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Review Article

Use of antioxidant-rich nutrients to slow the advancement of cataracts

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ABSTRACT

Cataracts occur due to opacification of the crystalline lens and are a leading cause of vision loss globally. The primary mechanism of cataract development is oxidative stress. Worldwide, nearly 17 million individuals are blind due to cataract, with 33% of cases of reduced vision. Current treatments mainly involve surgery, which may be associated with the risks of infection, astigmatism, glaucoma, pseudophakic bullous keratopathy, and retinal detachment. This study emphasizes prevention over treatment, using antioxidant-rich nutrients to delay or prevent cataract progression. This can shift the focus from conventional surgical treatment to a non-invasive, accessible approach. Eating antioxidant-rich fruits and vegetables is the effective method to obtain essential nutrients. Antioxidants, such as vitamins C and E and β-carotene, and enzymes, including superoxide dismutase, catalase, and glutathione peroxidase, can alleviate oxidative stress, help neutralize reactive oxygen species, and protect the integrity of the crystalline lens. This review aims to highlight the potential of antioxidant-rich nutrients in preventing or slowing the progression of cataract.

Keywords: Antioxidant nutrients, Cataract, Crystalline lens, Oxidative stress

INTRODUCTION

Cataract is caused by the opacification of the crystalline lens. The major cause of visual loss is cataract; the leading cause of blindness is oxidative stress; the root cause for both is cataract formation.[1]

Under physiological conditions, endogenous antioxidants (Enzymatic defenses like glutathione peroxidase, catalase, dismutase, and superoxide) and non-enzymatic antioxidants (Vitamin C and Vitamin E) neutralise ROS, resulting in maintaining redox homeostasis. Oxidative stress rises when ROS production overcomes these defense mechanisms, resulting in oxidative modification of nucleic acids, proteins, lipids and other macromolecules.[2] Many pathophysiological alterations are linked to the condition of diabetes mellitus.[3] Diabetes is a well-known cause of cataract development; however, some are xenobiotics.[4]

Unstable molecules that cause free radical damage to cells are formed by exposure to UV radiation, smoking, and stress.[5] Reactive oxygen species is produced in the lens by a variety of exogenous substances such as radiation, pharmaceutical drugs, smoke, and endogenous substances such as NADPH oxidases and cellular respiration sources.[5] Antioxidants are among the compounds reported to prevent the progression of cataract. They react with free

radicals and non-radical species; afterwards, oxidative stress triggers a defense mechanism that protects intracellular and extracellular components by maintaining oxidation-reduction balance.[6] A mere decade can delay cataract formation, with advantages that include reducing the need for surgery by half, providing financial benefits, delaying surgical complications (e.g., pseudophakic bullous keratopathy, glaucoma, retinal detachment, astigmatism) and enhancing both quality of life and lens productivity.[7]

This review emphasizes prevention over treatment by utilizing antioxidant-rich nutrients to slow the advancement of cataract. This can shift the focus from conventional surgical treatment to a non-invasive, accessible approach. To consume antioxidants rich fruits and vegetables is the most effective way to alleviate the global burden of cataracts and to enhance visual health.

Vitamin C, E, β-carotene, and enzymes, including superoxide dismutase, catalase, and glutathione peroxidase, help in neutralizing reactive oxygen species and protect the crystalline lens integrity. This review elaborates on the molecular pathways that regulate antioxidant activity at the cellular level, including Nrf2/Keap1 signaling, gap junctions, and the unfolded protein response. It further introduces the impact of genetic polymorphisms and epigenetic modifications on the antioxidant enzyme function, suggesting a personalized nutrition approach to cataract prevention. Supporting its claims with both human and animal studies; the review also bridges scientific insight with practical application by listing dietary sources of key antioxidants.

Overall, the novelty lies in integrating clinical evidence, molecular biology, genetics, and nutritional science to promote antioxidant-rich nutrients as a cost-effective, patient-friendly, and preventive strategy against cataracts, particularly beneficial for aging populations and underserved regions.

GLOBAL PREVALENCE

Globally, about 2.2 billion people affected by either distance or near vision-related problems, according to World Health Organization reports and alarmingly, around 1 billion population is suffering from vision impairment.[8] Today, these cases remain untreated and could be addressed.[9,10] These individuals are divided into various groups: 826 million have unassessed presbyopia, and 202 million from preventable blindness. Preventable blindness further subdivided into additional categories: 94 million individuals (46.53%) affected by cataracts and 884 lakhs (43.76%) with unaddressed refractive errors. Additionally, 80 lakhs individuals (3.96%) suffer from age-related macular degeneration, 77 lakhs (3.81%) are diagnosed with glaucoma, and 3.9 million (1.93%) are affected by diabetic retinopathy.[11] Worldwide, cataract is found as the cause of blindness in nearly 94 million people, that can be cured by various surgeries such as SICS and phacoemulsification.[12] By 2020, it was projected that due to cataract 33.6 million (45%) people will become blind, including 15 million adults with the age of 50 years and older.[11] In China, 22% of individuals of age group 45 to 89 years old were found affected by cataracts.[13] Reports indicate that the prevalence of cataracts among the older age group is between 53% and 58%. In 2020, this prevalence becomes higher (53.1%) in the individuals of age group 70 to 79 in comparison of age group 60 to 69 (22.1%).[14]

CRYSTALLINE LENS

The ocular lens is an exclusive part of the body that focuses all the light rays on the retina. The crystalline lens is an avascular organ, enclosed within the flexible capsule. It grows during life with new cells created from the single layer of epithelial cells located proximally inside the anterior capsule. The human lens is a transparent biconvex structure that is directly derived from the surface ectoderm. It consists of lens zonules, capsule, epithelium, and fibers, from the lateral to the medial part. Lens zonules play an important role in accommodation and are composed of cysteine; they regress with advancing age.[15] Structure and sectional view of lens is shown in Figure 1.[16]

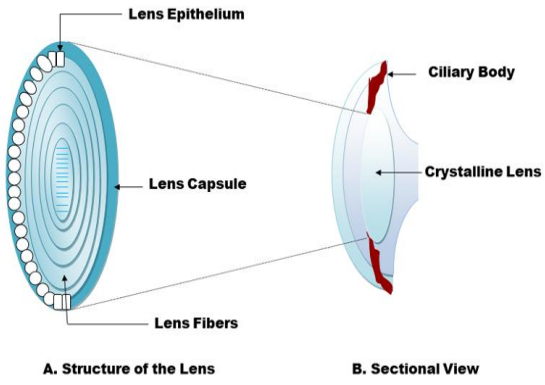

Figure 1: (A) Structure of the lens. (B) The diagrammatic sectional view of the eye.

CATARACT

It refers to the development of any opacity within the crystalline lens or its capsule. Mechanisms of cataract formation include protein aggregation and cross-linking; oxidative stress and lipid peroxidation; calcium and phosphate deposition; water and ion imbalance; and apoptosis in the eye that block the lens's ability to correctly transfer images to the retina.

The retina converts light entering the lens into signals. The optic nerve receives the signals from the retina and forwards them to the brain. Cataracts hinder the light's focus onto the retina. Stages include the incipient stage, an early and minor opacification, followed by the intumescent, mature, and then hyper-mature stage if it is not treated earlier.

Causes and clinical features

There are several causes of cataract, which can be attributed to age, increased exposure to UV rays, trauma, inflammation, medication effects such as steroids, genetic disorders, antioxidant deficiency, radiation treatment, and a sedentary lifestyle, such as smoking and an unhealthy diet. Some clinical symptoms can be felt by a person, such as reduced visual acuity, blurred vision due to opacification of the lens, double vision, glare, and halos.

Risk factors of cataract development

Several risk factors contributed to the development of cataracts. Nuclear type smokers are more prone to cataract formation with excessive alcohol intake, more UV exposure, diabetic mellitus patients, and exposure to radiation such as X-rays.[17,9]

Classification

Cataract are classified on the basis of etiological and morphological.[10]

Cataracts can be classified etiologically into congenital and developmental cataracts and acquired cataracts. Acquired cataracts include senile, traumatic, complicated, and cataracts associated with systemic diseases. Cataracts associated with systemic diseases include metabolic cataracts, such as diabetic, galactosemic, hypocalcemic, and those associated with Wilson's disease and Lowe's syndrome; syndermatotic cataracts; cataracts associated with myotonic dystrophy, neurofibromatosis type 2, osseous diseases, Down's syndrome, and Treacher–Collins syndrome. Other acquired cataracts include electric cataracts, radiation cataracts, toxic cataracts, corticosteroid-induced cataracts, miotic-induced cataracts, and cataracts induced by copper (chalcosis) and iron (siderosis).

On the basis of morphological classification, cataracts are classified into capsular cataracts (anterior and posterior), subcapsular cataracts (anterior and posterior), cortical cataracts, nuclear cataracts, and polar cataracts (anterior and posterior).

OXIDATIVE STRESS

Figure 2 shows that damage of cells and tissues occurs due to an imbalance between free radicals and antioxidants within the body; this condition is called oxidative stress. It is caused by obesity, smoking, alcohol intake, steroid medications, and exposure to UV radiation. It was reported that oxidative stress resulted from overproduction of reactive oxygen species in the body.[18] The antioxidant enzyme system consists of non-enzymatic and enzymatic components that protect against oxidative damage and maintain lens transparency.[19] Superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPX) are the enzymatic components.[20] CAT and GPX play an important role in protecting the eyes against oxidative stress.[21]

Genetic disparities in the antioxidant genes coding for the GPX and CAT enzymes may influence disease risk primarily by decreasing or impairing enzymatic activity and altering ROS detoxification at various levels.[22] Many studies has reported the relationship between SNPs in the coding region of the GPX or CAT genes and the development of cataract formation.[23] It has been suggested that the polymorphisms of CAT–21A/T and GPX1–198C/T reduce the antioxidant capacity of the lens; these gene polymorphisms are risk factors for various types of diseases.[24]

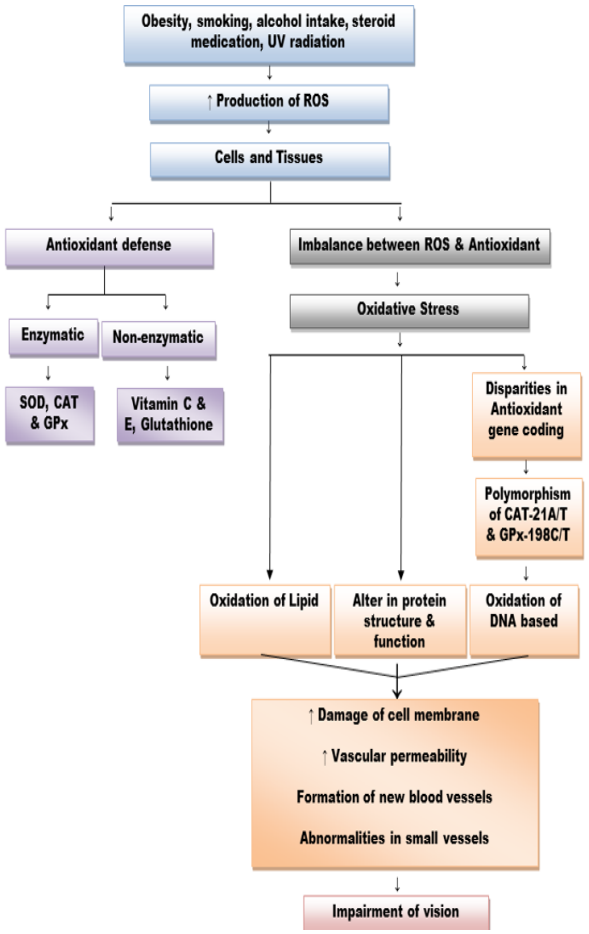

Figure 2: This shows how the vision is impaired.[25]

ANTIOXIDANT SYSTEMS

In the human lens, the antioxidant system is divided into two distinct categories: Enzymatic and Non-enzymatic antioxidants, in given table 1 shows the function of antioxidants.

Table: 1 Function of enzymatic and non-enzymatic antioxidant.

Enzymatic antioxidants		Non-enzymatic antioxidants	
SOD	It catalyses the dismutation of reactive into stable molecules, that is, superoxide into oxygen.[26]	GSH	Roles in the prevention of cataract formation.[28]
	It prevents the epithelial cells of the lens from apoptosis.[27]		In the human lens, it is found in high amounts and is involved in detoxifying numerous harmful oxidants.[29]
			It is upregulated and is involved in preventing apoptosis of lens epithelial cells by exogenous drugs.[30]
CAT	Breaks H_2O_2 into water and oxygen.[31]	Vitamin C and Vitamin E	Reduce oxidative stress in lens epithelial cells.[33]
	It suppresses the transforming growth factor-B (TGF-B), which helps in the prevention of lens opacification.[32]		
	It also suppresses lens epithelial cells surface blebbing and apoptosis.[32]		
GPX	Function is the same as CAT		
	It is a vital antioxidant enzyme in human cells.[34]		
	In mice, the formation of age-related cataract is due to the alteration of genes by the loss of expression of GPX.[35]		

Genetic and environmental factors can change the function of antioxidant enzymes of the lens epithelial cells (LECs), which enhances the development of cataract. According to a report, severity of disease due to genetic factors is about 50% in comparison of environmental factors (15%). It was observed by examining the nuclear cataract of monozygotic and dizygotic twins.[36]

A recent study suggests that a reduction in GSTP1 and TXNRD2 antioxidant genes by hypermethylation of DNA in LECs leads to early development of cataract.[37] On the one hand, UV radiation promotes apoptosis of LECs.[38]

While genetic factors cannot be in our control but by, limiting environmental factors may help reduce the progression of cataract development. Nevertheless, these recommendations should be approached with caution, as there is a lack of strong evidence-based support.

Role of endogenous antioxidant system

In geriatric age, oxidative stress and oxidative-stress-induced advanced glycation end products (AGEs) and advanced lipoxidation end products (ALE) products have been implicated (Figure 3).[39]

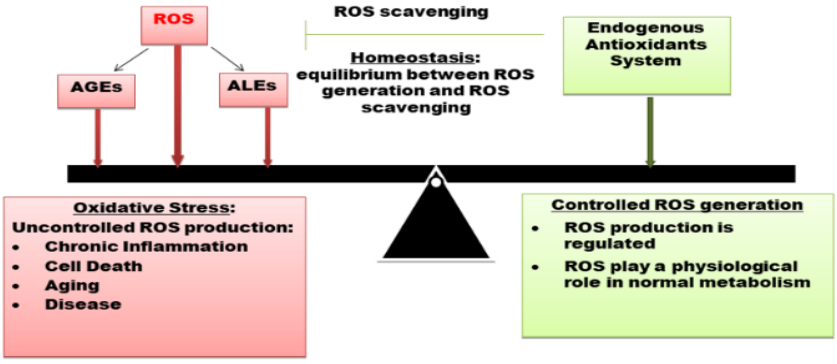

Figure 3: This figure illustrates the counterbalance between endogenous antioxidant mechanisms and the production of reactive oxygen species and their byproducts.

Antioxidant mechanism in human lens epithelium

Against oxidative stress, a lens contains a complex network of gap junctions made up of three types of connexin isoforms- Cx43, Cx46, Cx50, that play a primary defense mechanism by enabling the intracellular communication and transfer of small antioxidant molecules up to 1kDa within the lens.[40,41] These connexins regulate lens microcirculation; maintain hydrostatic pressure under fluid stress and also transport antioxidants and glucose.[42,43] Lens antioxidant defense is regulated by a transcription factor that controls the production of glutathione, superoxide dismutase, and catalase antioxidant enzymes, as well as heat shock proteins (HSPs) by nuclear factor-erythroid 2-related factor 2 (Nrf2).[44,45]

In Figure 4, it is shown that through the ubiquitin-proteasome system, during a standard physiological state, Nrf2 binds with its inhibitor, which is kelch-like ECH-associated protein 1 (Keap 1), resulting in degradation.[46] To activate the transcription of antioxidant enzymes in an oxidative environment, Nrf2 detaches from Keap1 and moves towards the nucleus, and binds to the promoter region with the associated response element (ARE).[47,48]

Stress signals from the endoplasmic reticulum (ER) influence these regulatory pathways.[48]

A study demonstrated that oxidative stress induced by hydrogen peroxide activates both Nrf2 and activating transcription factor 4 (ATF4), which facilitates the expression of ARE-regulated antioxidant enzymes.48 TXNIP regulates mitochondrial ROS and apoptosis in lens epithelial cells. BVRA modulates HO-1 and protects against senescence. ERK1/2 signalling interacts with Nrf2 in oxidative stress responses.[44,49] The activation of stress from the endoplasmic reticulum stress pathway induces the UPR (unfolded protein response) pathway, which prevents the build-up of misfolded proteins through three main signalling pathways: Protein kinase RNA-like Endoplasmic reticulum kinase (PERK), activating transcription factor 6 (ATF6) and inositol-requiring enzyme 1 (IRE1).[50]

The unfolded protein pathway controls the activation of various factors that enhance the transcription of genes in the nucleus, which is responsible for the folding of proteins and the disruption of misfolded proteins. In general, a malfunction in the antioxidant defense mechanism of the human lens can facilitate the lens opacification.

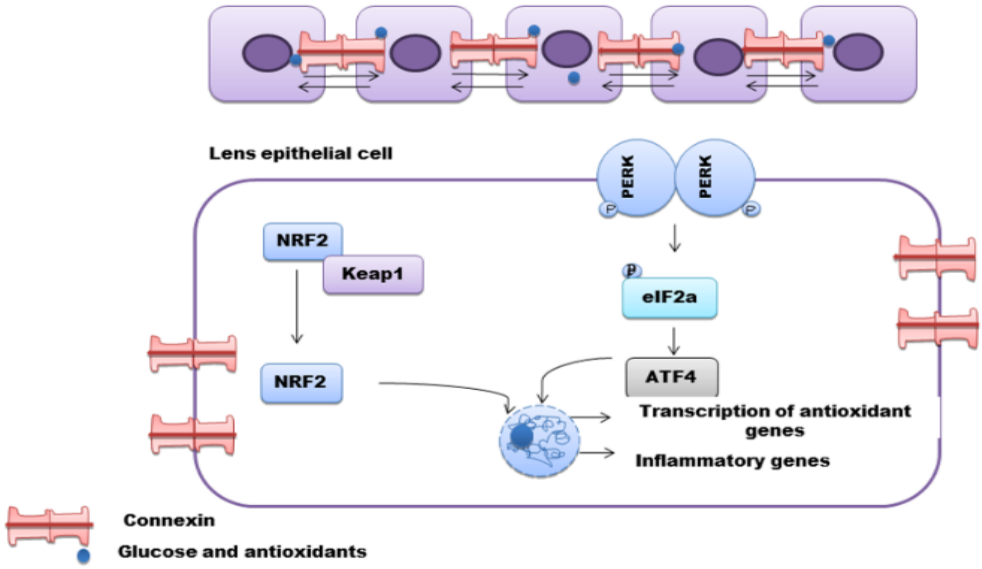

Figure 4: Diagrammatic representation of the antioxidant pathway within the lens epithelial cells.[51]

NATURAL SOURCES OF ANTIOXIDANTS ENZYMES AND THEIR EFFECTS IN CATARACT PREVENTION

In the human crystalline lens, it maintains transparency and protects from oxidative stress. Enzymes include superoxide dismutase, catalase, and Glutathione peroxidase, which help in the neutralization of reactive oxygen species. These antioxidants prevent the proteins present in the lens from being damaged and delay cataract formation, especially age-related cataract and in diabetic conditions. When superoxide anion reacts with antioxidant enzymes that is superoxide dismutase forms water and oxygen, as shown in Figure 5. Vitamin E, Vitamin C, polyphenols and carotenoids are non-enzymatic dietary antioxidants. Endogenous antioxidant enzymes are synthesised intracellularly and regulated by pathways such as Nrf2/Keap1, TXNIP/Trx2, and BVRA/HO-1.[52]

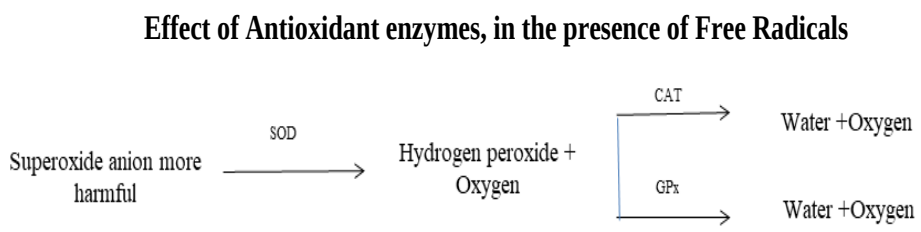

Figure 5: Effects of antioxidant enzymes.

NATURAL SOURCES OF ANTIOXIDANT VITAMINS AND THEIR EFFECTS IN CATARACT PREVENTION

In the human lens, vitamins C and E are the antioxidants. Vitamin C (vit. C) naturally neutralizes ROS and free radicals and also absorbs ultraviolet light and prevents oxidative damage.[53,54] Vitamin E helps during lipid oxidation and the transmission of free radical reactions by reducing reactive oxygen species formation.[55] In Table 2, it is shown that antioxidant vitamins are easily found from natural sources. And in Figure 6, it is shown that superoxide anion reacts with vitamin C to produce a dehydroascorbate, which is an oxidized form of ascorbic acid (vitamin C). But according to a population-based cohort study, high intake of vitamin C and E increase the risk of cataract development also.[56]

Table 2: Natural sources of antioxidant vitamins.

Antioxidant vitamin	Example
Vitamin C (water-soluble antioxidant)	Citrus fruits, potatoes, and tomatoes.[57]
Vitamin E (fat-soluble antioxidant)	Nuts, seeds, green leafy vegetables.[55]
ß Carotene	Carrot, spinach, lettuce, tomatoes, sweet potatoes, broccoli, mango, and maize.

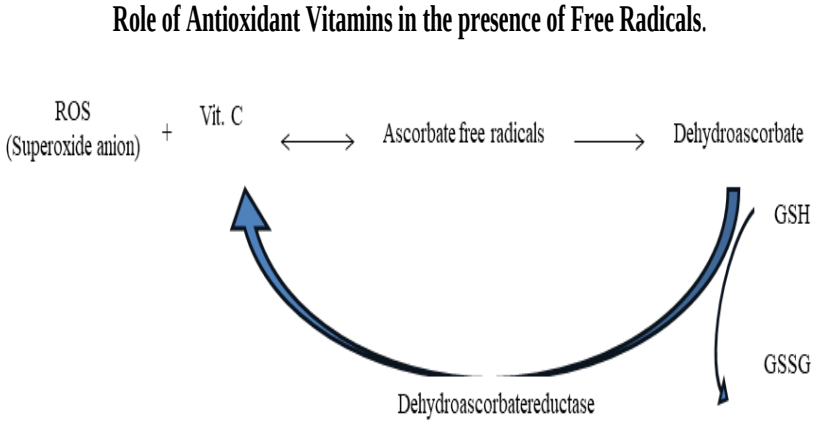

Figure 6: Effects of antioxidant vitamins.

REVIEW OF EXPERIMENTAL AND CLINICAL FINDINGS

In 2021, individuals were selected from outpatient department with inclusion criteria including senile cataract, of both sexes, and their age was between 40-75 years. A 10 ml venous blood sample was collected after an overnight fasting period of 12 hours; care was taken to avoid hemolysis.[7] And then estimate, the Serum ß carotene by Bradley & Hornbeck method, in which a protein mixture of retinal and carotene was prepared, and after that a protein is precipitated with 95% ethanol and then retinal and protein mixture is extracted into light petroleum and under spectrophotometrically at 450 nm, intensity of the yellow colour of the carotene was noted (Figure 6A). And also estimate, the Plasma Ascorbic Acid by photometric method, in which the ascorbic acid was oxidised by copper and form dehydroascorbic acid; it is then react with 2,4-dinitrophenylhydrazine to form bis-hydrazine which is red in colour, measured at 520 nm (Figure 6B). And also estimate the Serum Vitamin E by Baker & Frank method, in which the tocopherols in the serum were estimated by their ability to reduce their ferric ion to ferrous ion and the final absorbance reading was taken at 520 nm. Estimation of Plasma Superoxide Dismutase is by generation of chromogen by using phenazine methosulphate and nitroblue tetrazolium (NBT) in the presence of the Superoxide dismutase enzyme. And also estimate the

Serum Malondialdehyde (MDA) in this, the polyunsaturated fatty acid interacts with molecular oxygen to undergo destructive free radical-mediated antioxidation, followed by the formation of aldehyde and peroxide compounds during this process. In the laboratory, under acidic healing conditions, it breaks down into MDA, then reacts with thiobarbaturic acid (TBA) in an acidic environment to form a coloured product than read spectrophotometrically at 532 nm (Figure 6C). The final conclusion of this was that lowering the level of vitamin C and E in the blood was the key contributors in the formation of senile cataract. However, smoking is the major source of oxidative stress, as it substantially lowers the protective antioxidant levels in regular smokers.

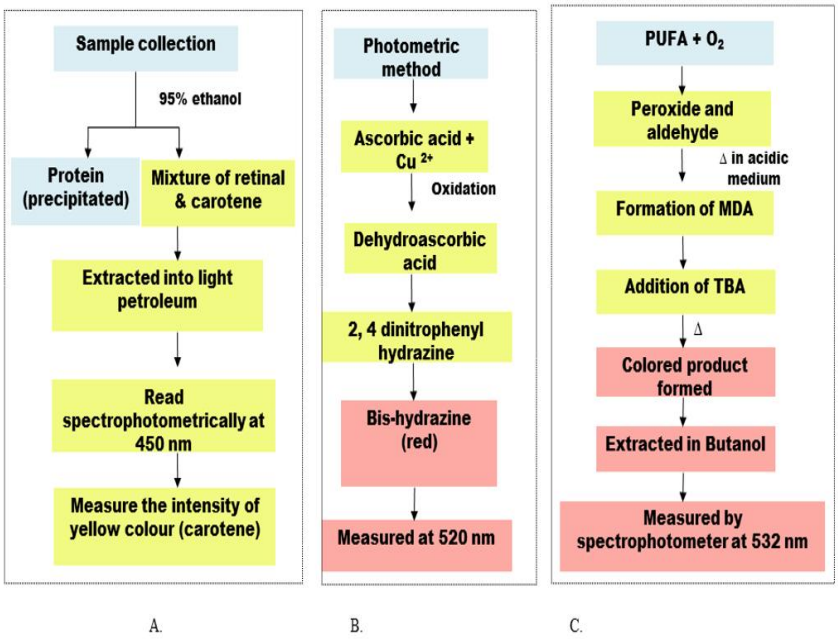

Figure 7: Flow chart of estimation of: (A) beta-carotene (B) Plasma ascorbic acid (C) Serum malondialdehyde.

And in other study, which is held in 2022, 30 rats (Rattus norvegicus) with inclusion criteria included about 8-10 weeks of age, and their weight in grams was in 150-200 in range and the gender was male sex, divided into 5 groups P1, P2, P3, P4 & P5.[58]

P1-Not give Vitamin C injection & were not induced by sodium selenite, P2-only single dose of sodium selenite, P3-Vitamin C injection (up to 1 week) as well as sodium selenite, P4-Vitamin C injection (up to 1 week) after induction of sodium selenite and in P5 group -Vitamin C injection up to 2 weeks after sodium selenite induction.

The mice were monitored daily for signs of distress and infection. Finally, the conclusion of their experiment was that a group receives vitamins C has reduced malondialdehyde levels (a marker of oxidative stress) and is better than others which does not receive Vitamin C and their difference were not statistically significant.

Another study, that is systemic literature review, was conducted in 2015 and shows that cataract is caused by the process of lipid peroxidation and calcium homeostasis imbalance, which is held due to the exposure of UV radiation.[59] Though, large cohort studies and randomised trials show mixed or even adverse effects, including increased cataract risk with high vitamin C intake.[60]

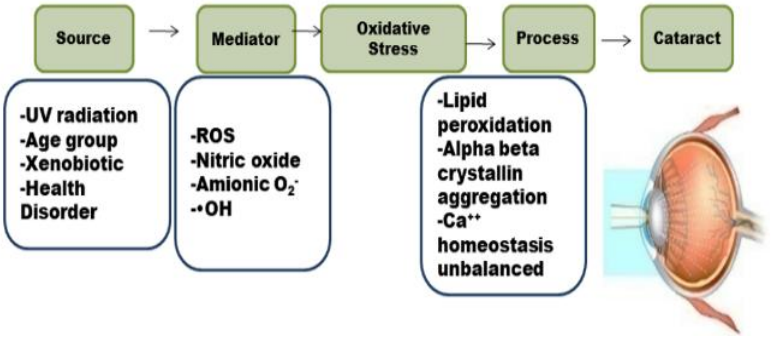

Figure 8: Cataract progression with ROS mediators.

TREATMENT OF CATARACT

Anti-glare glasses help in reducing glare-related symptoms. Surgery becomes necessary when it hinders daily tasks like driving or reading. Treatment options include Intra Capsular Cataract Extraction, Extra Capsular Cataract Extraction, Small Incision Cataract Surgery, Phacoemulsification, Microincision Cataract Surgery, and Femtosecond laser-assisted cataract surgery. But cataract can be prevented through various means, such as wearing sunglasses outdoors to shield your eyes from UVB radiation, quitting smoking, and consuming antioxidant-rich fruits and vegetables, maintaining a healthy weight, managing diabetes and other health issues, and having regular eye examinations.

CONCLUSION

This study highlights that globally, cataracts are the primary cause of vision loss. A key factor in cataractogenesis is the disruption of antioxidant regulatory systems, coupled with irregularities in cell proliferation and apoptosis within the lens epithelial cells. These irregularities lead to the formation of protein clumps, light-scattering effects, and molecular cross-linking that characterize cataracts. Over the years, numerous studies have been conducted to understand the role of antioxidants in cataractogenesis, prevention. Observational studies suggest a possible link between delaying cataract progression and the use of antioxidants, while a preclinical study shows that antioxidants help in the prevention of posterior capsular opacification and the proliferation of LECs. The advancement of technologies like gene therapy indicates non-surgical treatment strategies. However, this study does not focus on therapies or treatment; its main focus is on prevention, with individuals normally utilizing antioxidant-rich nutrients in their regular dietary plan.

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