ADDIS ABABA UNIVERSITY

COLLEGE OF NATURAL AND COMPUTATIONAL SCIENCES

DEPARTMENT OF ZOOLOGICAL SCIENCES

Frequency of Color vision Deficiency among Students of Andode Secondary School in Bole sub- city of Addis Ababa, Ethiopia

By

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JULY, 2021

ADDIS ABABA, ETHIOPIA

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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

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Approved By Board of Examiners

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Examiner	Signature	Date

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Abstract

The visual system, in the human eye receives physical stimuli in the form of light and sends these stimuli as electrical signals to the brain, which interprets them as images. The complex color perceptions, human eye which are initiated by rods and cones in the retina and completed by impulse integration in the brain. The color blindness is inability of identifying colors and its prevalence varies between populations. The aim of the present study was to determine frequency of color vision deficiency among students of Andode Secondary School in Bole sub- city of Addis Ababa, Ethiopia. The study was conducted from March to May 2021. 948 students from grade 9, 10, 11, and12 participated in the study. Among these, 568 were females and 380 were males and their ages range from 15 to 23 years. Students who have volunteered and a written consent from their parents or guardians were included in the study. The test of color vision was used Ishihara 24 plates and subjects were asked to sit in a room with sufficient light and read the figures/symbols on the plates from a computer screen placed 75 cm away from the subject. The data were manually arranged and frequencies were calculated. Among the 948 students tested, 26(2.74%) have color vision defect, and these included 19(2%) males and 7 (0.74%) females . These 19 males with color blindness include, 2 (0.21%) achromatopsia, 12 (1.27%) deutan, 4(0.42%) protan and1(0.11%) unclassified(combined form). Female colorblind includes achromatopsia 2(0.21%), deutan 3(0.32%), protan 1(0.11%) and 1(0.11%) unclassified. The plate number 16and 17 Students can read are grouped under unclassified different from what red-green colorblind students read. The prevalence of color blindness was observed higher in males than in females. Teaching institutions screening students is recommended for color vision deficiency, so that they can provide the necessary assistance to such students.

Key words: Color Vision deficiency, Ishihara test plates, Monochromatic, Dichromate, Trichromate, Protan, Deutan and Tritan.

1. Background of the Study

The sense organ responsible for vision or sight is the eye. The human eye consists of three layers of tissues 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. Animals detect changes in their environments by means of their sense organs. Vision is helps to communicate between the individual and the external environment (Neitz and Maureen, 2010).

Low level of light respond to rods but they do not detect colors whereas cones work only in bright light and respond to colors. Depending on the wave length of the visible spectrum of light sensitive are three types of cones . Those cones which absorb long wavelengths maximally at 560nm are referred to as L-cones and they absorb the red wavelength part of light. Another type of cones has maximum absorbance of light at 530nm which is in the region of the green spectrum. These are known as M-cones. The third type is known as S-cones and have maximum absorption at 426nm which is in the bluish range of the light spectrum (Misha, 2004).The L-, M- and S- cone stands for long, medium and short wavelength absorbing cones, respectively. In people with normal color perception, all the three types of cones are present and all are functional Christine (1991).

Each type of receptor has its own special pigment for absorbing the respective wavelength of light. Each type of receptor consists of a transmembrane protein called opsin coupled to the prosthetic group called retinal. Retinal, a non-protein organic molecule, is a derivative of vitamin A and this explains why night blindness is one sign of vitamin A deficiency (Neitz and Maureen, 2010). Most people can identify colors, but there are some people who have problems in identifying colors. Simple tasks such as selecting ripe fruit, choosing clothing, and reading traffic lights can be more challenging (Mulusew and Yilkal, 2013). Color blindness may also make some educational activities more difficult. People with total colorblindness (achromatopsia) may also have decreased visual acuity and be uncomfortable in bright environments. Most people can identify colors, but there are some people who have problems in identifying colors .Color vision deficiency is not a total loss of

color vision. The person who has trouble seeing red, green, blue or a mix of these color is referred to as colorblind (Rahman, 1998).

Color vision defects can be inherited or acquired (Okabe and Ito, 2008). Inherited color deficiency is commonly caused by mutations of genes that encode the light-absorbing photo pigment molecules in cones. The frequency of acquired deficiency is greater than congenital color vision defects (CCVD) and increases with advancing age Acquired color deficiency arises through drug or chemical toxicity, disease or trauma, pathologies, intracranial abnormalities, and certain systemic diseases. (Graham et al., 1980; Jaeger, 1994).

An inherited colorblindness the most common problem in the development of one or more of the three sets of color-sensing cones in the eye. Males are more likely to be color blind than females, as the genes responsible for the most common forms of colorblindness are on the X chromosome. As females have two X chromosomes, defect in one is typically compensated for by the other, while males only have one X chromosome (Graham et al., 1980; Jaeger, 1994).

The most common form color blindness is red-green followed by blue-yellow color blindness and total color blindness. Total colorblindness 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. Total color blindness is the lack of the ability to distinguish colors at all. It occurs when any two or all the three of the cone pigments are missing (Betsy, 2003). This is caused by dysfunctional, abnormally shaped cones or the absence of pigments (Betsy, 2003). Being color blind may make people ineligible for certain jobs in certain countries. This may include being a pilot; train driver, crane operator, and working in the armed force (Moudgil et al., 2016).

Screening methods of color blind individuals in a population are different. The most common one is the use of Ishihara plate testing in which the individuals are asked to tell the numbers inscribed on the Ishihara plate 38 and their response is recorded. Individuals who have color vision defect cannot recognize the figure written in the plate or they may tell wrong number. People with color vision defect may be found in any societies (Haile, 2014). However, studies show that the frequency varies from country to country and even among ethnic groups within a

given society. Some studies indicate that the prevalence of red-green color blindness in the world is about 8% and0. 5% among males and females, respectively (Moudgil et al., 2016).

The color vision deficiency prevalence in Ethiopia is very low(Haile,2014). 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).

A problem of color vision can affect 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. The aim of this study was to determine the frequency of color vision deficiency among students of Andode Secondary Schools, Bole sub-city Addis Ababa in order to create awareness about the existence of color vision problem among school children in general bring to the attention of educators the impact of colorblindness on learning-teaching process.

1.1 Objective of the Study

1 .1.1 General Objective

The general objective of the study is to determine the allelic frequency of color vision deficiency among students of Andode Secondary School, Bole sub-city Addis Ababa.

1.1.2 Specific Objectives

To determine the frequency of color vision deficiency among students of Andode Secondary school

To determine the types of color vision deficiency prevailing among the students

To determine the frequency of allelic, genotypic and phenotypic frequency of the major types of color vision deficiency among the students population

To determine color vision deficiency difference between male and female students

2. Literature Review

2.1. Color Blindness

John Dalton, an English chemist, he wrote the first scientific paper and to report the disorder of color blindness in 1798 (Niroula and Saha, 2010).Thus, color blindness is also called Daltonism, after John Dalton. 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). John Dalton was color blind himself. He realized that his condition had to be hereditary. John Dalton described his own color blindness in 1794. In common with his brother, he confused scarlet with green and pink with blue. Dalton supposed that his vitreous humor was tinted blue, selectively absorbing longer wavelengths.

Later, after the death of Dalton, two scientists, Thomas Young and Herman Von Helmholtz proposed trichromatic color vision and discovered that his perception about the cause of colorblindness was wrong (Mughal, et al., 2013). Three types of cone could be grouped based on depending 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 and found cone cell in the retina is sensitive to the wave lengths of blue, green and red(Mughal, et al., 2013).

2.2. The Eye as Sense Organ of Sight

A physical in the form of light received by human eye and sends these stimuli as electrical impulse to the brain. The brain interprets the impulse as images. The eye has three layers. These are sclera, choroid and retina .Each layer of the eye has its own specific functions (Colin et al., 2010).

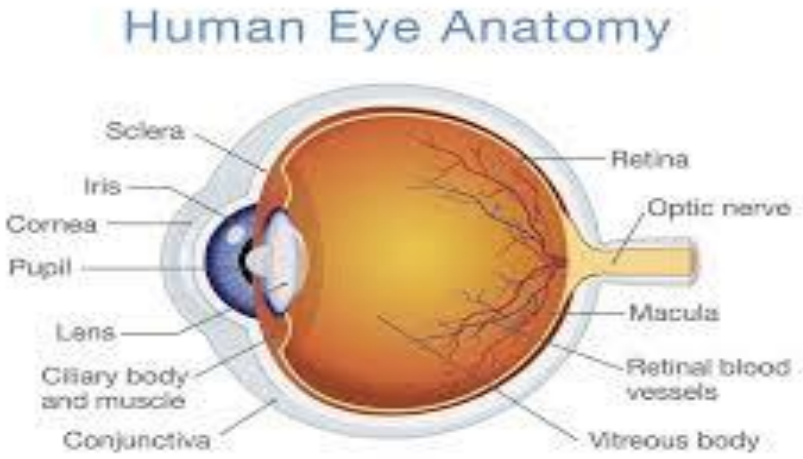

Figure 1: The internal structure of the human eye (Candice, 2015)

Photons of light stimulated by cones and rods. Retina contains light receptors cones and rods. The inner most layer of the eye is the retina. Sun light contains tiny packages of energy called photons. Some of the photons contain high energy. Photons of light contain many energy levels, only some of which we can see. 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 (violate) to 750 nanometers (red) of the wave length of photons. Light that passes through the pupil is focused by the lens on to the retina at back of the eye. The retina contains millions of rods and cones (Moudgil et al., 2016).

Rods are found on the edge of the retina. Rods cells shape looks- like rod. Rods absorb light at 500nm (Neitz and Maureen, 2010). All rod cells are functionally similar, and do not give color vision. Rods are sensitive to dim light and give twilight vision. Rod cells are more numerous than cone cell. The human retina contains about 120 million rod cells (Neitz and Maureen, 2010). Cones are cone shaped cells and are required for bright light or day light vision. 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 (Mustafi et al., 2009).

Cone cells are less in number than rod cells. Cone cells shape looks- like cone .There are around 6 million cones in the retina. They are typically 40-50µm long and their diameter varies from 0.50 to4.0µm.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. At the back of the eye is the retina that contains photoreceptor. These photoreceptor cells contain photo pigments, light sensitive molecules that are made up of a protein called opsin and a cofactor retinal that helps it work. The photo pigment of the rods is known as rhodopsin, and that of the cones is known as photo sin (Mustafi, 2009).

2.2.1 Photo Pigments of Rods and Cones

The human eye is visual pigment in a fragment of carotene and it is called cisretinal. The pigments in rods are derived from plant pigment called carotenoids. The pigment is attached to a protein called opsin to form a light detecting complex called rhodopsin. This gene has a considerable sequence similarly to the genes that encodes the opsin protein of the cone (Mustafi, 2009).

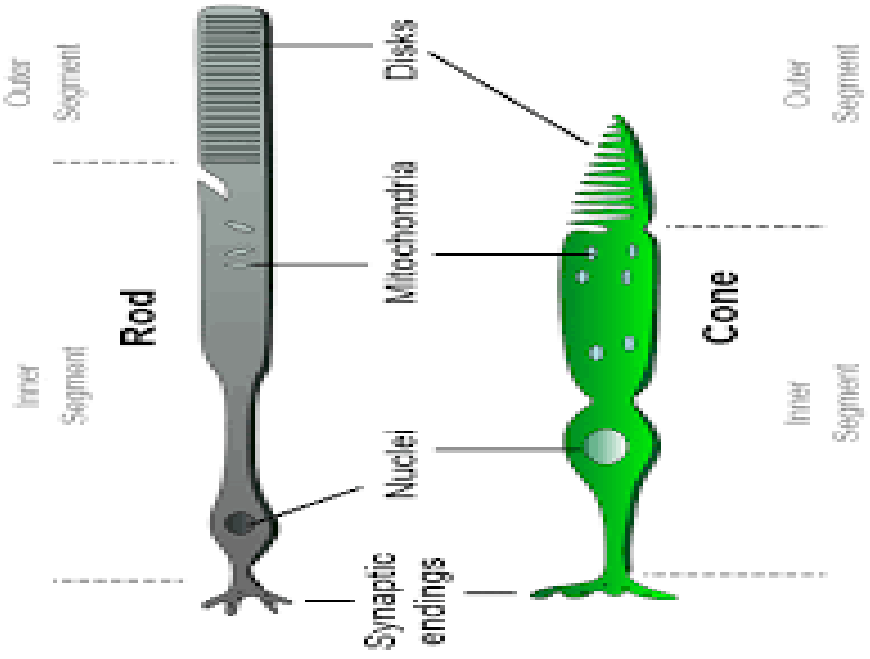

Figure 2: The structure of rods and cones (Mustafi, 2009)

Two components of photo pigments are atrans 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 wavelength of light (Neitz and Maureen, 2010).

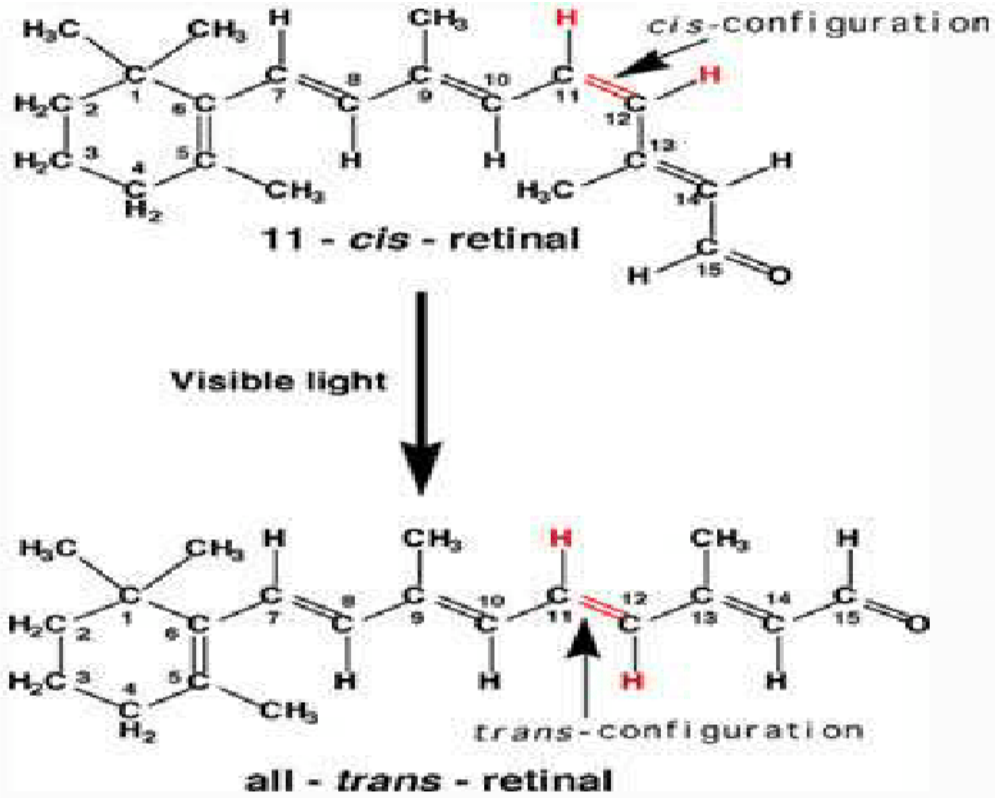

Figure 3: Configurational change of retinal from 11cis-retinal from to all trans-retinal form

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 carbon-12 which results in the rotation of all other carbons distal to it. The change in the shape of the pigment induces change in the shape of the protein opsin to which the pigment is bound, initiating a chain of events that leads to the generation of the nerve impulse (Candice, 2015).

2.3 Color Vision and Types of Deficiency

The eye perceived colors using the photoreceptors found in the retina. Color vision requires the presence of more than one photo pigment. The light absorbing molecules in photoreceptors are photo pigments (Neitz and Maureen, 2010). Cones are one of the two types of photoreceptors which help 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 (Nathans et al., 2011). The cone opsin, along with retinal, makes up the pigment. The cones have less stacked disks in their outer membrane, therefore, contain fewer photo pigments, and this characteristic makes them less sensitive to light, whereas in rods there are more photo pigmens 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 (Maureen and Jay, 2000).

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

(i) Rod monochromacy: Inability to distinguish any colors as a result of absence or nonfunctioning of cones, only the rods are functional. People with rod monochromacy see the world in black, white and gray. It is known to be an autosomal recessive disease and recent studies show that it is encoded on chromosome 2 as well as on chromosome 8. The source of rod monochromacy is not well studied (Karim and Saleem, 2013). 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 (Karim and Saleem, 2013).

(**ii) Cone monchromacy**: This condition 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. It is encoded at the same loci as red-green color blindness on the X chromosomes studied (Karim and Saleem, 2013).

B. Dichromatism: Two type's cones are functioning. But the third cone being nonfunctional. This is a moderately severe color vision. The gene mutation results in the absence of visual pigment of one cone type, pigments 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. 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**: Missing of red photoreceptor cones, thus removing the ability to see red color. (The absence of normal function of the L-cones it is a severe form of color blindness). It affects about 1% of males and 0.02% of females worldwide (Betsy, 2003).

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

iii) Tritanopia: 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 photo pigment is encoded by as gene which resides on chromosome 7, an autosomal chromosome. Tritan color vision defects are due to autosomal dominant. Tritanopia is 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 present (Dasupuram et al., 2013). The defect affects about 0.002% males and 0.001% females worldwide.

C Anomalous trichromatism: Inherited type of 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).

(**i) Tritanomaly:** It is a rare, hereditary color vision deficiency affecting blue yellow hue discrimination. The malfunctioning of the S-cone (blue absorbing cone). It is related to a gene on chromosome 7.Here the S cone is malfunctioning but not missing (Mohammed, 2015).

(**ii) Deuteranomaly:** the green absorbing cone **(**the malfunctioning of the M-cone) (Bansal et al., 2005). It affects red-green color discrimination in about 5% of European males. 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 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 a mild color vision defect in which an altered spectral sensitivity of red retinal receptors results in poor red-green hue discrimination. The malfunctioning of the L-cone (red absorbing). It is hereditary, sex-linked and present in about 1% European males. The difference with protanopia is that in this case the L-cone is present but it is malfunctioning, whereas in the former the photo pigment in the L-cone is completely missing (Mohammed, 2015).

2.4 Causes of Acquired and Inherited Color Vision Deficiency

Color blindness may be caused certain diseases, drugs and chemicals or Contact with certain chemicals such as fertilizers and styrene have been known to cause loss of color vision (Maureen and Jay, 2000). Damage by exposure to ultraviolet light (10-300nm) may also cause colorblindness. Color vision can also decline with age. Aging the ability to see colors can gradually lessen with age. Other causes for color vision deficiency include: medications drugs used to treat heart problems, high blood pressure, and infections, nervous disorders and psychological problems can affect color vision (Maureen and Jay, 2000). It can also be caused

by accidents and other trauma which produce swelling of the brain in the occipital lobe. Usually, color deficiency is an inherited condition caused by a common X-linked recessive gene, which is passed from a mother to her son. But disease or injury that damages the optic nerve or retina can also cause loss of color recognition (Maureen and Jay, 2000).

Color deficits can cause by some diseases such as diabetes, glaucoma, macular degeneration, Alzheimer's disease, Parkinson's disease, multiple sclerosis, chronic alcoholism, leukemia and sickle cell anemia.

Typically X-linked recessive inheritances carry color blindness (Maureen and Jay, 2000). 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 photo pigment molecules in the cones.

Women are typically just carriers of the color-deficient gene, though approximately 0.5% of women have color vision deficiency. In many cases, genetics cause color deficiency about 8% of white males is born with some degree of color deficiency. The severity of inherited color vision deficiency generally remains constant throughout life and does not lead to additional vision loss or blindness (Akhtar, 2015).

2.5 The Genetics of Color Vision Deficiency

Mutations capable of causing color blindness originate from at least 19 different chromosomes and 56 different genes. 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). Red-green color blindness is the most common types of genetic abnormalities are found on the long arm of the X chromosome. The gene for blue absorbing pigment is located on chromosome 7 (Akhtar, 2015). Yellow may be perceived as grey or purple and blue is perceived as grey or dark. 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).

Table 1: The different types of color blindness and their associated chromosomes (Colblindor, 2006)

Type of color vision deficiency	Chromosome bearing the responsible gene
Deuteranopia	X-chromosome
Deuteranomaly	X-chromosome
Protanopia	X-chromosome
Tritanopia(blue-color blind)	Chromosome 7
Tritanomaly(weak blue-color blind)	Chromosome 7

Traits that are determined by alleles carried on the X chromosomes are referred to as X-linked. One example of an X-linked trait is red-green color blindness (Kiula et al 2011). In humans there are 23 pairs of chromosomes. The 22 pairs of chromosomes are same in both sexes. The 23rd pair of human chromosomes consists of sex chromosomes and the two chromosomes of the pair are different for men and women. The sex chromosomes are known as X and Y, and the latter is small in size than the former females carry the combination of XX and men carry the(Kiula et al 2011).

Agirl to be color blind, both her X chromosomes should contain the recessive allele. This makes the colorblindness less common in females. On the other hand color blindness is more common in males because, if the single X chromosome carries the gene causing colorblindness, he will be color blind (XY) as there is no corresponding gene on the Y chromosome that would mask the expression of the defective recessive gene on the X chromosome. Male can either have X+Y or XcY, and will have normal and colorblind phenotype, respectively (Colblindor,2006).

The transmission pattern of colorblindness due to genes on X-chromosomes from the mother and father and the expression of the gene in male and female offspring can be illustrated by using Punnet square as presented. Suppose one uses the symbol + and Xc for X chromosome carrying normal and colorblind alleles, respectively. (Colblindor, 2006). A

female, being with two X chromosomes will have X+X or X+c or XcXc genotypes. This gives normal, normal (but carrier) and colorblind phenotype, respectively. (Colblindor, 2006).

Most people with color vision deficiency can see colors. Red-green is the most common form of color deficiency .This does not mean that people with this deficiency cannot see these colors altogether, they simply have a harder time differentiating between them, which can depend on the darkness or lightness of the colors (Bellot, 2010).

Blue-yellow is another form of color deficiency. This is a rarer and more severe form of color vision loss than just red-green deficiency because people with blue-yellow deficiency frequently have red-green blindness, too. In both cases, people with color-vision deficiency often see neutral or gray areas where color should appear (Falconer, 1960).

Achromatopsia,a condition people, who are totally color deficient, can only see things as black and white or in shades of gray. Color vision deficiency can range from mild to severe, depending on the cause. It affects both eyes if it is inherited and usually just one if it is caused by injury or illness (Falconer, 1960).

The frequency 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 population genetics (Falconer, 1960).

In a population genetics, allelic frequencies are designated as p and q. If the frequency of allele A=p, allele a=q. The random mating among individuals will give rise to genotypic frequencies of p2 AA, 2pq AA and q2 aa. The dominant phenotype (AA + Aa) =p2 +2pq and recessive phenotype (aa) =q2 (Falconer, 1960) .The above formula works for both male and female population in the trait is autosomal and only in females, if the trait is X-linked. In males allelic frequency = genotypic frequency= phenotypic frequency. Thus p=A, q=a holds for all the three cases (Shorrocks, 1978).

2.6 Tests for Color Vision Deficiency

People who have family history of color vision deficiency are required to identify colors accurately or those who have problems in identifying colors should be tested. (Ananya, 2016). Identifying colors should be checked for color vision impairment. The patient is then asked to look for numbers among the various colored dots. Individuals with normal color vision see a number, while those with a deficiency do not see it. On some plates, a person with normal color vision sees one number, while a person with a deficiency sees a different number. However, additional testing may be needed to determine the exact nature and degree of color deficiency (Ananya, 2016). There are different methods of testing individuals for colorblindness. These include Ishihara plate tests, anomaloscope test, arrangement test and lanterns (Ananya, 2016).

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

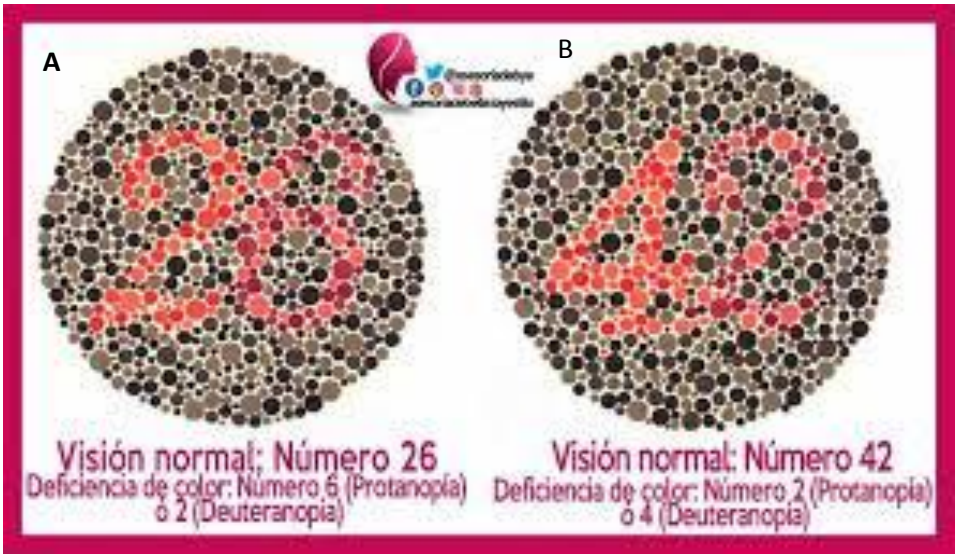

Figure 4: Samples of Ishihara test plates used for color blindness test (Shinobu Ishihara, 1972)

In Figure 4, a normal vision individual can read the number in plate A and plate B as 26 and 42, respectively. On the other hand, individuals who have color vision

problem read them as 6(Protanopia), 02(Deuteratopia) in plate A and 2(Protanopia), 04(Deuteratopia) in plate B respectively or they may not recognize any number.

B. Anomaloscope: The most accurate method distinction between dichromate and anomalous trichromats. Mixtures 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. **Ishihara plate test:** The Ishihara 38 and 24 plate test is the most commonly used color vision deficiency. Color vision deficiency 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).

2.7 Symptoms of Color Vision Deficiency

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

See some colors but not others. The individuals may not know the different form of color.

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).

People with red-green deficiency aren't aware of their problem because they've learned to see the right color. For example, tree leaves are green, so they call the color they see green. Early detection of color deficiency is vital since many learning materials rely heavily on color perception or color-coding (Moudgil et al, 2016). Also, parents may not suspect their children have the condition until a situation causes confusion or misunderstanding (Moudgil et al, 2016).

2.8 Treatment of Color Vision Deficiency

Problems of inherited color vision cannot be treated or corrected. Using gene technology a hope for a ‘cure’ of inherited color vision deficiency .This will involve injecting in vitro synthesized photo pigment into the eye. At the moment, this has only been proved to work in monkeys (Elie, 2009). Using specially tinted eyeglasses or wearing a red-tinted contact lens on one eye can

increase some people's ability to differentiate between colors, though nothing can make them truly see the deficient color (Kristin, 2015).

Labeling and organizing clothing, furniture or other colored objects (with the help of friends or family) for ease of recognition. Remembering the order of things rather than their color. For example, a traffic light has red on top, yellow in the middle and green on the bottom. Color vision deficiency can be frustrating and may limit participation in some occupations, but in most cases, it is not a serious threat to vision. With time, patience and practice, people can adapt. Although in the very early stages, several gene therapies that have restored color vision in animal models are being developed for humans (Kristin, 2015).

Depending on the cause some acquired color vision problems can be treated. 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 contacted lenses. These may help to see differences between colors. But these lenses do not provide normal color vision and can distort objects. People with severe color vision problems can see differences between colors better when there is less glare and brightness (Kristin, 2015)

2.9 Impact of Color Vision Deficiency

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. Achild with color vision problems may have to rely on size difference alone (Moudgil, 2016). Color vision defects affected people’s choices of career and many had been excluded from a chosen occupation (Richeson and Nussbaum, 2004). For example, there may be restriction on car driving.

3. Statement of the Problem

Colorblindness has major impact on day -today life experience. Persons with color blindness may not be able to identify between green and red traffic signals. Color blind person may face challenges at work for driving car and technician working in color industries (Falconer, 1960).

Research conducted to assess the frequency of color vision deficiency is limited in school children in Ethiopia. Colorblindness many make it harder to learn and read .Many of them undetected as they simply adapted to the environment to certain extent and also because of unawareness of the disease. Many tasks that we do each day rely on our being able to separate things by their color.

4. Significance of the Study

In Ethiopia there are only few studies that have recorded the frequency of colorblindness. This study helps to determine the existence of color vision deficiency and frequency among students of Andode Secondary schools in Addis Ababa. A color vision defect can affect the students learning process. It may make it harder to learn and read, and may have a negative impact in their future choice of career. Therefore, this study would contribute useful information about the frequency of color vision deficiency in school children in particular. This will initiate to take action to identify and help such children in schools.

5. Materials and Methods

Study area and period

The study was carried out in Andode Secondary School Bole sub city, Addis Ababa, Ethiopia. In 2020/2021 the total number of students enrolled in Andode Secondary School was 1363 of which 575 were males and788 were females. The study was conducted from March to May,2021 .Andode Secondary School is established in 2010 which is found in Bole sub city, Addis Ababa is the capital city of Ethiopia. The city is divided in to 11 sub cities and 106 woredas. Bole is one of the 11sub cities of Addis Ababa and the south eastern part of the city. It borders with districts of Yeka, Kirkos, Nifas silk lafto and Akaky /kality.

Sample size and Sample Size determination

In the study area a total of 948 study subjects were participated. Among these, 568 were females and 380 were males. Twenty five students were selected from the maximum number of thirty two students in each class .The total sample size was estimated using a single population proportion formula; the prevalence of low vision in Ethiopia (P) was taken to be 4.2% from the previous study (Haile, 2014), with a confidence interval of 95% and a marginal error of 2%. Contingency of 25% for the nonresponse rate was added.

The sample size for the study was determined by the following formula ((Berhane et al., 2007). **Where:**

$$n = \frac{g \times Z^2 \times P \times q}{d^2}$$

n = Sample size

P= proportion in the population having the particular trait in Addis Ababa Region (0.042).

d= Possible maximum error/Margin of error/= 0.02

q = 1-p =1-0.042= 0.96

z = 1.96 at 95% Confidence Interval (CI)

g=Design effect=1.9

$$n = \frac{1.96 \times 1.96^2 \times 0.042 \times 0.96}{0.02^2} = 759$$

To avoid non-response rate, 25% was added, so that the total sample was 759 + 189=948

The final sample size was estimated to be 948 with 25% adjusted non-response rate the total sample size was estimated using a single population proportion formula (Berhane et al., 2007).To increase the chance of color vision deficiency from students, twenty five percent of the sample size was added to the normal sample. None response rate the failure to obtain information from a designated individual for any reason(death, absence or refusal to replay.

Sampling technique and Source Population

The researcher selected twenty six students from the maximum number of thirty two students in each class by using simple random Sampling technique to collect data and used to estimate the sample size. Each individual subject has an equal chance of being selected. Stratified Sampling technique was applied for the selection of male and female participants. This is because of unequal number of male and female students in the School. First the students divided in male and female grouped. Second the female students grouped in to four and the male students also grouped in to four then selected tree students from each group. Hence, from the total sample 380 were males and 568 were females. Therefore 948 students were included in the research. The study source were students of Andode Secondary School grade 9 th,10 th ,11 th and 12th in Addis Ababa Administration Government School.

Inclusion criteria

Volunteer students who have a written consent from their parents or adult guardians in Andode Secondary School were included. Students participated with normal sight in the test for color vision defect.

Exclusion criteria

Students, who have no written consent from parents, were excluded. Students with special eye problems were obtained from the special need office in the Schools and they were excluded from the study

5.1 Procedures of Data Collection

Permissions of the school administrators were obtained before the test was conducted. Data were collected on the Andode high school. Signed consent of the parents /guardians was obtained for children under 18 years of age. Or the consent letter was written to parents. Then students were given orientation about color blindness and the objective of the study. Volunteered students who participate were asked to give their consents by signing on the consent form prepared for this purpose (see appendix I).The 24 Ishihara plate test was carried out in a properly lighted room. Each study subject was asked to read the figures in the Ishihara plate on a computer screen 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).

5.2 Instrument of Data Collection

To identify the presence of color blindness Ishihra test plates were used and the type of color blindness present among individuals. A book of Shinobu Ishihara (1972) was used to decide the type of color blindness (see appendix II). First, plates 1 – 24 of the Ishihara's test plate were presented to the subjects. Plates 1-17 each contain a number and plates 18-24 contain one or two wiggly lines.

The individual must identify the correct number, or correctly trace the wiggly lines to pass each test. If 10 or more plates were read correctly, the color vision is regarded as normal, but if only 9 or less than 9 plates are read correctly, the color vision is regarded as deficient. Subjects who were classified as color deficient were re-tested by using plate's number 16 and 17. To identify the type of the defect these plates are used (Shinobu Ishihara, 1972).This helps to classify the red-green defective dichromats as deutans or protans based on whether they were able to read plate 16 or 17 correctly. Normal color vision person reads 26 in plate number 16 and 42 in plate 17. In protanopia and protanomlia only 6(plate number 16) and 2(plate number 17) are read, in the case of mild protanomalous both numerals on each plate are read but the 6(plate number 16) and 2(plate number 17) are clearly than the other numerals. In Deuteranopia and strong Deuteranomalia only 2(plate number 16) and 4(plate number 17) are read and in the case of mild

Deuteranomalia both numerals on each plates are read, but the 2 (plate number 16) and 4(plate number 17) are clearer than the other numerals (Ishihara1972).

Color blind individual who cannot read more than two plates were classified as totally colorblind (monochromats).Individuals that are grouped under unclassified can read plate number 16 and 17 differently from what red green color blind 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 II).Students were regarded to read numbers or identify shape of figure inserted in the plates. The plates were preferred to students on a laptop screen. The student was sit in a welled or an appropriated distance from the plates.

5.3 Ethical Consideration

The study was conducted after ethical approval was obtained from the Institutional Review Board (CNS-IRB), College of Natural and Computational Sciences Addis Ababa University. The informed consent form was translated into Amharic version for simple understanding by students and parents.

5.4. Statistical Analysis

A statistical data analysis was to determine the frequency distribution among male and female students in Adode secondary school. The data were arranged and checked before the analysis. Arranged data means meaningful order can analyze it more effectively. The collected data were entered manually into a computer on excel sheet and the various population genetic parameters frequencies such as allelic frequency, phenotype frequency and genotype frequency were calculated.

The data analysis of Andode Secondary School Students was done gender wise and the frequency of colorblindness was determined on. For males, since they are hemizygous for the X-linked genes, the frequency of p and q were determined directly from the data. The collected data were presented by using table, qualitative and quantitative methods of expression. (Falconer, 1960).

6. Results

Out of the total of 948 students tested for colorblindness, 26 students (2.74%) were found to be colorblind among these 19 males (2%) and 7 (0.74%) were females. These 19 males with color blindness include, 2 (0.21%) achromatopsia, 12 (1.27%) deutan, 4(0.42%) protan and 1(0.11%) unclassified (combined form).Female colorblind includes achromatopsia 2(0.21%), deutan 3(0.32%), protan 1(0.11%) and 1(0.11%) unclassified. Students that are grouped under unclassified can read plate number 16 and 17different from what red-green colorblind students read. If those unclassified students tested using the other kind of colorblindness test such as anomaloscope their specific type of defect may be determined.

Table 2: Distribution of students by sex

Category	Sex				Total	
	F		M			
Adode	No	percent	No	percent		Percent
Grade 9th	171	18.04%	99	10.44%	270	28.48%
Grade10th	141	14.87%	127	13.40%	268	28.27%
Grade11th	145	15.30%	81	8.54%	226	23.84%
Grade12th	111	11.71%	73	7.70%	184	19.41%
Total	568	59.92%	380	40.08%	948	100%

Among the 948 subjects' tested, 922 (97.26%) of the participants were found to have normal color vision and 26(2.74%) was color vision defect which includes 7 (0.74%) females and 19 (2.00%) males.

Table 3: Frequency of CVD among Andode Secondary School students

Grade levels	Student status	Sex		
Grade 9th		Male	Female	Total
	Normal	95	161	256
	Deficient	4(0.42)	1(0.11%)	5(0.53%)
Total		99	162	261
Grade10th	Normal	121	139	268
	Deficient	6(0.63%)	2(021%)	8 (0.84%)
Total		127	141	268
Grade11th	Normal	85	130	210
	Deficient	5(0.53%)	2(0.21%)	7(0.74%)
Total		90	132	222
Grade12th	Normal	81	110	191
	Deficient	4(0.42%)	2(0.21%)	6(0.63%)
Total		85	112	197

948 students were tested with CVD. Among them, 4(0.42%) were males from grade 9th, 1(0.11%) were females from grade 9th 6(0.63%) were males from grade10th, 2(021%) were females from grade 10th, 5(0.53%) were males from grade 11th, 2(0.21%) were females from grade11th, 4(0.42%) were males from grade 12th and 2 (0.21%) were females from grade 12th respectively color blind. Among those deficiencies the highest record were obtained from grade 10th students (0.84%).

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

No of tested	Normal color vision	Type of color vision defect	Sex		Total
			F	M	
948	922(97.26%)	Achromatopsia	2(0.21%)	2 (0.21%)	4(0.42%)
		Deuteranopia	3(0.3%)	12(1.27)	15(1.58)
		Protanopia	1(0.1%)	4(0.42%)	5(0.53%)
		unclassified	1(0.1%)	1(0.09%)	2 (0.21%)
		Tritanopia	0	0	0
		Total	7(0.7%)	19(2%)	26(2.74%)

The 7 (0.74%) colorblind female students were further classified into achromatopsia (n=2, 0.21%), deutan (n=, 0.32%), protan (n=1, 0.11%) and Unclassified (n=1, 0.11%). The 19 (2%) males were further classified intoachromatopsia (n=2, 0.21%), deutan (n=12, 1.27%), protan (n= 4,0.42%)and Unclassified (n=1, 0.11%).

The unclassified individuals have color vision defects like the others, but they were not able to read plate number 16 and 17 or read different number from what protans and deutans can read. The frequency of red-green colorblindness (deutan and protan`s) in females was 0.43%. Among colorblind females, Deuteranopia was more prevalent.

1.69 %(1.27%+0.42%). the frequency of red-green colorblindness in males was higher than in females by 1.26%. Males have higher CVD frequency which reinforces the fact of X-linked recessive nature of the trait (i.e., the single X-chromosome in males is predominant to color blindness, while females with two X-chromosomes can act as dosage compensation and decreases the risk of the disease).

Deutan males are more frequent than the other color blind subjects. The frequency of the different type of colorblindness in this study shows that the percentage of dichromats was more than monochromats (Achromatopsia). Monochromats referred to total color blindness. Among dichromats,deutans have higher frequency than protans.When we consider the males

and females separately, the percentage of colorblindness among males and females was different. Out of 380 males tested, 19 were colorblind. Which makes19/380or 5%of the total males tested.

Only 7 females were colorblind out of total 568 females tested, which makes7/568 or1.23% of the total females tested. For the two common types of red-green color vision deficiency, Protanopia and deuteronopia phenotypic, allelic and genotypic frequencies were calculated for male and female populations separately. For the sake of Simplicity, we represented the protanopia locus gene (OPN1LW) by L for normal and l for the mutant allele. OPN1LW gene provides instructions for making a protein that is essential for normal color vision. This protein is found in the retina. The calculation shows that in males the dominant normal allele (L), had frequency of P= 0.9895 and the recessive (l) frequency of q=0.0105. In females, the dominant phenotype frequency=p2+2pq=0.9982 and the frequency of recessive phenotype, q2 =0.0018.The allelic frequency is 0.9576 for (L) and 0.0424 for (l). The genotypic frequency for LL (p2) =0.9170, heterozygous Ll=2pq=0.0812 and homozygous recessive is q2 =0.0018.The 7 (0.74%) colorblind female students were further classified into protanopia and deuteranopia.

Table 5: Phenotypic, allelic and genotypic frequencies of protanopia among the tested subjects by se

Trait		Item	Sex	
Protanopia	Sample size		Male	Female
		Normal	376	567
		Protan	4	1
		Total	380	568
	Phenotypic Frequency	Normal	0.9895	0.9982
		Protan	0.0105	0.0018
		Total	1	1
	Allelic frequency	L	0.9895	0.9576
		I	0.0105	0.0424
		Total	1	1
	Genotypic frequency	L	0.9895	0.9576
		I	0.0105	0.0424
		Total	1	1
		LL	0.9895	0.9170
		LI	0.0105	0.0812
		II	0.0105	0.0018
		Total	1	1

The frequencies were calculated phenotypic, allelic and genotypic for male and female populations separately for the two common types of red-green color vision deficiency, Protanopia and deuteranope. For the frequency allelic, phenotypic and genotypic simplicity, were represented the protanopia locus gene (OPN1LW) by L for normal and l for the mutant allele. The calculation shows that in males the dominant normal allele (L), had frequency of P=0.9684 and the recessive (l) frequency of q=0.0316.

The dominant phenotype frequency in females, = p2+2pq=0.9947 and the frequency of recessive phenotype, q2 =0.0053. The allelic frequency is 0.9272 for (L) and 0.0728for (l). The genotypic frequency for LL (p2) =0.8597, heterozygous Ll=2pq=0.1350 and homozygous recessive is q2 =0.0053.

For deteranopia method phenotypic frequencies are calculated in the same way as described above for protanopia. In males, the frequency of M was 0.9684 and m was 0.0316. These

frequencies are also true for genotypic and phenotypic frequencies of the trait. In females, the frequency of normal vision is 0.9947 and deutans is 0.0053. The allelic frequencies calculated based on the above phenotypic frequencies gave 0.9272 and 0.0728 frequency of M and m alleles, respectively. Based on the above allelic frequency of M= P= 0.9272 and m=q =0.0728, the genotypic frequencies are calculated as MM (p2) = $(0.9272)^2$, Mm (2xpq) = (2x0.9272x0.0728) and mm=q2 $(0.0728)^2$. These gave 0.8597, 0.1350 and 0.0053 frequencies for the three genotypes, respectively.

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

Trait		Item	Sex	
Deuteranopia	Sample size		Male	Female
		Normal	368	565
		Deutan	12	3
		Total	380	568
	Phenotypic Frequency	Normal	0.9684	0.9947
		Deutan	0.0316	0.0053
		Total	1	1
	Allelic frequency	L	0.9684	0.9272
		I	0.0316	0.0728
		Total	1	1
	Genotypic frequency	M	0.9684	0.9272
		M	0.0316	0.0728
		Total	1	1
		MM	0.9377	0.8597
		Mm	0.0316	0.1350
		Mm	0.0316	0.0053
		Total	1	1

The female genotypic frequency were obtained by using the formula p2 and p2+ 2pq for the homozygous dominant and heterozygous, respectively. The genotypic frequency of the recessive trait can be obtained by q2.The normal allele frequency can be obtained by the formula p + q=1.So by subtracting one from q we can get p.

7. Discussion

By using 24 Ishihara plate tests the study was conducted to determine the frequency of colorblindness in Andode Secondary school in a total of 948 students (380) males and (568) females. Teachers are in direct contact with the students and in order to guide their students for their future career. Individuals with the defect also do not have awareness about their problems. Screening students for color blindness is essential to help them with the defect in the classroom and to guide them in their future carrier.

An abnormal condition characterized by the inability to clearly distinguish different colors of the spectrum is called color blindness. Color blind individuals may face difficulties at work as seen for technician working in color industries. Small sample size was used out on the present study when it is compared to other studies that were performed in different parts of the world. The frequency of colorblindness is different in different countries. The frequency of colorblindness is about 8% in males and 0.4% in females (Fareed'et al, 2015).

Male students' frequency of colorblindness in this study (2%) was higher than reported from Pakistan 1.9% (Fareed et al., 2015). In some European Countries even higher frequency was reported (Mulusew, Yilikal, 2013). The frequency of color blindness among female students in the present study (0.74%) was found to be similar with some other studies such Pakistan (0.74%,).But it is higher than reports made in India(0.63%), Aligarh (0.71%), Denmark (0.54%), Greenland(0.4%), New Zealand (0%)and even lower than reports done in Iraq(3.2%),Japan (3.6%)and Black Americans (3.7%) (Fareed et al., 2015).

Among non-Europeans frequency of colorblindness is lower than in person 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 frequency is reported; 7.8% School boys in Germany, 7.95% among males in Greek and 7.33 is young Turkish men were reported to have color vision defect (Mulusew, Yilikal, 2013).Male students frequency of colorblindness in this study (2%) and 0.74% female students were higher than reported from Uganda (1.5%) and 0.6%, Congo (1.7%)and 0.4%,Nigeria 2.8%) and 0.7% , Tanzania(1.8%) and 0.7% as reviewed by(Niroula and Saha,1990). But it is lower than reported in Libya (2.2%) and Algeria (4.7%) in males (Fareed et al., 2015).

Frequency of colorblindness study in Ethiopia is very few. On such study reported frequency of colorblindness as 4.2% among males and 0.2% among females (Mulusew, Yilikal, 2013). The studies indicate that Ethiopians have a much higher incidence of color blindness (4.2%) than other sub-Saharan population such as Uganda and Congo. In addition to this, other studies also indicate the frequency of color blindness in Ethiopia such as study conducted by Mulusew and Yilikal 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, 0.54% protan, 0.58% unclassified, and 0.19% cases of total color blindness. The frequency of color blindness was also studied by Haile Fentahun on school children in Addis Ababa by using Ishihara test 38 plate edition on a sample of 378 of which (4.2%) were color blind. Among these, 1.3% were females and 2.9% were males (Haile, 2014). The frequency that is reported in Ethiopia is higher than the present study.

The color blindness frequency 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 color blind. Among these, 0.6% was females and 3.6% were males (Gashaw and Teshome, 2014). The present finding in males (2%) is lower than and females (0.74%) are higher that of the finding of (Gashaw and Teshome, 2014).Because 24 Ishihara test plate was used. Color blindness frequency was also studied by Tsega Habte on School children in Akaki /Kality Sub- city of Addis Ababa by using Ishihara test 24 plate edition on a sample of 1081of which (3.05%) were color blind. Among these, 0.83% were females and 2.22% were males (Tsega Habte, 2017).

The present finding in males (2%) and females (0.74%) were lower than that of the finding of (Tsega Habte, 2017). Recently color blindness also studied by Helen Kidane on School children in Addis Ababa (Kirkos Sub-city) by using Ishihara test 24 plate edition on a sample of 1012 of which (2.17%) were color blind. Among these, 0.49% were females and 1.68% were males (Helen Kidane, 2019). The present finding in males (2%) and females (0.74%) were higher than that of the finding of (Helen Kidane, 2019).

2% males and 0.74 females of colorblindness revealed on the present study, which is lower than studies done in the following regions or place in Ethiopia such as Addis Ababa (Akaki –Kality

(Tsega Habte, 2017), Amhara region (Sekela Woreda), (Woldeamanuel, 2018). Amhara region (Gish abay), (Yilikal Adamu & Abebe Zelalem, 2016), Somali region, Harari region (Woldeamanuel, 2018). and Debub (Wolkite (Gashaw and Teshome, 2014) in the (table10.) But it is higher than reports made in Addis Ababa (Kirkos Sub-city), (Helen Kidane, 2019). And Oromia region (Woldeamanuel, 2018).

The overall prevalence of CVD in the present study was higher than when compared to the previous studies done in Ethiopia such as in Addis Ababa (Kirkos Sub-city), 2.17, by Helen Kidane, 2019) and Oromia region, 1.9%, by Woldeamanuel, 2018). But it is lower than when compared to the previous studies done in Addis Ababa (Akaki –Kality), 3.05%, by Tsega Habte, 2017, Addis Ababa (Arada Sub-city ,4.2%, by Haile, 2014, Amhara region (Sekela Woreda), 4.1%, by Woldeamanuel, 2018, Amhara region(Gish abay town), 3.24%, by Yilikal Adamu 2016, Debub (Wolkite town), 4.2%, by Gashaw and Teshome, 2014, Somali region, 3.3%, by Woldeamanuel, 2018 and Harari region, 5.8%, by (Woldeamanuel, 2018). The overall colorblindness frequency of the highest report (5.8%) is recorded in Harari Region and the lower report is recorded in Oromia Region (Woldeamanuel, 2018).

8. Conclusions

The present finding showed the existence of protanopia, deuteranopia, achromatosopia and unclassified types of colorblindness among Students of Andode Secondary School in Bole sub-city of Addis Ababa, Ethiopia .The frequency of deutan is higher than the other type of colorblindness. Red-green colorblindness (protanopia and deuteranopia) was the most common type of colorblindness which is in agreement with various reports for different populations.

The combined frequency of colorblindness for males and females students' were2.74%. This results show more men suffer from it than women. In this study, all of the students tested have no information about CVD and all of them were unaware of their status.

9. Recommendation

An early life of an individual Color vision defect should be tested particularly male, to make informed decision on future carrier. Early detection of color blindness allows parents and teachers to make Necessary adjustments to the teaching learning process of affected children. But, in case of our country, much has to be done to screen Children for color blindness.

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.

Students who color blind are seriously advised to reconsider some of their professional ambitions that may require CVD test (e.g. pilot, paint industry, artist) and to take necessary precautions in daily activities (e.g. traffic lights, gardening, outfit selection).

Ministry of Education and other Stakeholders give emphasis to such deficiencies as color blindness.

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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 in Andode secondary school. This study was provided good information about the heritable color vision defects for various institutions. During the study consent was obtained from guardians and/or parents for children younger than 18 years. For this study I have been requested to take ISHIHARA's color vision test. I have been informed by Melese Baye (a MSc student at AAU in Biology) here in referred to as investigator about a study of Frequency of color vision deficiency among students of Andode Secondary School in bole sub- city of Addis Ababa, Ethiopia. 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 natural 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. A.A.U E-mail: cnsethical@gmail.com.

1.2 *የጥናቱ ስነምግባር ደንብ*

የጥናቱ ርዕስ፡ Frequency of color vision deficiency among students of Andode Secondary School in Bole sub- city of Addis Ababa, Ethiopia

የአጥኚዉ ስም እና አድራሻ፡ መለስ ባየ cell phone 0955419146

E-mail: melesebaye122127@ gmail.com

ስነህይወት ፋክልቲ፣ አዲስ አበባ ዩኒቨርሲቲ

ይህንን ጥናት በሚመለከት ለተሳታፊ ግለሰቦች የተገለጹና ተሳታፊዎችም 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** ISHIHARA Color Vision Test) **ቻርቶችን በመጠቀም ነዉ፤**
3. **ከተሳታፊዉ የሚፈላገዉ ተሳትፎ በቻርቱ ዉስጥ የተፃፉትን ቁጥሮች እና ምልክቶች በማንበብ/መለየት ብቻ ነዉ፤**
4. **ጥናቱ በተመርማሪዉ (ተሳታፊ ግለሰብ) 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 ስነህይወት ፋክልቲ የስነምግባር ኮሚቴዉ ተመርምሮ የጥናት ስነምግባርን የሚያሟላ መሆኑ የተረጋገጠና ፈቃድ የገኘ ነዉ ፤**
8. **አንድ የጥናቱ ተሳታፊ ቅሬታ ቢኖረዉ ለኮሚቴዉ ቅሬታዉን ሊያመለክት ይችላል።።**

እኔም በዚህ ጥናት በፍላጎቴ ለመሳተፍ እና ለመደገፍ ተስማምቻለሁ።።

ማሳሰቢያ፡ ማንም ቅሬታ ያለዉ የጥናቱ ተሳታፊ የስነምግባር ኮሚቴዉን ቀጥሎ በተመለከተዉ አድራሻ ማግኘት ይችላል።። A.A.U E-mail: cnsethical@gmail.co

1.3 Consent 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 frequency of color vision deficiency 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 approve 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፡፡ የጥናቱንም አላማ በሚገባ የተረዳሁ ሲሆን፤ የምሰጠውም መረጃ ለዚህ ጥናት ብቻ የሚውል በመሆኑ በልጄ ላይም ሆነ በኔ ላይ ምንም አይነት ጉዳት እንደማያደርስ እና የምሰጣቸው ማንኛውም መረጃዎች በሚስጥር እንደሚጠበቁ ስለተገነዘብኩ በጥናቱ ልጄ እንዲሳተፍ መወሰኔን በፊርማዬ አረጋግጣለሁ፡፡

የጥናቱ ተሳታፊ ወላጅ ወይም አሳዳጊ ስም----------

ፊርማ----------

የመረጃ ሰብሳቢው ስም----------

ፊርማ----------

ቀን---------/---------/----------

1.5 Data collection Form No._____ ቅፅ

College /School Name_____

ISHIHARA plates Test

Code	*plate No.*																								
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*1o*	*11*		*12*	*13*	*14*	*15*	*16*	*17*	*18*	*19*	*20*	*21*	*22*	*23*	*24*
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1.6 የቀለም እይታ ምርመራ የፈቃደኝነት ማረጋገጫ ቅፅ

እኔ ስሜ እንዲሁም ሌሎች መረጃዎቼ ከዚህ በታች የተገለፀው ለዚህ ምርምር ተሳታፊ ለመሆን ስወስን በአዲስ አበባ ዩኒቨርሲቲ የማስተርስተማሪ የሆኑት መለሰ ባየ የምርምሩን ህደትና ሁላንተናዊ ጠቀሜታዉን ገልያልኛል፡፡ የጥናቱ የስነምግባር ደንብንም ካነበቡልኝ በኋላ እኔም እንድሳታፍ ጠይቀዉኛል ፡፡ እኔም በተጠየቅኩበት መሰረት ያለምንም ግዴታ በሙሉ ፍቃደኝነት የተሳተፍኩ መሆኔን በፊርማዬ አረጋግጣለሁ፡፡የት/ቤት ስም_____

መለ.ቁ.	ስም ከነ አባት	ፆታ	ዕድሜ	ክፍል	ፊርማ
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1.7 Appendix II

Table I: 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	5				x	
4	29	70				x	
5	57	35				x	
6	5	2				x	
7	3	5				x	
8	15	17				x	
9	74	21				x	
10	2	X				x	
11	6	X				x	
12	97	X				x	
13	45	X				x	
14	5	X				x	
15	7	X				x	
16	16	X				x	
17	73	X				x	
18	X	5				x	
19	X	2				x	
20	X	45				x	
21	X	73				x	
		protan		deutan			
22	26	Strong	mild	Strong		Mild	
23	42	6	(2) 6	2		(6) 2	
24	35	2	(4) 2	4		(2) 4	

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

Declaration

I, 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: Melese Baye

Signature: _____

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

_____

Advisor: prof. Yalemtsehay Mekonnen