular tritanopia and unilateral tritanopia, supporting the interpretation that blue–yellow colour discrimination may be especially vulnerable in workers exposed to industrial solvents and related chemicals. However, the earlier manuscript statement that 59.8% of all participants had colour vision deficiency conflicts with the D-15 crosstab, which shows 37 abnormal cases out of 82 participants, equivalent to 45.1%. Therefore, the revised results are based on the crosstabulated D-15 data because it provides the clearest group-specific distribution.

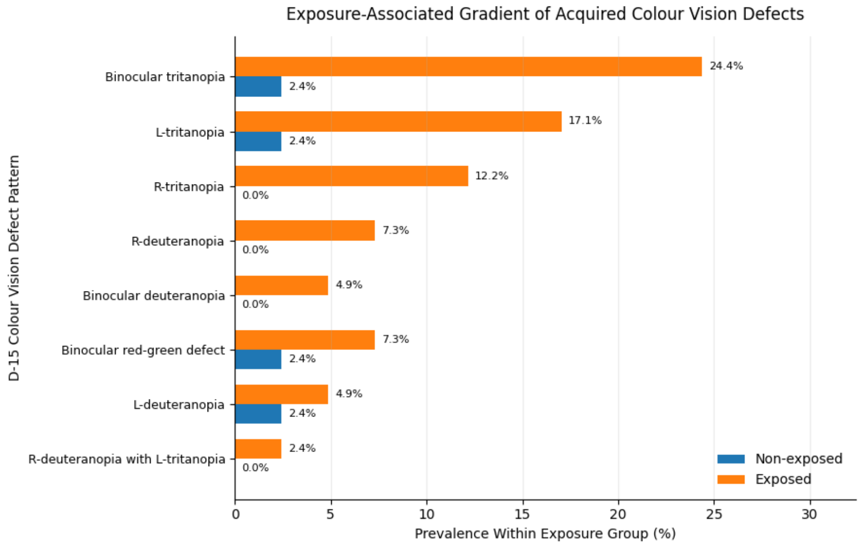

Figure 1 Exposure-Associated Gradient of Acquired Colour Vision Defects

Among exposed workers, tritan-type defects showed the clearest exposure-associated gradient, with binocular tritanopia present in 10/41 participants (24.4%) compared with 1/41 non-exposed participants (2.4%), followed by L-tritanopia in 7/41 exposed workers (17.1%) versus 1/41 non-exposed participants (2.4%) and R-tritanopia in 5/41 exposed workers (12.2%) versus none in the non-exposed group. This pattern indicates that blue–yellow colour discrimination defects were the dominant abnormality among chemically exposed workers, supporting a clinically meaningful association between occupational chemical exposure and acquired dyschromatopsia.

DISCUSSION

The present study found a strong association between occupational chemical exposure and acquired colour vision impairment among industrial workers. Abnormal Farnsworth D-15 findings were observed in 33 of 41 exposed workers (80.5%) compared with only 4 of 41 non-exposed participants (9.8%), indicating that colour vision abnormalities were more than eight times more frequent among exposed workers. The association was statistically significant, with Pearson's chi-square value of 42.866 and $p < 0.001$, and the large Cramer's V value of 0.723 suggests a strong relationship between exposure status and colour vision defect pattern. The most prominent abnormalities were tritan-type defects, particularly binocular tritanopia, L-tritanopia, and R-tritanopia, suggesting that blue–yellow colour discrimination was disproportionately affected in chemically exposed workers.

These findings are consistent with previous occupational toxicology literature indicating that organic solvents and other industrial chemicals may impair visual function through neurotoxic effects on retinal, optic nerve, or post-retinal visual pathways. Earlier studies have reported that acquired colour vision loss among solvent-exposed workers commonly affects the blue–yellow axis, which is clinically important because these defects may develop gradually and remain unnoticed until visual discrimination is substantially impaired (17–21). The predominance of tritan-type defects in the present study supports this pattern and strengthens the interpretation that the Farnsworth D-15 test may be useful as a practical screening tool for early detection of chemically induced dyschromatopsia in occupational settings (21).

The high frequency of abnormal colour vision among exposed workers is clinically relevant because colour discrimination contributes to safe performance in industrial environments where workers may need to identify warning signals, labels, chemical indicators, machinery controls, and safety-coded materials. Even mild or unilateral defects may compromise work accuracy in visually demanding tasks. The finding that many exposed workers had relatively shorter cumulative exposure duration, particularly within the 1–8 year category, suggests that screening should not be limited only to workers with long occupational histories. Instead, baseline and periodic colour vision assessment may be justified for employees working in solvent-rich or chemically hazardous environments (22).

The findings also have public health implications for occupational safety in Pakistan. Industrial workers may experience repeated exposure to mixtures of solvents such as toluene, xylene, styrene, propylene-based compounds, and carbon disulfide, yet routine visual surveillance is not always incorporated into workplace health programs. Because acquired colour vision impairment can serve as an early indicator of neurotoxicity, periodic screening using validated tools such as Ishihara plates and Farnsworth D-15 testing may help identify workers requiring further ophthalmological or occupational health evaluation. The results therefore support stronger preventive measures, including exposure reduction, appropriate ventilation, personal protective equipment, worker training, and regular visual monitoring.

Several limitations should be considered when interpreting these findings. The cross-sectional design does not establish causality or determine whether colour vision defects progressed over time or improved after exposure reduction. Chemical exposure was classified using occupational history and workplace exposure category rather than quantitative environmental monitoring, biological exposure indices, or dose-response modelling. Some D-15 categories had small cell counts, which may affect the stability of category-specific comparisons, although Fisher's exact test supported the overall association. The sample was also predominantly male, limiting generalizability to female workers. In addition, potential confounders such as smoking, nutritional status, lighting exposure, detailed PPE compliance, and subclinical ocular or neurological changes were not fully quantified. Future studies should use longitudinal designs, larger multisite samples, quantitative exposure assessment, and adjusted regression models to clarify dose-response relationships and temporal progression of chemically associated colour vision impairment (23).

Despite these limitations, the study provides useful evidence that industrial chemical exposure is strongly associated with acquired colour vision abnormalities, particularly tritan-type defects. The equal distribution of exposed and non-exposed participants strengthens the group comparison, and the use of Ishihara plates with the Farnsworth D-15 test provides clinically relevant screening for both congenital and acquired colour vision abnormalities. These findings support the integration of colour vision testing into occupational health surveillance programs for workers exposed to organic solvents and related industrial chemicals.

CONCLUSION

Occupational chemical exposure was strongly associated with acquired colour vision impairment among industrial workers, with exposed participants showing a markedly higher frequency of abnormal Farnsworth D-15 findings than non-exposed participants. Tritan-type defects were the dominant abnormality, indicating that blue–yellow colour discrimination may be particularly vulnerable in workers exposed to organic solvents and related industrial chemicals. These findings highlight the need for routine colour vision screening, improved workplace safety practices, exposure reduction strategies, appropriate use of protective equipment, and early referral of affected workers for ophthalmological evaluation. However, because the study was cross-sectional, the findings should be interpreted as evidence of association rather than causation.

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