MAXVISION Tablet: A Perfect Formulation Combines Powerful Antioxidants Helps to Reduce Eye Fatigue in Computer Professionals

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ABSTRACT

Age related macular degeneration (ARMD), Cataract and dry eye syndrome is multifactorial diseases associated with several risk factors and it is responsible for 50% of blindness worldwide. At present, the only remedy for cataract is surgery. However the incidence is so large that the available surgical facilities are unable to cope up with the problem because of postoperative complications such as posterior capsular opacification, endophthalmitis and uncorrected residual refractive error. In India alone around 30 million people suffer from cataract. Thus, the expense and unavailability of surgery mean that non-surgical medical therapy or nutritional treatment to inhibit the formation or slow the progression of cataracts is an important goal in experimental eye research to benefit patients and reduce the huge economic burden. The present Article reviews the role of MAX VISION tablet for eyes to prevent ARMD, Cataract and dry eye syndrome.

Keywords: Nutritional supplements, Eye disorders, Eye fatigue, Cataract, Maxvision tablet.

Introduction

MAX VISION tablet has perfect formulation to support the treatment of Age Related Macular Degeneration (ARMD) cataract & dry eye syndrome.

MAX VISION tablet helps in maintain healthy vision.

This formula combines powerful Carotenoids & antioxidants vitamins help’s to protect from the potentially damaging effects of free radicals also filters high energy radiation to support macular health.

Diseases and disorders of the eyes

Dry Eye Syndrome

Dry eye disease, also known as keratoconjunctivitis sicca, is a condition that results in the inflammation of the eye and the tear-producing glands. This inflammation decreases the eye’s ability to produce normal tears that protect the eye from irritation and keep it moisturized and lubricated. The tear film is composed of three intermingling layers: Oily layer - outermost layer prevents evaporation, Watery layer - middle layer moisturizes the eye, Mucous layer - inner layer allows adherence between the eye and the watery layer. A disruption of the function of any one or more of these layers can cause dry eye symptoms.
**Age-related macular degeneration (ARMD)**

ARMD is a degenerative condition of the macula (the central retina). It is the most common cause of vision loss in those 50 or older, and its prevalence increases with age. ARMD is caused by hardening of the arteries that nourish the retina. This deprives the sensitive retinal tissue of oxygen and nutrients that it needs to function and thrive. As a result, the central vision deteriorates.

Macular degeneration is a chronic disease that occurs when the macula of the eye breaks down or is damaged. The macula is part of the retina, which is located on the inside back wall of the eyeball and is responsible for central vision. Deterioration of the macula results in blurring and loss of central vision, which worsens over time, leading to blindness. This condition most commonly affects older adults so it is referred to as age-related macular degeneration. Macular degeneration is the leading cause of visual loss in people over 60 years and the second leading cause of blindness (after cataracts) in those over 65.

**There are two forms of macular degeneration:**

**Dry:** This is the most common form and is responsible for 90 percent of cases. It occurs when the macula breaks down and thins over time due to aging, free radical damage, and lack of blood and oxygen to the macula. Cellular debris accumulates under the retina and central vision slowly deteriorates over time.

**Wet:** Also known as hemorrhagic macular degeneration, this is less common but more serious, as it develops suddenly and progresses fast. It occurs when blood vessels grow under the macula, pushing against it and leaking fluid, which causes scarring of the macula and permanent damage to central vision.

Early detection and intervention can help to reduce visual loss from macular degeneration. It is possible to slow down the progression and prevent macular degeneration with lifestyle measures and supplements.

The symptoms of dry macular degeneration develop slowly over time while symptoms of the wet form develop suddenly and worsen rapidly. They include:

- Increasing need for light to do close work.
- Difficulty reading fine print.
- Words, straight lines, and signs appear crooked and wavy.
- Colours appear less bright.
- Blurring and loss of central vision; grey or blank spots in the centre of the visual field.
- Age (most common in those over 60).
- Environmental toxin exposure.
- Family history.
- Gender (women are at greater risk).
- Heart disease (high blood pressure, coronary artery disease, and stroke) Light-coloured eyes (green or blue).
- Nutritional deficiencies of vitamins A, C, E, and zinc.
Obesity.
Race (more common among Caucasians).
Smoking (the most preventable cause of macular degeneration).
UV light exposure.

**Smoking and Macular Degeneration**

Smoking a pack of cigarettes a day more than doubles a person’s risk of developing macular degeneration. Quitting lowers the risk, but they still have a higher risk than non-smokers.

There are a few surgical procedures, such as laser therapy, that can be done for wet macular degeneration, with limited success. These procedures prevent further damage to the macula and further visual loss, but they do not restore vision that is lost. Currently, there are no medical treatments for dry macular degeneration. A number of treatments are being investigated, including photocoagulation. Research has shown that antioxidant supplements can prevent worsening of this condition and further vision loss.

**Foods to Avoid**

Fast food and processed foods contain hydrogenated fats (trans-fats), saturated fats, and chemicals that can generate free radicals and have been associated with an increased risk of macular degeneration.

**Lifestyle Suggestions**

- Wear sunglasses with UV protection to shield your eyes from the sun’s harmful rays. Wide-brim hats are also recommended.
- Don’t smoke, and avoid exposure to second-hand smoke, as this is a major risk factor for macular degeneration.
- Use magnifying glasses to read, and have proper light in your home.
- Avoid driving at night and during bad weather conditions.

**Anatomy of Human Eye**

![Anatomy of human eye](image)

**Fig.1. Anatomy of human eye**
The crystalline lens is a transparent, biconvex structure in the eye that, along with the cornea, helps to refract light to be focused on the retina. The lens, by changing shape, functions to change the focal distance of the eye so that it can focus on objects at various distances, thus allowing a sharp real image of the object of interest to be formed on the retina. This adjustment of the lens is known as accommodation. It is similar to the focusing of a photographic camera via movement of its lenses. The lens is flatter on its anterior side.

The lens is also known as the *aquula* (Latin, *a little stream*, dim. of *aqua*, water) or *crystalline lens*. In humans, the refractive power of the lens in its natural environment is approximately 18 dioptres, roughly one-third of the eye’s total power.

**Position, size, and shape of lens**

The lens is part of the anterior segment of the eye. Anterior to the lens is the iris, which regulates the amount of light entering into the eye. The lens is suspended in place by the suspensory ligament of the lens, a ring of fibrous tissue that attaches to the lens at its equator [1-3] and connects it to the ciliary body. Posterior to the lens is the vitreous body, which, along with the aqueous humor on the anterior surface, bathes the lens. The lens has an ellipsoid, biconvex shape. The anterior surface is less curved than the posterior.

In the adult, the lens is typically circa 10 mm in diameter and has an axial length of about 4 mm, though it is important to note that the size and shape can change due to accommodation and because the lens continues to grow throughout a person’s lifetime.[4]. In many aquatic vertebrates, the lens is considerably thicker, almost spherical, to increase the refraction of light. This difference compensates for the smaller angle of refraction between the eye’s cornea and the watery medium, as they have similar refractive indices [5].

Even among terrestrial animals; however, the lens of primates such as humans is unusually flat. In reptiles and birds, the ciliary body touches the lens with a number of pads on its inner surface, in addition to the zonular fibres. These pads compress and release the lens to modify its shape while focusing on things at different distances; the zonular fibres perform this function in mammals. In fish and amphibians, the lens is fixed in shape, and focusing is instead achieved by moving the lens forwards or backwards within the eye [6].

**Structure and function of lens**

The lens has three main parts: the lens capsule, the lens epithelium, and the lens fibers. The lens capsule forms the outermost layer of the lens and the lens fibers form the bulk of the interior of the lens. The cells of the lens epithelium, located between the lens capsule and the outermost layer of lens fibers, are found only on the anterior side of the lens.

**Parts of lens are as follows:**

**Lens capsule**

The lens capsule is a smooth, transparent basement membrane that completely surrounds the lens. The capsule is elastic and is composed of collagen. It is synthesized by the lens epithelium and its main components are Type IV collagen and sulfated glycosaminoglycans (GAGs). The capsule is very elastic and so causes the lens to assume a
more globular shape when not under the tension of the zonular fibers, which connect the lens capsule to the ciliary body. The capsule varies from 2-28 micrometres in thickness, being thickest near the equator and thinnest near the posterior pole. The lens capsule may be involved with the higher anterior curvature than posterior of the lens [3].

**Lens epithelium**

The lens epithelium, located in the anterior portion of the lens between the lens capsule and the lens fibers, is a simple cuboidal epithelium. The cells of the lens epithelium regulate most of the homeostatic functions of the lens. As ions, nutrients, and liquid enter the lens from the aqueous humor, Na+/K+ ATPase pumps in the lens epithelial cells pump ions out of the lens to maintain appropriate lens osmolarity and volume, with equatorially positioned lens epithelium cells contributing most to this current. The activity of the Na+/K+ ATPases keeps water and current flowing through the lens from the poles and exiting through the equatorial regions [7]. The cells of the lens epithelium also serve as the progenitors for new lens fibers. It constantly lays down fibers in the embryo, fetus, infant, and adult, and continues to lay down fibers for lifelong growth [8].

**Lens fibers**

The lens fibers form the bulk of the lens. They are long, thin, transparent cells, firmly packed, with diameters typically between 4-7 micrometres and lengths of up to 12 mm long. The lens fibers stretch lengthwise from the posterior to the anterior poles and, when cut horizontally, are arranged in concentric layers rather like the layers of an onion. If cut along the equator, it appears as a honeycomb. The middle of each fiber lies on the equator. These tightly packed layers of lens fibers are referred to as laminae. The lens fibers are linked together via gap junctions and inter-digitations of the cells that resemble "ball and socket" forms. The lens is split into regions depending on the age of the lens fibers of a particular layer. Moving outwards from the central, oldest layer, the lens is split into an embryonic nucleus, the fetal nucleus, the adult nucleus, and the outer cortex. New lens fibers, generated from the lens epithelium, are added to the outer cortex. Mature lens fibers have no organelles or nuclei [8].

**Accommodation: changing the power of the lens**

The lens is flexible and its curvature is controlled by ciliary muscles through the zonules. By changing the curvature of the lens, one can focus the eye on objects at different distances from it.

This process is called accommodation. At short focal distance the ciliary muscle contracts, zonule fibers loosen, and the lens thickens, resulting in a rounder shape and thus high refractive power. Changing focus to an object at a greater distance requires the relaxation of the ciliary muscle, which in turn increases the tension on the zonules, flattening the lens and thus increasing the focal distance.

The refractive index of the lens varies from approximately 1.406 in the central layers down to 1.386 in less dense layers of the lens. This index gradient enhances the optical power of the lens. Aquatic animals must rely entirely on their lens both for focusing and to provide almost the entire refractive power of the eye as the water-cornea interface does not have a large enough difference in indices of refraction to provide significant refractive power.

As such, lenses in aquatic eyes tend to be much rounder and harder [8].
Crystallins and transparency

Crystallins are water-soluble proteins that compose over 90% of the protein within the lens. The three main crystallin types found in the human eye are α-, β-, and γ-crystallins. Crystallins tend to form soluble, high-molecular weight aggregates that pack tightly in lens fibers, thus increasing the index of refraction of the lens while maintaining its transparency. β and γ-crystallins are found primarily in the lens, while subunits of α-crystallin have been isolated from other parts of the eye and the body. α-crystallin proteins belong to a larger superfamily of molecular chaperone proteins, and so it is believed that the crystallin proteins were evolutionarily recruited from chaperone proteins for optical purposes. The chaperone functions of α-crystallin may also help maintain the lens proteins, which must last a human for his/her entire lifetime [9]. Another important factor in maintaining the transparency of the lens is the absence of light-scattering organelles such as the nucleus, endoplasmic reticulum, and mitochondria within the mature lens fibers. Lens fibers also have a very extensive cytoskeleton that maintains the lens fibers results in the lens growing more ellipsoid in shape; after about age 20, however, the lens grows rounder with time [10-11].

Nourishment of the lens

The lens is metabolically active and requires nourishment in order to maintain its growth and transparency. Compared to other tissues in the eye, however, the lens has considerably lower energy demands. By nine weeks into human development, the lens is surrounded and nourished by a net of vessels, the tunica vasculosalentis, which is derived from the hyaloid artery. Beginning in the fourth month of development, the hyaloid artery and its related vasculature begin to atrophy and completely disappear by birth [11].

In the postnatal eye, Cloquet’s canal marks the former location of the hyaloid artery. After regression of the hyaloid artery, the lens receives all its nourishment from the aqueous humor. Nutrients diffuse in and waste diffuses out through a constant flow of fluid from the anterior/posterior poles of the lens and out of the equatorial regions, a dynamic that is maintained by the Na+/K+ ATPase pumps located in the equatorially positioned cells of the lens epithelium [12].

Glucose is the primary energy source for the lens. As mature lens fibers do not have mitochondria, approximately 80% of the glucose is metabolized via anaerobic respiration. The remaining fraction of glucose is shunted primarily down the pentose phosphate pathway. The lack of aerobic respiration means that the lens consumes very little oxygen as well [13].

General overview of Cataract

Cataracts are described as an opacification (cloudiness) of the lens that leads to the scattering of light entering the eye and a loss of vision. Cataracts, which affect more than 50 million people [14] are the most common cause of blindness in the world. In first world countries, old age is the single largest cause of cataracts: only about 5% of Caucasian Americans aged 52-64 years have cataracts, whereas 18% of those aged 65-75 and 46% of those aged 75-85 are affected by cataracts.[15] As the average life span increases, the prevalence of cataract also increases. Cataract formation cannot be prevented or reversed [16], however it can be cured by surgical replacement of the
lens. There have been significant advances in surgical techniques and refinement of intraocular lens implants which have benefited cataract patients. In India alone around 30 million people suffer from cataract. Thus, the expense and unavailability of surgery mean that non-surgical medical therapy or nutritional treatment to inhibit the formation or slow the progression of cataracts is an important goal in experimental eye research to benefit patients and reduce the huge economic burden [17].

**Factors implicated in cataractogenesis**

Several risk factors have been identified in the pathogenesis of cataract. Apart from aging, smoking, diabetes, gender, steroids and nitric oxide are responsible for the development of cataract. These risk factors have been associated with different morphological type of cataract.

**Smoking:** Smoking is thought to increase the risk of cataract, at least in part by increasing the oxidative stress in the lens caused by the generated free radicals. In the presence of tobacco smoke, these free radicals may directly damage lens proteins and the fiber cell membrane in the lens [18-19]. Tobacco leaves contain a significant amount of cadmium (Cd), which is absorbed into the body when a person smokes or chews tobacco and this Cd replace the bivalent metals like Zinc (Zn), copper (Cu) and manganese from super oxide dismutase (SOD), a powerful antioxidant [20].

**Diabetes:** There are several ways that diabetes can affect the eyes but the most common cause of loss of vision is cataract. Cataractogenesis is one of earliest secondary complications of diabetes mellitus, a severe metabolic disorders characterized by hyperglycemia. Some mechanisms have been proposed for cataract formation in diabetes mellitus such as excessive tissue sorbitol concentrations, abnormal glycosylation of lens proteins and increased free radical production [21].

**Gender:** A number of epidemiological studies using cross sectional data have shown an increased prevalence of cataract in women compared with men [22]. The cause of the gender differences in cataract occurrences is not clear but could be related to the hormonal differences between women and men. Postmenopausal estrogen deficiency may be a factor. Recent epidemiologic data provided some evidence that estrogen and hormone replacement therapy may play a protective role in reducing the incidence of age related cataract [23].

**Steroids:** The association between steroid use and development of cataract is well established. There seems to be a consensus that higher the dose of steroid and longer the duration of use, the higher will be the risk for posterior sub capsular cataract [24]. Steroids cause an inhibition of the cation pump in the lens capsule and the resulting electrolyte/water imbalance is responsible for cataract formation [25].

**Nitric oxide:** O$_2^-$ in itself is not highly toxic but it may react with other molecules yielding more reactive compounds. For example, the reaction with nitric oxide (NO) generates peroxynitrite (ONOO-), which causes extensive cell damage and can also have an important role in diabetic cataract formation [26-27]. Apart from the above mentioned risk factors, genetic factors, socioeconomic status, illiteracy, malnutrition, diarrhea, myopia, renal failure, hypertension, sunlight, ultraviolet exposure, obesity, chemical burn, glaucoma and alcohol.[28,29] have also been implicated in cataractogenesis.
Fig. 2. Major risk factors implicated in cataractogenesis

**Etiology of cataract**

Developing anti-cataract agents has been difficult because cataract is not a single disease with a single aetiology. There are three major categories of cataract (nuclear, cortical, and posterior sub-capsule), each of which is multifactorial in aetiology and highly variable in severity and rate of progression. Further complicating the situation, the factors contributing to age-related cataractogenesis are a combination of pathological and normal aging processes, which have no obvious borders to distinguish them. Cataracts may be prevented if the mechanisms of formation are known. Based on the available knowledge of the biology of the normal lens and the cataractogenic process, three hypotheses have been proposed for the aetiology of cataract and three approaches have accordingly been adopted in the design of anti-cataract agents [17].

**Hypothesis for cataract**

The first hypothesis is that chronic oxidative stress is a major factor in the aetiology of age related cataract. Experimental evidence suggests that oxidative stress due to the generation of free radicals plays a role in the pathogenesis of cataracts and that the process can be prevented or ameliorated by antioxidants. Therefore, agents with anti-oxidative properties have received the most attention. Such compounds comprise of three categories including antioxidant vitamins (e.g., E, C, β-carotene); functional mimics of antioxidant enzymes; and a wide variety of low molecular weight compounds with antioxidant activity [30].

The second hypothesis is that phase separation phenomena are integral to cataract development. Phase separation results from non-covalent attractive interactions between proteins in concentrated solutions, creating protein-rich and protein poor regions. In the lens, formation of such domains creates light scattering, leading to cataract. Two putative phase separation inhibitors, pantethine and the radio protective phosphorothioate WR-77913, were tested in several acute animal models of cataract and displayed the delay of the onset of cataract [31].

The third hypothesis is the “protease hypothesis”. Calcium activated neutral enzymes, calpains, can induce proteolysis and truncate crystalline to precipitate and scatter light to form cataract [32]. Therefore, research on
calpain inhibitors is another approach to prevent or inhibit cataract formation. It has been reported that, when lambs with an inherited cataracts were treated with eye drops containing the calpain inhibitor SJA6017 for 4 months, progression of cataracts were slowed down in treated eyes compared with non-treated eyes [33].

**MOA OF MAX VISION tablet**

MAX VISION tablet helps to support the treatment of ARMD (Age Related Macular Degeneration), cataract, dry eye syndrome and helps in maintain healthy vision.

This formula combines powerful Carotenoids & antioxidants vitamins helps to protect from the potentially damaging effects of free radicals also filters high energy radiation to support macular health.

Numerous studies have found that those with high intakes of carotenoids have a lower risk of macular degeneration. Carotenoids are antioxidants found in yellow, orange, and dark green fruits and vegetables. They include lutein, and zeaxanthin. It is thought that these plant pigments protect the macula against UV light damage by dying the macula yellow (acting as natural sunglasses) and by neutralizing free radicals.

Vitamins A, C and E can protect against macular degeneration. In one study, those with the highest levels of these antioxidants had a 70 percent lower risk of developing macular degeneration.

**PHARMACOLOGY OF MAX VISION tablet**

MAX VISION tablet has perfect formulation to support the treatment of ARMD (Age Related Macular Degeneration) cataract & dry eye syndrome.

MAX VISION tablet helps in maintain healthy vision.

This formula combines powerful Carotenoids & antioxidants vitamins  help’s to protect from the potentially damaging effects of free radicals also filters high energy radiation to support macular health.

**Composition**

![Supplement Facts](image-url)
Supplement Facts

Presentation: Tablet

Indications

- Protect & Treat ARMD
- Cataract
- Dry Eye Syndrome Patient

Contra-indications: Product is contra-indicated in persons with Known hypersensitivity to any component of the product hypersensitivity to any component of the product.

Recommended usage: Adults: 1-2 MAX VISION tablet twice a day with water or liquid of choice twice daily

“Do not exceed the recommended daily dose”.

ISSN: 2581-5059  www.mjbas.com
Administration: Taken by oral route at any time with food.

Precautions: Do not exceed the recommended daily dose.

Warnings: If you are taking any prescribed medication or has any medical conditions always consults doctor or healthcare practitioner before taking this supplement.

Side Effects: Very mild side effects like nausea, headache and vomiting in some individuals may be observed.

Storage: Store in a cool, dry and dark place.

Summary & Conclusion

It is widely accepted that oxidative stress is a significant factor in the progression of cataractogenesis. Oxidative stress is associated with increased reactive oxygen species and is known to accelerate cataract formation since superoxide is converted to a toxic substance, namely hydrogen peroxide. This reaction is prevented by antioxidant enzymes, namely catalase, superoxide dismutase and glutathione peroxidase. Antioxidants are key prophylactic agents in preventing oxidation related cataractogenesis. A large number of epidemiological and interventional studies have been investigated for the role of dietary antioxidant supplement in the incidence of cataract. MAX VISION tablet has perfect formulation to support the treatment of ARMD (Age Related Macular Degeneration) cataract & dry eye syndrome.

Declarations

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Competing Interests Statement

The authors declare no competing financial, professional and personal interests.

Consent for publication

Authors declare that they consented for the publication of this research work.

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