# Prevalence of red-green color vision deficiency (cvd) among science students: a 10 years' survey

A.V. Panat<sup>1</sup> and D.A. Kulkarni<sup>2</sup>

Citation Panat AV and Kulkarni DA. 2016. Prevalence of red-green color vision deficiency (cvd) among science students: a 10 years' survey. Human Biology Review, 5 (1), 65-71.

<sup>1</sup>Arun V. Panat, M.Sc. M.Phil. Independent Researcher Retired Associate Professor and Former Head Department of Zoology Arts, Science and Commerce College Rahuri Dist. Ahemadnagar (M.S.) India . E mail: <u>avp\_panat@yahoo.co.in</u> and <u>avp.panat@gmail.com</u>

<sup>2</sup>Dilip A. Kulkarni M.S.(G.Surgeon) Sanjjivanee Hospital Rahuri Dist. Ahemadnagar E mail: dr.dilipkulkarni@yahoo.in

Corresponding author: Arun V. Panat, M.Sc. M.Phil. Independent Researcher Retired Associate Professor and Former Head Department of Zoology Arts, Science and Commerce College Rahuri Dist. Ahemadnagar (M.S.) India . E mail: <u>avp panat@yahoo.co.in</u> and <u>avp.panat@gmail.com</u>

# ABSTRACT:

Red-green Color vision deficiency (CVD) has a high prevalence and is often a handicap in everyday life. The fact that most of the students do not know they have CVD due to non testing of color vision. All Science faculty students of the ASC Rahuri College in the age range of 17-19 were screened for Red-green CVD by using Ishihara test plates from 2001 to 2010. Thus total 654 males and 292 female students were screened during 10 years. The collected data were analyzed for frequency estimation among males and females and then estimating mating types by using the Hardy-Weinberg Principle. Frequency of color blindness in males was 0.303 and that in females 0.288. Predicted mating types show a higher contribution of carrier females, and homozygous color blinds than expected. Estimated normal mating type is only 35.35%. Prevalence of Red-green CVD was found considerably high among science students and specifically among females which is unusual. Mating types involving carrier females, colorblind females, and colorblind males together show a very high contribution than normal ones.

**Key words:** 1) Color blindness 2) Science students 3) Red-green Color vision defect 4) Invisible ophthalmic disability

# **INTRODUCTION:**

Color vision deficiencies are a group of conditions that affect the perception of color. Color is specifically associated with electromagnetic radiation of a certain range of wavelengths visible to the human eye. Color is perceived by red, green and blue cone cells lining the retina and signals are generated. All these signals are mixed by the brain to create wide spectrum of colors that we perceive. Clinically color vision deficiency is classified in to 1) protan 2) deutan and 3) tritan types. Protan and Deutan defects are caused by recessive mutations of the genes located on the long arm of the X-chromosome within  $X_q$  28 band. Tritan defects are caused by mutations of the gene encoding blue retinal cone pigment present on the autosome number 7. Tritan defect is autosomal dominant and transmitted with incomplete penetrance (Deeb and Motulsky, 2005). The molecular nature of normal red-green color vision pigments and their defects were first revealed in 1986. Males with normal color vision have one red pigment gene and one or more green pigment genes. Red-green color vision defects from mild to severe are formed due to homologous recombination between red and green pigment genes leading either to deletions in green pigment genes or to full length hybrid or fusion genes (Nathans et al. 1986). Protan and Deutan defects can be unequivocally predicted by molecular analysis (Deeb et al. 1992). There are some reports of narrower foveal pit in subjects with congenital red-green CVD (Gupta et al. 2011). Studies on the prevalence of red-green color vision deficiencies are carried out worldwide and show variations between male and female sexes as well as between different populations (Tagarelli et al. 1999). In India the frequency of red-green CVD is variable and goes as high as 0.231 in some areas. Its frequency is estimated at 0.1 in the Maharashtra state (Bhasin, 2006). Individuals with defective color vision experience many problems with color in everyday life and at work. They take longer time for color identification than normal individuals (Cole 2004, Ramaswamy and Hovis 2009). It is therefore important for students to know the color vision status while molding their career in life. Some data are available for the prevalence of red-green CVD in different occupations. In Malaysia, 8.4 % and 0.3 % males and females are found color blind in the medical and paramedical profession (Balasundaram and Reddy 2006). In Nepal 5.8 % medical students were observed color weak (Pramanik et al. 2010). A CVD of 6.3 % was found among the practicing Nigerian dentists (Bamise et al. 2007) while 2.4% paramedical employees are found red-green color defective in Iran (Dargahi et al. 2010). Medical professionals encounter many difficulties due to color blindness in the practice of medicine (Spalding and Antony 2004). Only 3.2 % male and 1.81 % female population was found to be CVD in Pune district of the Maharashtra state. The same survey had found family aggregation of colorblindness in 28 families (Natu, 1987) The present study was undertaken for Science faculty students of the college, since colors play an important role in all disciplines of basic Sciences. For example in cytochemistry, histology, qualitative analysis, spectroscopy, chromatography etc. colors are important and hence normal vision is required. In many physiological experiments and dissections colors play a significant role in understanding.

#### **MATERIALS AND METHODS:**

Screening of students for red-green color deficiency is a regular theory and practical syllabus of Zoology at undergraduate level in the University of Pune. Naturally it leads to screening of students by using Ishihara plates available from the local practical text book of Biology and printed plates downloaded from the web site http://www.toledo-bend.com.These 8 plates help spot the most common red-green colorblindness even though the full test consists of a set of 38 plates. Every consenting student was informed that the collected data will be used for research purpose only and it will be maintained strictly confidential for protecting the privacy of everyone. The protocol was reviewed and approved by the research ethics committee of the college which functions independently as per the guidelines of the Helsinki declaration. The plates were held at about 75 cms from the student and tilted at right angle to the line of vision. The test was performed in a white painted room full of natural day light. However direct sunlight was avoided since it alters appearance of shades of the color. The time of 10 seconds was given to each student for reading the number. An assessment of the reading of plates determines the normality or defectiveness of color vision. In total of 8 plates, if 6 or more plates were read correctly, the color vision was regarded as normal. If 5 or less plates were read correctly, the color vision was regarded as red-green deficient. In this study, students were also given options to identify the numbers by observing them for longer time periods by holding plates in daylight as per their desire and relaxing the distance limit of 75 cms. This was to make sure beyond doubts we are not missing any colorblind student due to hasty inspection of Ishihara chart within 10 seconds. It is beyond our capacity to measure a degree of CVD as mild, moderate or severe and other sub classifications. In 10 years from 2001 to 2010, total 654 male and 292 female students of the age 17 to 19 admitted in the Science faculty of the ASC College at Rahuri were

screened. It was also ensured that a student was not having any serious ophthalmic problem. The frequency of color blinds was estimated in males and females from a data obtained. The same data were further analyzed for predicting mating types by using the Hardy-Weinberg Principle.

## **RESULTS**:

Table 1: Frequency of Red-green color vision deficiency among science students

Status	Males		Females		
	Number	Proportion	Number	Proportion	Expected
Colorblind	198	0.303 = q	084	$0.288 = q^2$	0.0918
Normal	456	0.697 = p	208	0.712	
Total	654		292		

Table -1 shows the frequency of colorblindness among males and females. The observed frequency of colorblindness in males and females is 0.303 and 0.288, respectively. Since males being hemizygous, the 'q' gene frequency in them would be the same as the proportion of colorblindness which is 0.303. Therefore the frequency of 'p' gene is 0.697. The expected frequency of colorblind females would be 0.0918 i.e.  $(0.303)^2$  whereas the observed frequency in females is 0.288 which is very high. There is a significant difference between expected and observed values of colorblind and normal females ( $\chi^2$ =132.59, df =1, p <0.001). The Hardy-Weinberg law enables the frequencies of different mating types to be predicted. Figure-1 is a prediction of different mating types using the Hardy-Weinberg law. In this figure the percentage of both the normal mating type is lesser than expected (35.35%). Mating types between both colorblind (2.512%) and normal male-colorblind female (6.07%) are unexpectedly high. The commonest mating type is the normal male and carrier female (29.29%).

## **DISCUSSION**:

From Table-1 the frequency of colorblind males is 0.303 which is a direct measure of this affliction since males are hemizygous due to a single X-chromosome. The frequency of colorblind females ( $q^2$ ) is 0.288 and the q is 0.536 which is an indirect estimate. Based on the male frequency the expected number of afflicted females is (0.0918 ×292) = 26.80 i.e. 27.

Actually the observed number of colorblind females is 84! It is expected that number of affected females is much lesser than that of the males but the quantitative agreement shows a very high deviation. The maximum likelihood estimate of q is 0.29 and this figure is closer to the male estimate and it is obvious because the male ratio is a direct measure of the allele frequency. This result indicates that in females, homozygous, carriers and compound heterozygotes must be contributing largely in the next generation. Table-1 already shows a high proportion of colorblind females (0.288). The same result is reflected in histograms of mating types (Figure-1).





Mating types of both male and female colorblind and colorblind female –normal male together constitute a large proportion than expected. The mating types between 1) normal male-carrier female and 2) colorblind male –carrier female together surpass the percentage of normal mating type. This result shows that mating types involving the defective gene are more than the normal ones. One cannot rule out the possibility of compound heterozygote females in 35.35% normal mating as well as colorblind male- normal female mating. Color vision of compound

heterozygote females cannot be distinguished from that of the normal vision (Carroll 2006) and for that family history investigation and genetic analysis are the only reliable ways. This primary investigation of colorblindness emphasizes the need for family history analysis and genetic analysis and the use of anamoloscope, flicker photometric electro retinogram for spectral sensitivity of the proband and modified Cambridge color tests. It is very difficult to explain the result of unusual high frequency of colorblind females but the possible explanation may lie in the theory of evolution. Small colonies of common surnames are everywhere in this area. The endogamy is common in India due to intra community marriages within castes. Gene flow between communities is restricted in many societies or castes and nearby villages. In such circumstances recessive founder or de novo mutation spreads rapidly showing higher frequency within a particular community (Bittles and Black, 2010). In the Founder effect relatively rare alleles have two possibilities i.e. they are either lost or they disperse throughout the population. Thus this may be the reason for high frequency of colorblindness in this population in both the sexes.

Conflict of interest: None. Funding agency: Nil

# **REFERENCES**:

- **1.** Bittles AH and.Black ML. 2010. Consanguinity, human evolution and complex diseases. *PNAS*.; 107(suppl. 1) : 1779-1786
- Balasundaram R and Reddy SC. 2006. Prevalence of color vision deficiency among medical students and health personnel. Malaysian Family Physician; 1(2 &3): 52-53
- Bamise CT, Esan TA, Akeredolu PA, Oluwatoyin O, Oziegbe EO. 2007. Color vision defect and tooth shade selection among Nigerian dental practitioners. *Rev. Clin. Pesq. Odontol.; set/dez*; 3(3): 175-182
- **4.** Bhasin M.K. 2006. Genetics of castes and tribes of India: A review of population differences in red and green color vision deficiency in India. *Int. J. Hum. Genet.* 6(1): 81-88.
- Carroll Joseph. 2006. Color-blindness detective story not so simple. *Clin.Exp.Optom.* 89:3: 184-186.
- Cole Barry L. 2004. The handicap of abnormal color vision. *Clin. Exp. Optom.* 87(4-5):258-275

- Dargahi Hossein, Einollahi Nihad and Dashti Nasrin. 2010. Colorblindness defect and medical laboratory technologist: unnoticed problems and the care for screening. *Acta. Medica. Iranica.* 48(3): 172-177.
- Deeb SS, Lindsey Delwin T, Hibiya Yuko, Sanocki Elizabeth, Joris Winderickx, Teller Davida Y, Motulsky Arno G. 1992. Genotype-Phenotype relationships in human Red/Green Color-Vision defects: Molecular and Psychophysical studies. *Am. J. Hum. Genet.* (51): 687-700.
- 9. Deeb SS, Motulsky AG. 2005. Red-Green Color Vision Defects. Sep 19 [Updated 2011 Sep 29]. In: Pagon RA, Adam MP, Bird TD, et al., editors. *GeneReviews™ [Internet]*. Seattle (WA): University of Washington, Seattle; 1993-2013. Available from: <a href="http://www.ncbi.nlm.nih.gov/books/NBK1301/">http://www.ncbi.nlm.nih.gov/books/NBK1301/</a> Accessed on 11/8/2011.
- **10.** Gupta A, G. Laxmi, Nittala M.G. and R. Raman. 2011. Structural and functional co-relates in color vision deficiency. *Eye.* 25: 909-917.
- **11.** Nathans J., Thomas D, Hogness DS. 1986. Molecular genetics of human color vision: The genes encoding blue, green and red pigments. *Science*. 232 : 193-202.
- 12. Natu Maya. 1987. Colorblindness A rural prevalence survey. *Ind.J. Ophthal.* 35(2): 71-73.
- **13.** Pramanik T, Sherpa MT and Shrestha R. 2010. Color vision deficiency among medical students: an unnoticed problem. *Nepal Med. Coll. J.* 12(2): 81-83
- Ramaswamy Shankaran and Hovis Jeffery K. 2009. Do color-Deficient Observers Take Longer to Complete a Color-related task? *Optom. Vis. Sci.* 86: 964-970.
- **15.** Spalding J. Antony B. 2004. Confessions of a color blind physician. *Clin.Exp. Optom.* 87(4-5): 344-349.
- **16.** Tagarelli A., Piro A, Tagerelli G. 1999. Genetic, Epidemiologic and social features of colorblindness. *Community Genet.* 2:30-35.
- 17. www.dfisica.ubi.pt/~hgil/P.V.../Ishihara/Ishihara.14.Plate.Instructions.pdf
- 18. <u>http://www.toledo-bend.com</u>