FREQUENCY OF COLOR BLINDNESS AMONG STUDENTS AND THIER AWARENESS ABOUT THEIR STATUS IN SELECTED PRIMARY SCHOOLS IN HARAMAYA, AWADAY AND HARAR TOWNS, EASTERN ETHIOPIA

MSc THESIS

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Frequency of Color Blindness Among Students And Their Awareness About Their Status In Selected Primary Schools in Haramaya, Awaday and Harar Towns, Eastern Ethiopia

A Thesis Submitted to the Department of Biology, Postgraduate Program Directorate

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As Thesis research advisors, we hereby certify that we have read and evaluated this Thesis entitled: **Frequency of Color Blindness among Students and their awareness about their Status in Selected Primary Schools in Haramaya, Awaday and Harar Towns, Eastern Ethiopia** Prepared under our guidance by **Besa Nagu**. We recommend that it be submitted as fulfilling the Thesis requirement.

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DEDICATION

This thesis paper is dedicated to my family, friends and relatives.

STATEMENT OF THE AUTHOR

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BIOGRAPHICAL SKETCH

The author was born on March 6, 1995 in Adami Tulu woreda, East Shewa Zone of Oromia region. She attended her elementary school education at Batu No1 Primary School in Batu town and her secondary schools education at Batu preparatory and Secondary School. In 2013, she completed her secondary school education successfully, passed the Ethiopian School Leaving Certificates Examination (ESLCE) and joined Wolaita Sodo University in 2013 to pursue her BSc study. After three years of rigorous study at Wolaita Sodo University, she graduated with a BSc degree in Biology in June, 2016, and directly joined Haramaya University, College of Natural and Computational Sciences, Department of Biology to pursue her M.Sc degree in Genetics in 2016.

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ABBREVIATIONS AND ACRONYMS

APS	Awaday Primary School
APS	Aboker Primary School
BPS	Bate Primary School
CVD	Color Vision Defect
CNS	Central Nervous System
DNA	Deoxy Ribo Nucleic Acid
FPS	Fendisha Primary School
HFMPS	Harar First model Primary School
ISCD	International Statistical Classification of Disease
LCR	Locus Control Region
MHUPS	Model School of Haramaya University Primary School
SPSS	Statistical Package for Social Science

TABLE OF CONTENTS

Continued

LIST OF TABLES

Table page

LIST OF FIGURES

Frequency of Color Blindness among Students and their Awareness about their Status in Selected Primary Schools in Haramaya, Awaday and Harar towns, Eastern Ethiopia

ABSTRACT

Color blindness, or color vision deficiency (CVD), is the inability or decreased ability to perceive color differences under normal lighting conditions. Human color vision is normally trichromatic i.e. the mixture of red, green, and blue lights. Impaired color vision, in the case of red-green color blindness, is genetically determined by X-linked recessive inheritance and thus occurs mainly in males. The general objective of the study is to estimate the frequency of Color Blindness among students and their Awareness about their color vision status in six selected Primary schools in Haramaya, Awaday and Harar towns (two from each town), Eastern Ethiopia. A total of 1158(male=female=579) school children of grade 3 to 8 were screened for color vision defect using Ishihara's test 14 plate editions. Some socio-demographic data of the participants such as age, grade, sex, self-declared ethnicity, religion were also collected using questionnaire. Hardy–Weinberg method ($p^2 + q^2 + 2pq = 1$) was applied to calculate the expected genotype and allele frequencies in females. Of the total 1158 participants 49(4.2%) were color deficient and 14 of them were females (1.2%) and 35 (3.0%) were males. The larger proportion (3.0%) of the CVD individuals were deutran while, 13(1.1%) were protan and 1(0.1%) was total color blind. The overall phenotypic frequency of color blindness in this study was: 2.3% among Oromo male > among Oromo females 1.12% > among Harar males 0.7% > 0.00% among Amhara males and Harar females. The highest frequency of allele X^A was found among Oromo (0.989) and the least frequency of allele X^A was found among Harar (0.00) and the highest Frequency of allele X^a were found among Oromo (0.011) the least Frequency of allele X^a were found among Harar (0.00). This study indicated that all of the color blind subjects were not aware of their color deficiency status. Further studies to be done to determine the severity of color blindness using Ishihara is also recommended.

Key words: *color blindness, frequency, Ishihara plate, Opsin gene and Primary school*

1. INTRODUCTION

Color blindness is an abnormal condition characterized by the inability to clearly distinguish different colors of a spectrum. Color blindness represents a group of conditions that affect the perception of color. Human color vision is normally trichromatic i.e. the mixture of red, green, and blue colors (Curcio *et al*., 1990). Color blindness is a visual problem. Visual system helps to appreciate the visible world around us. Objects do not have color as a physical attribute. In fact, color is light, which is carried as specific wavelengths that the eye absorbs and the brain converts into 'messages' so that we 'see' colors. An object that appears blue actually absorbs all the other color wavelengths except blue. The unabsorbed wavelength is reflected back to the eye and the brain interprets the object as blue (Krebs *et al.,* 2012). Color vision begins in the retina where different types of cones are sensitive to photons of different frequencies and it is analyzed through the comparison of cell activations in the retina and in the primary visual cortex (Krebs *et al.,* 2012). The color of any object we are looking at depends on the wavelength of light reflected by the object. Our brain recognizes the color of an object by interpreting the combination of signals coming to it from the three different color cones or color pigment (Silverthorn *et al.*, 2010).

As a consequence, there are different kinds of color blindness and most of them are congenital and permanent since they are inherited. Monochromacy is a group of color blindness that occurs when two or all the three of the cone pigments are missing and color and light vision is reduced to one dimension resulting in total color blindness. Total color blindness is rare, non-progressive inability to distinguish any color as a result of absence or nonfunctioning of retinal cones and people with this defect see everything as white, black, or some shade of gray (Tortora and Grabowski, 1996).

Another group of color blindness called dichromacy occurs when only one of the cone pigments is missing and color is reduced to two dimensions resulting in partial color blindness which includes red-green and blue-yellow color blindness. Red and green defects (also called Protan and Deutran, respectively) show the highest prevalence in the general population (Citrik *et al*., 2005). The cause of this condition is via development of a single or multiple arrays of retinal cones that identifies color in light and relays the information to the optic nerve system. Individuals who are red-green color deficient have trouble distinguishing between some shades of red, yellow, and green. Blue-yellow CVD also called tritan defects, which are rarer, cause problems with differentiating shades of blue and green and cause difficulty distinguishing dark blue from black. These two forms of color vision deficiencies disrupt color perception but do not affect the sharpness of vision.

The scientific basis for the CVD is that, Deoxy ribonucleic Acid (DNA) sequences of the red and green receptor gene are so similar, that it is easy for mutation to occur during the development of the egg, as genetic material is replicated and exchanged between chromosomes (Piantanida *et al*., 1986). Color is an extremely important component of the information that we gather with our eyes. CVD have a number of negative effects on learning, in getting driving licenses, and in many areas of daily activities of the person that involves distinguishing between colors. It has been estimated that 75-90% of all learning in the classroom comes to the students either wholly or partially via the visual pathway (Naresh, 1995). In classroom, blocks or other teaching tools may be color coded as well as being of different size. Most visually defected children show low compliance with the use of spectacle. Therefore, in children, CVD can affect school performance and poor performance at school may contribute to the child's self confidence and their career. Person with defective color vision are also at a disadvantage especially for employment purposes such as pilots, drivers, in defense services and in technical fields like engineering and medical profession (Naresh, 1995). Undiagnosed CVD could pose a handicap to the scholarly performance of an affected student (Gnadt and Amos, 1992).

For these reasons, studies of the prevalence of color blindness in a given populations are very important in giving directions in the management of the problem. So the present study was conducted to estimate the frequency of color blindness in six selected Primary Schools of Haramaya, Awaday and Harar towns of eastern Ethiopia,; where the information has not been generated so far. The results of this study would help in creating awareness of the need for early identification of color blindness and in suggesting possible management through advising the individuals to visit doctors for management options before the problem leads to other secondary effects. Although treatments are not available currently color blindness problems can be managed in different ways. Diagnosis of vision defects early in life may help children adjust better to tasks at school and may help adults understand their limitations at work. Screening for color blindness of children can also play an important role in preventing long-term visual deficiency.

General objective

The general objective of the study was to estimate the frequency of color blindness among students and their awareness about their color vision status in six selected primary schools in Haramaya, Awaday and Harar towns (two from each town), Eastern Ethiopia.

Specific objectives

- ✓ To estimate the frequencies of red-green color blindness, among primary school students of the selected study populations.
- ✓ To assess the awareness of students, about their status of color vision.
- ✓ To determine allelic and genotypic frequency of red-green color blindness, among the study population.
- ✓ To determine frequency difference of red-green color blindness among ethnic group of the study population.

2. LITERATURE REVIEW

2.1. Definition of color Blindness

Color blindness is the inability to distinguish certain colors. Molecular studies have shown that defects in color vision result from the malfunction, absence, or alteration of two (dichromatism), one monochromatism or all achromatism of the photopigments. (Diez *et al.,* 2001). Dichromats base their color vision on only two pigments. The class of Dichromats characterized by the entire absence of green cones is called deuteranopia, while those defects characterized by the absence of red cones are called protanopia and those characterized by the absence of blue cones are called tritanopia. Anomalous trichromacy is a relatively mild form of defective color vision. The terms protanomaly, deuteranomaly and tritanomaly is given when there is defect in red, green and blue pigments, respectively. Protanomaly and protanopia are collectively referred to as protan colour vision defects, and deuteranomaly and deuteranopia are referred to as deutran defects CVD (Cole, 2007).

Color blindness is the total inability or reduced capacity to distinguish between different colors under average lighting conditions. It is the primary means of integration between individuals and the external environments. It results from entrance of light into the eye and the interpretation of this stimulus by the brain. For a normal eye, light is focused to a spot on the retina. This message would then be sent to the brain to be interpreted as a message (Tonks, 1993).

The essential difference between the color blind and most people is that hues that appear different to most people look the same to a color blind person. In other words, having a color vision deficit means that the ability to discriminate hue, saturation, and brightness is reduced. To accommodate test users with these deficits, Pearson has developed assessments with more dramatic color contrast to address each of these three qualities of color (Arditi, 1999). Color blindness is permanent. The uses of drugs and vitamins or even attempts to retrain an individual to improve his perception of colors have met with very little success. Congenital color blindness cannot be treated since this type of color defects are non pathologic, incurable, and remain constant throughout life. “Color blind”

is a term of art; there is no actual blindness but there is a in the development of one or more sets of retinal cones that perceive color in light and transmit that information to the optic nerve (Simunovic, 2010).

2.2. Prevalence of Color blindness

The prevalence of congenital color blindness is about 8% in males and 0.4% in females, results either from alterations or absence in the absorption spectrum of photo pigment (Brick, 1993). The frequency of color blindness varies among different ethnic populations across the world. About 8%-10% of males of European descent are red-green colorblind while about 8%-10% of females of this group are carriers of the defective genes and transmit to their sons (Guyton and Hall, 2005). The red-green color blindness is very rare in females (generally≤1%, except in few populations). The high difference between men and women is resulting from the fact that the most common form, red-green color blindness, is a recessive sex-linked trait (Emslie-Smith *et al.*, 1998). In general, people of non-European descent have lower prevalence of color blindness and prevalence varies according to ethnic groups and geographical regions of the world inhabited by people of different ethnicity (Rahman *et al*., 1998).

Asian males have a prevalence of color vision defects of 4.9% compared to 0.64% in females while people of African, Native American or Mexican ancestry have an even lower prevalence: 3.1% in males and 0.7% in females (Jorgensen *et al*., 1990). In Africa, Sub-Saharan populations have a much lower frequencies (around 2.5% of males in some studied populations) of the red-green color blindness compared to the north-African populations (over 5-10% among some studied populations). In Ethiopia population, where there are several ethnic groups, the overall estimates of the frequencies of color blindness were reported to be 4.2% among males and 0.2% among females (Zein, 1990; Mulusew and Yilikal, 2013).

2.3. Inheritance of color blindness

Color blindness is an X-linked recessive inheritance trait. X-linked recessive is a mode of inheritance that a mutated gene on the X chromosome causes a phenotype to be expressed. The children of color blind fathers are rarely or never colorblind. The children of the sons of the colorblind father are not colorblind, but, the children of the daughters of a colorblind father are according to the Mendelian law of heredity, affected as follows: One-half of the sons will be colorblind. This law holds provided the father of the daughter's children is not color blind. Should he be so, half of the daughters will also be color blind. Now, if the color blind daughter of such a union has children all of the sons will be likewise affected. According to the Mendelian terminology color blindness is a characteristic dominant in the male, and recessive in the female, practically sex limited in its occurrence to the male, and also sex limited in its transmission by the female (Kennard, 2007).

The blue pigment gene is located on chromosome 7, while the red and green pigment genes are located on long arm of the X-chromosome (Xq28). The mothers who are carriers of the abnormal gene have a chance of 50% abnormal red-green color vision for sons. The CVD fathers transmit their X-chromosomes to daughters only, which leads to all daughters as carriers and sons with normal color vision. The defective genes for protan and deutran defects are situated at different loci on the X chromosome, and are therefore non-allelic (Nathans, 1986).

2.4. Classification of Color blindness

The two broad categories of 'red–green' defects are protan and deutran respectively. The protan defects are characterized by an absence or anomaly of L-cone function, This means that their trichromatic vision is not based on the classic L-, M-, and S-cone photopigments, but because the L-cone photopigments are lacking they rely on 2 M-cone photopigments and 1 S-cone photopigment. One M-cone, which processes green pigment, and the S-cone, blue pigment, are normally functioning, but the function of the L-cone is lost and is replaced by another M-cone. Protanopia is similar to deuteranopia except that those with the defect are missing the L-cone photopigment function. In most

cases of protanopia, the deletion of genes that could encode Lcone pigments is to blame for the color vision defect. When these genes are deleted variants are created in which the L-cone gene sequences are replaced by M-cone gene sequences, these variances are called chimeric genes (Neitz *et al.*, 2000).

These 2 M cone photopigments differ slightly in their spectral peak. Deuteranomaly is the most common type of the inherited color vision defects and has been shown to affect about 5% of men in the United States. In this type of anomalous trichromacy the M-cone photopigment is nonfunctional and the S-cone is joined by 2 spectral subtypes of L cones. (Neitz *et al.*, 2000). Those with this defect possess a reduced sensitivity to the color green because of the M-cone shift to L-cone photopigments. In approximately two thirds of men with deuteranomaly the M-genes are present, but have lost their function (Neitz *et al.*, 2000).

2.4.1. Acquired versus inherited color blindness

There are two ways a person can be visually color deficient: they can inherit color blindness at birth or they can acquire it after birth in life (Cohen, 1968). Inherited color blindness is much more common than acquired color blindness and develops from an alteration to the opsin genes. There are three types of inherited or congenital color vision deficiencies: monochromacy, dichromacy, and anomalous trichromacy. Monochromacy, also known as total color blindness, is the lack of ability to distinguish colors (and thus the person views everything as if it were on a black and white television); caused by cone defect or absence. Monochromacy occurs when two or all three of the cone pigments are missing and color vision is reduced to one dimension (Morgan *et al*., 1992). Rod Monochromacy (achromatopsia) is an exceedingly rare, non progressive inability to distinguish any colors as a result of absent or nonfunctioning retinal cones. It is associated with light sensitivity (photophobia), involuntary eye oscillations (nystagmus), and poor vision.

Cone Monochromacy is a result of having more than one type of dichromatic color blindness. People who have, for instance, both protanopia and tritanopia are considered to have cone Monochromacy. Since cone Monochromacy is the lack of/damage of more

than one cone in retinal environment, having two types of dichromacy would be an equivalent (Morgan *et al*., 1992). Dichromacy is a moderately severe color vision defect in which one of the three basic color mechanisms is absent or not functioning. It is hereditary and, in the case of protanopia or deuteranopia, sex-linked, affecting predominantly males. Dichromacy occurs when one of the cone pigments is missing and color is reduced to two dimensions. The resulting color deficiencies occur because opsin genes are lost, altered, or debilitated. These losses and alterations typically take place on the X-chromosome. This leads us into describing the different color vision defects and their causes (Sharpe, 2001). Acquired color vision defects are the less common forms and do not involve inherited alterations to the opsin genes. Color vision deficiency, commonly called color blindness, manifests itself in everyday life in the confusion of or blindness to one or more primary colors and its origins may be congenital or Aquired color vision defects are caused by toxins, inflammation or detachment of the retina, macular degeneration, optic nerve diseases, ageing and many other causes (Cohen, 1968). There are other forms of acquired color blindness related to things such as: fundus detachment, glaucoma, CNS diseases, macular degeneration, and optic atrophy (Bowmaker, 1998). Acquired color blindness is also mainly caused by ocular or neurological disease, drug toxicity or exposure to certain solvents. **(**Agamemnon *et al*., 2003).

Scientists have also discovered the color blindness can be chemical or physical damage of optic nerve. Nearly 8% of the male population and 4.5% of UK's population are colorblind (Franzco *et al.,* 2008). Color is routinely used to code and convey information as well as finding extensive application in the educational system. Currently, no treatment exists for congenital color vision defects. However, studies showed that diagnosis of these defects early in life may help children adjust better to tasks at school and may help adults understand their limitations at work.

2.5. Molecular Basis of Color blindness

The molecular genetics of rhodopsin are relatively simple. Rhodopsin is encoded by a single gene on chromosome 3, and that gene is expressed in all rod photoreceptors. In

contrast, it has been long understood that the organization of the visual pigment genes for human color vision would have to be complex enough to accommodate the production of 3 opsin types in 3 spectral classes of cone. From the inheritance of color vision defects, it was expected that an autosomal gene would encode the blue cone opsin, and the other 2 genes one for the red and another for the green cone opsin would be on the X chromosome (Piantanida, *et al* 1974).

The molecular genetics of color vision has turned out to be much more complex than originally suspected. This complexity derives in part from the fact that red and green opsin genes are adjacent to one another and they are about 98% identical. It seems that during human evolution, because of their close proximity and high similarity, the red and green genes were subject to frequent homologous recombination (Piantanida, *et al* 1974). This, perhaps in conjunction with relaxed natural selection against color vision defects in civilized humans, has given rise to a great deal of variability in the red and green photopigment genes. The rearrangements have included duplications of the red and green genes so that most people have extra pigment genes. Individual X chromosomes contain variable numbers of red and green genes, arranged in a tandemly repeated array. Nevertheless, in the face of the unanticipated complexity, much progress has been made toward understanding the relationship between color vision genotype and phenotype. During the past dozen years, consideration of the results from molecular genetics combined with those from physiology and psychophysics has brought about a revolution in how we think about the biological basis of color vision (Piantanida, *et al* 1974).

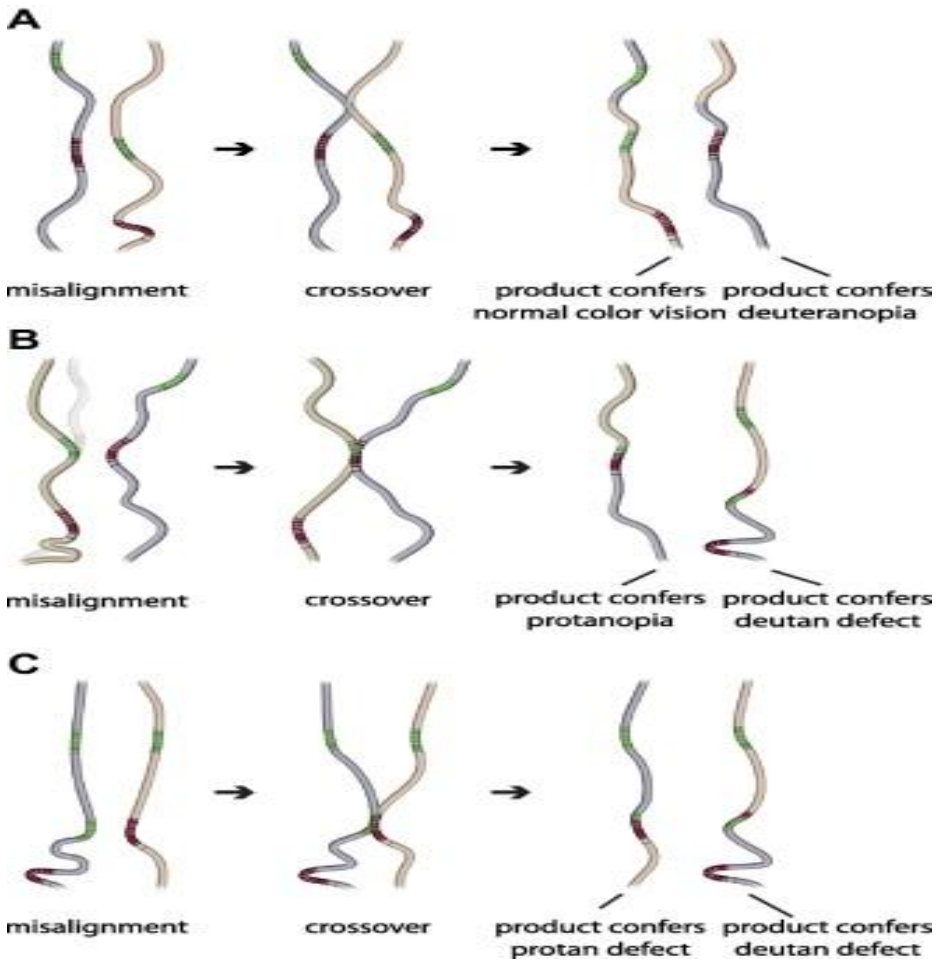
2.6. Effects of Color Blindness

Colour blindness is not physically debilitating, but it can have a major impact on one's day to day life. Person suffering from it may not be able to differentiate between red and green traffic signals, may face difficulties in job as seen for technician working in colour industries. Also colour testing is an integral part of the physical requirement for certain occupation. Children may have negative impact on school and learning and so on. One solution to this problem was the idea of placing the red traffic light above the green at stop signs (Phillips, 1995).

2.7. The Genetics of Color Vision Defects

Protan and deutran defects are characterized by the absence of a contribution to vision from L and M cones, respectively. The dichromatic forms, protanopia and deuteranopia, were caused by the replacement of the normal M gene with one that encoded an L pigment for deuteranopia and the replacement of the normal L gene with one that encoded an M pigment for protanopia. These gene replacements were thought to cause the inappropriate expression of M opsin in L cones for protan, and of L opsin in M cones for deutran. Anomalous trichromatic forms were thought to be caused by genes for anomalous photopigments replacing either the normal L or M pigment gene. Replacing one normal pigment with an anomalous one resulted in having peak sensitivities that were closer together than the peak sensitivities of the normal L and M cones (Piantanida et *al.,* 1974).

Red-green color vision defects 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**)**. Inherited color vision deficiencies can be explained by gene rearrangements that arose through unequal homologous recombination in females during meiosis (Piantanida et al., 1986). As illustrated in Figure 1. The DNA between the L and M genes is nearly identical to the DNA that follows the last gene in the array so an intergenic crossover (in-between the genes) is possible. Such crossover (Figure 1A) produces daughter chromosomes in which one daughter has one additional opsin gene compared to the parents, and the other chromosome has one fewer than the parents. As shown in figure 1A, the products are an X-chromosome with one opsin gene and a second with a tandem array of three opsin genes. A majority of human deuteranopia are single gene dichromats having been reduced to an L gene as the only opsin gene on the X-chromosome. The most frequent arrangement of opsin genes in humans with normal color vision is to have one L and two M genes arranged as shown in figure 1A.



Source: Vision research, 2011

Figure.1. Recombination produces an opsin array that causes color vision deficiencies.

Because the L and M genes are adjacent to each other on the X-chromosome and they are nearly identical, an L gene from the paternal chromosome can align with the M gene from the maternal chromosome, as shown in figure 1B. When X-chromosomes misalign a crossover within the L gene on one X-chromosome and an M gene on the other X-chromosome produces two new arrays, each of which will cause a CVD when inherited by a male. One will cause a protan defect; the other will cause a deutran defect. In the protan-causing array the one remaining opsin gene is a hybrid between the parental L and M genes. As long as the hybrid has exon 5 from a parental M gene, the encoded photopigment will fall into the M-class (Figure 1B). A male with a normal S-pigment

and one X-chromosome pigment gene encoding an M pigment is an obligate protanopia. The array associated with deutran CVD has a parental L gene as the first gene in the array. The second gene is a hybrid while the third gene is a parental M opsin gene. This array structure is the one most commonly found in deuteranomalous males, and it represented one of the most unexpected findings that have come from examining the molecular genetics associated with color vision deficiencies (Drummond *et al.,* 1989)

Genes and the cone photoreceptor mosaic

The significant lag in the appearance of LM versus S opsin protein and mRNA during development suggests that differentiation of S cones is independently controlled from LM cones; however, because these methods do not distinguish between L and M cones, they shed no light on whether L and M cones are independently controlled (Jacob *et al.*, 1993). For example, in squirrel monkeys, there are three alleles of the X-chromosome cone opsin gene. One encodes an opsin that forms a pigment that is similar in spectral peak to the human L pigment, another is similar to the human M pigment, and a third has a spectral peak that is intermediate between human L and M. All males of the species are dichromatic, having only one X-chromosome, and thus having S cones and a single cone type that absorbs in the middle-to-long wavelengths (Jacobs and Williams, 2006).

In humans, even though both L and M opsin genes reside on the X-chromosome, there is evidence that a stochastic mechanism also determines whether each individual cell expresses L versus. M opsin. In their early work to investigate the genetic mechanisms of blue cone monochromacy, Nathans and colleagues discovered a DNA element upstream of the L-opsin gene that is essential for transcription of the X-chromosome opsin genes (Nathans *et al.,* 1989). The DNA element was given the name Locus Control Region (LCR) and it is an enhancer that mediates cell-type specific expression of the X-chromosome opsin genes (Li, 2007 and Wang, 1992).

3. MATERIALS AND METHODS

3.1. Study Design

In this study, a cross-sectional study design was employed to assess the prevalence of red-green color blindness among students in selected primary schools in the three towns during November 2016- January, 2017.

3.2. Study Populations

The study population for this study was primary school students of six primary school selected from the three towns (two schools from each town or district), Bate primary school and Model school of Haramaya University (from Haramaya District), Awaday primary school and Fendisha primary school (from Awaday District) and Harar first model primary school and Aboker primary school (from Harar town). Both male and female students were equal proportion and range from grades 3 to 8 who are able to read numbers and who were in the schools during study period were the study population this student population was assumed to represent the population of the respective towns(Table 1)

Table 1. Distribution of participants in terms of school and sex

School	Sex	Student population	Sample population
MSUPS	Male	491	95
	Female	311	96
BPS	Male	432	98
	Female	454	97
APS	Male	386	97
	Female	360	97
FPS	Male	351	96
	Female	329	96
HFMPS	Male	502	97
	Female	400	97
APS	Male	309	96
	Female	449	96
Total	Male	2471	579
	Female	2303	579

MHUPS = Model School of Haramaya University Primary School, **BPS** = Bate Primary School

APS = Awaday Primary School, **FPS** = Fendisha Primary School

HFMPS = Harar First model Primary School, **APS** = Aboker Primary School

3.3. Sample Size and Sampling Techniques

1158 students were included in the study (579 from each of the two sexes). To get appropriate sample from selected schools, researcher was used random sampling method. From each town about 386 students were included which calculated by taking the Prevalence of 4.2% of red-green color blindness for males, obtained from previous studies in Ethiopia, (Zein, 1990; Mulusew and Yilikal, 2013), with 95% confidence interval, 2% margin of error, design effect of 2. A sample size was determined by Cochran's (1967) sampling formula.

n= $\frac{z2pq}{d2}$ Where

n = sample size

Z = z-score for 95% confidence level (1.96)

d = degree of accuracy (0.02)

p= estimate of the proportion (0.042)

q= 1-p=0.958

3.4. Data collection method

From each participant the following data were collected: red-green color blindness using Ishiara plate (Shinobu 1960) and information related to socio-demographic data and about the knowledge of the students about color blindness were collected using questionnaire (Appendix No. I and II). Participation was voluntarily and the objectives and benefits of the study were explained to the participants before consent to the test.

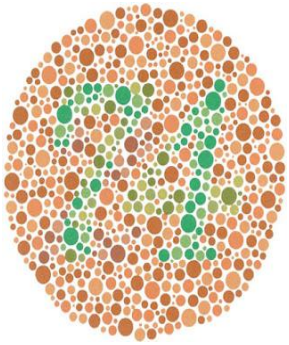
3.4.1. Ishihara's Test for Identification of color blindness

The identification of color blindness was based on Ishiara color test 14 plate Edition (Shinobu 1960). The tests were conducted in accordance with instructions accompanying the plates. Ishihara plates consist of a series of cards on which a colored background is printed in spots of different sizes. A number is printed against this background in spots of the same size (Figure2). To a normal subject the numbers at once becomes clear, but the color blind subject fails to distinguish it from the background. The Student was

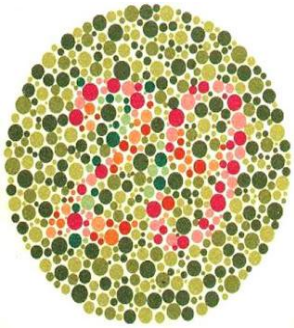
asked to read the numbers seen on the test plates and answer was noted down. The time given for telling the number on a plate was 5 sec (Balasundaram 2006). Assessment of the reading of the plate determines the normality or defectiveness of colour vision and also the type of color blindness. It was interpreted as per the instructions given on the booklet provided with Ishihara's type tests for color blindness so as to identify subject suffering from color blindness and also to differentiate the type of colour blindness. (Appendix VI).

The subjects were able to read the numerals at reading distance (75 cm). So, out of 14 plates, Plate numbers 1 to 11 were used to determine if any red-green color vision defects existed in a given subject. If 10 or more plates were read normally, the color vision was regarded as normal. If 7 or less than 7 plates were read normal, color vision was regarded as deficient, thereafter, plate numbers 12 to 14 were used to determine the precise type of color vision defects protan and deutran (Shah *et al.,* 2013*)*. The numerals which were seen on plate 1-11 were read without more than three seconds delay.

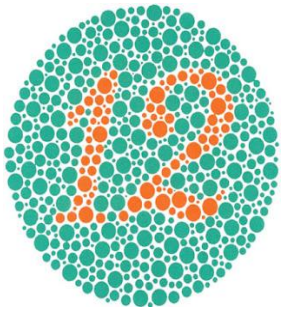
a) Plate No.12



b) Plate No.5



C) Plate No.4



d) Plate No.1

Source: Shinobu Ishihara, 1960

Figure.2. Selected plates used for color blindness

3.4.2. Socio-demographic and Color Vision Related Data

Some socio-demographic data of the participants such as age, grade, sex, self-declared ethnicity, religion were collected using questionnaire. In addition, to assess about their color vision deficiency knowledge, some questions such as whether they know about color blindness or not before this study were included in the questionnaires

3.5. Methods of Data Analyses

All data were entered into SPSS (stastical package for Social Science) version 20 for analysis as frequencies of study variables and cross tabulations. The results were calculated in the form of percentage and presented in the form table and graph.

Determination of allelic and genotypic frequency

Considering X^A =p, and X^a =q then Hardy–Weinberg method ($p^2 + q^2 + 2pq = 1$) was applied to calculate the expected genotype and allele frequency in female. Genotype and allele frequency of color blind was calculated separately for male and female. (Malik and Afzal, 2015).

For male: $q = \frac{\%\ \text{of colorblind phenotype}}{100}$ then, P = 1- q

For female: $q = \frac{\sqrt{\%\ \text{of color blind phenotype}}}{100}$ then, P = 1- q

The homozygosity (Ho) and heterozygosity (Ht) was determined using the formula:

Ho =$\sum Pi^2$ Where Pi is allele (X^Aor X^a).

Now, $H_t = 1 - \sum H_o$ (Malik and Afzal .2015).

3.6. Ethical Issues

In the whole research process much effort was made to follow required research ethical procedures at all stages from collection of data to reporting of the results as a thesis. Permission was obtained from the schools after getting letter of support from Department of Biology, Haramaya University.

4. RESULT AND DISCUSSION

4.1. Distribution of color blindness among sample population

Of the total 1158 participants 49(4.2%) were color deficient and 14 of them were females (1.2%) and 35 (3.0%) were males. Because the most common type of color blindness is red-green color blindness which is congenital and a sex-linked recessive trait, it is more common in males than females. All the studies invariably report a much higher frequency among the males as compared to the females which is only to be expected since color blindness is a genetic disorder transmitted through the X-linked recessive trait (Emslie-Smith *et al.*, 1988). In our study the frequency of color blindness was also higher in males than in females. The frequency of color blindness in our studies was found different in different ethnic groups and also efferent between sexes the highest phenotypic frequency was among Oromo male (2.3%) and the least was frequency was among Amhara males (0.00%) and among Harar females (0.00%). The overall phenotypic frequency of color blindness in this study was: 2.3% among Oromo male > among Oromo females 1.12% > among Harar males 0.7% > 0.00% among Amhara males and Harar females. Since the color blindness is genetically transmitted its distribution is likely to be variable in different ethnic groups. But the numbers of subjects in our studies was not adequate (Table 2).

Table 2. Phenotypic frequency of color blindness among male and female students of major ethnic groups

	Male	Female
Ethnic group	CVD	CVD
Oromo	27(2.3%)	13(1.12%)
Amhara	0(0.0%)	1 (0.09%)
Harar	8 (0.7%)	0 (0.0%)
Total	35(3.0%)	14(1.2%)

CVD= Color Vision Defect

The larger proportion (3.0%) of the color deficient subjects were deutran while, 13(1.1%) were protan and 1(0.1%) was total color blindness (Figure 3). Another study done Zein (Zein, 1990) in North-west Ethiopia in 1988 using the Ishihara 24 plate edition reported a color blindness frequency of 4.2% among males and 0.2% among females (overall prevalence being, 2.08% of which 1.6% were deutran and 0.45% were protan which are lower than our findings. But, the frequency of color blindness found in our study (4.2%) was less than other studies done in Africa including Libyans (5.99%), Algerians (6.56%), Tunisians (5.6%) and Moroccans (10.5%) (Sunderland and Rosa, 1976) and the study done in Nigerian dental practitioners (6.3%) (Cornelius *et al.*, 2007).

This finding is also similar with the study done in Addis Ababa Ethiopia in 2014 (Haile. 2014) that founds the prevalence of color blindness was 4.2%. Of these 2.9% involved deutran, 1.1% protan and 0.3% of totally color blind. Another study on licensed car drivers in Addis Ababa indicated a prevalence rate of 4.5 %; this is relatively the similar to the frequency of color blindness described in our finding (Abebe and Wondmikun, 2002). The most common type of color vision defect was deutran than protan. This study found that one individual (0.1%) had totally color blind. This type of color vision defect is reported to occur very rarely. In 2009, Mulusew *et al.*, found 0.2% totally color blind individuals for the first time in Ethiopia (Mulusew and Yilikal, 2009). The study done in immigrant populations in Punjab in 2012 (Khushdeep *et al.*, 2012) found a prevalence of 2.48% color blindness in male and 0.00% in female (0.78% protan, 1.28% deutran, 0.05% tritan and 0.25% unclassified) which is much lower than the result found in our study. This may be due to the fact that the study population came from different ethnic group (immigrant people from different area).

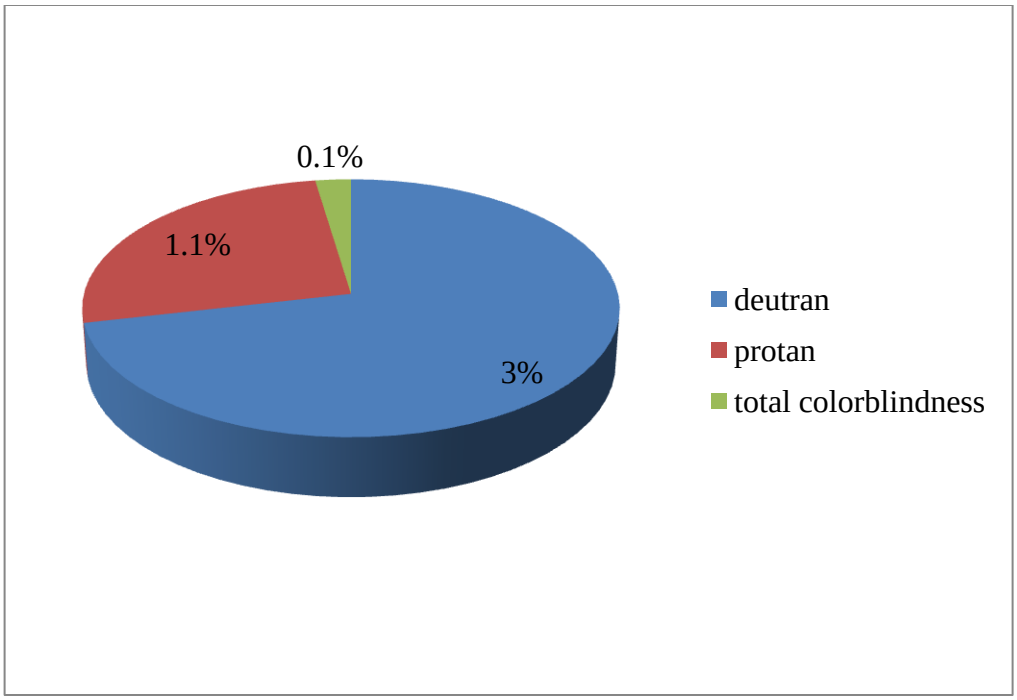


Figure.3. Percentage distribution of different types of color blindness among Study Population

4.2. Genotypic Frequency of Color Blindness

The females are present the three types of genotypes: homozygous dominant X^AX^A homozygous recessive X^aX^a and heterozygous X^AX^a, the heterozygosity X^AX^a was found the highest among Oromo students (0.010), and the least among Harar students (0.00). Since male with single X-chromosome, the genotypic frequencies are the same as of the allelic frequencies (allele X^A Y and allele X^a Y (Table 3)

Table 3. Genotypic frequency among male and female student's major ethnic groups

	Male		Female		
Ethnic group	X^A Y	X^a Y	X^AX^A	X^AX^a	X^aX^a
Oromo	0.973	0.027	0.978	0.010	0.001
Amhara	0.00	0.00	0.982	0.008	0.008
Harar	0.993	0. 007	0.00	0.00	0.00
Total	1.966	0.034	1.96	0.018	0.009

4.3. Allelic Frequency of Color Blindness

In females, X^A and X^a represent the dominant Alleles and recessive Alleles respectively. Among female students, the highest frequency of allele X^A was found among Oromo (0.989) and the least frequency of allele X^A were found among Harar (0.00) and the highest Frequency of allele X^a was found among Oromo (0.011) the least Frequency of allele X^a was found among Harar (0.00) (Figure 4).

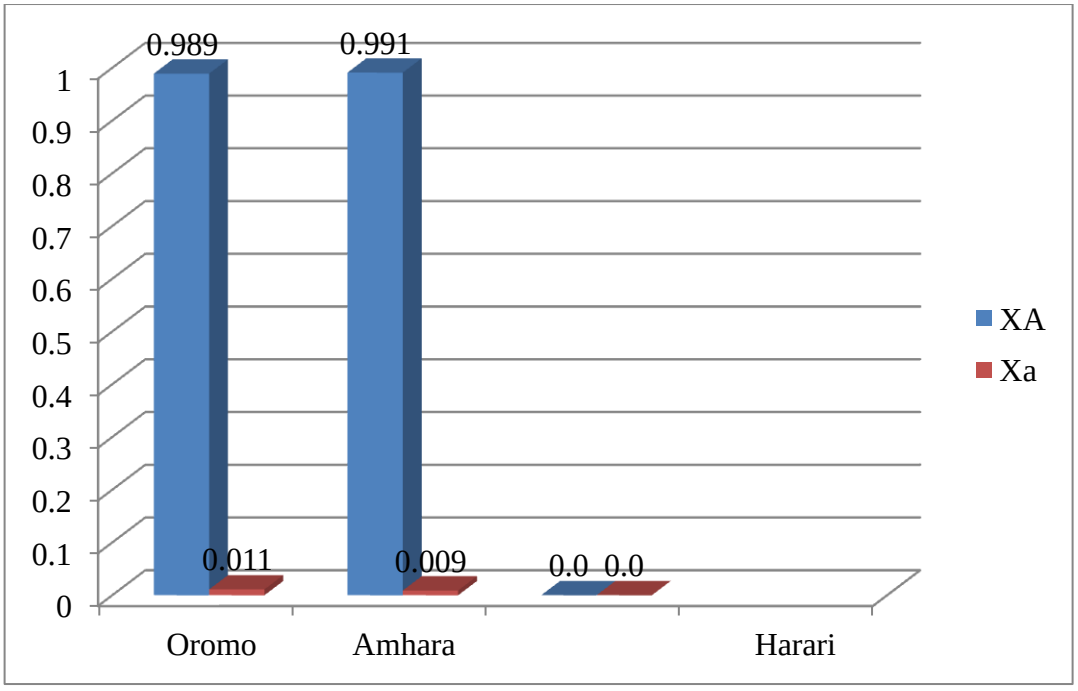


Figure: 4 Allelic frequency of color blindness among female students of three ethnic groups

In males, X^A Y and X^a Y present homozygous dominant and homozygous recessive genotypes respectively (Figure 6), where Y represents the Y-chromosome. The highest Allelic frequency of X^A Y for male students was found among Harar (0.993) and the least were among Amhara (0. 0). The highest frequency of allele X^a Y for male students was found among Oromo (0.027) and the least accounts for Amhara (0.00) (Figure 5).

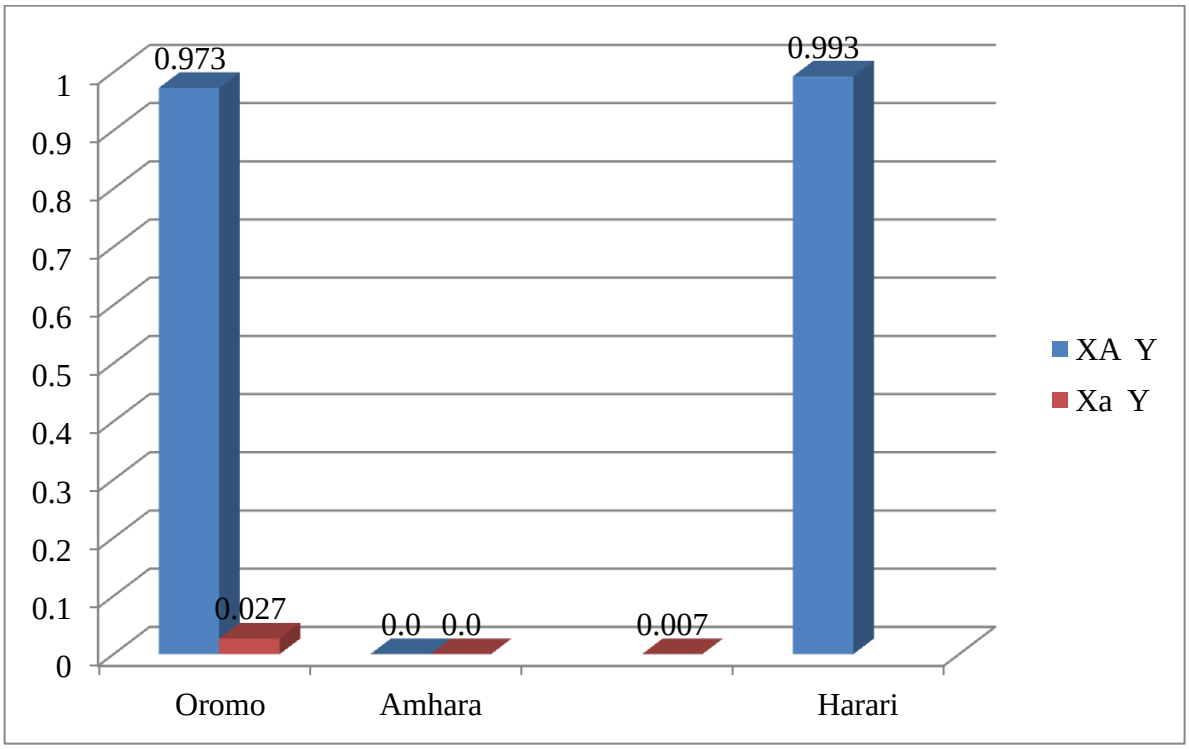


Figure: 5 Allelic frequency of color blindness among male students of major ethnic groups

Table 4. Distribution of different activities by participants

Previous eye disease	**N**	**%**
No	892	77.0
Yes	266	23.0
Total	1158	100.0
Previous awareness about color blindness		
No	1158	100.0
Yes	0	0.0
Total	1158	100.0
Trouble choosing the colors of plants, fruits or flowers		
No	1105	95.4
Yes	53	4.6
Total	1158	100.0
Trouble identifying colors of chalk on the black board?		
No	1099	94.9
Yes	59	5.1
Total	1158	100.0
Was your choice of study influenced by trouble linked to colour blindness		
No	1106	95.5
Yes	52	4.5
Total	1158	100.0
Trouble understanding colored diagrams		
No	1109	95.8
Yes	49	4.2
Total	**1158**	**100.0**

4.4. Awareness of participants on their status of color blindness

This finding indicated that all the subjects participated in this study were not aware of their color deficiency status. The subjects did not know their color vision status before this study, for the reason that the information about color blindness has not been generated in the study area.

5. SUMMARY AND CONCLUSSION

5.1. Summary

Of the total 1158 participants 49(4.2%) were color deficient and 14 of them were females (1.2%) and 35 (3.0%) were males. Because the most common type of color blindness is red-green color blindness which is congenital and a sex-linked recessive trait, it is more common in males than females. The larger proportion (3.0%) of the color deficient subjects were deutran while, 13(1.1%) were protan and 1(0.1%) was total color blindness. overall phenotypic frequency of color blindness in this study was: 2.3% among Oromo male > among Oromo females 1.12% > among Harar males 0.7% > 0.00% among Amhara males and Harar females. This finding indicated that all the subjects participated in this study were not aware of their color deficiency status. The subjects did not know their color vision status before this study.

5.2. Conclusion

Color vision deficiency, commonly called color blindness, manifests itself in everyday life in the confusion of or blindness to one or more primary colors and its origins may be congenital or acquired. The frequency of congenital color blindness is higher among males than females. This is because of color blindness is sex linked recessive trait. The distribution of different types of color blindness of the individuals of the present study was found by deferent frequency. Most of them were protan defect; none of the females were total color blindness. Color blindness is genetically transmitted its distribution is likely to be variable in different ethnic groups. Majority of color blind students were Oromo ethnic group.

5.3. Recommendations

From the present finding, the following research interventions are recommended for future work:

- In order to find out the exact frequency of color blindness among the different ethnic groups further study with a large sample is necessary.

- Further studies to be done to determine the severity of color blindness using Ishihara is also recommended.

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7. APPENDICES

Appendix I:

CONSENT FORM (English version)

Frequency of Color Blindness and their Awareness about their Status among Students of Selected Primary Schools In Haramaya, Awaday and Harar Towns, Eastern Ethiopia

Department of Biology (MSC in Genetics Program), Postgraduate Program Directorate, Haramaya University

As part of my graduate study, I am presently conducting a research on **Frequency of Color Blindness among Students and Their awareness about Their Status in Selected Primary Schools in Haramaya, Awaday and Harar Towns, Eastern Ethiopia** The purpose of the study is to estimate the Frequency of Color Blindness among Students and Their Awareness about Their Status in Selected Primary Schools in Haramaya, Awaday and Harar Towns, Eastern Ethiopia. Therefore I will ask you some question related to colour blindness Please be assured that the information will be confidential since participation is based on your willingness. However your kindly participation would play key role in the success of this study. In addition, no personal identification will be written and we assure you that whatever information you are providing will only be used for the research purpose and the data will be handled only by the researcher and would not be given to any third party.

Are you willing to participate in the study?

Agreed ____ _____ Not Agreed _____

Thank you for your time and cooperation.

Appendix II Questionnaires (English version)

Part I: Personal information

Code No: _____

1. Sex: Male □ Female □
2. Ethnicity:
3. Religion:
4. Age:

5. Grade: ---------- ----------
6. School: ---------- ----------

Part II: Day-to-day activity questions.

1. Have you got any eye disease before? Yes □ No □
2. Have you got any awareness about colour blindness before? Yes □ No □

3. Which colour you can't see clearly?

Green □ Red □ blue □ see all □ can't see all □

4. In your work and hobbies do you have trouble choosing the colors of materials?

Yes □ No □

5. Do you have trouble choosing the colors of plants, fruits or flowers?

Yes □ No □

6. Do you have trouble identifying colors of chalk on the black board?

Yes □ No □

7. Was your choice of study influenced by trouble linked to colour blindness?

Yes □ No □

8. Do you have trouble understanding colored diagrams? Yes □ No □

Appendix III

CONSENT FORM (Afan Oromo version)

Department of Biology (MSC in Genetics Program), Postgraduate Program Directorate, Haramaya University

Kaayyoon qorannoo kanaa frequency jaamina halluu barattoota manneen barnoota sadarkaa tokkoffaa Haramaayaa, Awaday fi Harar tilmaamuudha. Kanaafuu Gaaffilee waa'ee kanaan wal qabate isin gaafachuu waan barbaaduuf fedhii keessan irratti hundooftanii akka naaf deebiftan kabajaadhaan isin gaafadha. Hirmaannaan isin qorannaa kana keessatti gootan milkaa'ina qorannoo kanaatiif gahee guddaa qaba Eenyummaa ykn maqaa keessan barreessuun hin barbaachisu akkasumas odeeffannoon isin naaf kennitan dhimma qorannoo kanaatiif qofa fayyada.

Qorannoo kana keessatti hirmaachuudhaaf Eeyyamamaadha?

Eeyyamamaadha_____ Eeyyamamaa miti_____

Appendix IV

Questionnaires (Afan Oromo version)

Kutaa I: Odeeffannoo dhuunfaa

Koodii: _____

1. Saala: Dhiira □ Dubara □
2. Sabummaa:
3. Amantii:
4. Umrii:
5. Kutaa: ----------
6. Mana barnootaa: ----------

Kutaa II: Gaaffiilee gochaalee guyyaa guyyaatti raawwatamu

1. Kanaan dura dhukkuba ijaa kamiinuu qabamtee beektaa?

   Eeyyeen □ miti □

2. Kanaan dura hubannoo waa'ee jaamina halluu qabdaa?

   Eeyyeen □ miti □

3. Halluu isa kamiin sirritti arguu hin dandeessu?

   Magariisa □ diimaa □ cuquliisa □ hunda ni arga □ hunda hin argu □

4. Hojii kee keessatti meeshaalee halluu qaban addaan baafachuun si rakkisaa?

   Eeyyeen □ miti □

5. Halluuwwan biqilootaafi Kan daraaraa addaan baafachuun si rakkisaa?

   Eeyyeen □ miti □

6. Gabatee gurraacha irratti halluu boronqii addaan baafachuun si rakkisaa?

   Eeyyeen □ miti □

7. Qayyabannaan kee rakkoo jaamina halluu wojjiin wal qabateen si jalaa miidhamaa?

   Eeyyeen □ miti □

8. Fakkiiwwan halluu qaban addaan baaftee hubachuun si rakkisaa?

   Eeyyeen □ miti □

Yeroo keessan kennitanii waan na gargaataniif isin galateeffadha.

Appendix V: Examiner Protocol

1. Seat the subject comfortably in front of a desk.

a. The plate should be 75cm from the subject and tilted so that the plane of the paper is at a right angle to the line of sight. The test consists of a book of 14 plates in which you will be asked to identify a number presented on the plate. Some plates may not have a number, and if you do not see a number, please state, “I do not see a number.” You must respond within 5 seconds in order to have your answer counted. Do you have any questions?

4. Note the time.

5. Set the book on the stage and open the front cover to the first example plate.

6. Ask the subject, Please call out the number you see.

a. The subject must respond within 3 seconds in order to get credit for the plate.

7. Record the number that the subject said on the Subject Recording Sheet

a. If the subject cannot see a number, or does not respond within 5 seconds, record an "X."

8. Turn the page to the next plate and repeat for all 14 plates.

9. Record the elapsed time for the test on the recording sheet.

Appendix VI

Explanation of the plates

No.1. any subject, whether with normal or defective color vision read it as “12”. This plate is used mainly for preliminary explanation of the test process to the subjects. No.2. Normal subjects will read “8” and those with red-green deficiencies “3”, No. 3. Normal subjects will read “5” and those with red-green deficiencies “5”, No. 4. Normal subjects will read “29” and those with red-green deficiencies “70”, No. 5. Normal subjects will read “74” and those with red-green deficiencies “35”. No.6. Normal subjects will read “7” and those with red-green deficiencies “2”, No. 7. Normal subjects will read “45” and those with red-green deficiencies “5”, No. 8. Normal subjects will read “2” and those with red-green deficiencies “17”, No. 9. Normal subjects cannot read and those with red-green deficiencies “21”, No. 10. Normal subjects as “16”, but most of those with red green Deficiencies cannot, No. 11 Normal subjects cannot read and also most of those with red green Deficiencies cannot, No. 12. Normal subjects read as “35” but protan read as “5”, and deutran read as “3”, No. 13. Normal subjects read as “96” but protan read as “6” and deutran read as“9”, No. 14. In tracing the winding lines between the two lines the normal trace along Purple and red lines. In protan (red colorblind) only the purple line is traced, and in deutran (green colorblind) only the red line is traced.

Table 5: Numerals on each plate and answers which would be given by normal and color defective individuals

Number of Plate	Normal Person	Person with Red-Green Deficiencies	Person with Total Color Blindness and Weakness
1	12	12	12
2	8	3	X
3	5	5	X
4	29	70	X
5	74	35	X
6	7	2	X
7	45	5	X
8	2	17	X
9	X	21	X
10	16	X	X
11	X	X	X
		Protan	Deutran
12	35	5	3
13	96	6	9
14	X	Purple	Red

X= the plates cannot read.

Lakk 2/WB/21/1192

Guyyaa 13/4/09

Mana Barnoota Sos Ijaa **tiif**

Awwadaay

Dhimmii:- Mambarnoota irratti Risarchii hojjachuu ilaala.

Akkumma armaan olitti ibsamuuf yaalameetti Baratuun **Bassa Naguu University Harmaayaa** irra Risarchii hojjachuuf gama keenyaa waan Dhufaniif isins kanuma beektani Deegarsaa barbaachisuu akka gootanif isiin beeksifna.

Nagaya Wajjin!

Mustariya Aliyyi

UAanaa Waajjira Barnoota Bul/M/ Awwadaay

G/G

CRC. Sad.1ffaa

Lakk WIBIBI 291/2009

Guyyaa 09-03-09

Mana Barnoota Baatee sadarkaa 1ffaa tiif

Mana Barnoota Moodela Haramaayaa Yuunivarsiti tiif

Mana Barnoota Gadaa Sadarkaa 1ffaa tiif

Haramaayaa

Dhimmi:- Barattuu Bassaa Naguu deegarsaa Ragaa Barbaachisuu akka gootaniif ibsu ilaala.

Barattuun maqaan ishee armaan olitti ibsame "Resarch" gaggeessuu akka barbaadu xalayaan Yuunivarsitti Haramaayaa irraa nuuf barreeffame jira.

Kanaafuu isinis kanuma hubachuun deegarsa barbaachisuu akka gootaniif isin beeksifna.

Nagaya wajjiin

Amaan Daddafoo

Raaw/Hojii Karooraa Istaatiksii Barnootaa

G/G

Bassaa Naguu tiif

ሐረሪ ኡምመት ሑስኒ ሑኩመ

ታእሊም ቢሮ

የሐረሪ ሕዝብ ክልላዊ መንግስት
ትምህርት ቢሮ

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BIIROO BARNOOTAA

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ቁጥር [illegible]

ቀን 27/[illegible]/17

ለአንደኛ ደረጃ ት/ቤቶች

<u>ሀረር</u>

ጉዳዩ፦ <u>ድጋፍ መጠየቅን ይመለከታል</u>

ከላይ በርዕሱ እንደተገለፀው ተማሪ **<u>በሳ ነጉ</u>** በትምህርት ቤታችሁ ሪሰርች ለመስራት ወደ እናንተ ት/ቤት እንድስት ሥራ እንደተፈቀድላት ትብብር እንድታደርጉላት እንጠይቃለን።

ከሰላምታ ጋር

ዘከሪያ አብዱልአዚዝ

ሥ/ት/ዘ/ግ/አ/ተ/ዋና ሥራ ሂደት ባለቤት

Curriculum Owner

ግልባጭ ፡-

- በሳ ነጉ