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Frequency of Color Blindness among School Children in two Secondary Schools in Kirkos Sub- city of Addis Ababa, Ethiopia

A Thesis Submitted to the School of Graduate Studies of Addis Ababa University in Partial Fulfillment of the Requirements for the Degree of Master of Science in Biology

By

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November, 2019

ACKNOWLEDGEMENTS

First, I would like to give great thanks to the Almightily God who helps me in every aspects of my life.

Next I would like to express my gratitude to my advisor, Dr. Kifle Dagne, who supervised and advised me during the study. I would like to thank him for his support and constructive comments starting from the proposal development up to the completion of the thesis. So I want to thank him for everything he did for me.

I have also great respect to all the staff and students of Temenja Yaj and Shmelis Habte secondary schools, who helped me and who voluntarly, participated as study subjects in this study.

I am also indebted to the directors and staff members of Temenja Yaj and Shmelis Habte secondary schools for their positivity and cooperation during data collection. My special thanks go to the Biology teachers of Temenja Yaj Secondary School who managed the students where I collected the data.

The Ministry of Education deserves my acknowledgment for giving me the chance to join the training program and for its financial support that covered the research expense.

Addis Ababa University and the Department of Zoological Sciences are dully acknowledged for the training I received.

I would like to thank my husband Mebrahtu Gebrewahd, my sister Kdst Kidane, my brothers Hgus Gebretsadkan and Mearg Kidane for their support in my carrier.

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Abstract

Frequency of Color Blindness among School Children in two Secondary Schools in Kirkos Sub- city of Addis Ababa, Ethiopia

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In the human visual system, the eye receives physical stimuli in the form of light and sends these stimuli as electrical signals to the brain, which interprets them as images. In retina, there are two types of cells that detect light. They are called rods and cones. Rods detect only light and dark and are very sensitive to low light levels. Cone cells detect color and are concentrated near the center of our vision. There are three types' cones that see color: red, green and blue. The brain uses input from these cone cells to determine color perception. Color blindness can happen when one or more of the color cone cells are absent, or not functioning, or detect a different color than it does normally. The main aim of the present study was to determine the frequency of color blindness among school children of two schools in Addis Ababa. The study was conducted from March 20 to June 06, 2019. Simple random sampling method was used to estimate the sample size. Accordingly, 1012 students from grade 9 and 10 of the two schools were participated in the study. Among these, 606 were females and 406 were males and their ages range from 14 to 20 years. Color vision was tested by using Ishihara 24 plates and subject were asked to seat in a room with sufficient light and read the figures/symbols on the plates from a computer/printed paper placed 75 cm away from the subject. The data were manually arranged and frequencies were calculated. Among the 1012 students tested, 22(2.17%) have color vision defect; these included 5(0.49%) females and 17(1.68%) males. Total colorblindness (achromatopsia) was the more frequent 10(0.99%) color vision defect observed. It was followed by deuteranopia 8(0.79%), protanopia 3(0.29%). It was observed that the prevalence of color blindness is higher in males than in females.

Key words: Color Vision Deficiency, Trichromat, Dichromat, Monochromat, protan, Deutan, Tritan, Ishihara's color plates.

1. Introduction

1.1 Background

Animals detect changes in their environments by means of their sense organs. Vision is one of the senses with which individuals communicate with their external environments. The sense organ responsible for vision or sight is the eye. The visual system is the primary means of integration between individuals and the external environment (Bear et al., 2007).

Visual process is the series of actions that take place during visual perception. During visual process, image of an object seen by the eyes is focused on retina, resulting in the production of visual perception of that object (Sembulingam and Sembulingam, 2012).

The human eye consists of three layers of tissues, the outer, middle and inner layers. The inner layer is known as the retina. This layer contains the light sensitive cells known as the rods and cones. The other two layers have mainly protective and nutritive function (Neitz and Maureen, 2010).

Rods detect only light and dark and are very sensitive to low light levels. Cone cells detect color and are concentrated near the center of our vision. There are three types of cones based on the light spectrum they detect: red, green and blue. The brain uses input from these cone cells to determine our color perception (Neitz and Maureen, 2010).

Most people can identify different colors, but there are some people who have problems in identifying colors. The person who has trouble seeing red, green, blue or a mix of these color is referred to as colorblind. Color blindness is the inability or decreased ability to see color, or notice color differences under normal light. Color blindness affects a significant percentage of the population in many countries (Shah et al., 2013). Worldwide, around 8% of the male population is affected by color blindness, while less than 5% of the female

population is affected. Color blindness is commonly classified as Total, Red-Green and Blue-yellow color blindness (Neitz and Maureen, 2010).

Total color blindness is the inability to distinguish colors at all. It occurs when any two or all the three of the cone pigments are missing. Total color blindness is the rarest type of colorblindness and affects about 0.00001% of the population worldwide. People who are totally colorblind are unable to distinguish between any colors. This is caused by dysfunctional, abnormally shaped cones or the absence of cone pigments (Betsy, 2003).

Red-green color blind people are unable to distinguish between red and green colors. Red-green color blindness is the most common form of color blindness that affects about 8% of males and less than 5% of females. Blue-yellow color blindness is rare and not-gender specific type of color blindness. About 0.02% of the population worldwide is affected by this type of colorblindness. Red-green and blue –yellow types of color blindness occur when the photo pigments of certain cones are missing or they do not function properly (Bansal et al. 2005).

Most color vision problems are inherited, usually, as X-linked recessive. In some cases, people can have an acquired color vision problem due to such causes as aging, eye problems such as, cataracts, retinopathy, injury to the eye and side effects of some medicines (Alharif et al., 2014, 2015).

The photoreceptors (cones) contain light absorbing molecules known as photo pigments. Color vision requires the presence of more than one type of photo pigment (Neitz and Maureen, 2010). Based on the wave length of light they maximally absorb, cones are grouped into: S-cones (absorbing short-wave length), M-cones (absorbing medium-wave length) and L-cones (absorbing long-wave length) (Neitz and Maureen, 2010).The three versions of opsin in the cones absorb light at around 426nm (blue), 530nm (green) and 560nm (red), respectively, (Nathans et al., 2011). The cone opsin, along with retinol, makes up the photo-pigment. Stacked disks are the place where the photo-pigments

are found in cones and rods. Compared to rods, the cones have less number of stacked disks in their outer membrane and therefore, contain fewer photo-pigments, and this characteristic makes them less sensitive to light, whereas in rods there are more photo-pigments in the outer membrane. The three types of cones respond to variation in color in different ways and make possible trichromatic vision (Maureen and Jay, 2000).-

There are different methods of screening color blind individuals in a population. The most common one is the use of Ishihara plate testing in which the individuals are asked to tell the numbers or shape of figures inscribed on the Ishihara plate and their responses are analyzed. Individuals who have color vision defect cannot recognize the numbers in the plates or shapes drawn in the plate or they may tell wrong number or figures. People with color vision defect may be found in any society. However, studies show that the frequency varies from country to country and even among ethnic groups within a given society (Haile, 2014.)

In Ethiopia, information about the prevalence of color vision deficiency is very scares. According to some studies the prevalence of color blindness in Ethiopia is about 4.2% among males and 0.2% among females (Mulusew and Yilikal, 2013).

Color vision deficiency is a problem that affects the person's life. It may make it harder to learn and read, and the individual may not be able to have certain careers such as driving due to difficulty in distinguishing the red-green traffic light. Therefore the main aim of this study is to determine the frequency of color blindness among school children in two governmental schools in the Kirkos Sub-city of Addis Ababa. It is hoped that the result of the study would create awareness about the existence of color vision problem among school children in the country in general and in the two schools selected for study in particular so as to bring it to the attention of teachers for consideration in the teaching-learning process.

1.2. Statement of the problem

Colorblindness is not physically debilitating, but it can have a major impact on one's day-to-day life. Persons suffering from colorblindness may not be able to differentiate between red and green traffic signals. They may face difficulties at work as seen for technician working in color industries (Agarwal and Bansod, 2014)

Colorblindness may make it harder to learn and read, and the individual may not be able to have certain careers such as driving due to difficulty in distinguishing the red-green traffic light. Colorblindness may deter individuals from taking some professions that require good to perfect color vision including professions like airline pilot, air traffic controller, firefighter, police officer, train conductor, some ranks in armed forces, and some electrical/electronic engineers.

The information generated by this study would help education policy makers and other stake holders to take the problem of color vision deficiency in to consideration in their future policy planning.

1.3 OBJECTIVES

1.3.1 General Objective

To determine the frequency of color blindness among school children in the schools Temenja Yaj and Shimelis Habte.

1.3.2 Specific objectives

The specific objectives of this study are:

- To determine the frequency of color blindness among male and female students of the two schools;
- To determine the frequency of the different types of color blindness among the school children of the two schools;

- To determine the allelic, genotypic and phenotypic frequencies of the major types of color vision deficiencies among the children of the two schools;
- To determine if the data is at Hardy-Weinberg genetic equilibrium.

1.4 Significance of the study

This study helps to create awareness about the existence and prevalence of color vision deficiency among school children of the two governmental secondary schools in Addis Ababa. A color vision defect can affect the students learning process. It may make it harder to learn and read, particularly color illustration in teaching materials and may have a negative impact in their future choice of career. In Ethiopia, there are only few studies made on the frequency of color blindness. Therefore, this study will contribute useful information about the frequency of color vision deficiency in Ethiopian population in general and school children in particular. This would initiate to take action to identify and help such children in schools.

2. Literature review

2.1 Historical background of color blindness

John Dalton, an English chemist, was the first to report the disorder of color blindness. He wrote the first scientific paper on color blindness in 1798 (Niroula and Saha, 2010). Thus, color blindness is also called Daltonism, after John Dalton. John Dalton was color blind himself. He realized that his condition had to be hereditary. Dalton believed that a color liquid inside the eye ball was the reason for color blindness; acting like a tinted shield surrounding the eyeball (Niroula and Saha, 2010). Later, after the death of Dalton, scientist discovered that his perception about the cause of colorblindness was wrong; although he was the first to recognize color vision problems (Mughal, et al., 2013).

After the death of Dalton, two scientists, Thomas Young and Herman von Helmholtz, proposed trichromatic color vision. In 1802, Young suggested that there were three types of cone cells in the eye. Each type of cone cell is sensitive to light. By 1850, Hermann von Helmholtz expanded the theory further. He stated that the three types of cone cells could be grouped based on the wavelengths of light hitting the retina (Kaur et al., 2011). Short waves are blue, medium waves are green, and long waves are red. A Swedish physiologist, Gunnar Svaetichin, examined the outside layer of the retina of fish and found that cones of the retina are sensitive to the wavelengths of blue, green and red. In 1983 the theory of Young and Helmholtz was completed (Mughal et al., 2013).

2.2 The eye as sense organ of sight

The human eye receives physical stimuli in the form of light and sends these stimuli as electrical impulse to the brain. The brain interprets the impulse as images. The eye has three layers (Fig 1). These are scleara, choroid and retina and each layer of the eye has its own specific functions (Getahun and Abiy, 2014).

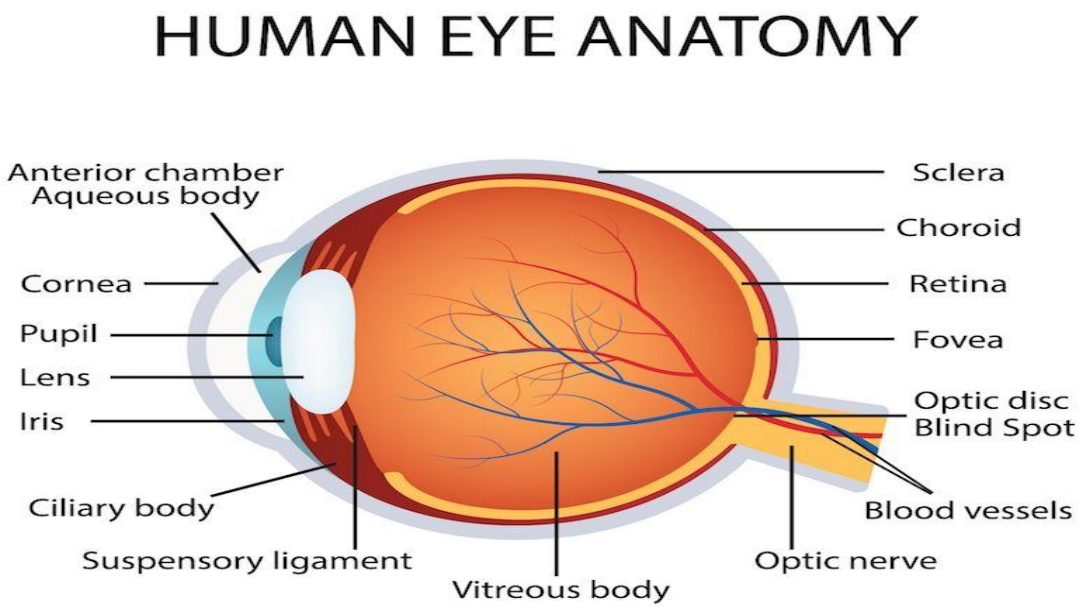


Figure 1: The internal structure of the human eye (Candice, 2015)

The inner most layer of the eye is the retina. Retina contains light receptors: cones and rods. Cones and rods respond to photons of light. Sun light contains tiny packages of energy called photons. Photons of light contain many energy levels, only some of which we can see. Some of the photons contain high energy. Our eye perceives photons carrying intermediate amount of energy as visible light. Visible light represents only a small portion of the range of photon energies in the sun light. The receptors of the eye absorb from 380 nanometers (violet) to 750 nanometers (red) of the wave length of photons. Light that enters the eye through the pupil is focused by the lens on the retina at the back of the eye. The retina contains millions of rods and cones (Moudgil et al., 2016).

2.2.1 The cones

Cones are cone shaped cells and are required for bright light or day light vision (Fig 2). Cone cells are long and narrow with a synaptic terminal, an inner segment, and an outer segment, as well as an interior nucleus and various mitochondria. The synaptic terminal forms a synapse with a neuron such as

bipolar cell. The inner and outer segments are connected by a cilium. The inner segment contains organelles and the cell nucleus, while the outer segment, which is pointed toward the back of the eye, contains the light absorbing materials.

Cone cells are less in number than rod cells. There are around 6 million cone cells in the retina. They are typically 40-50nm long and their diameter varies from 0.50 to 4.0 nm. They are concentrated at the center of retina called fovea. Cones function in day light and produce detailed image. They give color vision (Mustafi et al., 2009). Functionally, there are three kinds of cones which absorb the red, green and blue regions of the light spectrum, respectively.

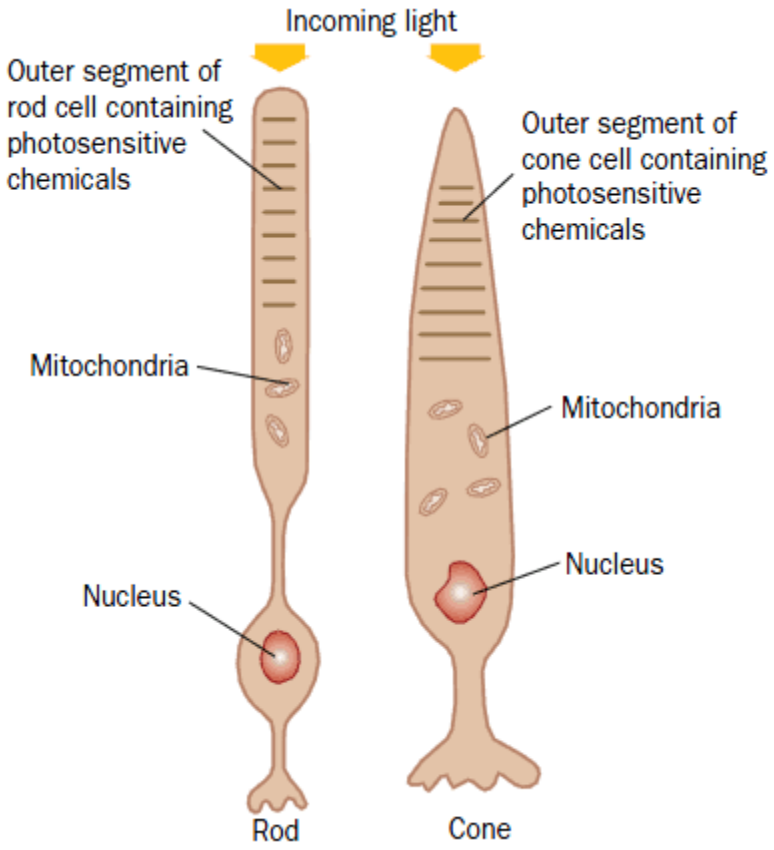


Figure 2: The structure of rods and cones (Mustafi, 2009)

2.2.2 The rods

These cells are known as rods because their shape looks-like rod (Fig.2). Rods are sensitive to dim light and give twilight vision. All rod cells are functionally similar, and do not give color vision. Rod cells are more numerous than cone cells. The human retina contains about 120 million rod cells (Neitz and Maureen, 2010). Rods are found on the edge of the retina. Rods absorb light at 500nm (Neitz and Maureen, 2010).

2.2.3 The photo pigments

Photo-pigments are unstable pigments that undergo a chemical change when they absorb light. The term is generally applied to the non-protein chromophore moiety of photosensitive chromoproteins, such as the pigments involved in photosynthesis and photoreception. In medical terminology, however the term "photo-pigment" commonly refers to the photoreceptor proteins of the retina.

The pigments in photoreceptor proteins either change their conformation or undergo photoreduction when they absorb a photon. This change in the conformation or redox state of the chromophore then affects the protein conformation or activity and triggers a signal transduction cascade.

Photoreceptor cells (rods and cones) contain photopigments, light sensitive molecules that are made up of two type of molecules a protein called opsin and a cofactor retinol. The light sensitive molecules of the rods is known as rhodopsin, and that of the cones is known as photopsin.

Opsins are a group of proteins, made light-sensitive, via the chromophore retinal (or a variant) found in photoreceptor cells of the retina. Five classical groups of opsins are involved in vision, mediating the conversion of a photon of light into an electro chemical signal, the first step in the visual transduction cascade. Another opsin found in the mammalian retina, melanopsin, is

involved in circadian rhythms and pupillary reflex but not in vision. Opsin proteins covalently bind to a vitamin-A based retinaldehyde chromophore through a Schiff base linkage to a lysine residue in the seventh transmembrane alpha helix. In vertebrates, the chromophore is either 11-*cis*-retinal or 11-*cis*-3,4-didehydroretinal and is found in the retinal binding pocket of the opsin. The absorption of a photon of light results in the photoisomerization of the retinal from the 11-*cis* to an all-*trans* conformation. The photoisomerization induces a conformational change in the opsin protein, causing the activation of the phototransduction cascade. The opsin remains insensitive to light when the retinal is converted to its *trans* form. Sensitivity is regenerated by the replacement of the all-*trans* retinal by a newly synthesized 11-*cis*-retinal provided from the retinal epithelial cells. Opsins are functional while bound to either chromophore, with 11-cis-3,4-bound opsinλ_{max} being at a longer wavelength than 11-cisretinal-bound opsin.(http:// en.m. Wikipedia.org> wiki). Photopsins are the photoreceptor proteins found in the cone cells of the retina that are the basis of color vision. (Litmann and Mitchell 1996).

2.2.3.1 Photo pigments of rods

The pigments in rods are derived from plant pigment called carotenoids. The visual pigment in the human eye is a fragment of carotene and it is called cis-retinal. The pigment is attached to a protein called opsin to form a light-detecting complex called rhodopsin. The opsin protein is encoded by a gene located on chromosome 3. This gene has a considerable sequence similarly to the genes that encode the opsin protein of the cone.

2.2.3.2 Photo pigments of cones

The photo-pigments of the cones like that of the rods, have two components: a trans membrane protein (opsin) and the chromophore, 11-cis-retinal. Like as in rods, the pigment in cones is a fragment of carotene called cis-retinal (derivative of vitamin A). The pigment is attached to a protein called opsin to form a light-detecting complex called photopsin. There are three different types of pigment which are sensitive to, blue, red or green wavelengthes of light.

These three pigment types contain the same retinal molecule but differ in opsin molecules (Neitz and Maureen, 2010).

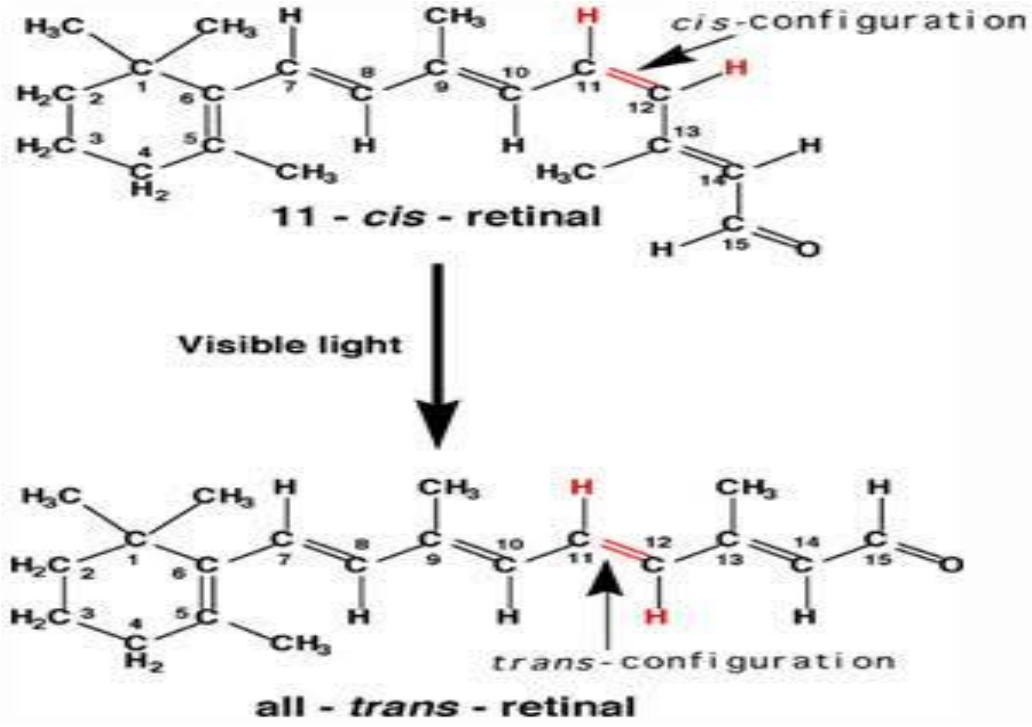


Figure 3: Configurational change of retinal from 11-cis-retinal form to all-trans-retinal form following absorption of light photon (Neitz and Maureen, 2010)

The retinal exists in the 11-cis-retinal form in the dark. When it receives a photon of light, the pigment undergoes a change in its shape and it becomes trans-retinal. This is due to the rotation of its carbon-12 which results in the rotation of all other carbons distal to it (Fig.3). The change in the shape of the pigment induces change in the shape of the protein opsin to which the pigment is bounded, initiating a chain of events that leads to the generation of the nerve impulse (Candice, 2015).

2.3 Vision

Vision is the sense by which the qualities of an object (as a color) that make up its appearance are perceived through a process in which light rays entering the eye are transformed into signals that pass to the brain.

Vision begins with light passing through the cornea and then the lens adjusts the focus. Both combine to produce a clear image on the photoreceptors of the retina. The information from the retina is sent to the optic lobe of the brain by the optic nerve in the form of electrical signals and the brain processes the impulse and allows us to see.

Rods are responsible for vision at low light levels (scotopic vision). They do not mediate color vision and have a low spatial acuity. Cones are active at higher light levels (photopic vision) and are capable of color vision and are responsible for high spatial acuity.

2.3.1 Color Vision

The eye is very important to perceive colors using the photoreceptors (cones) found in the retina. Color vision requires the presence of more than one type photopigments. Photopigments are the light absorbing molecules in photo receiptors (Neitz and Maureen, 2010). Cones are the photoreceptors that help us to see colors. There are three types of cones: S-cones (absorbing short-wave length), M-cones (absorbing medium-wave length) and L-cones (absorbing long-wave length) (Neitz and Maureen, 2010). The three versions of opsin in the cones absorb light at around 426nm (blue), 530nm (green) and 560nm (red), respectively (Fig.4)(Nathans et al., 2011). The cones have less stacked disks in their outer membrane and therefore, contain fewer photo-pigments, and this characteristic makes them less sensitive to light, whereas in rods there are more photopigments in the outer membrane. Stacked disks are the place where the photo pigments are found. The three types of cones respond to variation in color in different ways and make possible trichromatic vision (Manreen and Jay, 2000).

2.3.2 Normal color vision: A person with normal color vision can typically perceive up to 1 million different shades of colors. Normal color sighted individuals are trichromats, meaning that have three different color sensitive cons in their retina: red, green, and blue (Fig.4).

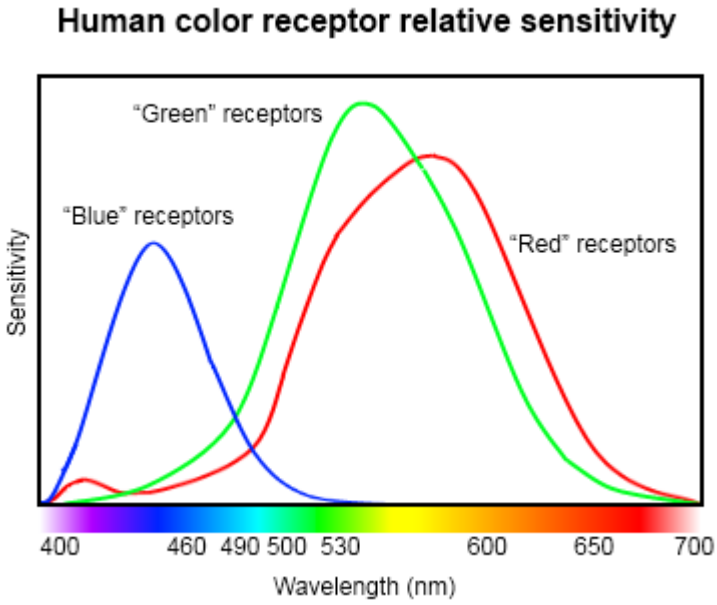


Figure 4: Cone-pigment absorption spectra (Maureen and Jay, 2000).

2.3.3 What is color vision deficiency?

Color vision deficiency describes the inability or decreased ability to identify colors or perceive color differences, under normal lighting conditions. It may vary from not being able to tell certain colors apart to not being able to identify any color (Steven, 2013). People born with color vision problems are unaware that what they see is different from what others see unless it is pointed out to them. Most people with color vision deficiency can see colors, but they have difficulty in differentiating between particular shades of red and green or blue and yellows (Mulusew and Yilkal, 2013).

2.3.3.1 Types of color vision deficiency

There are three types of inherited color vision deficiency:monochromacy, dichromacy and anomalous trichromacy (Neitz and Maureen, 2010). These are further categorized into sub-types. The effect of color vision deficiency can be categorized by the effect they have on our visual sense. The following are the different forms of color vision deficiency.

A. Monochromatism: This is a condition when there is no functional cone present or just only one type of functional cone is present. This is due to mutations in the genes encoding the proteins of the photoreceptor. This type of color vision deficiency is also known as "Total colorblindness" (Luo et al., 2015). It is the lack of the ability to distinguish colors (and thus the person views everything as if it were on a black and white television). In such people, only rods are functional and cones are non-functional. Individuals can perceive only in white, grey, and black tones (Karim and Saleem, 2013). It is a rare hereditary condition. Two subtypes of this deficiency are known:

(i)Rod monochromacy: It is an exceedingly rare inability to distinguish any colors as a result of absence or nonfunctioning of cones, only the rods are functional. This is frequently called achromatopsia, where none of the cone cells have functional photo pigments, so that in addition to the absence of color discrimination, vision in lights of normal intensity is difficult. People with rod monochromacy see the world in black, white and gray (Karim and Saleem, 2013).

(ii)Cone monchromacy: This is the condition where both cones and rods are present, but with only a single kind of cone functional. A cone monochromat can have good pattern vision at normal day light levels, but will not be able to distinguish colors. Blue cone monochromacy is caused by lack of functionality of L (long wave length) and M (medium wave length) absorbing cones.

B. Dichromatism: This is a moderately severe color vision; it is due to the presence of function of only two types of cones with the third cone being non-functional. The gene mutation results in the absence of visual pigment of one

cone type or pigment of either L or M or S. Dicromats exist in three different types according to which of the three cone types is not functioning. These are called protanopia, deuteranopia and tritanopia. The former two types of defects are controlled by two separate loci located side-by-side on the X-chromosome. The two types of defects together constitute what is known as red-green colorblindness. The locus controlling tritanopia is located on the autosomal chromosome number 7 (Table 1). These are the commonly inherited color blindness that affects a substantial portion of the human population. The three types of dichromatism are briefly described below.

(i) Protanopia: It is caused by the absence of normal function of the L-cones (missing of red photoreceptor cones), thus removing the ability to see red color. It is a severe form of color blindness. It affects about 1% of males and 0.02% of females worldwide (Betsy, 2003).

(ii)Deuteranopia: This is caused by the absence of function of M-cones, absorbing the green part of the light wavelength), giving rise to a moderate inability to discriminate green color (Dasupuram, 2013). It affects about 1% males and 0.01% females in the world.

(iii) Tritanopia (blue-yellow color vision deficiency): this is caused by mutation in the gene encoding opsin of the S-cone, removing the ability to see blue color (Kiula et al., 2011). The opsin protein of the photopigment is encoded by a gene which reside on chromosome 7, an autosomal chromosome. Tritan color vision defects are due to autosomal dominant. Thus tritanopia is expected to be equally frequent among males and females. It is a rare color vision problem, in which there are only two out of the three types of cones are present. The defect affects about 0.002% males and 0.001% females worldwide (Dasupuram et al., 2013).

C. Anomalous trichromatism: It is a type of inherited color vision deficiency, occurring when one of the three cone pigments is altered in its spectral sensitivity. Anomalous trichromatism also exists in three different types according to its malfunctioning cone type (Maureen and Jay, 2000). These are:-

(i) Trianomaly: This is due to the malfunctioning of the S-cone (blue absorbing cone). It is a rare, hereditary color vision deficiency affecting blue-green and yellow-red/pink hue discrimination. It is related to a gene on chromosome 7. Here the S-cone is malfunctioning but not missing (Mohammed, 2015).

(ii) Deuteranomaly: It is due to the malfunctioning of the M-cone (the green absorbing cone) (Bansal et al., 2005). It is caused by a shift in the green retinal receptors and it is, by far, the most common type of color vision deficiency. It affects red-green color discrimination in about 5% of European males. It is hereditary and sex-linked. The difference with deutranopia is that in this case the green sensitive cones are not missing but they are malfunctioning (Mohammed, 2015).

(iii) Protanomaly: It is caused by the malfunctioning of the L-cone (red absorbing). It is a mild color vision defect in which an altered spectral sensitivity of red retinal receptors results in poor red-green hue discrimination. It is hereditary, sex-linked and present in about 1% European males. The difference with protanopia is that in this casethe photo pigment in L-cone is present but it is malfunctioning, whereas in the former the function of the photopigment in the L-cone is completely missing (Mohammed, 2015).

Table-1. The different types of colorblindness and their associated chromosomes (Colblindor, 2006).

Types of color vision deficiency	Chromosome bearing the responsible gene
Deuteranopia	X- Chromosome
Deuteranomaly	X-Chromosome
Protanopia	X-Chromosome
Tritanopia	Chromosome 7
Tritanomaly	Chromosome 7

2.3.4 Causes of color vision deficiency

Color vision deficiency can be classified as acquired or inherited.

a. Acquired deficiency: Certain diseases, drugs and chemicals may cause color blindness. It can also be caused by accidents and other trauma which produce swelling of the brain in the occipital lobe. Damage by exposure to ultraviolet light (10-300nm) may also cause colorblindness. Color vision can also decline with age, most often because of cataract, a clouding and yellowing of the eye's lens (Maureen and Jay, 2000).

b. Inherited deficiency: Most of the inherited color vision deficiencies are due to the inheritance of mutated gene on the X-chromosome. This is a rearrangement or deletion of genes that encode the light absorbing photopigment molecules (opsins) in the cones. Colorblindness is typically an X-linked recessive inheritance (Maureen and Jay, 2000).

Of the three types of inherited color vision deficiencies, which are (monochromacy, dichromacy, and anomalous trichromacy) the most common type of color blindness is the red-green deficiency or deuteranopia. This deficiency is present in about 8% of males and 0.5% females of North European ancestry (Agarwal and Bansod, 2014). This color blindness is passed on via the X chromosome.

i. The genetics of color vision deficiency

The genes responsible for the most common types of genetic abnormalities, red-green color blindness, are located on the long arm of the X chromosome. The gene for blue absorbing pigment is located on chromosome 7 (Akhtar, 2015). Mutations capable of causing color blindness originate from at least 19 different chromosomes and 56 different genes. The two genes associated with red-green color vision defects are OPN1LW (opsin 1 long wave), encodingthe opsin of the red wavelength absorbing pigment and OPN1MW (opsin 1 middle wave), encoding the opsin of green wave length absorbing pigment (Neitz and Maureen, 2010). The OPN1LW and OPN1MW genes reside in a cluster with a head-to-tail configuration on the X-chromosome at Xq28.

The gene for the short wavelength (S) cone opsin is located on chromosome 7 at 7q32. There are 15 amino acid differences between OPN1LW and OPN1MW. There is a single gene for the red photopsin but there are multiple ones for the green cone opsin (Neitz and Mauren, 2010). OPN1LW and OPN1MW are nearly the same to one another, sharing more than 98% of nucleotide sequence identity, but they share only about 40% nucleotide sequence identity with OPN1SW.

Blue-yellow color vision deficiency is inherited as an autosomal dominant trait caused by mutation in the OPN1SW gene. It is a rare type of color deficiency where the affected person finds it difficult to differentiate between blue and yellow (Hesham et al., 2013). Yellow may be perceived as grey or purple. The blue-yellow deficiency is passed on through a non-sex chromosome and it is equally inherited through both parent and equally common among men and women (Neitz and Maureen, 2010).

ii. Transmission and Expression of X-linked color vision deficiency gene

Traits that are determined by alleles carried on the X chromosomes are referred to as X-linked. Red-green color blindness is a good example of such a trait (Kiula et al 2011). In humans, there are 23 pairs of chromosomes, 22 pairs of chromosomes are same in both sexes. They are referred to as autosomes. The 23rd pair of human chromosomes consists of sex chromosomes(X and Y) females carry XX and males XY combination of sex chromosomes (Bellot, 2010).

Therefore, for a female to be color blind, both her X chromosomes should contain the recessive allele. In other words she should receive the defective allele form both her parents. This makes colorblindness less common in females. Color blindness is more common in males because, a single dose of a single X chromosome carrying the defective gene can causes colorblindness, as there is no corresponding gene on the Y chromosome that would mask the expression of the defective recessive gene carried on the X chromosome (Colblindor, 2006).

The pattern of transmission and expression of colorblindness gene from the mother and father and the expression of the gene in male and female offspring can best be illustrated by using a Punnet square as presented in Fig. 5 below. Suppose one uses the symbol X^C and X^c for X chromosome carrying normal and colorblind allele, respectively. A female, being with two X chromosomes will have X^CX^C or X^CX^cor X^cX^c genotypes. This gives normal, normal (but carrier) and colorblind phenotype, respectively. As a male has only a single X chromosome, he can either have X^CY or X^cY, and will have normal and colorblind phenotype, respectively. Various mating combinations of parents with regard to their colorblind genotypes are presented in the Punnet square below.

Cases 1. The following is a mating between a normal father and a carrier mother, where, X^c represents colorblind causing recessive allele, X^C normal alleles, respectively:

	X^C	X^c
X^C	X^CX^C(Normal)	X^CX^c(Normal carrier)
Y	X^CY (Normal)	X^cY (Colorblind)

Case 2. Mating between normal father and colorblind mother

	X^c	X^c
X^C	X^CX^c(carrier)	X^CX^c(carrier)
Y	X^cY (Colorblind)	X^cY (Colorblind)

Case 3. A mating between a colorblind father and colorblind mother

	X^c	X^c
X^c	X^cX^c	X^cX^c
Y	X^cY	X^cY

Figure 5: Punnet square showing the pattern of transmission of X-linked colorblindness gene from maternal and paternal parents and expression of the gene among their children

A closer look at the above three punnet squares will show the following out comes of the crosses

Case 1.

The phonotypes of the progeny are:

- 1/4(or 25%) normal female
- 1/4 (or 25%) carrier female
- 1/4 (or 25%) normale male and 1/4 (or 25%) colorblind male

Case 2.

All females become carriers, but do not show colorblindness because the recessive allele of the mother is masked by the normal allele from the father, where as all males are colorblind.

Case 3.

All children (100%) become colorblind, all females receive double dose of the recessive allele, (one copy from each parent) and all male receive the recessive allele from the mother.

2.3.5 Prevalence of color vision deficiencies

Prevalence of hereditary trait is usually discussed in population context. Population may be defined as a group of individuals that share a common gene pool. A population may comprise of a whole species or a subgroup of a species. In other words, a population is a group of individuals that freely interbreed with each other (Falconer, 1960), be it the whole species or its sub-group. Any genetic study that is done at a population level, including the present study, is considered as a type of population genetics.

2.3.5.1 Allelic, genotypic and phenotypic frequencies

In a population genetics, the main interest is to determine the frequencies of alleles, genotypes and phenotypes of trait(s) of interest in a population and factors that maintain or change these frequencies. In this process of determining these frequencies a sample is drawn from a population and analyzed and the result is extrapolated to the population.

In a population, alleles at a given locus may or may not occur in equal frequencies. For Example, if A and a are two alleles of a particular gene at a locus in a population, the two alleles may be equal in frequency or A is more

frequent than a or vice-versa. When the frequencies of the two alleles are equal (A=0.5, a=0.5) the expected frequency of the genotypes =1/4AA, 1/2Aa, 1/4 aa, and phenotypic frequency = 3/4A- and ¼ aa. When the two alleles do not have equal frequency, both the genotypic and phenotypic frequencies will deviate from the above frequancies.

In a population genetics, conventionally allelic frequencies may be designated as P and q, where A=P and a=q. The random mating among individuals in the population (Fig.6) will give rise to genotypic frequencies of P^2AA, 2pq Aa and q^2aa (Table 2). The dominant phenotype (AA+ Aa) =p2 + 2pq and recessive phenotype (aa) = q^2 (Falconer, 1960).

Table-2: Random mating among individuals in population (Falconer, 1960).

Gamete	PA	qa
PA	P^2 AA	Pq Aa
qa	Pq Aa	q^2 aa

The above genotypic and phenotypic frequency works for both male and female populations if the trait is determined by an autosomal linked gene but in case of X-linked gene it applies only for females. In males, allelic frequency =genotypic frequency = phenotypic frequency. Thus P=A, q=a holds for all the three cases in males for X-linked genes (Shorrocks, 1978).

2.3.5.2 Prevalence of color vision deficiencies in different populations

The incidence of color blindness varies from race to race or between people of different ethnicity (Rahman,1998). Different studies show that the prevalence of color blindness in the world is 1 in 12 male (8%) and 1 in 200 women 0.5% (Haile, 2014). In small isolated communities, the prevalence may increase, including that of more rare forms as a matter of random chance or

founder effects. Examples of countries with such communities include rural areas of Hungary, Finland and some Scottish islands (Rahman, 1998).

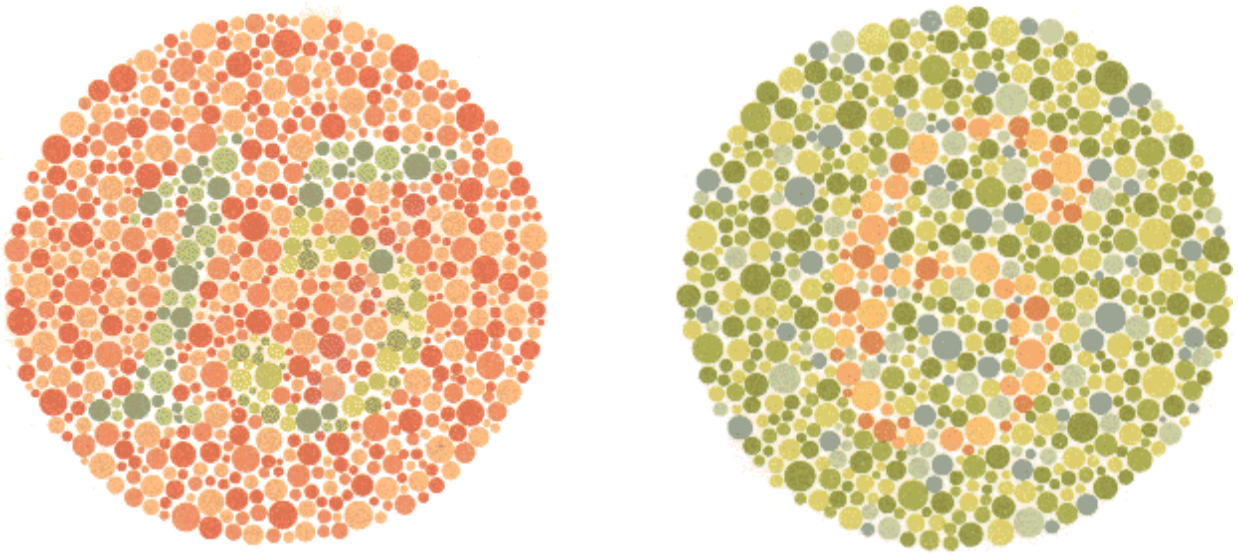
The study on prevalence of color blindness so far done on Ethiopian population is very scarce. According to these studies, the prevalence of congenital color blindness among Ethiopians was reported to be 4.2% among males and 0.2% among females (Mulusew and Yilika, 2013) in which protanopia and deuteranopia are the most common types of color blindness (Haile, 2014). The prevalence of color blindness among school children was reported to be 2.22% among males and 0.83% among females (Tsega, 2017). The prevalence of color blindness among school children was also reported to be 3.6% among males and 0.6% among females (Gashaw and Teshome, 2018).

2.3.6. Tests for color vision deficiency

Any person who is having difficulty in identifying colors should be checked for color vision impairment. Those who have family history of color vision deficiency are required to identify colors accurately or those who have problems in indentifying colors should be tested.

There are different methods of testing individuals for colorblindness. These include Ishihara plate tests, anomaloscope test, arrangement test and lanterns.

a. **Ishihara plate test**: The most commonly used test is Ishihara 38 and 24 plate test. The Ishihara 38 Plates color vision deficiency test was introduced by Dr. Shinobu Ishihara from Japan a long time ago. These plates are named after him and by far they are the best known test for red-green color blindness (Ananya, 2016). They are made up of many colored circles and contain numbers or symbol written in them, which can only be seen and recognized if one is not colorblind (Fig 6).



A. B.

Figure 6: Samples of Ishihara test plates used for color blindness test (Shinobu Ishihara, 1972).

In the above figure, a normal visioned individual can read the number in plate A and Plate B as 15 and 6, respectively. On the other hand, individuals who have color vision problem read them as 17 and X (the plate cannot be read), respectively or they may not recognize any number.

b. Anomaloscope: This method provides the most accurate distinction between dichromats and anomalous trichromats. A mixture of red and green light sources have to be matched with a yellow light source. Through the matching range, it is possible to discover all the different types of red-green color vision deficiency (Ubom, 2014).

c. Arrangement tests: This consists of certain number of colored discs or plates which have to be arranged in the correct order starting from a pilot plate (Ubmob, 2014).

2.3.6.1 Symptoms of color vision deficiency

The symptoms of color vision problems may vary, but may include the following:

The affected individuals may:

- Be able to see some colors but not others. For instance, they may not tell the difference between some reds and greens but can see blue and yellow easily
- Not be able to see many colors, so the individuals may not know that they see color differently from others.
- Be able to see only a few shades of color, while most people can see thousands of colors.
- In rare cases, some people see only black, white, and grey (Moudgil et al, 2016).

2.3.6.2 Treatment of color vision deficiency

Curing color blindness is currently impossible. About 99% of color blind males and females are color blind as a result of defective genetics on the X-chromosomes. There is a hope for a cure of inherited color vision deficiency using gene technology. This will involve injecting invitro synthesized photopigment into the eye. At the moment, this has only been proved to work in monkeys (Elie, 2009).

Some acquired color vision problems can be treated, depending on the cause. For example, if cataract is causing color vision problems, surgery to remove the cataract may restore normal color vision (Kristin, 2015).

The following may help to solve color blindness to some extent:

- Wearing color contact lenses. These may help to see differences between colors. But these lenses do not provide normal color vision and can distort objects.
- Wearing glasses that block glare. People with severe color vision problems can see differences between colors better when there is less glare and brightness

- Learning to look for cues like brightness or location, rather than colors. For example, learning the order of the three colored lights on traffic signals.
- Label coloring utensils (Pen, Pencils) with color name (Kristin, 2015).

2.3.7 Impact of color vision deficiency

Many people are affected by colorblindness but many of them are undetected as they simply adapted to the environment to certain extent and also because of unawareness of the deficiency. Many tasks that we do each day rely on our being able to separate things by their color. If people are not able to see the difference in color, they have to rely on other differences, which may be harder to pick. Color vision defects affected people's choices of career and many have been excluded from a chosen occupation (Richeson and Nussbaum, 2004). For example, there may be restriction on car driving.

Color vision deficiency causes learning difficulty. In the classroom, blocks or other teaching tools may be color coded as well as being of different size. A child with color vision problems may have to rely on size difference alone (Moudgil, 2016).

3. Materials and Methods

3.1 Schools Selected for study

The study was carried out in TemenjaYaj and ShimelisHabte secondary schools. The schools are found in Kirkos Sub-city of Addis Ababa.The total number of students enrolled in Temenja Yaj School in 2018/19 academic year was 763 of which 458 were females and 305 were males. In Shimelis Habte School the total number of students for the same academic year was 923 consisting of 551 females and 372 males.

Sample size

The total number of students of the two secondary schools are 1,686. Among these the researcher selected 24 students from a maximum number of 40 students in each class by using simple random sampling. Therefore 1,012 students were included in the research. Basically, for this study a large number of students participated in order to increase the chance of including among the study subjects, students who have got color blindness.

Regarding the selection of male and female participants, stratified sampling technique was applied. This is because of un equal number of male and female students in the target schools. First, divide the students in male and female group. Second, the male students grouped in to five and the female students also grouped in to five then selected three students from the each group. Hence, from1012 total sample, 406 were males and 606 were females.

Inclusion criteria

Temenja Yaj and Shimeles Habte secondary schools volunteer children who have a written consent from their parents or adult guardian were included. Also, students with normal sight participated in the test for color vision defect.

Exclusion criteria

Students, who were not willing to participate, as well as students with no written consent from parents or guardians were, excluded. Students who have vision problem were also excluded. For this purpose list of students with special eye problems was obtained from the special Need Office in the respective schools and they were excluded from the study.

Table 3. Student populations and number of students participated in the study from the two schools.

School	Total student population in each school	% of total	Sex		Sample size in each school	% of sample size
			F	M		
Shimelis Habte Secondary School	923	55%	333	223	556	33%
Temenja Yaj Secondary School	763	45%	273	183	456	27%
Total	1686	100%	606	406	1012	60%

3.2 Procedures of data collection

Data were collected in the respective compounds of the two high schools. Permission of the school administrators were obtained before the test was conducted. Signed consent of the parents\guardians was obtained for children under 18 years of age. Then orientation was given to the students about color blindness and the objective of the study. Those students who volunteered to participate were asked to give their consents by signing on the consent form prepared for this purpose (see appendix I).

The test was carried out in a properly lighted room. The test was done by using the 24 Ishihara test plates. The Ishihara plates were exposed on laptop screen one plate at a time. Each study subject was asked to read the figures in the Ishihara plate as they appeared on the computer screen and at a rate of 3 to 5

seconds per plate from a distance of 75cm. The result of the reading was immediately recorded in a form prepared for this purpose (see appendix II). In times when electric power was non available, color prints of the Ishihara plates were used to replace laptop screen.

3.3 Method of testing for the color vision deficiency

Ishihara test plates were used to identify the presence of color blindness and the type of color blindness present among individuals. First plate 1- 24 of the Ishihara's test plates were presented to the subjects one by one to read. Plate 1- 17 contain numerals and plates 18-24 contain one or two wiggly lines. If 10 or more plates, out of the 24 plates, are read correctly, the color vision is regarded as normal (Shinobu Ishihara, 1972), where as if only 9 or less plates are read correctly, the color vision is regarded as deficient. Subjects who were classified as color deficient were re-tested by using plates (No.16) and (No.17) in order to determine deutans and protans type of dichromats. A person with normal color vision reads 26 in plate 16 and 42 in plate 17. In protanopia and protanomalia only **6** (plate no.16) and **2** (plate No.17) are read, and in case of mild protanomalia both numerals on each plate are read but the **6** (plate No.

16) and **2** (Plate NO.17) are clearer than the other numerals. In deuteranopia and strong deuteranomalia only **2** (plate No. 16) and **4** (plate No.17) are read, and in case of mild deuteranomalia both numerals on each plate are read but the **2** (plate No.16) and **4** (plate No. 17) are clearer than the other numerals. (Ishihara 1972).

Among the color blind individuals, those who read not more than two plates are classified as totally colorblind (monochromats). Individuals that are grouped under unclassified can read plate number 16 and 17 differently from what red-green colorblind individuals read. If these individuals are further tested using the other types of tests such as anomaloscope, their specific type of defect may be determined (see appendix 2).

3.4 Statistical Analysis

The collected data were entered manually into a computer on excel sheet and frequencies of population genetic parameters were tested using Chi-square test and Hardy-Weinberg equilibrium. The data were arranged and checked before the analysis. The analysis was done gender wise and the prevalence of color blindness was determined on the data of individual school as well as on the combined data of the two schools.

4. Results

Among the 1012 subjects tested, 990(97.83%) of the participants were found to have normal color vision. The total number of students who had color vision defect was 22(2.17%) which includes 5(0.49%) females and 17(1.68%) males (Table 3).

Table 4. Frequency of different types of color blindness sex wise

No. tested	Normal color vision	Types of color vision defect	Sex		Total
			F	M	
1012	990(97.83%)	Achromatopsia (total colorblindnes)	_	10(0.99%)	10(0.99%)
		Deuteranopia	4(0.39%)	4(0.39%)	8(0.79%)
		Protanopia	1(0.099%)	2(0.19%)	3(0.29%)
		Unclassified	_	1(0.099%)	1(0.099%)
		Total	**5(0.49%)**	**17(1.68%)**	**22(2.17%)**

Colorblind females are further classified into 4(0.39%) deuteranopia and 1(0.099%)protanopia. The prevalence of red-green colorblindness (deutan and protans) in females was 0.49%. Females with deutrans types of colorblindness are relatively more prevalent than protan type. Achromatopsia (total colorblindness) was not observed in the studied female subjects.

The 17(1.68%) males are further classified into 10(0.99%) achromatopsia, 4(0.39%) deutan, 2(0.19%) protan and 1(0.099%) unclassified. The unclassified individual has color vision defects like the others, but they were not able to read plates number 16 and 17 or read different number from what protons and deutans can read in these plates. The prevalence of red-green colorblindness in males was 0.58%. The frequency of males with color vision deficiency was higher than in females by 1.19%. Achromatopsia males are more frequent than the other type of colorblind subjects. The prevalence of the different type of

colorblindness in this study shows that the percentage of monochromates was more than dichromats. Among dicromats, deutans (0.79%) had higher frequency than protons (0.30%).

When we consider the males and females separately, because the trait is X-linked, the percentage of colorblindness among males and females was different. Out of 406 males tested, 17 were colorblind. Colorblind males make 17/406 or 4.19% among the males. Only 5 females were colorblind out of the total 606 females tested, which makes 5/606 or 0.83% of the total females tested.

Deuteranopia and protanopia are the two major types of red-green colorblindness and are due to mutating in X- linked locus. Thus Phenotypic, allelic and genotypic frequencies were calculated for male and female populations separately for the two common types of red-green color vision deficiency, (Table 5 and 6). Even though the two classes of defects are X-linked, they are controlled by different loci. Thus, the frequency for the two traits are considered separately. For the sake of simplicity, we represented the protanopia locus gene (OPN1LW) by L for normal allele and by l for the mutant allele. The calculation shows that in males the dominant normal allele (L), had a frequency of P=0.9951 and the recessive (l) frequency of q= 0.0049.

In females, the dominant phenotype has a frequency=p^2+2pq=0.9983 and the frequency of recessive phenotype, q^2=0.0017. The allelic frequency is 0.9588 for (L) and 0.0412 for (l). The genotypic frequency for LL = (p^2) =0.9193, heterozygous Ll =2pq =0.0790 and homozygous recessive is q^2=0.0017 (Table 5), provided that the population is at Hardy-Weinberg genetic equilibrium.

Table 5. Phenotypic, allelic and genotypic frequencies of protanopia among the tested subjects by sex

Trait	Item		sex	
			Male	Female
Protanopia	Sample size	Normal	404	605
		Protan	2	1
		Total	406	606
	Phenotypic frequency	Normal	0.9951	0.9983
		Protan	0.0049	0.0017
		Total	1.00	1.00
	Allelic frequency	L	0.9991	0.9588
		l	0.0049	0.0412
		Total	1.00	1.00
	Genotypic frequency	L	0.9951	-
		l	0.0049	-
		Total	1.00	-
		LL	-	0.9193
		Ll	-	0.0790
		ll	-	0.0017
		Total	-	1.00

For ease of presentation, the alleles at OPN1WM locus of deutranopia are symbolized as M and m for normal and mutant alleles, respectively.

The allelic, genotypic and phenotypic frequencies for deutranopia were calculated in the same way as described above for protanopia. In males, the frequency of allele M was 0.9901 and m was 0.0099 (Table 6). These frequencies are also true for genotypic and phenotypic frequencies of the trait.

In females, the frequency of normal vision is 0.9934 and the frequency of deutrans is 0.0066. The allelic frequencies calculated based on the above phenotypic frequencies gave 0.9188 and 0.0812 frequency of M and m alleles, respectively. Based on the above allelic frequency of M=P=0.9188 and

m=q=0.0812, the genotypic frequencies are calculated as MM (p^2) = $(0.9188)^2$, Mm (2pq) =(2x(0.9188x0.0812)) and mm = $q^2(0.0812)^2$. These gave 0.8442, 0.1492 and 0.0066 frequencies for the three genotypes, respectively. The a bove genotypic frequencies hold true provided that the population is at Hardy-Weinberg genetic equilibrium.

Table 6. Phenotypic, allelic and genotypic frequency of deutranopia among the tested subjects by sex

Trait	Item		sex	
			Male	Female
Deuteranopia	Sample size	Normal	402	602
		Deutan	4	4
		Total	406	606
	Phenotypic frequency	Normal	0.9901	0.9934
		Deutan	0.0099	0.0066
		Total	1.00	1.00
	Allelic frequency	M	0.9901	0.9188
		m	0.0099	0.0812
		Total	1.00	1.00
	Genotypic frequency	M	0.9901	-
		m	0.0099	-
		Total	1.00	-
		MM	-	0.8442
		Mm	-	0.1492
		mm	-	0.0066
		Total	-	1.00

The female data on protanopia and deutranopia were subjected to chi-square test in order to determine whether the population is at Hardy- Weinberg genetic equilibrium regarding each of the two loci (Table 7).

The result showed that there is no significant deviation between the observed numbers and the calculated number assuming that the population is at Hardy-Weinberg equilibrium (p>0.99).

Table 7. Chi-square test on female data for Hardy-Weinberg equilibrium

A. Female data (protanopia)

Class	Observed number(o)	Expected number(e)	Deviation (d)	Dev^2/exp
Normal	605	604.9698	+0.0302	0.00000151
protan	1	1.0302	-0.0302	0.000885
Total	606	606	0.0000	X^2=0.00088651

P>0.99

B. Female data (deutranopia)

Class	Observed number(o)	Expected number(e)	Deviation (d)	Dev^2/exp
Normal	602	602.0004	-0.0004	0.00000000027
Deutan	4	3.9996	+0.0004	0.00000004
Total	606	606	0.0000	X^2=0.00000004027

P>0.99

The chi-square value shows that the observed result is in good agreement with the expected result indicating that the population is at Hardy-Weinberg equilibrium.

5. Discussion

Detection of color vision defect early in life of an individual is very important to make informed decision on future carrier. Early detection of colorblindness allows parents and teachers to make the necessary adjustments to the teaching learning process of affected children. But, in case of our country, much has not been done to screen children for colorblindness. Lack of awareness about the defect, and lack of facilities for testing the defect are among the problems in our country.

Available studies on the prevalence of colorblindness in Ethiopia are very few. One such study reported prevalence of colorblindness as 4.2% among males and 0.2% among females (Mulusew andYilikal, 2013). The study indicates that Ethiopians have a much higher incidence of colorblindness (4.2%) than other sub-Sahara population. Other studies (Mulusew and Yilikal 2013) also indicate the prevalence of colorblindness in Ethiopia who studied the prevalence of colorblindness in the school of Abeshage District, in central Ethiopia. In their study they use Ishihara,s test 38 plate editions on 850 sample size of which 4.2% cases of defective color vision were detected. Among these, 2.89% cases were deutans, 0.58% unclassified, and 0.19% cases of total colorblindness. The prevalence of colorblindness was also studied by Haile Fantahun on school children in Addis Ababa by using Ishihara test 38 plate edition on a sample of 378 subjects of which (4.2%) was colorblind. Among these, 1.3% were females and 2.9% were males (Haile, 2014). The prevalence of colorblindness in the two studies was similar. Recently colorblindness was also studied by Gashaw and Teshome on school children in Wolkite, Southern Ethiopia by using Ishihara test 38 plate edition on a sample of 844 of which 4.1% were colorblind. Among these, 0.6% were females and 3.6% were males (Gashaw and Teshome, 2018). Color blindness was also studied by Tsega Habte on school children in Akaki/Kality Sub-city of Addis Ababa by using Ishihara test plate 24 plate edition on a sample of 1081 of which (3.05%) was color

blind. Among these, 0.83% were females and 2.22% were males (Tsega, 2017). In the present finding, the frequency of colorblindness is lower than that of others because we used Ishihara 24 test plate and small sample size compared with that of Tsega.

A study on prevalence of colorblindness among university students in Ethiopia was also carried out by Bekele Serbessa and Kifile Dagne on a total of 1899 male and 238 female students. The allelic frequencies among male is 0.011 deutan and 0.0918 protan. In females deutan gene frequency is 0.0924 and protan is 0.065 (Bekele and Kifile, 2013, un published).

The prevalence colorblindness in males in this study was 1.68% which is similar with studies that were carried out in Pakistan (1.1%) and India (1.12%)(Karim, 2013). However, the prevalence of the colorblindness among male students in this study is lower than that was reported from Libya (2.2%), India (2.3%), Colombia (2.4%) and Saudi Arabia (2.9%) (Fareed et al., 2015).

In the present study the prevalence of colorblindness among female students (0.49%) is similar with Greenland (0.4%) and Iran(0.43%). However, the prevalence of colorblindness among female students in this study is lower than other studies done in Saudi Arabia (0.75%), Aligarh and Simla (0.8%), Spain (0.75%) and India (0.8%). The present study revealed 2.17% of colorblind people, which is higher than studies done in China (1.7%), Mexico(1.3%) and Russia(1.8%). However, it is lower than reports made from Britain(7.8%), USA(4.8%) and Canada(4.2%)(Karim, 2013).

Frequency of colorblindness among non-Europeans is lower than in persons of European ancestry in whom it is reported to be 6.0% for males and 0.25 % for females. In some European countries even higher prevalence is reported; 7.8% of school boys in Germany, 7.95% among males in Greek and 7.33% in young Turkish men were reported to have congenital color vision defect (Mulusew, Yilikal, 2013). The prevalence rate of female colorblindness reported from different parts of the world are Denmark 0.5%, Greenland 0.4%, Italy 0%,

Newzealand 0%, Iraq 3.2%, Iran 0.43%, Jordan 0.33%, Spain 0.75% and Saudi Arabia 0.75% (Shah, et al 2013), Japan 3.6% and Black Americans 3.7% (Niroula, Saha 2010). But similar with some findings recorded in different studies such as 1.9% and 2.1% in Nepal, 2.6% in Nigeria (Karim, 2013). But the prevalence that is performed in Ethiopia is higher than the present study. The prevalence of colorblindness was 4.1% (Gashaw, Teshome 2018) which is higher than the present study 2.17%.

The chi-square test applied to the female data regarding the prevalence of protanopia and deutranopia has shown that there is no statistical significant deviation between the observed data and that expected assuming that the population is at Hardy-Weinberg genetic equilibrium. This indicates that, regarding color vision, the population is mating at random and no evidence for the operation of natural selection and non-random mating in the population regarding the trait.

6. Conclusions and recommendations

6.1 Conclusion

The present study showed the existence of protanopia, deuteranopia and achromatosopia among the school children.

The prevalence of colorblindness in the combined male and female data was 2.17%, which is similar with the finding of some other studies in the world.

Among the observed colorblind individuals, 1.68% were males and 0.49% were females. Among the 22 individuals with color vision defects, 10(0.99%) were achromatopsia, 8(0.79%) deutan, 3(0.29%) protan and 1(0.099%) unclassified out of the total 1012 students tested. The study indicated that the prevalence of achromatopsia (Total colorblindness) is higher than the other types of colorblindness. But red-green colorblindness (Protanopia and deuteranopia) was the most common type of colorblindness similar to that repeated in other studies for different populations.

The study showed that the frequency of colorblindness is more common in males than females which are in agreement with other reports as well as with its mode of inheritance as an X-linked trait. Chi-square test showed no significant deviation from Hardy-Weinberg genetic equilibrium.

6.2 Recommendation

Based on the present findings the following recommendations are made:

- In Ethiopia there are no adequate studies done on colorblindness and therefore, it is recommendable to perform further study on prevalence of colorblindness on large sample size taking additional parameters such as ethnicity into consideration.
- Most people in Ethiopia do not have awareness about colorblindness. Therefore creating awareness about colorblindness defect among teachers, students, parents the public in general is very important.
- Teachers should be trained to perform color vision screening and to adjust their teaching methods so that children with color vision deficiency can be accommodated. A section on colorblindness should be included in biology textbook in order to create awareness among teachers and students.

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Appendix I

1.1 Information sheet and consent form for study subject

The aim of this study is to identify the frequency of color deficiency among students attending schools in Kirkos sub-city. This study will provide good information about the heritable color vision defects for various institutions. During the study consent will be obtained from guardians and/or parents for children younger than 18 years. In addition assent will be subsequently obtained from students. All participants found positive for color defect willgetadvices.

For this study I have been requested to take ISHIHARA's color vision test. I have been informed by Helen kidane (a Msc student at AAU in Biology) herein referred to as investigator about a study of prevalence of congenital color deficiency among students from some selected schools in Addis Ababa. I have been informed that I will get an advice for color vision defect after being tested and found to be positive. The investigator has briefed me that there are no risks associated with the procedure and result. The investigator also informed me that all the test results would be kept confidential. Moreover, I have also been well informed of my right to withdraw from participating in this project and that my actions will have no impact on the overall management of my conditions. I have been given enough time to think over before I signed this informed consent. It is therefore, with full understanding of the situation that I gave my informed consent and cooperates at my will in the course of the conduct of the study.

N. B. The ethical aspects of this study were approved by the Ethical Committee of Faculty of Life Science, AAU. If you have any complaints or reservations about any ethical aspect of your participation in this research, you may contact the Committee through its secretary. Any complaint you make will be treated confidential and investigated, and you will be informed of the outcome.

Addis Ababa University **E- mail:** cnsethical@gmail.com

1.2 የጥናቱ ስነ ምግባር ደንብ

የጥናቱ ርዕስ፡ Frequency of Color Blindness among School Children in two Secondary Schools in Kirkos Sub- city of Addis Ababa, Ethiopia

የአጥኚዉ ስም እና አድራሻ፡ ሄለን ኪዳነ

ስነ-ህይወት ፋክልቲ፤ አዲስ አበባ ዩኒቨርሲቲ

ይህንን ጥናት በሚመለከት ለተሳታፊ ግለሰቦች የተገለጹና ተሳታፊዎች ስምምነታቸዉን የሰጡበት ነጥቦች

1. የጥናቱ ዓላማ፡ በተፈጥሮ በአንደንድ ግለሰቦች ለይ የሚከሰተዉን ቀለማትን የመለየት ችግር (color vision deficiency) በኢትዮጵያ ዉስጥ ዓይነቱና መጠኑ ምን እንደሚመስል ለማወቅ በት/ቤት ተማሪዎች ላይ ጥናት ማካሄድ ነዉ፤
2. የአጠናኑ ዘዴ ለዚህ ዓይነት ጥናት የተዘጋጁ በአለም አቀፍ ደረጀ በአገልግሎት ለይ የሚገኙትን የ ISHIHARAColor Vision Test) ቻርቶችን በመጠቀምነዉ፤
3. ከተሳታፊዉ የሚፈላገዉ ተሳትፎ በቻርቱ ዉስጥ የተፃፉትን ቁጥሮች እና ምልከቶች በማንበብ /መለየት ብቻ ነዉ፤
4. ጥናቱ በተመርማሪዉ (ተሳታፊግለሰብ)ላይ ምንም ዓይነት አካላዊም ሆነ ሌላ ጉዳት አያስከትልም፤
5. የጥናቱ ዉጤት በሚስጥር ይያዛል፤
6. የአንድ ተመርማሪ ዉጤት አዉንታዊ (positive) ከሆነ ለግለሰቡ ልዩ ምክር ይሰጠዋል፤
7. ይህ ጥናት በአዲስ አባባ ዩኒቨርሲቲ ስነ ህይወት ፋክልቲ የስነ ምግባር ኮሚቴዉ ተመርምሮ የጥናት ስነ ምግባርን የሚያሟላ መሆኑ የተረጋገጠና ፈቃድ የገኘነዉ ፤
8. አንድ የጥናቱ ተሳታፊ ቅሬታ ቢኖረዉ ለኮሚቴዉ ቅሬታዉን ሊያመለክት ይችላል፡፡

እኔም በዚህ ጥናት በፍላጎቴ ለመሳተፍ እና ለመደገፍ ተስማምቻለሁ፡፡ ተሳትፎዬንም ለመግለጽ ለዚሁ ጥናት በተዘጋጀ የቀለም ዕይታ ምርመራ የፈቃደኝነት ማረጋገጫ ቅጽ ላይ በፊርማዬ አረጋግጠለሁ፡፡

ማሳሰቢያ፡ ማንም ቅሬታ ያለዉ የጥናቱ ተሳታፊ የስነ ምግባር ኮሚቴዉን ቀጥሎ በተመለከተዉ አድራሻ ማግኘት ይችላል፡፡

ስም፡ ሄለን ኪደነ፡ የስነ-ህይወት ትምህርት ክፍል፡ አ.አ ዩኒቨርስቲ

ስልክ፡ 0913836715፡ Email:Helenkidane134@gmail.com

Advisors: Dr. Kifile Dagne Tel.09 11 10 39 37 Email: dagnekifles@gmail.com

Advisors: Dr. KifileDagne Tel.0911103937 Email: dagnekifles@gmail.com

1.3 Concent form Code no.....

Information about the study has been explained to me by the investigator. I understood that the objective of this study is to determine the prevalence of color blindness in school children and the information given by the children will serve only for this study not for any other purpose. It has also been explained to me that children have the right to stop participation at any time in between and there is nothing they will lose if they refuse to participate. I agree that my children to participate in the study and I hereby approved my agreement with my signature.

Participant's name &
signature.....
.....

Date.....

Investigator's name &
signature.....
.....

Date.....

1.4 የስምምነት መጠየቂያ ቅጽ በአማርኛ

የጥናቱ ተሳታፊ መለያ ቁጥር_____

ጥናቱን በተመለከተ በቂ ማብራሪያ ተደርጎልኛል፡፡

የጥናቱን አላማ በሚገባ የተረዳሁት ሲሆን የምሰጠውም መረጃ ለዚህ ጥናት ብቻ የሚውል በመሆኑ በልጄ ላይም ሆነ በእኔ ላይ ምንም አይነት ጉዳት እንደ ማያደርስ እና የምሰጣቸውም ማንኛውም መረጃዎች በሚስጢር እንዲጠበቁ ስለተገነዘብኩ በጥናቱ ልጄ እንዲሳተፍ በመወሰኔን በፊርማዬ አረጋግጣለሁ፡፡

የጥናቱ ተሳታፊ ወላጅ ወይም አሳዳጊስም_____

ፊርማ_____

የመረጃ ሰብሳቢው ስም_____

ፊርማ_____

ቀን____/ _____/ _____

1.5 የቀለም እይታ ምርመራ የፈቃደኝነት ማረጋገጫ ቅፅ

እኔ ስሜ እንዲሁም ሌሎች መረጃዎቼ ከዚህ በታች የተገለፀው ለዚህ ምርምር ተሳታፊ ለመሆን ስወስን በአዲስ አበባ ዩኒቨርሲቲ የማስተርስ ተማሪ የሆኑት ወ/ሮ ሄለን ኪዳነ የምርምሩን ህደትና ሁላንተናዊ ጠቀሜታዉን ገልፆልኛል፡፡ የጥናቱ የስነምግባር ደንብንም ካነበቡልኝ በኋላ እኔም እንድሳታፍ ጠይቀዉኛል፡፡ እኔም በተጠየቅኩበት መሰረት ያለምንም ግዴታ በሙሉ ፍቃደኝነት የተሳተፍኩ መሆኔን በፊርማዬ አረጋግጣለሁ፡፡

የት/ቤት ስም_____

መለያ ቁ.	**ስም ከነ አባት**	**ፆታ**	**ዕድሜ**	**ክፍል**	**ፊርማ**
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1.6 Data collection Form No _____ **ቅፅ ቁጥር፡**

_____

College /School Name_____

ISHIHARA plates Test

Code	Pla																							Remark
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	
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Appendix II

Table 1: Numerals on each plate and answers which would be given by normal color vision and color defective individuals (Shinobu Ishihara, 1972)

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	6	70				x
4	29	2				x
5	5	5				x
6	15	17				x
7	74	21				x
8	6	x				x
9	45	x				x
10	5	x				x
11	7	x				x
12	16	x				x
13	73	x				x
14	x	5				x
15	x	45				x
		protan		deutan		
		Strong	Mild	Strong	Mild	
16	26	6	(2) 6	2	2 (6)	
17	42	2	(4) 2	4	4 (2)	

The mark **x** shows that the plate cannot be read. The numerals in parenthesis show that they can be read but they are comparatively unclear.

Declaration

1. The undersigned, declare that this Thesis is my original work and has not been presented for a degree in any other University. All sources of materials used for the Thesis have been duly acknowledged.

Name: HELEN KIDANE

Signature: ……

This Thesis has been submitted for examination with my approval as a University advisor.

Advisor: Dr. Kifile Dagne

Signature: ……

November 19, 2019