# International Journal of Health Sciences and Research

ISSN: 2249-9571 www.ijhsr.org

Review Article

### The Aging Eye and Vision: A Review

Esenwah E.C, Azuamah Y.C, Okorie M.E, Ikoro N.C.

Department of Optometry, Federal University of Technology, Owerri, Imo State, Nigeria.

Corresponding Author: Esenwah E.C

Received: 07/05//2014 Revised: 04/06/2014 Accepted: 06/06/2014

#### **ABSTRACT**

Aging involves anatomical, physiological and neurological changes in the body. It occurs naturally and is sometimes, accelerated by preventable factors such as economic stress and psychological difficulties; and by diseases, such as diabetes mellitus, hypertension and cancer. The eye, as part of the body, is also affected by this aging process, resulting in changes in its anatomy, physiology and neurological system. These changes include a decrease in corneal sensitivity, a decrease in the anterior chamber depth, atrophy of the dilator fibers of the iris and senile miosis. Others include reduction in lens transparency, presence of vitreous floaters and slow response to light stimuli. The eye is also known to lose its ability to focus on near objects, develops opacities of the crystalline lens, risk increased intraocular pressure, visual field loss, reduction in tear production, and experiences increased glare on exposure to very bright light. Pathological changes such as age related macular degeneration, diabetic retinopathy in people with diabetes mellitus and hypertensive retinopathy in people with hypertension, have also been reported to occur. Adequate intake of antioxidants has been reported to prevent excess free radicals formation that lead to the rapid deterioration of vision as the eye ages. Older adults are advised against smoking, excessive intake of alcohol and diets rich in cholesterol. They should always go for regular eye check-up.

Keywords: Aging eye, Vision

### INTRODUCTION

is primarily Man visually motivated machine. Just like our body, our eyes and vision change every passing day. As we get older and approaching 60 years, we will begin to observe that our eyes function quite differently from ways they were functioning when we were in our 30s. The changes observed, involve the eye itself, the surrounding muscles and the central nervous system that control the ocular functions. While some of these changes are considered normal, others are considered pathogenic or disease-related. Consequently,

while many normal eye-related changes occur in all healthy eyes, many people also suffer from disease-related changes that further impair their vision. Older eyes are more susceptible to some age-related diseases such as macular degeneration, cataracts, glaucoma and dry eye syndrome than younger eyes. (1)

Systemic health problems such as diabetes and hypertension also make the elderly more at-risk for eye diseases such as diabetic and hypertensive retinopathies. Unfortunately, most of elderly distinguish population cannot between normal vision loss and disease-related vision loss and as such, fail to seek professional care in time. (2)

**Aging:** Aging is a term difficult to define, primarily because, its meaning varies from one Individual/Organisation to the other. (1) Agulanna and Agulanna<sup>(2)</sup> defined aging as an "inevitable process of decline in psychological biological, and functioning of a person, which culminate in death." On the other hand, Abanobi<sup>(3)</sup> stated that "a person can be regarded as aging from the moment of conception or from the individual reaches moment the maturation," while the United Nations defined an "aging person" as that "who has attained the age of 60 years or more."

As at today, sociologist and demographers, are yet to agreed just when to begin to consider a person as an older adult. However, as a figure, age 65 years, is commonly found in many publications. The U.S. Census Bureau sometimes start at age 60 years for older adults. Below is a table showing the stages of life. 3

| Table 1: The stages of life Cycle. |                |                       |
|------------------------------------|----------------|-----------------------|
| S/No                               | Stage          | Approximate Age (Yrs) |
| 1                                  | Infancy        | 0-1                   |
| 2                                  | Preschool      | 2-5                   |
| 3                                  | Childhood      | 6-12                  |
| 4                                  | Adolescence    | 13-17                 |
| 5                                  | Early Maturity | 18-24                 |
| 6                                  | Maturity       | 25-40                 |
| 7                                  | Middle Age     | 41-55                 |
| 8                                  | Later Maturity | 56-75                 |
| 9                                  | Old Age        | Over 75               |

As mentioned earlier, certain developmental changes start to occur in us as we begin to age. They include normal and expected alterations in growth patterns, sexual maturation, the graying of hair color and loss of skin (dermal) elasticity, i.e., the so-called bagginess, wrinkles and creases. Indeed, wrinkling always occurs at right angles to the pull of the underlying muscles, which accounts for the radial pattern around the mouth and eyes. (2) Nevertheless, it should be noted that people vary in the rate

at which they manifest the indicators of aging. According to Abanobi, (3) these variations may be partly due to genetic make-up and partly due to lifestyle as some people remain youthful well into older adults, while others look far older than their age.

Types of Aging: Aging can be classified into primary and the secondary. Primary aging refers to the anatomical and physiological changes associated with the aging process (normal aging). This does not depend on any concomitant or co-existing disease Primary mechanism. aging has been reported to begin at about the age of 30 years and continues steadily, thereafter. Our body organs reach their peak of efficiency and reserve at about the age of 20 years and remain relatively stable until about the age of 30 years. Thereafter, a steady and gradual decline is experienced in functional activity and ability. At age 75-80 years, most physiological structures have lost about 50% of their original functional capabilities. (1)

On the other hand, secondary aging refers to the aging process that has been accelerated by controllable or preventable factors such problems, as social psychological difficulties, economic stress diseases that include diabetes, and hypertension, cardiovascular, cerebrovascular. and rheumatic cancer disorders. Other factors include smoking, excessive use of alcohol, poor nutrition and lack of proper exercise. Indeed, preventive health care must be at the forefront of all health care programs for older adults. (1)

*Vision:* According to Dorland's Illustrated Medical Dictionary, <sup>(4)</sup> Vision, is the "special sense by which objects in the external environment are perceived by means of light they give off or reflect which stimulates the photoreceptors in the retina." This phenomenon is what we refer to as the act of seeing or visual acuity, i.e., clarity or clearness. Visual acuity levels show marked

changes throughout a person's life. By the 7<sup>th</sup> decade, a progressive loss in the time dark required for adaptation experienced. (5) These resultant dysfunctional visual abilities have been reported to be predominantly responsible for loss freedom, independence mobility, and deterioration of the lifestyles of older adults. (6) According to Siegel Davidson, (7) impaired vision can also lead to isolation, depression, sometimes disorientation and confusion for the older adult. In addition, there is difficulty in ambulation leading to injuries from falls, motor accidents and general diminished productivity.

### Vision and Aging: Effects of Primary Aging on Vision

While, not everyone of the same age group will experience the same level of visual changes and symptoms, some common changes associated with the normal aging process are reviewed below.

Cornea: The cornea which forms the anterior 1/6<sup>th</sup> of the outer coat of the eyeball serves as the window of the eye with both protective and optical functions. As we age, the corneal sensitivity to touch decreases. The threshold for touch almost doubles between the ages of 10 and 80 years. The cause for this decrease is not known. (8,9) Consequently, considerably less pressure is required to damage the corneal epithelium in adults older than 60 years. This reduced sensitivity and corneal fragility increases the risk of corneal abrasion and ulcer.The cornea is also known to be thicker with age, resulting in extra light scatter inside the aging eye. It also affects the focusing power of the cornea.

Anterior Chamber: The depth of the anterior chamber (AC) has been reported to decrease from an average of 3.6mm in the age range of 15-20 years to an average of 3.0mm in the 70 years and above age group, due to the growth of the crystalline

lens. (10) Sometimes, this decrease in depth of the AC makes the angle of the AC at the root of the iris more acute, thus increasing the possibility of interference with aqueous outflow resulting to glaucoma. If this does not happen, the decrease depth increases the refractive power of the eye, making it relatively more myopic. (10)

Iris: This forms a wall or diaphragm between the anterior and posterior chambers of the eye with a central opening called the It controls the amount of light entering the eye and the depth of focus. As we age, there is an increased rigidity of the iris blood vessels and an atrophy of the dilator muscle fibers responsible for the control of our pupil size and reaction to light. This loss in strength causes the pupil to become smaller (senile miosis) and less responsive to changes in ambient lighting. (10) This linear loss with age varies from 0.43mm per decade for low photopic light levels to 0.15mm per decade for high photopic light levels. (11) These changes are dependent on sex and refractive error. Consequently, people in their 60s tend to need three times more ambient light for comfortable reading than those in their 20s. In addition, they are more likely to be dazzled by bright light and glare when emerging from a dimly lit room to a bright environment.

Crystalline Lens: The human lens continues to grow throughout life. (10) The axial thickness of the lens has been reported to increase linearly by about 28% by age 70 years from what it was at the age of about 15-20 years, i.e., from about 3.6mm to 4.6mm at age 70 years. While the nuclear thickness remains constant, the cortical thickness increases. This results in the lens losing its flexibility, thus becoming rigid. This loss of form, overtime, results in the inability of the eye to focus in detail on objects at normal close range. This change is

usually experienced at about the age 37-40 years. (5)

The miosis arising as a result of the atrophy of the dilator muscle fibers and the growth of the lens, do alter the visual performance of the eye. Thus, the amount of light reaching the retina of a normal 60 year old is only about  $1/3^{\rm rd}$  of that reaching the retina of a 20 year old. Implying that an older person must use significantly more light to achieve the same level of retinal illumination as that achieved by a younger person. This explains why, the visual performance of an older person is usually impaired at twilight. Again, as the lens thickens, it begins the selective absorption of light. (12) Flourogens begin to accumulate, just as proteins of high molecular weight increase towards the lens nucleus, leading to the yellowing of the lens overtime. This yellowing of lens reduces its transparency and causes lens opacities that serve as scatter points for light. These yellow lens pigments absorb short wavelengths more than the long ones, resulting in older adults having decreased sensitivity at the violet end of the spectrum. Consequently, objects appear yellow, while it becomes difficult for them to differentiate blue from green, and dark grey from dark brown. (10)

Vitreous: In a healthy young eye, the vitreous is a clear gel-like substance with the consistency of egg-white. It is essentially protein and hyaluric acid. Its functions are mechanical by maintaining the lens and retina in their normal positions and optical by providing a clear medium through which light rays pass unhindered to the retina. With age, the vitreous becomes thinner and more water-like (liquefaction) and at the same time undergoes syneresis. (10) Thus, pockets of vitreous develop within the eye creating lumps of cellular debris called floaters or muscaevolitantes. Vitreous floaters ordinarily settle at the bottom of the eye in a normal eye, but as the vitreous continues to

liquefy, they start moving about with speed and amplitude. Though, they do not impair vision, they are certainly a continuous source of worry and irritation to older adults, particularly, when reading. Another change observed in the vitreous is what we call posterior vitreous detachment. The thinning or the liquefaction and syneresis of the vitreous make it to pull away from the retina at the back of the eye. While, this detachment does not impair vision, its symptoms which include increased floaters, flashes of light, distorted and blurred images, are a handful of worries and anxiety for older adults.

*The Retina:* Neural refers to the parts of the nervous system which includes the brain, the spinal cord and the nerves. Our visual properties are totally dependent upon neurons (which are the specialized cells that make up the nervous system), as they pass information of whatever we perceive or see to the brain for processing, before we can appreciate whatever object is before us. (10)

The retina is made up of several layers of neurons and is an extension of the brain. The back layer of the retina also contains photoreceptor cells. These are specialized cells that transform light energy into neural signals. As we age, these retinal cells thin externally at the periphery leading to the irregular orientation of the surviving cells, thereby causing glare. It should be noted that other parts of the brain experience cell loss with age as well. Because neurons in the brain do not regenerate, these cell deaths result in reduced abilities of the eye to perceive different aspects of visual stimuli, leading to slow response time. Consequently, the older eye responds more slowly to light stimuli than younger eye.

**Retinal Pigment Epithelium:** This darkly pigmented tissue behind the retina provides the retina with rich nutrients as well as serving to absorb excess light and prevent scatter within the eye. As we age, the cells

of the retinal pigment epithelium become irregular making them less able to absorb excess light and control light scatter resulting to increase glare. It should be noted that both the anatomical and neural changes occur independent of each other. Sometimes however, they occur together resulting in reduced ocular motility, leading to reading difficulty.

**Presbyopia:** As we approach the milestone age of 37-40 years, you will begin to observe some difficulty to focus on near objects, particularly reading materials. This difficulty, we involuntarily/unconsciously try to compensate for, by pushing the print further and further away from ourselves. This is due to the hardening of the lens with age, making it to lose some of its ability to relax and contract, thereby making it difficult to vary the focal points of the eye. This loss in ability to focus on near objects due to age is referred to as presbyopia. (13)

Cataract: This is any opacity of the crystalline lens sufficient to cause visual impairment. (14) Age- related cataract (senile cataract), can be in the form of nuclear, cortical or posterior subcapsular cataract. All 3 types cause blurred vision and blindness. The main symptoms are slowly progressive, painless, reduced vision, glare, yellowing of vision due to the modification of light reaching the retina as a result of nuclear sclerosis formation. Sometime, the elderly may complain of renewed ability to read without glasses despite a decrease in distance visual acuity. This is due to the slow progression of the cataract (nuclear sclerosis) which creates acquired myopia or second sight in the process. (15)

Nicholas and Patel<sup>(15)</sup> reported that the disorganization of the lens fibre architecture, denaturation of the lens protein in the lens fibre cytoplasm, followed by the aggregation of adjacent protein molecules to form clumps are responsible for cataract formation. These changes, according to

WHO<sup>(16)</sup> are exacerbated by the increased radiant energy reaching the earth due to Ozone layer depletion.

Glaucoma: This refers to a rise in the intraocular pressure (IOP) of the eye above normal level, accompanied by its clinical features. There are two main categories of glaucoma, namely, primary-comprising (POAG) and closed-angle open-angle glaucoma, and secondary glaucoma. The most common type of glaucoma is the POAG and the condition is more of signs rather than symptoms. Hence, patients usually do not report for early treatment until they are almost blind. At this stage, very little can be done to restore vision.

The main symptoms include increased IOP, atrophy of the optic disc with characteristic excavation (cupping) and visual field defects. Everybody is at risk of developing glaucoma as we get older. Indeed, statistics show that the risk of getting glaucoma rises from 1% at the age of 40 years to 12% at the age of 80 years. (17) These risks are as a result of the shallowing of the depth of the AC due to the growth of the crystalline lens that makes the root of the iris more acute; the increased deposition of extracellular materials on the trabecular meshwork with age; and the degeneration of the endothelial cells lining the canal of the schlemm, which has been reported to be at the rate of 430 cells per year after age 40 years. All three factors contribute to decreased aqueous outflow at the trabecular meshwork resulting in IOP build-up. (17)

*Dry Eyes:* As we age, our tear glands can no longer produce enough tears to lubricate the eye. When they do, they are of poor quality. The symptoms include itching, burning sensation, foreign body sensation, intolerance to dust and smoke and occasionally, excessive tearing. The changes tend to be seen more in women (particularly after menopause) than in men. (17)

Decreased Color Vision: The fovea is the most sensitive part of the retina where fine details are resolved. It contains densely packed color-sensitive photoreceptors called cones. As the eye ages, the experiences cell loss some and consequently, loss of important color information. Hence, colors become less bright and the contrast between different colors becomes less noticeable, such that blue color may appear faded or washed-out. This is a problem for older artists, electricians. and Color seamstresses discrimination is also affected as the ageing lens starts getting discolored (yellowing) due to cataract formation.

Visual Field Loss: The visual field is the projected area of vision with the line of sight in the primary position. It covers the area extending 60superiorly, 70 inferiorly, 95 temporally and 60 nasally. This gives a totalhorizontal visual field of 190, a monocular visual field of 155, and a binocular overlap of 120. The normal aging eye loses about 20° to 30° peripheral visual field by the age of 70years. This is an average of 1°-3° per decade. This loss of peripheral field increases the risk of automobile accidents in the elderly.

Glare: This is a common complaint among the elderly when they are exposed to headlights at night or sun reflection from the windshields or pavements during the day. This makes it more difficult for them to drive. Glare is caused by the changes in the crystalline lens which causes light to scatter rather than being focused, precisely at a point on the retina. (17)

Age Related Macular Degeneration (ARMD): Also referred to as senile macular degeneration, it is caused by arteriosclerosis or insufficient blood supply through the vessels of the choroids underlying the macular area. Lack of important nutrients in diet may also be a factor. The initial complaints are reading difficulty and

inability to see things straight or straight lines appear broken. There are two types of ARMD, (14) namely:-

dry (Atrophic) ARMD - which involves the gradual destruction of cones in the macular area and wet (Exudative) ARMD- when the retina respond to the loss of blood circulation by forming new blood vessels leading to neovascularization.

The symptoms of ARMD include blurring or distortion of objects, straight lines appearing wavy or missing, vision is generally poor in low contrast or glare conditions such as in early morning, early evening or night.

Effects of Secondary (pathologic) Aging on Vision

Diabetic Retinopathy: Clinically, diabetic retinopathy can be classified into two stages, the early stage known as background retinopathy and the later and more serious stage known as proliferative retinopathy. Recently however, two additional categories have been added. They are pre-proliferative retinopathy and maculopathy. (13) The most important risk factor is the duration of the diabetes in the individual. While it is rare to develop diabetic retinopathy within 5 years of the onset of diabetes, about 5% of the sufferers of Type 2 diabetes present with background diabetic retinopathy. On the other hand, anybody diagnosed as having diabetes before the age of 30 years has a 50% chance of developing diabetic retinopathy within 10 years and 90% chance after 30 years. (14) While good metabolic control of diabetes will not prevent diabetic retinopathy, poorly controlled diabetics develop diabetic retinopathy much sooner than well-controlled ones.

Miscellaneous risk factors include pregnancy, systemic hypertension, renal disease and anemia. While pregnancy may not be controlled, the later three factors must be well controlled in diabetics to avoid the adverse effects of diabetic retinopathy. (14)

## Clinical Features of Background Diabetic Retinopathy include:

*Micro-aneurysm* located in the inner nuclear layer of the retina. They appear as small round dots usually temporal to the macula.

Hemorrhages that have "dot" and "blot" configuration. Also presence of flame-shaped hemorrhages.

*Hard exudates* which appear as yellow and waxy with distinct margins

Retinal Edema characterized by retinal thickening which obscures the underlying retinal pigment epithelium.

# Clinical Features of Pre-Proliferative Diabetic Retinopathy include:

Vascular Changes—Veins begin to appear as "bead", hop and sausage" — resembling a branch retinal artery occlusion.

Dark blot hemorrhages – represents hemorrhagic retinal infarcts.

*Multiple Cotton-wool spots* – white and opaque appearance.

Intra-retinalmicrovascular abnormalities – resembles focal areas of flat retinal neovascularization.

## Clinical Features of ProliferativeDiabetic Retinopathy include:

*Neovascularization* – new vessels proliferate on the optic new head, new vessels at disc.

Vitreous detachment – usually the result of the strong attachments of the cortical vitreous gel to areas of fibrovascular proliferation.

Hemorrhages— bleeding into the vitreous gel.

Symptoms of Diabetic Retinopathy include blurred distance vision as a result of the increase in the refractive index of the crystalline lens due to increase in blood glucose level. Usually, the vision returns to normal when the blood glucose level returns to normal. There may be no symptoms in the early stages of background diabetic retinopathy. However, once tiny hemorrhages begin to appear, the individual may begin to complain of blurred vision, which "comes and goes." If macula edema is present, there will be constant vision degradation. In the later stages, i.e., the stage of proliferative diabetic retinopathy, widespread hemorrhages cause severe vision disturbance. The individual may observe red objects in front of the eye. This is due to the hemorrhaging blood seeping into the space between the retina and the vitreous humor.

Hypertensive Retinopathy: The narrowing of the retinal arterioles is usually the primary response of the eye to systemic hypertension. In sustained hypertension, the blood-retinal barrier is disrupted in small areas, resulting in increased vascular permeability.

The fundus picture is characterized by: (i) Vasoconstriction (ii) Leakage and (iii) arteriosclerosis

- i. *Vasoconstriction:* Generalized and focal arteriolar narrowing. In severe hypertension, obstruction of the pericapillary arterioles may occur leading to the development of cotton-wool spots.
- ii. **Leakages:** Caused by abnormal vascular permeability which leads to the development of flame-shaped hemorrhages, retinal edema and hard exudates in the Henle's layer of the fovea may lead to a macular star configuration.
- iii. Arteriosclerosis: Results in the thickening of the retinal vessel wall which presents as "arterio-venous crossings." Though, the presence of these arterio-venous crossings alone does not indicate the severity of the hypertension, it however tells us that the hypertension has been present in the individual for many years.

The retinal changes that occur in hypertensive retinopathy can be grouped into four categories. (19)

*Group 1*: Mild to moderate generalized arteriolar attenuation or sclerosis.

Group 2: Moderate to marked generalized sclerosis of the retinal arterioles, exaggeration of the light reflex, arteriolar constriction associated with deflection of veins at the arterio-venous crossings (Salus' sign).

Group 3: Copper-wiring appearance of the arterioles, banking of veins distal to the arterio-venous crossings (Bonnet's sign), tapering of veins on either side of the crossings (Gunn's sign).

*Group 4*: Consists of all the characteristics in group 3 plus the silver-wiring of arterioles and disc swelling (papilloedema).

Apart from all the above features, there are other ocular manifestations associated with systemic hypertension. They include branch retinal vein occlusion, branch retinal artery occlusion, retinal arterial macro-aneurysm, ischaemic optic neuropathy, ocular motor nerve palsies and exudative retinal detachment. Uncontrolled systemic hypertension also has adverse effects on diabetic retinopathy.

### CONCLUSION AND RECOMMENDATIONS

Research Reports (20) have suggested that nutrition has a role to play in all agerelated vision problems except in glaucoma. Inadequate antioxidant intake can lead to excess free radical formation that has direct impact cataracts and macular on degenerations. Free radicals also play some roles in the formation of arterial plaques resulting in retinal vein and/or arterial occlusions. Excess fat intake can cause increased blockage of arteries also leading to retinal artery and/or vein occlusions. Obesity is a risk factor for diabetes and diabetic retinopathy. Indeed, the overall health and nutritional status of the older adult must be of concern to the maintenance of good vision. Ryan, et al., (21) established that poor nutrition was a major problem to older adults in the US. He said that about 40% of those above 65 years had inadequate nutritional intake.

Older adults should be advised against smoking, excessive intake of alcohol and diets rich in cholesterol. They should always go for regular eye check-up. Indeed, annual eye examinations are strongly recommended for older adults particularly, particularly, where there are previous family histories of eye diseases such as glaucoma, age-related macular degeneration, diabetes, etc. Prevention, they say, is better than cure.

#### REFERENCES

- 1. Schmitt, E.P. and Castillo, R.E. (2007): Primary Care in Geriatrics: An Overview. Rosenbloom and Morgan's Vision and Aging. St Louis: Butterworth Heinemann.
- Agulanna, E.C. and Agulanna, G.G. (2003): Issues on Ageing.Management of Retirement and Ageing. Owerri: JoeMankpa Publishers.
- 3. Abanobi, O.C. (2010): Aging (as a Biological Phenomenon). Core Concepts in Epidemiology & Public Health Practice-A Quick Reference Manual. Owerri: Opinion Research and Communications Inc.,
- 4. Dorland's Illustrated Medical Dictionary (2007): 31<sup>st</sup> ed. Philadelphia: Saunders Elsevier.
- 5. Marial, G.L. and Onosley, C. (1988): Vision through my aging eyesrevisited. Journal of American Optometric Association, 59: 288-94.
- 6. Morse, A.R. and Fried, D.B. (1986): Vision rehabilitation and aging.

  Journal of Visual Impairment and Blindness, 80:803-4.
- 7. Siegel, J.A. and Davidson, M. (1984):
  Demographic and socio-economic aspects of aging in the United States,
  Washington DC, United States
  Government Printing Office. p.108.

- 8. Balazsi, A., Roofman, J. and Drance, S. (1984): The effect of age on the nerve fiber population of the human optic nerve. Am J Ophthalmol, 97:760-6.
- 9. Millodot, M. (1977): The influence of age on the sensitivity of the cornea. Invest Ophthalmol Vis Sci, 16:240-72.
- Haegerstrom-Portnoy, G. and Morgan, M.W. (2007): Normal Age-Related Vision Changes. Rosenbloom and Morgans Vision and Aging. St Louis: Butterworth Heinemann.
- 11. Winn,B.,Whitaker, D. and Elliot, D.B. (1994): Factors affecting light-adapted pupil size in normal human subjects. Invest Ophthalmol Vis Sci, 35: 1132-7.
- 12. Mellorio, J. (1987): Yellowing of the human lens: nuclear and cortical contributions. Vis Res 27: 1581-7.
- 13. Grosvenor, T. (2007): Primary Care Optometry. St. Louis: Butterworth Heinemann Elsevier.
- 14. Kanski, J.J. (1997): Diabetic Retinopathy. Clinical Ophthalmology. 3<sup>rd</sup> ed. Oxford: Butterworth-Heinemann.

- 15. Nicholas, P.B. and Patel, C.K. (1994): The Status of Vitamins for the treatment of Cataract. The Optician, 208:5473.
- 16. WHO(1997): Blindness and Visual Disability: Part VII of fact sheets N147:pp. 271-289
- 17. Giese, T. and Shelley, B. (2007): Anterior Segment Diseases in the Older Adult. RosenbloomandMorgan's Vision and Aging. St. Louis: Butterworth Heinemann.
- 18. Borish, I.M. (2006): Borish's Clinical Refraction. St. Louis: Butterworth Heinemann.
- 19. Miller, S.J.H. (1996): Diseases of the Retina. Parsons' Disease of the Eye. 18<sup>th</sup> Edition. Edinburgh: Churchill Livingstone.
- 20. Caffery, B. (2007): Nutrition and Older Adults. RosenbloomandMorgan's Vision and Aging. St. Louis: Butterworth, Heinemann.
- 21. Ryan, A.S., Craig, L.D. and Finn, S.C. (1992): Nutrition intakes and dietary patterns of Older Americans: a national study. J. Gerontol, 47:45-50.

How to cite this article: Esenwah E.C, Azuamah Y.C, Okorie M.E. et. al. The aging eye and vision: a review. Int J Health Sci Res. 2014;4(7):218-226.

\*\*\*\*\*\*\*

#### International Journal of Health Sciences & Research (IJHSR)

#### Publish your work in this journal

The International Journal of Health Sciences & Research is a multidisciplinary indexed open access double-blind peer-reviewed international journal that publishes original research articles from all areas of health sciences and allied branches. This monthly journal is characterised by rapid publication of reviews, original research and case reports across all the fields of health sciences. The details of journal are available on its official website (www.ijhsr.org).

Submit your manuscript by email: editor.ijhsr@gmail.com OR editor.ijhsr@yahoo.com