Addis Ababa University
School of Graduate Studies

The Prevalence of Colorblindness among School Children in Akaki/Kality Sub-city of Addis Ababa

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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

By

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Abstract

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. The human eye has complex color perceptions, which are initiated by cones in the retina and completed by impulse integration in the brain. Most people see the world in color, but there are people who do not perceive colors. These people may see colors differently than what most people perceive. The inability of identifying colors is known as color blindness and its prevalence varies between populations. The main aim of the present study was to determine the prevalence of color blindness among children in Beseka and Kality secondary schools. The study was conducted from March to May 2017.Random sampling method was used to estimate the sample size. Accordingly, 1081 students from grade 9 and 10 of the two schools participated in the study. Among these, 542 were females and 539 were males and their ages range from 14 to 20 years. All the school age children in the selected schools who have volunteered and a written consent from their parents or guardians were included in the study. Color vision was tested by using Ishihara 24 plates (Shinobu, 1972) and subjects were asked to seat 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 1081 students tested, 33(3.05%) have color vision defect, and these included 9 (0.83%) females and 24(2.22%) males. It was observed that the prevalence of color blindness is higher in males than in females. It was recommended that schools and other teaching institutions screen students for color vision deficiency, so that they can provide the necessary assistance to such students.

Key words: Color Vision Deficiency, Ishihara's Color plates, Protan, deutan, Tritan, Ishihara colr plates

1. Introduction

Animals detect changes in their environments by means of their sense organs. Among these, vision is the one which helps to communicate between the individual and the external environment .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 (Neitz and Maureen, 2010).

Rods respond to low level of light but they do not detect colors whereas cones work only in bright light and respond to colors. There are three types of cones depending on the wave length of the visible spectrum of light to which they are sensitive. 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. This refers to as trichromatic vision (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).

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Most people can identify 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 colorblindness, while less than 5 % of the female population is affected. Colorblindness is commonly classified as Total, Red-Green and Blue-yellow colorblindness (Rahman, 1998).

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. 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. This is caused by dysfunctional, abnormally shaped cones or the absence of pigments (Betsy, 2003).

Red-green colorblind people are unable to distinguish between red and green colors. Red-green colorblindness is the most common form of color blindness that affects about 8% of males and less than 5% of females. Blue – yellow colorblindness is rare and not-gender specific type of colorblindness. About 0.02% of the population is affected by this type of colorblindness. Red – green and blue-yellow types of color blindness occur when the photopigments 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).

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 inscribed on the Ishihara plate

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. 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% and 5% among males and females, respectively (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).

A color vision problem 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.

Therefore the main aim of this study was to determine the prevalence of color blindness in school children in two governmental schools in the Akaki/Kality Sub-city of Addis Ababa in order to create awareness about the existence of color vision problem among school children in general and in the two studied schools in particular so as to bring to the attention of educators the impact of colorblindness on learning-teaching process.

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, scientists 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, were the first to propose 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 arranged 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. A Swedish physiologist, named Gunnar Svaetichin examined the outside layer of the retina of fish. He found the cones of the retina are sensitive to wavelengths of blue, green and red. In 1983 the theory of Young and Helmholtz's was complete (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. These are sclera, choroid and retina (Fig.1) Each layer of the eye has its own specific functions (Colin et al., 2010).

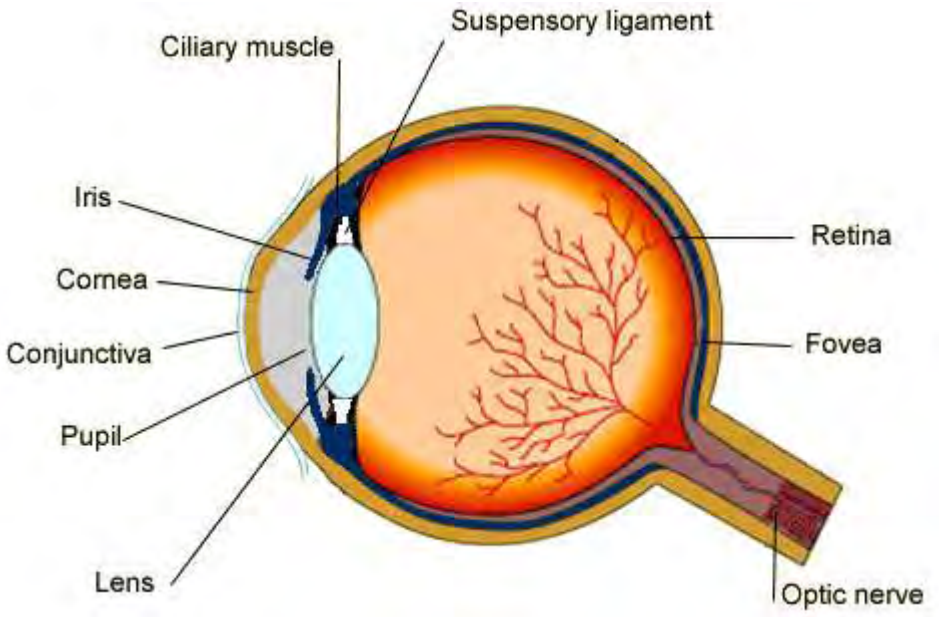


Fig 1: The internal structure of the human eye (Candice, 2015)

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 (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).

2.2.1. 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 cell. 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).

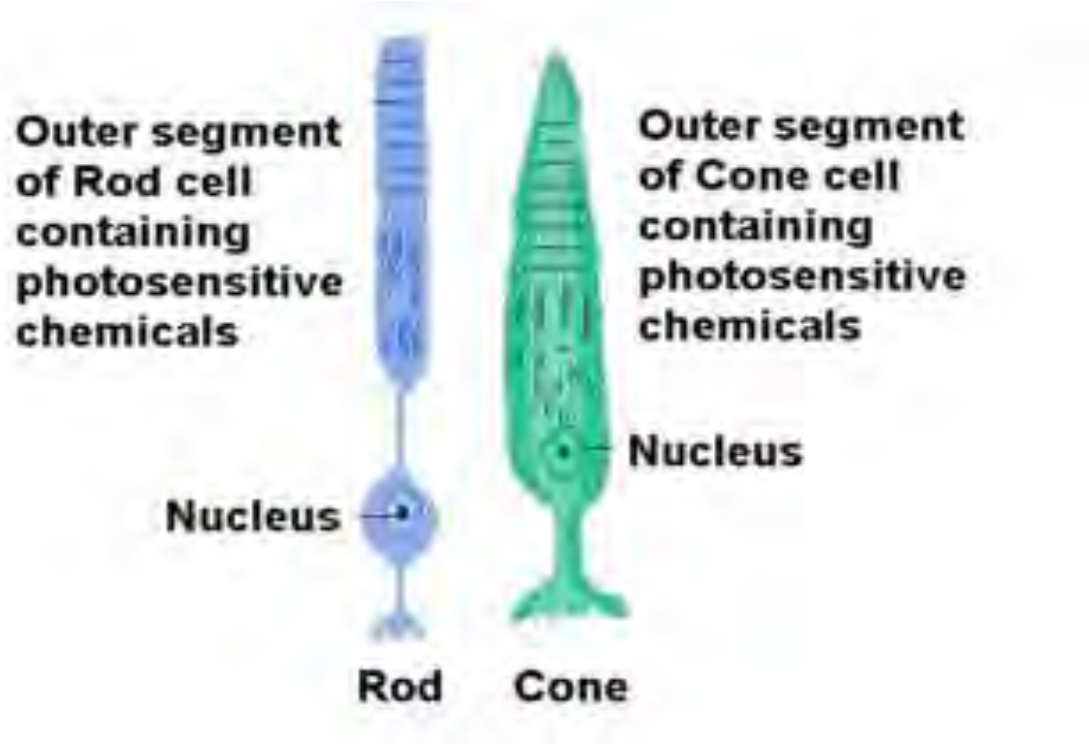


Fig 2: The structure of cone and rod (Mustafi, 2009)

2.2.2. The cones

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. 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 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.

2.2.3 The photopigments

At the back of the eye is the retina that contains photoreceptor. These photoreceptor cells contain photopigments, light sensitive molecules that are made up of a protein called opsin and a cofactor retinal that helps it work. The photopigment of the rods is known as rhodopsin, and that of the cones is known as photopsin (Mustafi, 2009).

.2.2.3.1 Photopigments 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 encodes the opsin protein of the cone (Mustafi, 2009)**.**

2.2.3.2 Photopigments of cones

Photopigments 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 wavelength of light (Neitz and Maureen, 2010).

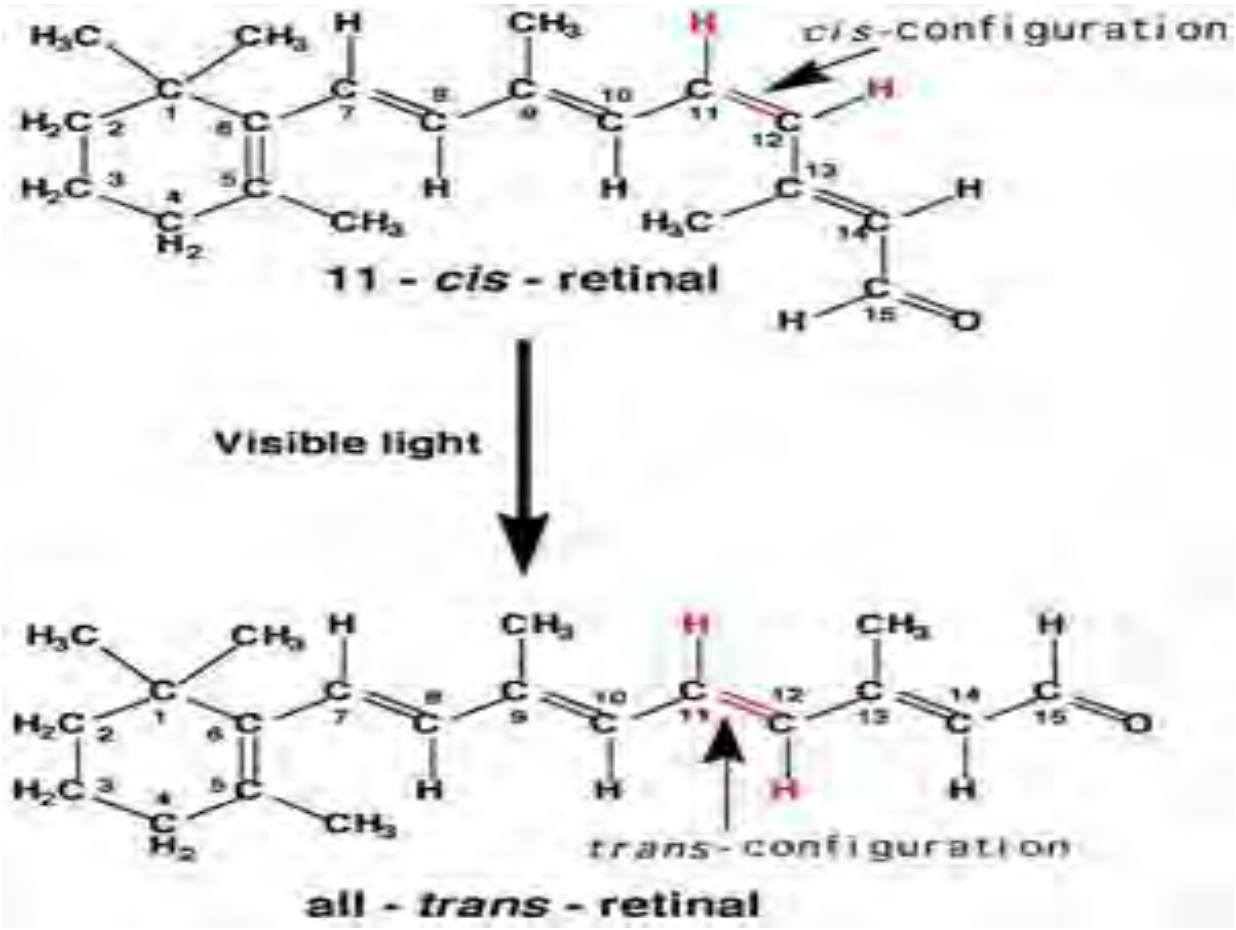


Fig 3: Configurational change of retinal from 11-cis-retinal form to all-trans-retinal form following absorption of light photon (Neitz and Maureen, 2010)

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 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 bound, initiating a chain of events that leads to the generation of the nerve impulse (Candice, 2015).

2.3. Vision

Vision begins with light passing through the cornea and then the lenses adjust 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 lope of the brain by optic nerve in the form of electrical signals and the brain process the impulse and allows us to see.

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 photopigments. Photopigments are the light absorbing molecules in photoreceptors (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 (Fig.4) (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 photopigments, and this characteristic makes them less sensitive to light, whereas in rods there are more photopigmens in the outer membrane. Stacked disks are the place where the photopigments are found. The three types of cones respond to variation in color in different ways and make possible trichromatic vision (Maureen and Jay, 2000).

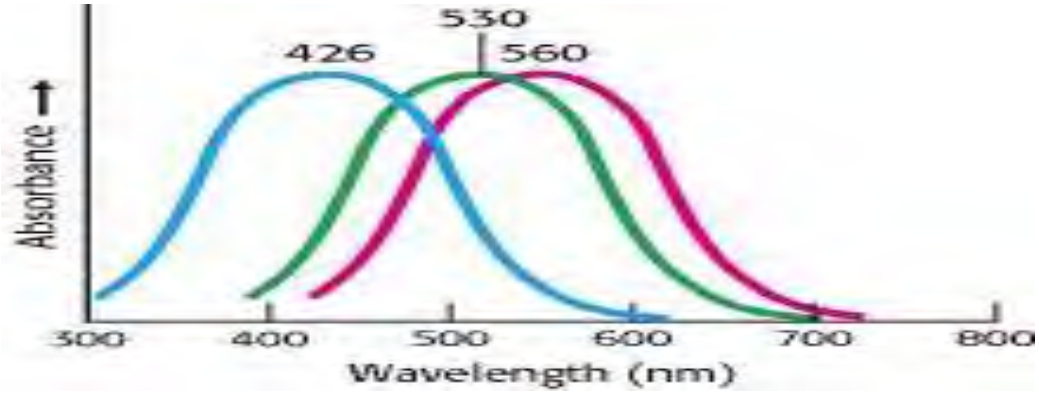


Figure 4: Cone-pigment absorption spectra (Maureen and Jay, 2000).

2.3.2. 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 differentiating between particular shades of red and green or blue and yellows (Mulusew and Yilkal, 2013).

2.3.2.1. 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 in to 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 photopigments, 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. 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).

(*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. It is encoded at the same loci as red-green color blindness on the X chromosomes.

b. Dichromatism: This is a moderately severe color vision; it is due to the presence of function of only two type's cones with the third cone being non-functional. 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 (Table1). 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 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 (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. 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 worldwid.

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*) Tritanomaly: - This is due to the malfunctioning of the S-cone (blue absorbing cone).It is a rare, hereditary color vision deficiency affecting blue-yellow 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 case the L-cone is present but it is malfunctioning, whereas in the former the photopigment in the L-cone is completely missing (Mohammed, 2015).

2.3.3. Causes of color vision deficiency

Color vision deficiency can be classified as acquired or inherited.

2.3.3.1 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).

2.3.3.2. 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 in the cones. Colorblindness is typically an X-linked recessive inheritance (Maureen and Jay, 2000).

Of the three types of inherited or congenital color vision deficiencies, which are (monochromacy, dichromacy,and anomalous trichromacy) the most common 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 an ancestry (Agarwal and Nishant, 2014).This type of color blindness is passed on via the X chromosome.

2.3.3.2.1. The genetics of color vision deficiency

The genes responsible for the most common types of genetic abnormalities, red-green color blindness, are found 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), encoding the red wavelength absorbing pigment and OPN1MW (opsin 1 middle wave), encoding the green wave length absorbing pigments (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 cone opsin but there are multiple ones for the green pigment (Neitz and Maureen, 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(Table 1).

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 and blue is perceived as gray 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	Chromosome 7
Tritanomaly	Chromosome 7

2.3.3.2.2. Transmission and Expression of X-linked color vision deficiency

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 combination XY (Bellot, 2010).

Therefore, for a girl 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 (Colblindor,2006).

The pattern of transmission 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 best be illustrated by using Punnet square as presented in Fig.5 below. Suppose one uses the symbol X^+ and X^c for X chromosome carrying normal and colorblind alleles, respectively. A female, being with two X chromosomes will have X^+X^+ or $X^+ X^c$ or $X^c X^c$ 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^+Y or $X^c Y$, 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.Normal father and carrier mother

Where, X^c represents colorblindness, X^+ normal alleles, respectively.

	X^+	X^c
X^+	X^+X^+(Normal)	X^+X^c (Normal carrier)
Y	X^+Y(Normal)	X^cY(Colorblind)

The phenotype of the progeny will:

- One female (25%) normal,
- One female (25%) is normal, but carrier,
- One male (25%) colorblind and one male (25 %) normal.

Case 2.

Normal father and colorblind mother.

	X^c	X^c
X^+	X^+X^c(carrier)	X^+X^c(carrier)
Y	X^cY(colorblind)	X^cY(colorblind)

All females become carriers and all males colorblind.

Case 3.

Colorblind father and colorblind mother

	X^c	X^c
X^c	X^c X^c	X^c X^c
Y	$X\ ^cY$	$X\ ^cY$

All children become colorblind (100%)

Fig 5: Punnet square to show the pattern of the inheritance of X-linked colorblindness gene from maternal and paternal parents to their children

2.3.4. 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 (Falconer, 1960). Any genetic study that is done at a population level, including the present study is considered as population genetics.

2.3.4.1 Allelic, genotypic and phenotypic frequencies

In a population genetic study, the main interest is to determine the frequencies of alleles, genotypes and phenotypes of trait of interest in a population and factors that change the frequencies. In this process a sample is 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 occurring at a locus in a population, the two alleles may be equal or A is more frequent than a or vice-versa. Likewise, the resulting three genotypes, AA, Aa and aa may be different from what we expect from Aa X Aa cross, ¼ AA +1/2 Aa +1/4 aa, or the expected

phenotypic frequency of ¾ A- and ¼ aa. In a population the genotypic and the phenotypic frequencies are determined by the frequency of the alleles.

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 p^2 AA, 2pq Aa and q^2 aa. The dominant phenotype (AA + Aa) =p^2 +2pq and recessive phenotype (aa) =q^2 (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).

2.3.4.2. Prevalence of color vision deficiencies in different populations

The incidence of color blindness varies from race to race or between people of different ethnicity. Since color blindness is genetically transmitted, its distribution is likely to be variable in different ethnic groups (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 countries where there are isolated communities with limited gene pools, the prevalence may be increased, including that of more rare forms. Examples of countries with such communities include rural areas of Hungary, Finland and some Scottish islands (Rahman, 1998).

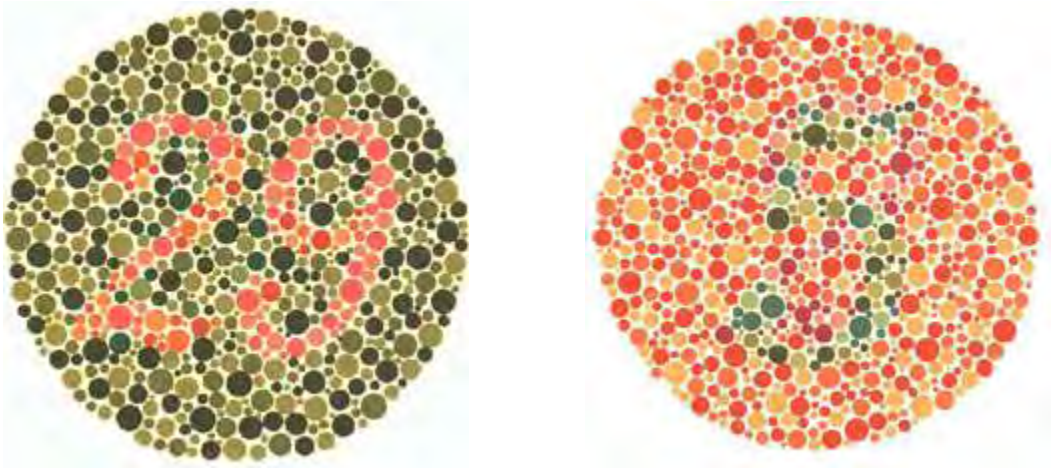
The study on prevalence of color blindness done so far on Ethiopian population is very scarce, and only two published studies are available to date. According to these studies, the prevalence of congenital color blindness among Ethiopians was reported to be 4.2% among males and 0.2% among females (Mulusew and Yilikal, 2013) in which protanopia and deuteranopia are the most common types of color blindness (Haile, 2014).

2.3.5. 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 the Ishihara 38 and 24 plate test. Ishihara 38 plates 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). They are made up of many colored circles and contain written in them numbers or symbol, which can only be seen if one, is not colorblind (Fig 6).



A. Plate No. 3 B. Plate No. 4

Fig 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 29 and 5, respectively. On the other hand, individuals

who have color vision problem read them as 70 and 2, 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 (Ubom, 2014).

2.3.5.1. Symptoms and treatments of color vision deficiency

2.3.5.1.1 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).

2.3.5.1.2. Treatment of color vision deficiency.

Inherited color vision problems cannot be treated or corrected. There is a hope for a 'cure' of inherited color vision deficiency using gene technology. This

will involve injecting *in vitro* 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 contacted 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).

2.3.6. Impact of color vision deficiency

Many people are affected by colorblindness but 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. 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 had 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. Significance of the study

This study helps for determining the existence of color vision deficiency among school children of two governmental 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. In Ethiopia there are only few studies made on the prevalence of colorblindness. Therefore, this study will contribute useful information about the prevalence of color vision deficiency in Akaki/Kality population in general and school children in particular. This would initiate to take action to identify and help such children in schools.

4. Objectives

4.1. General objective

The general objective of the study was:

- To determine the prevalence of color blindness among children in Beseka and Kality Secondary schools selected for the study.

4.2. Specific objectives

The specific objectives of this study were:

- To determine the prevalence of color blindness among male and female students of the two schools;
- To find out the prevalence of the different types of colorblindness 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.

5. Materials and methods

5.1. Study area and period

The study was carried out in Beseka and Kality secondary schools. It was conducted from February to March 2017.The schools are found in Akaki/Kality sub-city of Addis Ababa. The sub-city is situated in the south of Addis Ababa.

Sample size

A total of 1081 study subjects participated in the study. Among these, 542 were females and 539 were males.

Inclusion criteria

Beseka and Kality secondary school volunteer children who have a written consent from their parents or adult guardians were included. Students with normal sight participated in the test for color vision defect.

Exclusion criteria

Students who were not willing to participate, or have no written consent from parents, were excluded. List of students with special eye problems was obtained from the Special Need Office in the schools and they were excluded from the study.

Table 2. Student populations and number of students participated in the study.

School	Total student population in each school	% of total	Sample size in each school	% of sample size
Beseka Secondary School	1581	51%	619	20.0%
Kality Secondary School	1500	49 %	462	15.0 %
Total	3081	100%	1081	35.0%

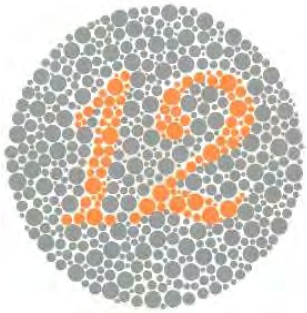
5.2. Procedures of data collection

Data were collected on the respective compounds of the two respective high schools. Permissions 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 by using the 24 Ishihara test plate. 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).

5.3. Method of testing for color vision deficiency

Ishihra test plates were used to identify the presence of color blindness and the type of color blindness present among individuals. In order to decide the type of color blindness a format from a book of Shinobu Ishihara (1972) was used (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. To pass each test the individual must identify the correct number, or correctly trace the wiggly lines. 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 plates number 16 and 17.These plates are used to identify the type of the defect (see appendix II) (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. Among the color blind individuals, those who read not more than two plates are classified as totally colorblind (monochromats).



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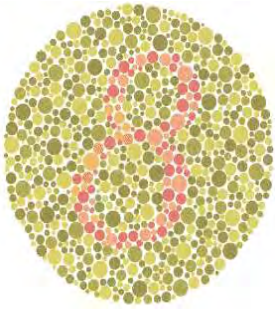

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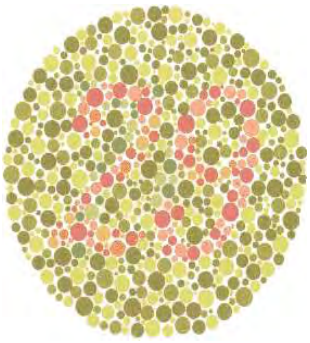

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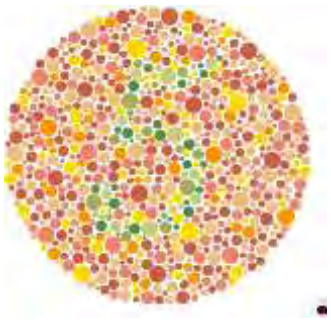

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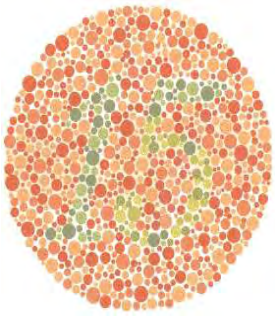

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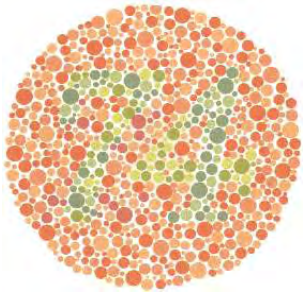

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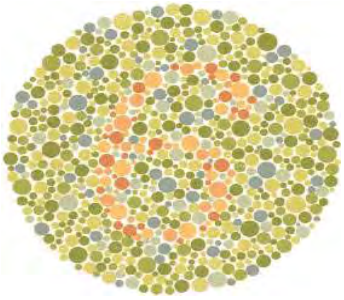

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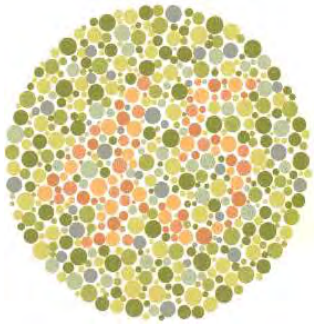

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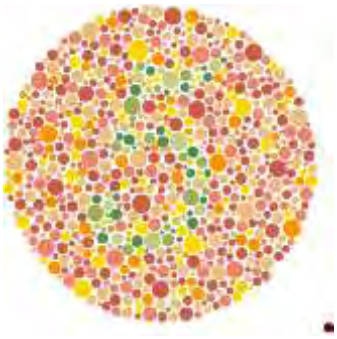

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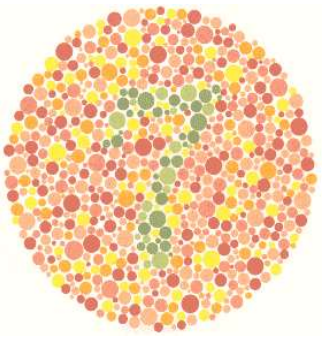

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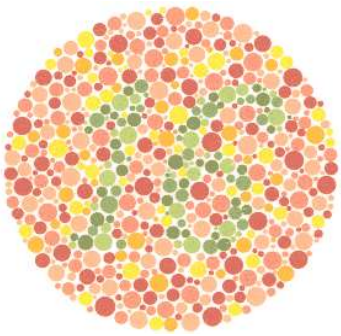

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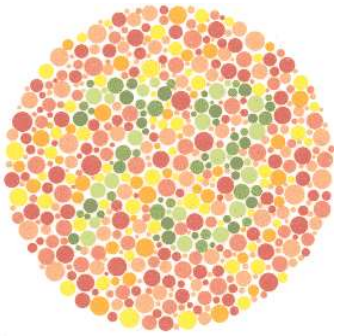

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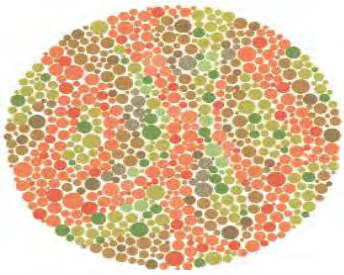

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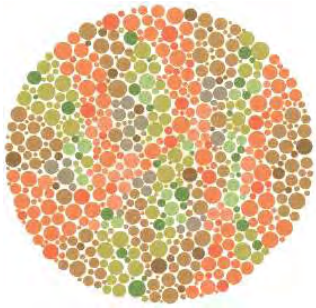
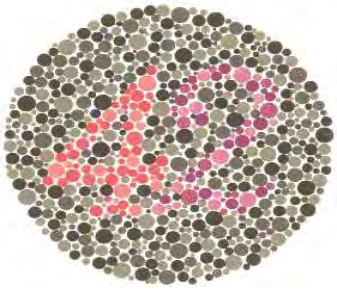


n. Plate No.15

o. Plate No.16-26



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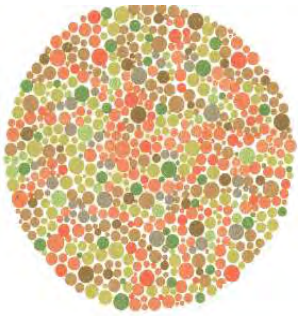
q. Plate No.18



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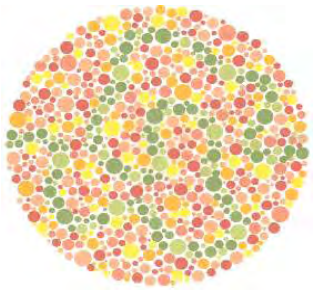

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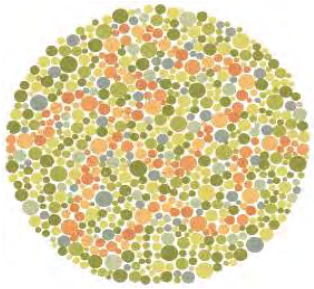
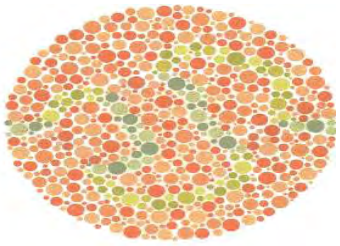
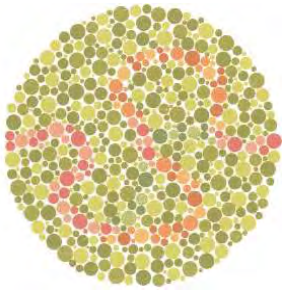


t. Plate No-21



u. Plate No.22



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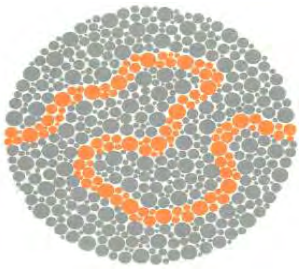

w. Plate No.24

Fig 7: Samples of plates used for color blindness test (Shinobu Ishihara, 1972)

Fig 8: Student testing for colorblindness using Ishihara plate test.

5.4. Statistical Analysis

The collected data were entered manually into a computer on excel sheet and frequencies of the various population genetic parameters were calculated. The data were arranged and checked before the analysis. The analysis was done gender wise and the prevalence of colorblindness was determined on the data of individual school as well as on the combined data of the two schools.

Ethical clearance

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

6. Limitations of the study

- Ishihara plate test is used only for red-green color test. Thus, it is difficult to estimate other color defects like tritanopia.
- Children and parents did not show willingness to tell their ethnicity; so it was not possible to determine prevalence of the defect on ethnic basis.
- Some parents and children did not volunteer to participate in the study.
- Time constraint was one of the limitations of the study.

7. Results

In the present study, out of the total of 1081 students tested for colorblindness, total 33 students (3.05%) were found to be colorblind which include 24 males (2.22%) and 9 (0.83%) in females. These 24 males with color blindness include, 4 (0.37%) achromatopsia, 13 (1.20%) deutan, 6(0.56%) protan and 1(0.09%) unclassified. Female colorblind includes achromatopsia 1 (0.09%), deutan3 (0.28%), protan 4(0.37%) and 1(0.09%) unclassified. Students that are grouped under unclassified can read plate number 16 and 17 different from what red-green colorblind students read. If those students tested using the other kind of colorblindness test such as anomaloscope their specific type of defect may be determined.

Table 3.Distribution of participants by sex and school

School	Sex				Total	Percent
	F		M			
	No	percent	No	percent		
Beseka	290	53.51%	329	61.04%	619	57.3%
Kality	252	46.49%	210	38.96%	462	42.7%
Total	542	100	539	100	1081	100%

Among the 1081 subjects' tested, 1048 (96.95%) of the participants were found to have normal color vision. The total number of students who had color vision defect was 33(3.05%) which includes 9 (0.83%) females and 24 (2.22%) males (Table 5).

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

No. tested	Normal color vision	Type of color vision defect	Sex		Total
			F	M	
1081	1048 (96.95%)	Achromatopsia (total colorblindness)	1(0.09%)	4 (0.37%)	5 (0.46%)
		Deuteranopia	3 (0.28%)	13 (1.20%)	16 (1.48%)
		Protanopia	4 (0.37%)	6 (0.56%)	10 (0.93%)
		Unclassified	1(0.09%)	1(0.09%)	2 (0.18%)
		Total	9 (0.83%)	24 (2.22%)	33 (3.05%)

The 9 (0.83%) colorblind female students were further classified into achromatopsia (n=1, 0.09%), deutan (n=3, 0.28%), protan (n=4, 0.37%) and unclassified (n=1, 0.09%). The 24 (2.22%) males were further classified into achromatopsia (n=4, 0.37%), deutan (n=13, 1.20%), protan (n= 6, 0.57%) and unclassified (n=1, 0.09%). 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 prevalence of red-green colorblindness (deutan and protans) in females was 0.66%. Among colorblind females, protons were more prevalent.

The prevalence of red-green colorblindness in males was 1.77%.The frequency of males with color vision deficiency was higher than in females by 1.38%. Deutan males are more frequent than the other color blind subjects. The prevalence of the different type of colorblindness in this study shows that the

percentage of dichromats was more than monochromats. 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 539 males tested, 24 were colorblind. Colorblind males make 24/539 or 4.45% among males. Only 9 females were colorblind out of total 542 females tested, which makes 9/542 or 1.66% of the total females tested.

Phenotypic, allelic and genotypic frequencies were calculated for male and female populations separately for the two common types of red-green color vision deficiency, Protanopia and deuteronopia (Table 6 and 7).For the sake of simplicity, we 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=0.9889 and the recessive (l) frequency of q=0.0111.

In females, the dominant phenotype frequency=p^2+2pq=0.9926 and the frequency of recessive phenotype, q^2 =0.0074.The allelic frequency is 0.9140 for (L) and 0.0860 for (l). The genotypic frequency for LL ($p^{2)}$ =0.8354, heterozygous Ll=2pq=0.1572 and homozygous recessive is q^2 =0.0074(Table 6).

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

Trait	Item		Sex	
			Male	Female
Protanopia	Sample size	Normal	533	538
		Protan	6	4
		Total	539	542
	Phenotypic frequency	Normal	0.9889	0.9926
		Protan	0.0111	0.0074
		Total	1.00	1.00
	Allelic frequency	L	0.9889	0.9140
		l	0.0111	0.0860
		Total	1.00	1.00
	Genotypic frequency	L	0.9889	-
		l	0.0111	-
		Total	1.00	-
		LL	-	0.8354
		Ll	-	0.1572
		ll	-	0.0074
		Total	-	1.00

The allelic, genotypic and phenotypic frequencies for deteranopia were calculated in the same way as described above for protanopia. In males, the frequency of M was 0.9759 and m was 0.0241. These frequencies are also true for genotypic and phenotypic frequencies of the trait.

In females, the frequency of normal vision is 0.9945 and deutans is 0.0055.The allelic frequencies calculated based on the above phenotypic frequencies gave 0.9258 and 0.0742 frequency of M and m alleles, respectively. Based on the above allelic frequency of M=p=0.9258 and m=q=0.0742, the genotypic frequencies are calculated as MM (p^2) = $(0.9258)^2$, Mm (2xpq) = (2x0.9258x0.0742) and mm=q^2 $(0.0742)^2$. These gave 0.8571, 0.1374 and 0.0055 frequencies for the three genotypes, respectively.

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

Trait	Item		Sex	
			Male	Female
Deuteranopia	Sample size	Normal	526	539
		Deutan	13	3
		Total	539	542
	Phenotypic frequency	Normal	0.9759	0.9945
		Deutan	0.0241	0.0055
		Total	1.00	1.00
	Allelic Frequency	M	0.9759	0.9258
		m	0.0241	0.0742
		Total	1.00	1.00
	Genotypic frequency	M	0.9759	-
		m	0.0241	-
		Total	1.00	-
		MM	-	0.8571
		Mm	-	0.1374
		mm	-	0.0055
		Total	-	1.00

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

The female data on protanopia and deuteranopia were subjected to chi-square analysis 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.

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	538	537.9892	+0.0108	0.00000021
Protan	4	4.0108	-0.0108	0.0000291
Total	542	542	0.0000	X^2 =0.0000293

p>0.99

B. Female data (deutranopia)

Class	Observed number(o)	Expected number(e)	Deviation(d)	Dev^2/exp
Normal	539	539.019	-0.019	0.00000067
Deutan	3	2.981	+0.019	0.000121
Total	542	542	0.0000	X^2 =0.000122

P>0.99

The chi-square value shows that the observed result is in good agreement with the expected result indicating the population from which the data was taken is at Hardy-Weinberg genetic equilibrum. In population genetics the allelic frequencies may or may not occur in the same frequencies, so in the case of the present data the allelic frequencies were not similar. The two alleles add up in to one according to Hardy –Weinberg genetic equilibrium.

8. Discussion

The study was conducted to determine the prevalence of colorblindness in Beseka and Kality secondary schools in a total of 1081 students (539 boys and girls 542) by using 24 Ishihara plate tests. Colorblindness can make educational activities difficult such as in identifying colors and to understand colorful figures. Most text-books have colorful illustrations, so students who are colorblind may have difficulties in recognizing the main points on the illustrative figures. Teachers should know about it, because 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.

The present study was carried out on a relatively small sample size when it is compared to other studies that were performed in different parts of the world. The prevalence of colorblindness is different in different countries. The prevalence of congenital colorblindness is about 8% in males and 0.4% in females (Fareed'et al, 2015).

The prevalence of colorblindness among male students in this study was higher than reported from Pakistan (Fareed et al., 2015). In some European countries even higher prevalence was reported (Mulusew, Yilikal, 2013). In the present study the prevalence of color blindness among female students (0.83%) were found to be similar with some other studies and even lower than reports done in Iraq, Japan and Black Americans (Fareed et al., 2015). Table 8 compares color blindness reported in various studies.

Table 8 .The prevalence of color blindness reported in various studies

Name of countries	Prevalence of color blindness(percent)	
	Males	Females
Libya	2.2%	unavailable
India	2.3%	0.8%
Colombia	2.4%	unavailable
Saudi Arabia	2.9%	0.75%
Germany	7.8%	unavailable
Greek	7.95%	unavailable
Turkey	7.33%	unavailable
Aligarh	unavailable	0.8%
Simla	unavailable	0.8%
Spain	unavailable	0.75%
Denmark	unavailable	0.54%,
Greenland	unavailable	0.4%
Italy	unavailable	0%
Newzealand	unavailable	0%
Iraq	unavailable	3.2%,
Iran	unavailable	0.43%,
Jordan	unavailable	0.33%,
Spain	unavailable	0.75%
Japan	unavailable	3.6%,
Black Americans	unavailable	3.7%

The present study revealed an overall 3.05% of colorblindness people, which is higher than studies done in the following countries indicated in the table. But it is lower than reports made in Britain, USA and Canada (table 10) (Karim, 2013).

Table 9.The overall prevalence of colorblindness reported in different countries.

Name of countries	Prevalence of color blindness(percent)
China	1.7%
Mexico	1.3%
Russia	1.8%
Britain,	7.8%
USA	4.8%
Canada	4.2%

Prevalence of color blindness 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.

The study on prevalence of colorblindness in Ethiopia is very few. One such study reported prevalence 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. In addition to this, other studies also indicate the prevalence 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, 0.58% unclassified, and 0.19% cases of total color blindness. The prevalence 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%) was color blind. Among these, 1.3% was females and 2.9% were males (Haile, 2014). The prevalence that is performed in Ethiopia is higher than the present study.

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 allelleic frequencies among male is 0.011deutan and 0.0918 protan. In females deutan gene frequency is 0.0924 and protan is 0.065(Bekele and Kifile, unpublished).

The present study result is more or less similar to that of Bekele and Kifile (unpublished). In the present study the allelic frequency among male was 0.0241 deutan and 0.9759 for normal; in females allelic frequency for deutan was 0.0742 and for normal it is 0.9258.The frequencies of protan and normal visioned were 0.0111 and 0.9889, respectively in males. In females frequencies of 0.0860 protan and 0.9140 normal were observed. Though it has not been possible to compare the present result and the result of Bekele Serbessa for statistical significance, overall no large deviation between the two results is discernible.

The chi-square test applied to the female data regarding the prevalence of protanopia and deutranopia has shown that there is no statistically significant deviation between the observed data and that expected assuming that the population is at Hardy-Weinberg genetic equilibrium.

9. Conclusions and recommendations

9.1 Conclusion

The present study showed

- The existence of protanopia, deuteranopia and achromatosopia among school children.
- The prevalence of colorblindness combined for males and females was 3.05%, which is similar with the finding of some other studies on colorblindness in Ethiopia and elsewhere.
- The prevalence 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 prevalence of colorblindness is more common in males than females.

9.2. Recommendation

- Color vision defect should be tested early in life of an individual particularly boys, 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.
- To document the incidence of color blindness among different ethnic groups, further study in large sample is necessary.
- Labeling coloring utensils with color name should be practiced to help children with the defect.
- 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.
- The special Need Office situated at each school should extend their awareness and assistance to colorblind students as well.
- A section on colorblindness should be included in biology textbooks in order to create awareness among teachers and students.

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Appendix I

Information sheet and consent form for study subject

The aim of this study is to identify the prevalence of color deficiency among students attending schools in Akaki/Kality subcity. 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 will get advices.

For this study I have been requested to take ISHIHARA's color vision test. I have been informed by Tsega Habte(a Msc student at AAU in Biology) here in 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 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

<u>የጥናቱ ስነ ምግባር ደንብ</u>

<u>የጥናቱ ርዕስ፡</u> Prevalence of Congenital Color Deficiencies among Students from Selected Secondary-Schools in Addis Ababa.

<u>የአጥኚዉ ስም እና አድራሻ፡ ጸጋ ሃብቴ</u>

<u>ስነህይወት ፋክልቲ፤ አዲስ አበባ ዩኒቨርሲቲ</u>

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፡፡ ተሳትፎዬንም ለመግለጽ ለዚሁ ጥናት በተዘጋጀ የቀለም ዕይታ ምርመራ የፈቃደኝነት ማጋገ ቅጹ ላይ በፊርማዬ አረጋግጠለሁ፡፡

<u>ማሳሰቢያ፡</u> ማንም ቅሬታ ያለዉ የጥናቱ ተሳታፊ የስነምግባር ኮሚቴዉን ቀጥሎ በተመለከተዉ አድራሻ ማግኘት ይችላል፡፡

ስም፡ ጸጋ ሀብቴ፡ የስነህይወት ትምህርት ክፍል ፡ ጽ.ጽ ዩንቨርሰቲ

ስልክ፡0911371653፡ ኢሜል፡Tsegahabite@gmail.com

Advisors:Dr.kifile Dagne Tel.0911103937 Email:dagnekiflesl@gmail.com

Bekele Serbessa Tel.0913041722 Email:Bekeleserbessa@gmail.com

Assent 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 approve my agreement with my signature.

Participant's name & signature----------
Date----------

Investigator's name & signature----------
Date----------

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---------/---------/----------

<u>የቀለም እይታ ምርመራ የፈቃደኝነት ማረጋገጫ ቅፅ</u>

እኔ ስሜ እንዲሁም ሌሎች መረጃዎቼ ከዚህ በታች የተገለፀው ለዚህ ምርምር ተሳታፊ ለመሆን ስወስን በአዲስ አበባ ዩኒቨርሲቲ **የማስተርስ ተማሪ የሆኑት ወ/ሮ ጸጋ ዘብቴ** 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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Appendix II

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

Number of Plate	Normal Person	Person with Red-Green Deficiencies		Person with Total Color Blindness and Weakness
1	12	12		12
2	8	3		×
3	6	5		×
4	29	70		×
5	57	35		×
6	5	2		×
7	3	5		×
8	15	17		×
9	74	21		×
10	2	×		×
11	6	×		×
12	97	×		×
13	45	×		×
14	5	×		×
15	7	×		×
16	16	×		×
17	73	×		×
18	×	5		×
19	×	2		×
20	×	45		×
21	×	73		×
		protan	deutan	
22	26	6	2	
23	42	2	4	
24	35	5	3	

X-the plate cannot read

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: Tsega Habte

Signature: _____

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

_____

Dr. Kifile Dagne

COLLEGE OF NATURAL & COMPUTATIONAL SCIENCES
Addis Ababa University

የተፈጥሮና ኮምፒቲሽናል ሣይንስ ኮሌጅ
አዲስ አበባ ዩኒቨርሲቲ

OFFICE OF THE DEAN
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Ref. No. ቁጥር CNSDO/359/2017
Date ቀን March 23/2017

To

College of Natural Science Inst s meeting held on 7/03/2017 Minute No. IRB/026 proposal entitled **"Prevalence of colorblindness among school children in Akaki/Kality sub-city of Addis Ababa"** by Tsega Habte from the Department of Zoology Sciences.

The proposal was approved for implementation.

With regards,

Shibru Temesgen /Dr./
Dean College of Natural & Computational Science

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