Red- green Color Vision Deficiency among Male Medical Students in University of Gezira, Sudan

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DECLARATION

I declare that none of the work referred to in this dissertation had been submitted in support of application for another degree or qualification at this or any other university or institution and it had been done in the department of physiology, university of Gezira.
بسم الله الرحمن الرحيم

قال تعالى:

(ولا تقنع ماليس لك به علم إلا إن الصم والبحر والجناح قلل أولئك خان عنه)

(36) الآية (63) سورة الإسراء

(ومعولا)
Dedication

I dedicate this simple work to:

My father and my mother Asma’a Hamza

My husband and son, Moayed

My brother and sisters

To Refqa
Acknowledgement

Praise and thanks to “Allah” the most gracious and the most merciful for giving me health, strength and patience to complete this study.

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Most importantly, none of this could have happened without my parents, husband, sisters, brother and my husband’s family. Every time I was ready to quit, they did not let me and I am forever grateful. This thesis stands as a testament to their unconditional love and encouragement. Special thanks to the medical students of Gezira University who were very patient and helpful namely Ahmed Faisal and Ahmed Rashid and to every student that was included in this study.
Red-green Color Vision Deficiency among Male Medical Students in University of Gezira, Sudan

Mariam omer Qurashi Babikir

Abstract

Color–blindness is the inability to perceive differences between some colors that other people can distinguish. The ability to differentiate colors is essential for our smooth daily activities. Medical students and medical practitioners who lack this ability partially or totally, experience a wide range of difficulties in their practice of medicine with a potentiality of errors. Some common difficulties reported by medical practitioners and medical students were in recognizing widespread body color changes (pallor, cyanosis, jaundice, rashes, erythema of skin), colorful charts, slides, test-strips for blood and urine, body products: blood or bile in urine, faeces, sputum, vomitus, microscopy, mouth and throat conditions, impressions presented in the Ishihara chart, titration end-points, tissue identification (surgery) etc. The aim of this project was to study the color vision deficiency (CVD) among male medical students at University of Gezira, Sudan, and to inform the affected students about their deficiency so as to deal with it probably. The prevalence of CVD among male medical students at University of Gezira was examined using the Ishihara color vision deficiency test (38 plates). The Ishihara color vision deficiency test was shown to 301 male medical students whom were selected randomly using the cluster random sampling with one stage. The students were asked to read the impressions in the color chart and compared with the right ones. The impression perceived by a person with normal color vision was different from the impression perceived by a person with color vision deficiency. The data analysis was performed using SPSS 16. The prevalence of CVD was found to be 3% which is less than the reports of prevalence of CVD from United Kingdom and USA. Medical students must be made aware of their color vision deficiency and its effects on their work. Screening will enable the student and later the doctor to become aware of limitations in their powers of observation and deal with it; the patient will be protected from harm and litigation may be avoided when doctors have adapted their practice to their deficiencies. Screening must take place to new enrolled medical and health sciences students.
دراسة عمى الألوان (الأحمر – الأخضر) وسط طلاب كلية الطب (الذكور) – جامعة الجزيرة – السودان

مرمت عمر قرشي بابكر

ملخص الدراسة

عمى الألوان هو عدم القدرة على تمييز بعض الألوان التي يمكن أن يميزها الأشخاص الطبيعيون. يعتبر تمييز الألوان أمرًا مهمًا في جميع مناحي الحياة. يواجه طلاب الطب والأطباء الذين يفتقرون لهذه القدرة كلياً أو جزئياً الكثير من المصاعب في ممارستهم لمهنة الطب مع التعرض المستمر للأخطاء. تمثل بعض المشاكل التي تواجه طالب الطب والطبيب المصاب بعمى الألوان في معرفة التغييرات اللونية التي تصب جسم الإنسان مثل (الشحوب، الإزرع، البقع، البرقان، الطفح الجلدي)، وأيضاً تمييز اللوحات الملونة، الشرائح، شرائح الاختبار للدم والبول، منتجات الجسم (وجود دم أو صفراء في البول، البراز، البصاق و الفقي)، نقاط النهاية في المعايير وتحديد الأنسجة في الجراحة، إلخ. هدف هذه الدراسة هو تحديد نسبة طلاب كلية الطب (الذكور) المصابون بعمى الألوان بجامعة الجزيرة – السودان وجعلهم ملمين بمشكلتهم ليتمكنوا من التعامل معها صحيحاً.

تم الكشف عن عمى الألوان باستخدام اختبار الإيشيهارا لعمى الألوان (38 لوحة). عرض اختبار الإيشيهارا لعمى الألوان على 301 طالب طب اختيروا عشوائياً باستخدام أخذ العينات العشوائية العقودية ذات المرحلة الواحدة. طلب من الطلاب قراءة الأرقام الموجودة في لوحات اختبار إيشيهارا (تختلف الأرقام التي يراه الشخص المصاب بعمى الألوان عن تلك التي يراه الشخص الطبيعي). تم تحليل النتائج باستخدام الحزم الإحصائية للعلوم الاجتماعية (16) ووجد أن 3% من طلاب كلية الطب (الذكور) بجامعة الجزيرة – السودان مصابون بعمى الألوان وهي أقل من النسبة المسجلة في بريطانيا و الولايات المتحدة. لا بد من توعية طلاب كلية الطب المصابون بعمى الألوان بالمشاكل التي يمكن أن يواجهوها بما في عالمهم فحص عمى الألوان يمكن طالب كلية الطب الذي سيغدو طبيباً من معرفة القيود على ملاحظاته وابتكار طرق للتغلب عليها.

هكذا يمكن حماية المرضى من التشخيص الخاطئ. يجب فحص عمى الألوان مع الكشف الطبي الذي يجرى على الطلاب المراد قبولهم للدراسة بكليات الطب وجميع كليات القطاع الصحي.
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List of abbreviations

CVD: Color vision deficiency.

CCVD: Congenital color vision deficiency.

D: dioptres.

GMP: Guanosine 3'-monophosphate.

GDP: Guanosine diphosphate.

GTP: Guanosine triphosphate.

Introduction and literature review

1.1 Background:

God blessed us with a world rich in colors. The lush green wide fields, the brilliant colors of the fall foliage, the gorgeous blue of the sea, the majestic colorful horizon during sunrise and sunset gives immense pleasure to our eyes. The ability of appreciation of color is essential for our smooth daily activities. Especially medical students and medical practitioners, who lack this faculty partially or totally, experience a wide range of difficulties in their practice of medicine with a potentiality of errors. It is very difficult for them to appreciate and evaluate the presence and extent of colored clinical signs eg, fresh blood in vomitus or stool, position of bacilli in sputum stained by Ziehl-Neelsen method or biopsy sample stained by H-E stain (Pramanik, et al, 2010).

1.1.1 Structural features of the eye:

The eye has three coats: An outer fibrous layer: the cornea, sclera and the lamina cribrosa. A middle vascular layer (uveal tract): the iris, ciliary body and choroid (figure 1.1). The ciliary body secretes aqueous humour, which is iso osmotic with plasma, and constitutes the blood–aqueous barrier. The ciliary muscle allows accommodation of vision by producing changes in lens shape. An inner nervous layer: the retinal pigmented epithelium and the neuroretina (photoreceptors – rods and cones; and retinal neurons). The retinal pigmented epithelium is involved in photoreceptor renewal and in the scavenging of free radicals (Banerjee, 2005).
1.1.2 Optical characteristics of the eye:

The eye can be considered optically as a positive double-lens arrangement that casts a real image on a light-sensitive surface, the retina. The lens system comprises: The anterior corneal surface (cornea–air interface), with +43 dioptres (D). It has a fixed focal length. The anterior and posterior surfaces of the lens, with +15 D in the accommodated eye. The iris controls light entry to the retina and reduces intra-ocular light scatter. The lens has the properties of: Transparency, Lamellar structure, Pliability (Banerjee, 2005).

Variation in refractive index from the inner core (lower) to the less dense cortex (higher), constituting a gradient-index system (gradient in the index of refraction). An adjustable focal length to allow for focussing on objects at different distances. This accommodation is achieved by alteration in shape of the lens, produced by the ciliary muscles attaching to the peripheral suspensory ligament. Ciliary muscle relaxation causes flattening of the lens, increasing its focal length. Focussing on a closer object is achieved by ciliary muscle contraction. The closest point on which the eye can focus is the near point. The amplitude of accommodation decreases with age (presbyopia). Overall, the refractive power of the eye depends on: The refractive power of the cornea, which in turn depends upon its shape; the depth of the anterior chamber, the refractive power of the crystalline lens; the axial length of the eye (Banerjee, 2005).

The eye consists of three compartments:

The anterior chamber. This contains aqueous humour, which is secreted by the ciliary body into the posterior chamber (between the iris and the lens) and flows through the pupil into the anterior chamber. The fluid is absorbed into the trabecular meshwork, the uveoscleral system and into the
episcleral blood vessels. The normal intra-ocular pressure is 10–21mm Hg. The posterior chamber and the vestibular chamber (Banerjee, 2005).

There is a protective tear film in front of the eye, comprising an oily surface layer, an aqueous layer and a deep mucoid layer. The pre-corneal tear film has the following properties: Lubrication of the eyelids; smoothening of the optical surface; nutrient transfer to the corneal epithelium; dilution and removal of irritants; antibacterial activity: secretory IgA (Banerjee, 2005).

Figure (1.1): Structural features of the eye

1.1.3 Physiology of vision:

1.1.3.1 Visual cycle:

The visual cycle involves the following mechanisms: Retinal rods and cones are photoreceptor cells specialized for the transduction of light stimuli into nerve impulses. The peripheral retina has more rods and fewer cones. The fovea has no rods, only cones which are densely packed, and is specialized for high-resolution vision. The functional regions of the rods and cones include:
An outer segment specialized for photo-transduction; An inner segment with nucleus and most of its biosynthetic machinery: ion pumps, transporters, ribosomes, mitochondria, endoplasmic reticulum; A synaptic terminal that makes contact with the photoreceptor’s target cells; A stalk or cilium connects outer and inner segments; Photo-transduction results from a cascade of biochemical events in the outer segment of the photoreceptors (Figure 1.2). Cones detect bright light and colour and provide high resolution. Each cone cell has one of three different colour-sensitive pigments: blue: 430 nm; green: 540 nm; red: 575 nm. Rods perceive dim light but do not detect colour, being responsible for black and white vision. They also help in the recognition of movement.

![Figure (1.2): Structure of a rod and a cone.](image)

The disc membrane is rich in rhodopsin, an integral seven transmembrane span protein. Rhodopsin contains 11-cis-retinal, a chromophore, as a prosthetic group, which forms a Schiff
base with a free amino group. Retinal, the aldehyde of vitamin A, is the light-sensitive pigment. The discs are loaded with guanosine 3-monophosphate (cyclic GMP). The inner segment contains the Na+ pump that is essential in generation of the resting potential. The chromophore 11-cis-retinal absorbs light in the visible range (400–700 nm). When one photon of the appropriate energy is captured by 11-cis-retinal, the configuration is changed to 11-trans-retinal by an isomerization process. 11-trans-retinal cannot form a stable complex with rhodopsin. As a result, free 11-trans-retinal and opsin (rhodopsin minus retinal) are formed. An intermediate, meta-rhodopsin II binds to and activates transducin, a G-protein, facilitating a GDP-GTP exchange. The alpha subunit of transducin exchanges its bound GDP for GTP and dissociates from the beta and gamma subunits. The free T-GTP activates a specific cyclic nucleotide phosphodiesterase, which catalyses the hydrolysis of cyclic GMP to 5-GMP. In the dark or resting state, cyclic GMP is known to bind to cyclic nucleotide gated Na+-channels in the plasma membrane of the rod and stimulates their opening. This causes release of an inhibitory neurotransmitter from their synaptic terminals. The decrease in concentration of intracellular cyclic GMP affected by phosphodiesterase activity ensures that it dissociates from the channels, allowing them to close and causing a hyperpolarization of rod plasma membrane. Hence the light stimulus has been converted into a nervous electric signal. The system is desensitized to allow further photons to be detected because closure of the Na+-channel prevents entry of Ca²⁺, to which the Na+ channel is also permeable. The consequent decrease in cytosolic Ca²⁺ concentration ensures its release from the Ca²⁺ binding protein recoverin. The Ca²⁺ free recoverin stimulates guanyl cyclase, promoting cyclic GMP formation and opening of cyclic GMP-gated channels. Changes in intracellular Ca²⁺ underlie light adaptation in the photoreceptors (Banerjee, 2005) (figure 1.3).
**Dark**

- Retinal in 11-cis form
- High concentration of cGMP
- Keeps Na⁺ channels in outer segment open
- Depolarization of photoreceptor
  - (Spreads to synaptic terminal)
  - Opens Ca²⁺ channels in synaptic terminal
  - Release of neurotransmitter
  - Hyperpolarization of on-center bipolar (and subsequently) ganglion cells
  - Depolarization of off-center bipolar (and subsequently) ganglion cells
  - Action potentials in off-center ganglion cells
  - Propagation to visual cortex

**Light**

- Absorption of light
- Retinal changes to all-trans form, activating photopigment
- Activates transducin
- Activates phosphodiesterase
- Decreases concentration of cGMP
- Na⁺ channels in outer segment close
- Hyperpolarization of photoreceptor (the receptor potential)
  - (Spreads to synaptic terminal)
  - Closes Ca²⁺ channels in synaptic terminal
  - Release of neurotransmitter
  - Further retinal processing in bipolar and ganglion cells
  - Depolarization on-center bipolar cell
  - Action potentials in on-center ganglion cells
  - Propagation to visual cortex

**(a)** In response to the dark

**(b)** In response to a light stimulus
Figure (1.3): Phototransduction, further retinal processing, and initiation of action potentials in the visual pathway. (a) Events occurring in the retina and visual pathway in response to the dark. (b) Events occurring in the retina and visual pathway in response to a light stimulus.

1.1.3.2 Central visual pathways:

The retinal image is an inversion of the visual field. The visual field is what each eye sees. Partial decussation of optic nerve fibres in the optic chiasm leads to each optic tract consisting of fibres arising in the temporal retina of the ipsilateral eye and fibres arising in the nasal retina of the contralateral eye. The pretectal area of the midbrain controls the pupillary reflexes. The superior colliculus controls saccadic eye movements. The lateral geniculate nucleus in the thalamus possesses visual information. Neurons in the lateral geniculate nucleus have concentric receptive fields. There is point-to-point representation of the retina in the visual cortex, which can be described as being retinotopically mapped (figure 1.4). The primary visual cortex transforms concentric receptive fields into linear segments and boundaries. The primary visual cortex is functionally organized into narrow vertical columns of cells that run from the pial surface to the white matter. These columns subserve a variety of functions: ocular dominance, specific line orientation, direction of movement, spatial frequency and image disparity. The most effective stimulus shapes are slits, dark bars, and edges. The cortical columns represent cells with the same receptive-field axis of orientation). The columnar units are linked by horizontal connections (Banerjee, 2005).
1.3.3 Color Vision:

1.3.3.1 Characteristics of Color:

Colors have three attributes: hue, intensity, and saturation (degree of freedom from dilution with white). For any color there is a complementary color that, when properly mixed with it, produces a sensation of white. Black is the sensation produced by the absence of light, but it is probably a
positive sensation because the blind eye does not "see black;" rather, it "sees nothing." (Kim Barrett, et al., 2010).

Another observation of basic importance is the demonstration that the sensation of white, any spectral color, and even the extraspectral color, purple, can be produced by mixing various proportions of red light (wavelength 723–647 nm), green light (575–492 nm), and blue light (492–450 nm). Red, green, and blue are therefore called the primary colors (Kim Barrett, et al., 2010). A third important point is that the color perceived depends in part on the color of other objects in the visual field. Thus, for example, a red object is seen as red if the field is illuminated with green or blue light, but as pale pink or white if the field is illuminated with red light (Kim Barrett, et al., 2010).

### 1.1.3.3.2 Retinal Mechanisms

The Young–Helmholtz theory of color vision in humans postulates the existence of three kinds of cones; each containing a different photopigment and that are maximally sensitive to one of the three primary colors, with the sensation of any given color being determined by the relative frequency of the impulses from each of these cone systems. The correctness of this theory has been demonstrated by the identification and chemical characterization of each of the three pigments. One pigment (the blue-sensitive, or short-wave, pigment) absorbs light maximally in the blue-violet portion of the spectrum. Another (the green-sensitive, or middle-wave, pigment) absorbs maximally in the green portion. The third (the red-sensitive, or long-wave, pigment) absorbs maximally in the yellow portion (Kim Barrett, et al., 2010). Blue, green, and red are the primary colors, but the cones with their maximal sensitivity in the yellow portion of the spectrum
are sensitive enough in the red portion to respond to red light at a lower threshold than green. This is all the Young–Helmholtz theory requires (Kim. Barrett, et al., 2010).

The gene for human rhodopsin is on chromosome 3, and the gene for the blue-sensitive S cone pigment is on chromosome 7. The other two cone pigments are encoded by genes arranged in tandem on the q arm of the X chromosome. The green-sensitive M and red-sensitive L pigments are very similar in structure; their opsins show 96% homology of amino acid sequences, whereas each of these pigments has only about 43% homology with the opsin of blue-sensitive pigment, and all three have about 41% homology with rhodopsin. Many mammals are dichromats; that is, they have only two cone pigments, a short-wave and a long-wave pigment. Old World monkeys, apes, and humans are trichromats, with separate middle- and long-wave pigments—in all probability because there was duplication of the ancestral long-wave gene followed by divergence (Kim. Barrett, et al., 2010).

There are variations in the red, long-wave pigment in humans. It has been known for some time that responses to the Rayleigh match, the amounts of red and green light that a subject mixes to match a monochromatic orange, are bimodal. This correlates with new evidence that 62% of otherwise color-normal individuals have serine at site 180 of their long-wave cone opsin, whereas 38% have alanine. The absorption curve of the subjects with serine at position 180 peaks at 556.7 nm, and they are more sensitive to red light, whereas the absorption curve of the subjects with alanine at position 180 peaks at 552.4 nm (Kim. Barrett, et al., 2010) (figure1.5).
1.3.3.3 Neural Mechanisms

Color is mediated by ganglion cells that subtract or add input from one type of cone to input from another type. Processing in the ganglion cells and the lateral geniculate nucleus produces impulses that pass along three types of neural pathways that project to V1: a red–green pathway that signals differences between L- and M-cone responses, a blue–yellow pathway that signals differences between S-cone and the sum of L- and M-cone responses, and a luminance pathway that signals the sum of L- and M-cone responses. These pathways project to the blobs and the deep portion of layer 4C of V1. From the blobs and layer 4, color information is projected to V8. However, it is not known how V8 converts color input into the sensation of color (Kim, Barrett, et al., 2010).
1.1.3.3.4 Color Blindness:

Color – blindness is the inability to perceive differences between some colors that other people can distinguish. It is most often of genetic nature, but might also occur because of eye, nerve or brain damage or due to use of chemicals (Cumberland, et al., 2005; Cole BL, 2004).

Color blind people are not actually blind, but are color deficient, so more appropriate term to be used for color blindness is color vision deficiency (CVD). The first known scientific paper on CVD was written by John Dalton, who himself was color blind, so CVD is also called ‘Daltonism’, after John Dalton (Dalton, 1798). The prefixes "prot-," "deuter-," and "trit-" refer to defects of the red, green, and blue cone systems, respectively. Individuals with normal color vision are called trichromats. Dichromats are individuals with only two cone systems; they may have protanopia, deuteranopia, or tritanopia (Kim. Barrett, et al., 2010).

Monochromats have only one cone system. Dichromats can match their color spectrum by mixing only two primary colors, and monochromats match theirs by varying the intensity of only one. Abnormal color vision is present as an inherited abnormality in Caucasian populations in about 8% of the males and 0.4% of the females. Tritanopia is rare and shows no sexual selectivity. However, about 2% of the color-blind males are dichromats who have protanopia or deuteranopia, and about 6% are anomalous trichromats in whom the red-sensitive or the green-sensitive pigment is shifted in its spectral sensitivity (Kim. Barrett, et al., 2010).

These abnormalities are inherited as recessive and X-linked characteristics. Color blindness is present in males if the X chromosome has the abnormal gene. Females show a defect only when both X chromosomes contain the abnormal gene. However, female children of a man with X-linked color blindness are carriers of the color blindness and pass the defect on to half of their
sons (Kim. Barrett, et al., 2010). Therefore, X-linked color blindness skips generations and appears in males of every second generation as shown in figure (1.6). Color blindness can also occur in individuals with lesions of area V8 of the visual cortex since this region appears to be uniquely concerned with color vision in humans. This deficit is called achromatopsia (Kim. Barrett, et al., 2010). Transient blue-green color weakness occurs as a side effect in individuals taking sildenafil (Viagra) for the treatment of erectile dysfunction because the drug inhibits the retinal as well as the penile form of phosphodiesterase (Kim. Barrett, et al., 2010).

Most cases of congenital color vision deficiency are characterized by a red-green deficiency which may be of two types; first, a protan type which may be absolute (protanopia) or partial (protanomalia), and secondly, a deutan type which may be absolute (deuteranopia) or partial (deuteranomalia) (Ishihara, 2006).

In protanopia the visible range of spectrum is shorter at the red end compared with that of the normal, and that part of the spectrum which appears to the normals as blue-green appears to those with protanopia as grey. The whole visible range of the spectrum in protanopia consists of two areas which are separated from each other by this grey part. Each area appears to those with protanopia as one system of color with different brightness and saturation within each area, the color in one area being different from that of the other. The slight tinge of purple which is the complementary color of blue-green appears also as grey (Ishihara, 2006).

In deuteranopia, that part of the spectrum which appears to the normal as green, appears as grey, and the visible range of the spectrum is divided by this zone into two areas, each of which appears to be of one system of color. The visible range of the spectrum is not contracted, in contrast to protanopia. Purple-red which is the complementary color of green appears also as grey (Ishihara, 2006).
In protanomalia and deuteranomalia, there is no part of the spectrum which appears as grey, but the part of the spectrum which appears to those with protanopia as grey, appears to those with protanomalia as a greyish indistinct color, and likewise, the grey part of the spectrum seen by the person with deuteranopia appears to those with deuteranomalia as an indistinct color close to grey (Ishihara, 2006).

Consequently, one of the peculiarities of red-green deficiencies is that blue and yellow colors appear to be remarkably clear compared with red and green colors.

In the congenital color vision deficiencies, although very rare, there is total color blindness which may be typical or atypical. The subject who suffers from typical total color blindness shows a complete failure to discriminate any color variations, usually with an associated impairment in central vision with photophobia and nystagmus (Ishihara, 2006).

In the atypical total color blindness, the color sensitivity to red and green, as well as to yellow and blue is so low that only very clear colors can be perceived; but, except for the color sensitivity, there is no abnormality in the visual function. The plates in the Ishihara’s book form an easy method of establishing the diagnosis on such cases and in distinguishing them from cases of red-green deficiencies (Ishihara, 2006).

Furthermore, a failure in appreciation of blue and yellow may be termed tritanomalia if partial, and tritanopia if absolute, but, even if such cases do exist, they are extremely rare (Ishihara, 2006).
Figure (1.6): Inheritance of the sex-linked red-green color vision deficiencies. The genes for the L and M cone photopigments are carried on the X chromosome. X’ designates the X chromosome carrying the abnormal gene for color vision. The yellow boxes represent carriers and the red boxes those with abnormal color vision.

1.1.3.3.5 The role of color in medical diagnosis:

It is not surprising that medical practitioners with color vision deficiencies report difficulties in clinical work. The colors of the human body and its products in health and disease are commonly unsaturated (for example, pallor, jaundice, cyanosis and many rashes) or dark (for example, blood and motions) and these are the colors that those with a deficiency find difficult to discriminate, name and match, not just for red and green but across most of the spectrum. The names so commonly used in medicine indicate the importance of color to the physician: melaena, rubella and scarlet fever are examples, yet it is difficult to be precise in many cases about the part that observations of color play in arriving at a diagnosis. Color seems to be more a sign that there
is something wrong that needs attention rather than forming an integral part of the diagnosis, although the actual color may give a clue to the nature of the pathology (Spalding, 2004). Some specific points concerning the work of the medical practitioner will illustrate the significance of this analysis in practice. The physician uses a scanning process as part of the examination of patients and their body products. It is known that it can be impossible for a person with a severe colour vision deficiency to distinguish by scanning some objects or variations in colour against certain backgrounds if they subtend an angle of less than two degrees at the eye (Smith VC and Pokorny J, 1977). In addition, cues to colors, so often used by those with a deficiency, are of no help if the color cannot be discriminated from its background in the first place. With total body color changes there can be an inability to recognize normality and to detect the milder cases of cyanosis and jaundice, and pallor even when severe. Sometimes these signs are pivotal, in that if the sign is missed, the correct course of action will not be taken.

![Figure (1.7): Transformations to deuteranopic (centre) and protanopic (right) color perceptions of a red eye.](image)

### 1.1.3.3.6 Color vision tests

There are several methods of clinical tests for color vision, such as anomaloscope, panel test (Farnsworth-Munsell 100 hue test, Farnsworth D-15 test) and pseudoisochromatic plates.

#### 1.1.3.3.6.1 The anomaloscope

The anomaloscope is an accurate technique which was first designed for clinical use in 1907 (Dreyer, 1969). This test is based upon a matching of a mixture of red light of 670nm wavelength with green light of 546nm wave length with standard yellow color of 589nm wavelength. Normal viewers and individuals with red - green deficiency use characteristic
proportions of red and green to match the yellow. On the one side of the test results, one can
group color deficiency in the subtypes protons and deutans, and on the other side, a
classification of anomalous trichromacy and dichromates can be made (Dreyer, 1969).
Gunkel (1981) considered the Nagel anomaloscope as the instrument of choice for evaluating
red- green deficiency.

1.1.3.3.6.2 Pseudoisochromatic plates:

More simple but less accurate tests can be widely employed in clinics, the most popular of
which are pseudoisochromatic plates such as Ishihara and Hard-Rand-Rittler (H-R-R) plates.
The pseudoisochromatic plates depict colored numbers of figures that stand out from a
background of dots. Individuals with color deficiency will either see no pattern at all or an
alternative pattern. Both tests can detect the great majority of those of red-green color
deficiency. Each of these tests however, fails occasionally to detect mild degrees of red-green
deficiency. Crone (1961) reported that the Ishihara plates were excellent for differntiation
between normals and abnormals but were unsuitable for quantitative differntiation. The
H-R-R plates, on contrary, were unsuitable for differentiating between normals and
abnormals but were very good for quantitative differetiation and for distinguishing between
protan and deutan types. Eventhough the pseudoisochromatic plates tests have many short-
comings, they are quick to perform and sensitive for screening color deficient persons. In the
present study, Ishihara plates were used as a test device for identification and classification of
color deficiency.

1.2 Literature review

A study held in the Department of Physiology, Nepal Medical College, Jorpati, Kathmandu,
Nepal by Pramanik, et al., (2010), revealed that, among the study population (the 1st and 2nd
year medical students of Nepal Medical College and Teaching Hospital n = 120), 5.83% of
the volunteers were color deficient. Amongst the color-deficient volunteers, 57.0% were protanopic while 43.0% were deuteranopic.

Spalding (1999), showed that the prevalence of congenital form among male doctors in the United Kingdom was probably about the same as for the population at large; i.e. 8%. However, the data is insufficient for any estimate to be made of the small number of female doctors and for the acquired forms of CVD. Because of certain features of their work, general practitioners may have special problems.

Furthermore, Hossein Dargahi, et al., (2010) studied the color blindness prevalence among Hospitals’ Clinical Laboratories' Employees and Students in Tehran University of Medical Sciences (TUMS). They found that 2.4% of TUMS Medical Laboratory Sciences’ Students and Hospitals' Clinical Laboratories' Employees are color-blind.

"poole and others (1997)" found that 13% of histopathologist and 10% of medical laboratory technologists in the United Kingdom have deficient color vision which make more errors in slide interpretation than those with normal color vision. They concluded that histopathologists and medical laboratory technologists and technicians should have their color vision tested.

Inuma and Handa (1976) found that the prevalence of congenital color vision deficiency(CCVD) in Caucasians is 8% for men and 0.4% for women. Evidence indicates that the prevalence is the same in the medical profession (Currier, (1994); Olson, (1971); Poole, et al., (1997); Rigby,et al., (1991); Koningsberger (1994); Arden, (1995)), so it is likely that in 1995 there were about 5,800 doctors in practice in the UK with CCVD( (Department of Health, (1996) ; National Health Service in Scotland, (1995); Welsh Office (1996)).
(Spalding, 2004) revealed a wide range of difficulties experienced by color vision defective doctors in their practice of medicine with a potentiality for errors.

1.3 Justification:

Color-deficient medical students will be faced by many difficulties in recognizing-widespread body color changes (pallor, cyanosis, jaundice, rashes, erythema of skin), colorful charts, slides, test-strips for blood and urine, body products: blood or bile in urine, faeces, sputum, vomitus, microscopy, mouth and throat conditions, impressions presented in the Ishihara chart, titration end-points, tissue identification (surgery) etc.

Medical students must be made aware of their congenital color vision deficiency and its effects on their work. Screening will enable the student and later the doctor to become aware of limitations in their powers of observation and devise ways of overcoming them; the patient will be protected from harm and litigation may be avoided when doctors have adapted their practice to their deficiency.

1.4 Objectives:

1.4.1 General objective:

The aim of this study is to determine the prevalence of color vision deficiency among the medical students in Gezira University –Sudan.

1.4.2 Specific objectives:

a. To determine the prevalence of CVD among male medical students.

b. To correlate the CVD with history of eye refractive errors.

c. To test the correlation between history of familial color vision defects and CVD.

d. To test the correlation between taking sufficient amount of vitamin A and CVD.
Subjects and methods

2.1 Study area:

The research was conducted at Faculty of Medicine, Gezira University, Wad Madani, Sudan. University of Gezira is one of the largest universities in Sudan, which was designed to meet the requirements of rural areas’ problems. It is situated in Wad Madani which is the second largest city in Sudan. Wad Madani occupied the center of Gezira state where Gezira Scheme is found. The faculty of medicine has 1321 students, 473 of them are males.

2.2 Target population:

2.2.1 Inclusion criteria:

Male medical students at University of Gezira, Sudan were included in this study.

2.2.2 Exclusion criteria:

The students of other faculties were excluded.

2.3 The study plan:

The study was carried out among the male medical students at University of Gezira, Sudan using Ishihara chart which is one of the simplest screening methods for CVD used throughout the world now. Ishihara Chart was shown to all participants and they were asked to read the numbers in the color chart under day light conditions at normal reading distance.
2.3.1 Ishihara test:

This series of plates is designed to provide a test which gives a quick and accurate assessment of color vision deficiency of congenital origin. This is the commonest form of color vision disturbance (Ishihara, 2006).

2.3.1.1 How to use the test:

The plates are designed to be used in a room with adequate daylight. The introduction of direct sunlight or the use of electric light may produce some discrepancy in the results because of an alteration in the appearance of shades of color. When it is convenient only to use electric light, it should be adjusted as far as possible to resemble the effect of natural daylight. The plates are held 75 cm from the subject and tilted so that the plane of the paper is at right angle to the line of the vision. The correct position of each plate is indicated by the number which is printed at the back of the plate. The numbers which are seen on plates 1-25 are stated, and each answer should be given without more than three seconds delay. If the subject is unable to read numbers, plates 26 – 38 are used and the winding lines between the two (X’s) are traced with the brush. Each tracing should be completed within ten seconds (Ishihara, 2006).

It is not necessary in all cases to use the whole series of plates. Plates 22, 23, 24 and 25 may be omitted if the test is designed merely to separate the color defectives from those with normal color appreciation. In a large scale examination the test may be simplified to an examination of six plates only; No. 1, one of Nos. 2, 3, 4, 5 one of Nos. 6, 7, 8, 9 one of Nos. 10, 11, 12, 13, one of Nos. 14, 15, 16, 17, and one of Nos. 18, 19, 20, 21. (Ishihara, 1972).

It may be necessary to vary the order of the plates if it is suspected that there is a deliberate deception on the part of the subject (Ishihara, 2006).
2.3.1.2 Explanation of the plates:

The series used in this study is made up of the following 38 plates.

No. 1. Both normal and those with all sort of CVD read it as 12.

**Numbers (Nos).** 2~5. The normal person read them as 8 (No. 2), 6 (No. 3), 29 (No. 4) and 57 (No 5). Those with red-green deficiencies read them as 3 (No. 2), 5 (No. 3), 70 (No. 4) and 35 (No 5). Those with total color blindness and weakness are not able to read any number.

**Nos.** 6~9. The normal person read them as 5 (No.6), 3 (No. 7), 15 (No. 8) and 74 (No. 9). Those with red-green deficiencies read them as 2 (No.6), 5 (No. 7), 17 (No. 8) and 21 (No. 9). Those with total color blindness and weakness are not able to read any number.

**Nos.** 10~13. The normal person read them as 2 (No.10), 6 (No. 11), 97 (No. 12) and 45 (No. 13). The majority of those with color vision deficiencies are not able to read them or read them incorrectly.

**Nos. 14~17.** The normal person read them as 5 (No.14), 7 (No. 15), 16 (No. 16) and 73 (No. 17). The majority of those with color vision deficiencies are not able to read them or read them incorrectly.

**Nos. 18~21.** The majority of those with red-green color vision deficiencies read them as 5 (No.18), 2 (No. 19), 45 (No. 20) and 73 (No. 21). The majority of the normal person and those with total color blindness and weakness are not able to read any number.

**Nos. 22~25.** The normal person read them as 26 (No. 22), 42(No. 23), 35(No. 24) and 96 (No. 25). In protanopia and strong protanomalia only 6 (No. 22), 2 (No. 23), 5 (No. 24), and 6 (No. 25) are read, and in case of mild protanomalia both number on each plates are read, but 6 (No. 22), 2 (No. 23), 5 (No. 24), and 6 (No. 25) are clearer than the other numbers. In deuteranopia and strong deuteranomalia only 2 (No. 22), 4 (No. 23), 3 (No. 24), and 9 (No. 25) are read, and in case of mild deuteranomalia both number on each plate are read, but the 2 (No. 22), 4 (No. 23), 3 (No. 24), and 9 (No. 25) are clearer than the other numbers (Ishihara, 2006) (fig 2.1).
Figure 2.1: (a) plate 1(everyone should see number12). (b) plate 2 (Normal view: 8 Red-green deficiency: 3) (c) plate 23(Normal person sees 24, Protanopia or protanomaly: 2 and Deuteranopia or deuteranomaly: 4).
Table (2.1): Explanation of the Ishihara plates.

<table>
<thead>
<tr>
<th>Number of plate</th>
<th>Normal person</th>
<th>Person with Red-Green deficiencies</th>
<th>Person with total color blindness and weakness</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>2</td>
<td>8</td>
<td>3</td>
<td>X</td>
</tr>
<tr>
<td>3</td>
<td>6</td>
<td>5</td>
<td>X</td>
</tr>
<tr>
<td>4</td>
<td>29</td>
<td>70</td>
<td>X</td>
</tr>
<tr>
<td>5</td>
<td>57</td>
<td>35</td>
<td>X</td>
</tr>
<tr>
<td>6</td>
<td>5</td>
<td>2</td>
<td>X</td>
</tr>
<tr>
<td>7</td>
<td>3</td>
<td>5</td>
<td>X</td>
</tr>
<tr>
<td>8</td>
<td>15</td>
<td>17</td>
<td>X</td>
</tr>
<tr>
<td>9</td>
<td>74</td>
<td>21</td>
<td>X</td>
</tr>
<tr>
<td>10</td>
<td>2</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>11</td>
<td>6</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>12</td>
<td>97</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>13</td>
<td>45</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>14</td>
<td>5</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>15</td>
<td>7</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>16</td>
<td>16</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>17</td>
<td>73</td>
<td>X</td>
<td>X</td>
</tr>
<tr>
<td>18</td>
<td>X</td>
<td>5</td>
<td>X</td>
</tr>
<tr>
<td>19</td>
<td>X</td>
<td>2</td>
<td>X</td>
</tr>
<tr>
<td>20</td>
<td>X</td>
<td>45</td>
<td>X</td>
</tr>
<tr>
<td>21</td>
<td>X</td>
<td>73</td>
<td>X</td>
</tr>
<tr>
<td>Protan</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strong</td>
<td>Mild</td>
<td></td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>26</td>
<td>6</td>
<td>(2)6</td>
</tr>
<tr>
<td>23</td>
<td>42</td>
<td>2</td>
<td>(4)2</td>
</tr>
<tr>
<td>24</td>
<td>35</td>
<td>5</td>
<td>(3)5</td>
</tr>
<tr>
<td>25</td>
<td>96</td>
<td>6</td>
<td>(9)6</td>
</tr>
<tr>
<td>Deutan</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strong</td>
<td>Mild</td>
<td></td>
<td></td>
</tr>
<tr>
<td>22</td>
<td>2</td>
<td>2</td>
<td>2(6)</td>
</tr>
<tr>
<td>23</td>
<td>4</td>
<td>4</td>
<td>4(2)</td>
</tr>
<tr>
<td>24</td>
<td>3</td>
<td>3</td>
<td>3(5)</td>
</tr>
<tr>
<td>25</td>
<td>9</td>
<td>9</td>
<td>9(6)</td>
</tr>
</tbody>
</table>
2.3.1.3 Analysis of the results:

Assessment of the readings of plates 1 to 21 determines the normality or defectiveness of color vision. If 17 or more plates are read normally, the color vision is regarded as normal. If only 13 or less than 13 plates are read normally, the color vision is regarded as deficient. However in references to plates 18, 19, 20 and 21, only those who read the numbers 5, 2, 45 and 73 and read them easier than those in plates 14, 10, 13 and 17 are recorded as abnormal (table 2.1).

2.4 Questionnaire:

A questionnaire was constructed to obtain the following data from the students:

Personal data, Visual problems (myopia, hyperopia, astigmatism, cataracts, etc).

History of familial CVD, eye surgery and eye trauma.

Exposure to chemical agents that may cause acquired CVD like; digoxin, barbiturates, anti-tuberculous drugs and others.

History of diabetes mellitus and hypertension.

Taking sufficient amount of vitamin A by mentioning the sources in which it is found to the student.
RESULTS

A total of three hundred and one male medical students at University of Gezira with mean age (20.22 ± 2.48) years old, participated in this study.

3.1 Percentage of CVD among medical students:

Among 301 male medical students, nine were found to have color vision deficiency (3%) of the total (figure 3.1).

![Prevalence of CVD among male medical students](image)

Figure (3.1): Prevalence of CVD among male medical students.

3.2 Correlation between eye refractive errors and CVD:

33.6% of male medical students who were included in this study (101 students), were found to have power refractive errors. Of whom 63.4% have Myopia, 10.9% have hyperopia, 2% have astigmatism, 2% have cataract, 1% have glaucoma and 12% have other problems (figure 3.2).
There was no significant correlation between eye refractive errors and CVD as shown in table (3.1).

![Percentage of visual problems among male medical students](image)

**Figure (3.2):** percentage of visual problems among male medical students.

**Table (3.1):** The correlation between eye refractive errors and CVD.

<table>
<thead>
<tr>
<th>Eye refractive errors</th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>97</td>
<td>195</td>
<td>292</td>
</tr>
<tr>
<td>CVD</td>
<td>4</td>
<td>5</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>101</td>
<td>200</td>
<td>301</td>
</tr>
</tbody>
</table>

Chi-Square=.493

sig=.482
3.2 Correlation between history of family color vision defect and CVD in the students:

No significant correlation was found between history of familial color vision defect and CVD as shown in Table (3.2) and figure (3.3).

Table (3.2): Correlation between family color vision defect and CVD:

<table>
<thead>
<tr>
<th>History of familial CVD</th>
<th>Yes</th>
<th>NO</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>26</td>
<td>248</td>
<td>274</td>
</tr>
<tr>
<td>CVD</td>
<td>2</td>
<td>7</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>28</td>
<td>255</td>
<td>283</td>
</tr>
</tbody>
</table>

Chi-Square=1.585         sig=.208

Figure (3.3): the correlation between history of familial CVD and CVD.
3.3 Correlation between history of eye surgery and CVD:

There was no significant correlation between history of eye surgery and CVD. Table (3.3)

Table (3.3): Correlation between history of eye surgery and CVD:

<table>
<thead>
<tr>
<th>History of eye surgery</th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>9</td>
<td>258</td>
<td>267</td>
</tr>
<tr>
<td>CVD</td>
<td>0</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>9</td>
<td>267</td>
<td>276</td>
</tr>
</tbody>
</table>

Chi-Square=.314          sig=.575

3.4 Correlation between history of eye trauma and CVD:

There was no significant correlation between history of eye trauma and CVD. Table (3.4)

Table (3.4): Correlation between history of eye trauma and CVD

<table>
<thead>
<tr>
<th>History of eye trauma</th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>9</td>
<td>223</td>
<td>232</td>
</tr>
<tr>
<td>CVD</td>
<td>0</td>
<td>7</td>
<td>7</td>
</tr>
<tr>
<td>Total</td>
<td>9</td>
<td>230</td>
<td>239</td>
</tr>
</tbody>
</table>

Chi-Square=.882          sig=.595
3.5 Correlation between exposure to chemical agents and CVD:

There was no significant correlation between exposure to chemical agents and CVD. (Table 3.5)

Table (3.5): Correlation between exposure to chemical agents and CVD

<table>
<thead>
<tr>
<th>Exposure to chemical agents</th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>14</td>
<td>260</td>
<td>274</td>
</tr>
<tr>
<td>CVD</td>
<td>0</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td>269</td>
<td>283</td>
</tr>
</tbody>
</table>

Chi-Square=.484                      sig=.487

3.6 Correlation between hypertension, diabetes mellitus and CVD:

There was no significant correlation between hypertension and diabetes mellitus and CVD.

Table (3.6)

Table (3.6): Correlation between hypertension and diabetes mellitus and CVD.

<table>
<thead>
<tr>
<th></th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>8</td>
<td>278</td>
<td>286</td>
</tr>
<tr>
<td>CVD</td>
<td>0</td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>8</td>
<td>287</td>
<td>295</td>
</tr>
</tbody>
</table>

Chi-Square=.259                      sig=.611
3.7 Correlation between taking sufficient amount of vitamin (A) and CVD:

There was no significant correlation between diabetes mellitus and sickle cell anemia and CVD. Table (3.7) and figure (3.4).

Table (3.7): Correlation between taking sufficient amount of vitamin (A) and CVD

<table>
<thead>
<tr>
<th>Taking sufficient amount of vitamin A?</th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>220</td>
<td>67</td>
<td>287</td>
</tr>
<tr>
<td>CVD</td>
<td>8</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>Total</td>
<td>228</td>
<td>68</td>
<td>296</td>
</tr>
</tbody>
</table>

Chi-Square=.738 sig=.390

Figure (3.4): Correlation between taking sufficient amount of vitamin A.
Discussion

Those who have color vision deficiency will be better able to adapt and make more inherited career choices, if they know about their deficiency (Cole BL, 2007).

Medical practitioners with a moderate or severe color vision deficiency can have a wide range of difficulties in clinical work. These difficulties are a potential cause of errors. It is not known how frequently errors occur but it seems inevitable that they do occur (Spalding, 2004). With appropriate advice, it is likely that most are avertable. There has been virtually no open debate on this subject and only a little research (Spalding, 2004).

This cross-sectional descriptive study examined the CVD in 301 male medical students at University of Gezira - Sudan. This is the first study to determine the prevalence of CVD among medical students at University of Gezira and in Sudan. The findings from this study are expected to highlight the problem which is an unnoticed one. The prevalence of CVD among male medical students at university of Gezira was found to be 3%. This percent is in a close approximation of the findings of Hossein Dargahi, et al., (2010), who revealed that 2.4% of Hospitals' Clinical Laboratories' Employees and Students in Tehran University of Medical Sciences (TUMS) are color deficient. A study held in the Department of Physiology, Nepal Medical College, Jorpati, Kathmandu, Nepal by Pramanik, et al., (2010), revealed that, among the study population (the 1st and 2nd year medical students of Nepal Medical College and Teaching Hospital) (n = 120), 5.83% of the volunteers were color deficient. Spalding, 1999 found that 8% of the male doctors in the United Kingdom were color deficient. Evidence indicates that in 1995 there were about 5,800 doctors in practice in the UK with CCVD( (Department of Health, (1996) ; National Health Service in Scotland, (1995); Welsh Office (1996)). In USA, prevalence of CVD in junior medical students was 12.8% (Logan JS, 1977).

However, the results obtained in this study are less than the reports of prevalence of CVD from United Kingdom and USA, but there would be difficulties of color-deficient students in their practice of medicine with a potentiality of errors "poole, et al.," determined that 13% of histopathologist and 10% of medical laboratory technologists in the United Kingdom have deficient color vision which make more errors in slide interpretation than those with normal color vision.
Study of Campbell et al, (2005) revealed that the physicians with color vision deficiencies could not identify and outline properly the clinical sign in 10 photographs of which 8 were vomit or stool (of which 6 showed fresh blood), one skin rash and for 1 to mark the position of bacilli in sputum stained by Ziehl-Neelson method, whereas physicians with normal color vision did it easily.

33.6% of male medical students who were included in this study (101 students), were found to have eye refractive errors. Of whom 63.4% had Myopia, 10.9% had hyperopia, 2% had astigmatism, 2% had cataract, 1% had glaucoma and 12% had other problems as shown in figure (4.2). When the correlation between CVD and eye refractive errors tested using chi-square test, there was no significant correlation between the two variables found.

From the total 301 students, 28 had history of familial CVD. Using chi-square test, there was no significant correlation between the two variables.
Conclusion and Recommendations

5.1 Conclusion:

The prevalence of CVD among male medical students at university of Gezira- Sudan was found to be 3%. This result was less than those reported in UK and USA which may be due to the smaller sample size used in this study (n=301) or due to racial differences as it is an X linked disease.
5.2 Recommendations

1. The students who were found to have CVD must receive proper advice to overcome their problem.

2. Screening should be done to all medical students and clinical practitioners to make them aware of their disability.

3. Color vision tests should be done to all students applying to study medicine or medical sciences.

4. More research should be done in female medical students to determine the prevalence among them.

5. For the color vision deficient doctors, disciplines such as anaesthesiology, emergency medicine, pathology, microbiology, dermatology, and surgery may pose difficulties but psychiatry and neurology are less problematic.
References:


- **Welsh Office,(1996).** Health Statistics Analysis Unit.