Colour vision deficiency among students in Lagos State, Nigeria

Olalekan A Oduntan,1 Khathutshelo P Mashige,1 Franklin E Kio2

1. Department of Optometry, School of Health Sciences, University of KwaZulu-Natal, Durban, 4000, South Africa.
2. Department of Optometry, Faculty of Life Sciences, University of Benin, Benin City, Nigeria.

Abstract

Background: Congenital colour vision defects are x-linked inherited, non-progressive and untreatable disorders that describe poor colour discrimination.

Objective: To determine the prevalence of congenital colour vision deficiency among students in Lagos, Nigeria.

Methods: A school-based cross-sectional, cluster sample study was conducted to test the colour vision of 2326 primary and high school students. Inclusion criteria were Snellen VA 20/20 or better and absence of known ocular pathologies. Colour vision deficiency (CVD) was evaluated with the Richmond-HRR colour vision test plates.

Results: There were 1014 (43.6%) males and 1312 (56.4%) females with a mean age of 13.40 ± 2.40 years (range = 7−22 years). The prevalence of CVD was 58 (2.5%), which was higher in males 49 (4.8%) than females 9 (0.7%). The prevalence of congenital CVD was significantly associated with males (p = 0.00), but not with females (p = 0.22). Of the 58 cases of CVD, 17 (0.7%) had protan deficiency, 38 (1.6%) had deutan deficiency and three (0.1%) were unclassified.

Conclusion: The prevalence of congenital CVD among students in Lagos is comparable to findings in other parts of Nigeria but differs from other parts of the country. These results strengthen the need to establish school vision screening.

Keywords: Colour vision deficiency, Richmond-HRR, prevalence, red-green defects, Nigeria.

DOI: https://dx.doi.org/10.4314/ahs.v19i2.48

Cite as: Oduntan OA, Mashige KP, Kio FE. Colour vision deficiency among students in Lagos State, Nigeria. Afri Health Sci.2019;19(2):2230-2236. https://dx.doi.org/10.4314/ahs.v19i2.48

Introduction

Human vision relies on the ability to perceive a narrow window of electromagnetic radiation, and the sense of colour depends on the ability to discriminate among different wavelengths stimuli.1 Colour vision is a function of three types of retinal cones, each with its specific-wavelength sensitivity; blue (tritan) at 414-424 nm, green (deutan) at 522-539 nm and red (protan) at 549-570 nm.2 The various types of photoreception mediating vision and their functions have been discussed by many authors.1,2 Colour vision deficiencies (CVD) can be congenital or acquired and congenital defects are inherited (genetic in origin), while acquired CVD are usually associated with other ocular and systemic conditions such as media opacities, macular diseases, optic neuropathies, and diabetes mellitus.3 Congenital colour vision defects are non-progressive and untreatable disorders, for which screening is done to enable children to understand the implications of their condition for a variety of life circumstances, including occupation.3 The historic aspects of colour vision defects have been reported4 and the genetic aspects published,2 and will therefore not be presented in this article. Abnormalities in or the absence of colour vision is often used to classify colour vision deficiency. When the colour deficiency is a result of abnormality of red-sensitive cones, the condition is known as protanomaly, and when the defect is caused by absence of the red sensitive cells in the macula, the condition is called protanopia.2 Individuals with an abnormality of red-sensitive cones, and with an absence of these cones, are referred to as protanom-
alous trichromats and protanopes, respectively. Those with an abnormality of green-sensitive cones have deuteranomaly, and with absent green cones have deuteranopia. The green colour deficient individuals are referred to as deuteranomalous trichromats. Those with blue-green deficiency caused by abnormality of blue-sensitive cones have a colour efficiency called tritanomaly, and when the blue-sensitive cones are absent it is called tritanopia. The individuals with abnormality of blue-green sensitive cones are referred to as tritanomalous trichromats and those with absence of the cones are called tritanopes.

According to Pease, occupations in the armed forces, aviation, electrical, railroad and maritime have colour standard required for employment, while other professions, such as geology, graphic designs and healthcare professions, require normal colour vision for effective, efficient and safe performance. Bacon found that the colour differentiation was needed for teaching and learning chemistry, physics and biology in secondary school. Gordon suggested that CVD affects the activities of children in school, leading to some psychological effect. It is therefore important that children know of their colour vision status, be advised on how to deal with the condition and what profession they might choose in order not to face occupational difficulties.

The prevalence of CVD have been shown in many published studies to vary with respect to race, ethnicity and gender. In addition, the few studies that have been conducted on colour vision in other parts of Nigeria have yielded varying results, suggesting that data from different areas and ethnicities should be studied. Furthermore, the patterns across Africa are not uniform, with 1.8% in the Congo and 1.9% in Uganda, and 3.5% in Sudan. Such studies will provide useful information for the health professionals and policy makers, as well as parents of children with colour vision impairment.

Methods

This was an explorative, cross-sectional and quantitative study to determine the prevalence and types of CVD among primary and high school students in Lagos State, Nigeria. Lagos, located in the South-Western part of Nigeria, is the largest state in the country (population of approximately 21 million) and one of the largest in Africa. It is culturally and ethnically diverse, attracting residents from across the country. It is the commercial capital of Nigeria and most of the inhabitants are Black. There are two public Universities in Lagos, as well as primary and high schools, many of which are public institutions. Lagos is divided into 20 Local Government areas (LGA) for administrative purposes due to the population size and physical extent of the state.

Sampling procedure and sample size

A multistage sampling technique was used to identify the four local government community areas of Agege, Alimosho, Ifako/Ijaiye and Ikeja. Thereafter, simple random sampling was used to select eight schools from a total of 42 schools in these areas. Finally, a stratified multistage cluster random sampling was used to select participants. The minimum sample size for the study was determined using the formula for a prevalence study.

\[
N = \frac{Z^2 \times (P) \times (1 - P)}{C^2}
\]

Where \(N = \) minimum required sample size, \(Z = \) value of \(z\) statistic at 95% confidence level = 1.96, \(P = \) assumed prevalence of congenital colour vision defects = 8% for maximum sample size, \(C = \) maximum acceptable sampling error = 1.6%.

Data collection

A pilot study was conducted among 100 participants outside the study area to establish any need for modification of the test procedures. The assessment consisted of data regarding socio-demographic details, visual acuity, retinoscopy, subjective refraction, pen torch examination, direct ophthalmoscope observation and colour vision testing. Visual acuity assessment was performed with a Tumbling E Snellen's chart in a well-illuminated outdoor environment, and all those with spectacles had their visual acuities assessed while wearing them. Thereafter, an ocular examination was done for the students with a retinoscope followed by subjective refraction, pen torch and direct ophthalmoscope through undilated pupils. Retinoscopy and subjective refraction were for refractive error determination and other tests were for ocular health examination. Colour vision was assessed using the Richmond-HRR (Richmond Products Inc), which was administered by a qualified optometrist under conventional fluorescent light and performed monocularly, with
all findings being recorded on the record sheet. The tests consists of 24 plates, with the first four being used for demonstration, and the remaining 20 being divided into three to test for problems CVD.

The procedures were explained to the students, with the demonstration plates (plates 1-4) being used to show them how the test and scoring worked. The students were asked to report how many symbols could be seen, what they were and where they were in the four corner areas. Except for the students who were colour vision deficient or malingering, they should see “OX” and “X∆” in colour on the first two plates respectively, one coloured “O” in the third and no coloured symbol on the fourth.

The students were then told that the following 20 plates constituted the test, and that plate 5 contained one, two or no symbols. The screening plates (5-10) were then administered and the students were asked how many coloured symbols were seen, what and where they were. The responses were recorded as X O in the box provided for plate 5 on the scoring sheet, with the locations of the symbols being also recorded, as indicated by the students. If they correctly answered all three questions, a tick was placed beside the box by the examiner to indicate correct responses. However, if they made an error in answering any of the three questions, no tick mark was made.

A similar procedure was used with plates 6-10, turning the pages at about 3-second intervals, asking the students to answer the same questions as each page is turned. If all the six boxes were ticked to show correct responses, the student had normal colour vision and no more testing needed to be done. However, if plates 5 or 6 were not ticked, the student had defective blue-yellow vision and the examiner proceeded to show plates 11-20. If any of plates 7-10 were not ticked, the student had defective red-green vision and the examiner proceeded to show plates 21-24. If any of plates 7-10 were not ticked, the student had defective red-green vision and the examiner proceeded to show plates 11-20. If any plates of both screening groups (5-6 and 7-10) were not checked, the student was tested on all remaining plates (11-24). These 14 plates (21-24) provide diagnostic information as to the extent (mild, medium or strong) and type of defect (protan, deutan, tritan). Further information as applied to this procedure was adhered to as contained in the manual (HRR Instruction manual).18

Analysis
The data were coded and analysed anonymously using the Statistical Package for Social Sciences (SPSS) programme (SPSS for Windows, version 19; SPSS Inc., Chicago, Illinois, USA). Analysis was done with the assistance of a qualified statistician with descriptive and inferential statistics performed to compute prevalence and distribution of CVD with age and gender. A p value of less than 0.05 was considered statistically significant.

Ethical approval
Ethical approval to conduct the study was obtained from the Research Committee, Faculty of Life Sciences, University of Benin. Permission to conduct the study was also obtained from the Department of Education, Lagos State and the principals of the selected primary and high schools. Parents and/or legal guardians of the students who participated in this study signed consent forms, while the students provided informed assent. Confidentiality of data was maintained, and those found to have CVD were advised about their condition, and how it may affect their future choice of occupation or profession, as well as any other conditions that were identified during the various tests. Similarly, the parents/legal guardians of students who were found to have CVD were given feedback about their children’s colour vision status.

Results
A total of 2326 primary and high school students participated in the study, their ages ranging from 7 to 22 years with a mean of 13.40 ± 2.40 years. There were 1014 (43.6%) male and 1312 (56.4%) female students, of whom 2268 (97.5%) had normal colour vision and 58 (2.5%) [95% CI: 1.1-3.6] had CVD. The prevalence of CVD was 49 (4.8%) [95% CI: 3.6-6.1] in males and 9 (0.7%) [95% CI: 0.4-0.9] in females. There was a statistically significant difference in the prevalence of CVD and male students (p = 0.00), but not statistically significant (p = 0.22) in the females. Of the 58 cases of CVD, 17 (0.7%) were protan, 38 (1.6%) were deutan and three (0.1%) were unclassified. The prevalence of CVD was compared among younger and older students, with the differences among age groups not reaching statistical significance overall (p = 0.08). Table 1 shows the number and percentage of students with CVD stratified by age group and gender.
Table 1: Prevalence of CVD according to age and gender

| Category | Total | Protan n (%) | Deutan n (%) | Unclassified n (%) | CVD n (%) | 95%CI     | P-value |
|----------|-------|--------------|--------------|-------------------|-----------|-----------|---------|
| Age (years) |       |              |              |                   |           |           |         |
| 7 – 11   | 966   | 8(0.8)       | 11(1.1)      | 3(0.3)            | 12(2.2)   | [0.6-3.2] | 0.08    |
| 12 – 16  | 643   | 3(0.4)       | 12(1.9)      | 0                 | 15(2.3)   | [0.8-3.3] |         |
| 17 – 21  | 717   | 6(0.9)       | 15(2.0)      | 0                 | 21(2.9)   | [0.9-3.8] |         |
| Gender   |       |              |              |                   |           |           | 0.00    |
| Male     | 1014  | 15(1.4)      | 31(3.2)      | 3(0.2)            | 49(4.8)   | [3.6-6.1] |         |
| Female   | 1312  | 2(0.2)       | 7(0.5)       | 0                 | 9(0.7)    | [0.4-0.9] |         |
| Total    | 2326  | 17(0.7)      | 38(1.6)      | 3(0.2)            | 58(2.5)   | [1.1-3.6] |         |

Out of 58 affected students, 26 were mild deutans (1.12%), 10 were mild protans (0.43%) and three (0.1%) could not be classified in any of sub-groups of red-green colour vision defects. The type and severity of CVD among the 58 (2.5%) students is shown in Table 2.

Table 2: Types and severity of CVD among the students

| Type of colour blindness | Number of students (n) | Prevalence (%) |
|--------------------------|------------------------|----------------|
| Mild protans             | 10                     | 0.43           |
| Mild deutans             | 26                     | 1.12           |
| Moderate protans         | 3                      | 0.13           |
| Moderate deutans         | 7                      | 0.30           |
| Strong protans           | 4                      | 0.17           |
| Strong deutans           | 5                      | 0.14           |
| Unclassified             | 3                      | 0.10           |

Discussion

This study presents a detailed description of CVD for the first time among male and female primary and high school students in Lagos State, and thus provides the basic epidemiology of colour blindness in this region. Colour vision deficiency assessments enable patients to follow adaptive strategies that could minimise the risks associated with the disorder. Testing was done using the Richmond-HRR test, which is generally considered to be efficient for screening congenital CVD. In addition, the HRR test can reliably detect, categorise and grade the severity of the protan, deutan and tritan colour vision deficiencies. The Richmond-HRR is therefore not only a useful and simple diagnostic device, it also has sufficient sensitivity and specificity to allow investigators to use the results in a clinically meaningful way.

The significance of normal vision involves absolute colour matching for many occupations. For example, the traffic light signals are less obvious for deutans and protans, while deutan and protan individuals working with telecommunications and electric cables can recognise the blue and white wires but will be uncertain about the red, orange, brown and green. Steward and Cole also reported that approximately 30% of people with abnormal colour vision had trouble judging the ripeness of fruit. The above suggests that colour perception is integral to an individual’s understanding and engaging with the visual world, and those with these defects can experience hardships in everyday life. However, adaptive strategies and behaviours can help to deal with potential difficulties that CVD individuals face in both their professional and personal lives.
The distribution of CVD was fairly consistent across the age categories (7-11: 2.2%, 12-16:2.3%, 17-22: 2.9%). Although this shows an increase in the prevalence of the defect with increasing age, the difference was not statistically significant (p=0.08). As CVD is a hereditary defect, the prevalence in different age groups is statistically insignificant (p > 0.05). Table 3 provides an overview of CVD prevalence data in selected studies and an opportunity to compare our findings with those of other age, race and ethnic groups.

Table 3: Characteristics of colour vision defects reported compared with the findings of our study

| Study            | Ethnicity      | Instrument used | Number     | Age (years) | Overall prevalence | Prevalence (95% CI) |
|------------------|----------------|-----------------|------------|-------------|--------------------|---------------------|
| Africa           |                |                 |            |             |                    |                     |
| Present study    | Nigerian       | Richmond-HRR    | 1014/1312  | 7-22        | 2.5                | M/F                 |
| Ugalahi et al15  | Nigerian       | Ishihara, FM D-15 | 769/866  | 13.9±1.9   | 2.3                | 4.8                 | 0.7                 |
| Abah et al15     | Nigerian       | Ishihara        | 149/178   | 5-17        | 1.5                | N/R                 | N/R                 |
| Tabuni et al13   | Nigerian       | Ishihara        | N/R       | 5-17        | 2.6                | N/R                 | N/R                 |
| Zein25           | Ethiopian      | Ishihara        | 954/1054  | 8-24        | 2.08               | 4.2(2.93-5.47)       | 0.20(0.45)          |
| Rahman et al23   | Libyan         | Ishihara        | 163/179   | 17-24       | N/R                | 1.84                | 0                   |
| Pickford and     | South African  | Ishihara        | N/R       | N/R         | 3.337              | 0.233               |
| Pickford22       | African Zulu   |                 |           |             |                    |                     |
| Applemans13      | Congolese      | Ishihara        | N/R       | N/R         | 1.8                | N/R                 | N/R                 |
| Simon14          | Ugandan        | N/R             | N/R       | N/R         | 1.9                | N/R                 | N/R                 |
| Alrasheed et al15| Sudanese       | Ishihara        | 544/556   | 10-80       | 3.5                | 6.8(5.2-8.4)         | 0.6(1.0-2.2)        |
| Asia/Middle East |                |                 |            |             |                    |                     |
| Qian et al15     | Chinese        | Ishihara, FM 100 | 5819     | N/R         | 4.46               | 0.65                |
| Shah et al17     | Indian         | Ishihara        | 2674      | N/R         | 8.73               | 1.69                |
| Moaress et al16  | Iranian        | Ishihara        | 1136/922  | 12-14       | 8.18               | 0.43                |
| Al-Aqtum and Al- | Jordanian      | Ishihara        | 1200/218  | 18-27       | 8.7(4.97-12.47)    | 0.33(0.01-0.65)      |
| Qusmeleh25       |                |                 |           |             |                    |                     |
| Oroiwo and       | Saudi Arabian  | Ishihara        | 838/800   | 6-19        | 5.85(4.26-7.44)    | 0.75(0.15-1.35)      |
| Alothabi28       |                |                 |           |             |                    |                     |
| Mian et al27     | Punjabi        | Ishihara        | 214       | N/R         | 4.89(0.0-0.0)      | N/R                 |
| Chia et al28     | Singaporean    | Ishihara        | 1249      | 13-15       | 5.3                | 0.2                 |
| Europe           |                |                 |            |             |                    |                     |
| Nom11            | Danish         | Ishihara        | 173/186   | N/R         | 8.67(4.48-12.86)   | 0.54(0.1-1.59)       |
| Inuit            |                | Ishihara        | 290/250   | N/R         | 1(0.19)            | 0.40(0.3-1.18)       |
| Malaspina et al8 | Caucasian      | Ishihara        | 3285      | 13-20       | 6.10(5.2-6.92)     | N/R                 |
| Rebato and Basque| Basque         | Ishihara        | 174/218   | 15-25       | 4.02(1.1-6.94)     | 0.46(0.1-1.36)       |
| Calderon12       |                |                 |           |             |                    |                     |
| Oceania          |                |                 |            |             |                    |                     |
| Groversen13      | Caucasian      | Ishihara        | 817       | N/R         | 6.50(4.81-8.19)    | N/R                 |
| Polynesian       | Ishihara       | 571             | N/R       | N/R         | 2.60(1.29-3.91)    | N/R                 |

The prevalence of CVD detected in the present study was 2.5% (58 of 2326 students), comparable to the 2.3% reported in Ibadan, SouthWest Nigeria10 and the 2.6% reported in Port Harcourt, Southern Nigeria12 but higher than the 1.5% found in Zaria, Northern Nigeria11 (Table 3). Ethnically based studies that were conducted in Asia, Europe and Oceania reported higher prevalence of CVD than the current study, which could be due to racial differences. This suggests that CVD varies among races and geographical regions of the world.

In this study, the prevalence of CVD was higher among males (4.8%) than females (0.7%) with a significant association between gender and CVD (p < 0.05). This is an expected finding as CVD is a genetic disorder transmitted through the sex-linked recessive X chromosome.232 There were no congenital tritans observed in this study; this type of CVD is reported to occur very rarely, with a prevalence of 1:15 000 to 1:50 000 (0.002-0.007%) of the population.1,2 Although in some careers a CVD does not debar entry, it can be an impediment, specifically in those occupations that involve colour matching such as in industries (paint, textile, plastic, decorates, furniture), transport (rail, road, aviation, maritime), defense (police, armed force, fire and rescue services) and other occupations (electricians, technicians, telecommunications, me-
Early detection of CVD is therefore important in making decisions about future career choices. It is also important for parents and teachers to make necessary adjustments during teaching to ensure effective learning of those with CVD.

All the colour vision deficient students in this study were not aware of their status, which could negatively affect their daily lives and future careers choices. It is suggested that students diagnosed with CVD be counselled concerning the effects of defective colour vision on activities of daily living, learning progress and occupations that require critical colour judgment. Eye care practitioners and occupational therapists should advise CVD patients at an early age to find adaptive strategies that will enable them to make appropriate choices about activities of daily living and future occupations. Finally, colour vision testing should form part of routine eye examination, as its assessment also helps to determine the functional and structural integrity of the visual system. The findings highlight the need to include vision screening as part of a comprehensive school health programme, specifically in poor communities that are unlikely to be able to afford to pay for such services. Colour vision deficiency is only one of many vision problems that could affect students and impact on their activities of daily living and school work. Every effort needs to be made to keep students in school to enable them to maximise their adult life opportunities, and to prevent avoidable dropouts due to vision problems that could have been addressed with a simple eye test.

Conflict of interest
None declared.

References
1. Wissinger B, Sharpe LT. Genetics of perception '98. New Aspects of an Old Theme: The Genetic Basis of Human Color Vision. Am J Hum Genet. 1998; 63(5):1257-1262.
2. Neitz J, Neitz M. The genetics of normal and defective colour vision. Vis Res. 2011; 51(7):633–651.
3. Cumberland P, Rahi JS, Pechkam CS. Impact of congenital colour vision deficiency on education and unintentional injuries: findings from the 1958 British birth cohort. Brit J Ophthalmol. 2004; 329(7474):1074-1075.
4. Pease PL. Color Vision. In: Benjamin WJ. Borish's Clinical Refraction: 2nd Ed. Butterworth-Heinemann, St. Louis, 2006. p. 289-348.
5. Campbell JL, Griffin L, Spalding JAB, Mir FA. The effect of abnormal colour vision on the ability to identify and outline coloured clinical signs and to count stained bacilli in sputum. Clin Exp Optom. 2004; 88(6):376-381.
6. Bacon L. Color vision defect—an educational handicap. Med Officer. 1971; 125:199-209.
7. Gordon N. Colour blindness. Pub Health. 1998; 112(2):81-84.
8. Malaspina P, Biondi G, Santillo C. Color blindness (CB) distribution in the male population of Albanian and Croatian communities of Molise, Italy. Gene Geogr. 1989;3(1):53–63.
9. Qian YS, Abudureheman Z, Aximu A, Muhamat P, Yasen G, Aili M, Chu RY. Comparison of congenital color vision deficiencies prevalence between Han and Uygur high school students. Zhonghua YanbKe Za Zhi. 2009; 45(2):131–134.
10. Ugalia MO, Fasina O, Ogun OA, Ajayi BG. Prevalence of congenital colour vision deficiency among secondary school students in Ibadan, South-West Nigeria. Niger Postgrad Med J. 2016; 23(1):93-96.
11. Abah ER, Oladigbolu KK, Samaila E, Gani-Ikilama A. Ocular disorders in children in Zaria children's school. Niger J Clin Pract. 2011; 14(4):473-476.
12. Tabansi PN, Anochie IC, Nkanginieme KE, Pedro-Egbe CN. Screening for congenital color vision deficiency in primary children in Port Harcourt city; teachers' knowledge and performance. Niger J Med. 2008; 17:428-432.
13. Applemans M. Color defects among natives of Congo. Bull Soc Belge Ophthalmon. 1953; 103: 226-229.
14. Simon T. Color defects among natives of Uganda. E Afr Med J. 1951; 28: 75-78.
15. Alrasheed SH, Awad ME, Abdulbagi AA, Abdu M. Congenital and acquired colour vision deficiency among population in North Kordofan State of Sudan. *Sudanese J Ophthalmol*. 2017; 9(1): 22-27.

16. World Population Review. Lagos Population. Available from: http://worldpopulationreview.com/world-cities/lagos-population. Accessed on 08/09/2017.

17. Kirkwood BR, Sterne JA. Essentials of Medical Statistics. 2nd ed. Massachusetts: Blackwell Science Limited; 2003. p. 420.

18. Richmond Products. Eye Examinations and Testing Products. HRR instruction manual. Richmond Products Inc 4400 Silver Ave SE: Albuquerque, NM 87108.

19. Cole BL, Lian KY, Lakkis C. The new Richmond HRR pseudoisochromatic test for colour vision is better than the Ishihara test. *Clin Exp Optom*. 2006; 86(2):73-80.

20. Cole BL. Assessment of inherited colour vision defects in clinical practice. *Clin Exp Optom*. 2007; 90(3):157-175.

21. Steward SM, Cole BL. What do colour vision defectives say about everyday tasks? *Optom Vis Sci*. 1989; 66(5):288-295.

22. Zein ZA. Gene frequency and type of color blindness in Ethiopians. *Ethiop Med J*. 1990; 28(2):73-75.

23. Rahman SA, Singh PN, Nanda PK. Comparison of the incidence of colour blindness between sections of Libyan and Indian populations. *Indian J Physiol Pharmacol*. 1998; 42(2):271-275.

24. Pickford RW, Pickford R. Frequency of colour vision defects among Zulus in Natal. *J Biosoc Sci*. 1981; 13(2):241-248.

25. Shah A, Hussain R, Fareed M, Afzal M. Prevalence of red-green color vision defects among Muslim males and females of Manipur, India. *Iran J Public Health*. 2013; 42(1):16-24.

26. Modarres M, Mirsamadi M, Peyman GA. Prevalence of congenital colour deficiencies in secondary-school students in Tehran. *Int Ophthalmol*. 1996; 20(4): 221-222.

27. Al-Aqtum MT and Al-Qawasmeh MH. Prevalence of colour blindness in young Jordanians. *Ophthalmologica*. 2001; 215(1): 39-42.

28. Oriowo M, Alotaibi AZ. Colour vision screening among Saudi Arabian children. *S Afr Optom*. 2008; 67(2): 56-61.

29. Mian A, Ali M, Rafique S. Frequencies of colour blindness in different ethnic groups of Quetta (Pakistan). *Pak J Zool*. 1991; 23: 153-155.

30. Chia A, Gazzard G, Tong L, Zhang X, Sim EL, Fong A, et al. Red-green colour blindness in Singaporean children. *Clin Exp Ophthalmol*. 2008; 36(5):464-467.

31. Norn M. Prevalence of congenital colour blindness among Inuit in East Greenland. *Acta Ophthalmol Scan*. 1997; 75(2):206-209.

32. Rebato E, Calderon R. Incidence of red/green colour blindness in the Basque population. *Anthropol Anz*. 1990; 48(2): 145-148.

33. Grosvenor T. The incidence of red-green colour deficiency in New Zealand’s Maoris and “Islanders”. *Am J Optom Arch Am Acad Optom*. 1970; 47(6): 445-450.

34. Fareed M, Anwar MA, Afzal M. Prevalence and gene frequency of color vision impairments among children of six populations from North Indian region. *Genes & Diseases*. 2015; 2(2): 211-218