Eye health

Description

To: Jesus

Use this as an aide to your own research and share with your doctor as appropriate.

You can use drugs.com or other trusted health websites to look up the latest information on prescription drugs, herbs, foods or other treatments possible side & interaction effects often by typing in the name of two drugs or drug/herb and interaction effects into a search engine.

Eye Health

Night vision

Black currant & bilberry

Black currant & bilberry have anthocyanosides like c**yanidin-3-glucoside (C3G)** that appear to help to see in low light & shifting lights & prevent visual fatigue. Bilberry & pycnogenol may also help cataracts.

Superfruit black currant supplies active C3G to support dark adaptation and night vision performance

Yellow sunglasses

Wearing yellow undarkened sunglasses (blueblockers) block blue light & appear to significantly enhance night vision by reducing blue light that scatters vision & turns off night vision temporarily.

Steroid injections directly into the eye may be 2x as effective against uveitis than adjacent.

What may cause macular degeneration-
Chris Knoppe

Refined oils
Cooking polyunsaturated fat
Refined grains
Transfat
Refined sugars
NRG, heart disease, diabetes, obesity, lung cancer, copd, lung infections, asthma, macular
degeneration

YouTube player

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What May Help

kale, spinach, avocados & egg yolks may have lutein & zeaxanthin for eyes

beta carotene
vitamin D3
coQ10
acetyl-L-carnitine
acetyl-L-carnosine
vitC
noni
acai

dry eyes-
spirulina

curcumin
vitA
vitD
GAMMA LINOLENIC ACID GLA
mucus specific anti-inflammatory
80% effective
statins appear to increase cataracts
pinkeye- boric acid in warm water, belladonna (homeopathic) worked better

contact lenses- peroxide kills blinding acanthamoeba parasite- never use tap water to rinse
macular degeneration AMD

Contact lenses appear to contain 5,000 higher than safe levels PFAS (flouride)
nationalworld.com/health/soft-contact-lenses-found-to-contain-toxic-forever-chemicals-4155461

800mg+/day calcium 2x/\ age related macular degeneration AMD
http://www.lef.org/protocols/eye_ear/macular_degeneration_07.htm
wet needs avastin

The macula or macula lutea (from Latin macula, “spot” + lutea, “yellow”) is a highly pigmented
yellow spot near the center of the retina of the human eye, providing the clearest, most distinct
vision needed in reading, driving, seeing fine detail, and recognizing facial features.

Age-related macular degeneration (AMD) is a devastating condition characterized by the
deterioration of the macula in which central vision becomes severely impaired. There are two
forms of macular degeneration: atrophic (dry) and neovascular (wet). Both forms of the disease
may affect both eyes simultaneously.

Age-related declines in the retinal carotenoid pigment content,

coupled with photo damage
induced by harmful Ultraviolet (UV) rays, give rise to this debilitating condition. The progression
and severity of macular degeneration, as with all age-related diseases, are exacerbated by
factors such as oxidative stress, inflammation, high blood sugar, and poor vascular health.

Scientifically studied natural compounds which help restore waning carotenoid levels within the
macula, boost the antioxidant defenses of the eye, and support healthy circulation offer an
effective adjunct to conventional treatment that may greatly improve the outlook for those with
AMD.

This protocol will explore the pathology, weigh the risks and benefits of conventional treatment,
and reveal exciting new scientific findings on innovative natural approaches for ameliorating the
effects of AMD.

Prevalence

AMD is the leading cause of irreversible visual impairment and blindness among North
Americans and Europeans 60 and older. According to the National Institute of Health, more
Americans are affected by AMD than cataracts and glaucoma combined. The eye-health
organization Macular Degeneration Partnership estimates that as many as 15 million Americans
currently exhibit evidence of macular degeneration (www.amd.org).

Approximately 85-90 percent of AMD cases are the dry form. Wet AMD, which represents only
10-15 percent of AMD cases, is responsible for more than 80 percent of blindness. AMD is
equally common in men and women, and has a heritable nature (Klein 2011; Haddad 2006).

A positive development is that the estimated prevalence of AMD in Americans 40 and older has
decreased from 9.4% in the years 1988-1994 to 6.5% in the years 2005-2008 (Klein 2011).
Pathology of AMD

The retina is the innermost layer of the eye, which contains nerves that communicate sight.
Behind the retina is the choroid, which supplies the blood to the macula and retina. In the
atrophic (dry) form of AMD, cellular debris called drusen accumulate between the retina and the
choroid. The macular degeneration progresses slowly with vision

lost painlessly. In the wet form of AMD, blood vessels below the retina undergo abnormal growth into the retina beneath the macula.

These newly formed blood vessels frequently bleed, causing the macula to bulge or form a mound, often surrounded by small hemorrhages and tissue scarring. The results are a distortion in central vision and the appearance of dark spots. Whereas the progression of atrophic AMD may take place over years, neovascular AMD can progress in mere months or even weeks (de Jong 2006).

While the exact causes of AMD are not fully understood, recent scientific evidence points to chronic vascular disease, including cardiovascular disease, as a potential cause. Scientists believe that slow degradation of the blood vessels in the choroid, which provides blood to the retina, may lead to macular degeneration.

A complementary theory suggests an alteration in the dynamics of the choroidal blood circulation as an important pathophysiological mechanism. Blockages within the choroidal blood vessels, possibly due to vascular disease, lead to increased ocular rigidity and decreased efficiency in the choroidal blood circulation system. Specifically, the increased capillary resistance (due to blockages) causes elevated pressure, resulting in

the extracellular release of
proteins and lipids that form deposits known as drusen (Kaufmen 2003).

Cholesterol exists within the drusen. Researchers suggest that the formation of AMD lesions
and their aftermath may be a pathological response to the retention of a sub-endothelial
apolipoprotein B, similar to a widely accepted model of atherosclerotic coronary artery disease
(Curcio 2010). As such, researchers have now found that bio-markers predictive of
cardiovascular risk (e.g., elevated homocysteine and C-reactive protein (CRP) levels) are risk
factors for AMD (Seddon 2006).

Small drusen are extremely common, with approximately 80% of the general population over 30
manifesting at least one. The depositing of large drusen (= 63?m) are characteristic of atrophic
AMD, in which this drusen causes thinning of macular tissue, experienced as blurry or distorted
vision with possible blank spots in central vision. Drusen continue to accumulate and aggregate
with advancing age; those over 75 are 16 times more likely to develop aggregated large drusen
compared to those 43-54 (Klein 2007).

Along with drusen formation, there may be deterioration in the elastin and collagen in Bruch's membrane—the barrier between the retina and the choroids—causing calcification and fragmentation.

This, coupled with an increase in a protein called vascular endothelial growth factor (VEGF), allows capillaries (or very small blood vessels) to grow up from the choroid into the retina, ultimately leading to blood and protein leakage below the macula (wet form AMD) (Friedman 2004; Bird 2010).

Other theories postulate that abnormalities in the enzymatic activity of aged retinal pigment epithelium (RPE) cells lead to the accumulation of metabolic byproducts. When the RPE cells become engorged, their normal cellular metabolism is obstructed, resulting in extracellular excretions that produce drusen and lead to neovascularization.

People who have a close relative with AMD have a 50% higher risk of eventually developing it compared to 12% for other people. Scientists believe a newly discovered genetic association will better help predict those at risk and ultimately lead to better treatments (Patel 2008).

Risk Factors of AMD

Cigarette Smoking. An increased incidence of neovascular and atrophic AMD has been consistently demonstrated among smokers (Thornton 2005;

Chakravarthy 2010).
The macular pigment (MP) optical density in 34 cigarette smokers was compared against the
MP optical density in 34 non-smokers matched for age, sex, and dietary patterns. It was found
that tobacco users had significantly less MP than control subjects. Further, smoking frequency
(cigarettes per day) was inversely related to MP density (Hammond 1996).

In a study investigating the relationship between smoking and the risk of developing AMD in
Caucasians, 435 cases with end stage AMD were compared to 280 controls. The authors
demonstrated a strong association between the risk of both dry and wet form AMD and the
amount of cigarette smoking.

More specifically, for subjects with 40 pack years (number of pack years = packs smoked per day [x] years as a smoker) of smoking, the odds ratio (probability of
the condition occurring) was 2.75 compared with non-smokers. Both types of AMD showed a
similar relation; smoking more than 40 pack years of cigarettes was associated with an odds
ratio of 3.43 for dry AMD and 2.49 for wet AMD. Stopping smoking was associated with reduced
odds of AMD. Also, the risk in those who had not smoked for over 20 years was comparable to
non-smokers. The risk profile was similar for males and females. Passive smoking exposure
was also associated with an increased risk of AMD in non-smokers (Khan 2006).

Oxidative Stress

The retina is particularly susceptible to oxidative stress because of its high consumption of oxygen, high proportion of polyunsaturated fatty acids, and exposure to visible light. In vitro studies have consistently shown that photochemical retinal injury is attributable to oxidative stress. Furthermore, there is strong evidence suggesting that lipofuscin (a photoreactive substance) is derived, at least in part, from oxidatively damaged photoreceptor outer segments (Drobek-Slowik 2007). While naturally occurring antioxidants typically manage this, environmental factors and stress can decrease circulating antioxidants. For example, levels of the endogenous antioxidant glutathione decrease as people age, making the lens nucleus and retina susceptible to oxidative stress (Babizhayev 2010).

Vitamin C, normally highly concentrated in the aqueous humor and corneal epithelium, helps absorb damaging ultraviolet radiation, protect the basal layer of the epithelium, and prevent AMD (Brubaker 2000). L-carnosine and vitamin E also mitigate oxidative stress and free-radical damage (Babizhayev 2010).

Inflammation

Injury and inflammation to the pigmented layer of the retina (retinal pigment epithelium or RPE) as well as the choroid cause an altered and

abnormal diffusion of nutrients
to the retina and RPE, possibly precipitating further RPE and retinal damage (Zarbin 2004).
Animal studies show that oxidative stress-induced injury to the RPE results in an
immune-mediated chronic inflammatory response, drusen formation, and RPE atrophy
(Hollyfield 2008).

Research has identified specific genetic changes, which can lead to an inappropriate
inflammatory response and set the stage for AMD onset (Augustin 2009). Other studies looking
at whether inflammatory markers predicted AMD risk found that higher levels of C-reactive
protein (CRP) were predictive of AMD after controlling for genotype, demographic and
behavioral risk factors (Seddon 2010; Boekhoorn 2007).

Phototoxicity

Another risk factor for AMD is phototoxicity caused by exposure to blue and
ultraviolet (UV) radiation, both of which adversely affect the functioning of RPE cells. Cultured
human RPE cells are susceptible to apoptotic cell death induced by Ultraviolet B (UVB)
irradiation. Absorption of UV light by the innermost layer of the choroid can largely prevent the
cytotoxic effect. (Krohne 2009). Exposure to sunlight without protective sunglasses is a risk
factor for AMD (Fletcher 2008).

Hypertension

A study of 5,875 Latino men and women identified a pronounced risk for wet
AMD if diastolic blood pressure was high, or if individuals had uncontrolled diastolic
hypertension (Fraser-Bell 2008). Prolonged treatment of hypertension with a thiazide diuretic,
however, was associated with a more significant incidence of neovascular AMD, possibly due to
the known phototoxic effects of thiazide diuretics (De la Marnierre 2003).

Low Carotenoid Intake

Insufficient intake of the following carotenoids is linked to AMD: lutein,
zeaxanthin, and meso-zeaxanthin. Lutein, zeaxanthin, and meso-zeaxanthin are carotenoids
present in the retina and positively affect MP density (Ahmed 2005).

Lutein and zeaxanthin help
to prevent AMD by maintaining denser MP, resulting in less retinal tearing or degeneration
(Stahl 2005). The therapeutic efficacy of lutein and zeaxanthin in AMD is significant, according
to the Lutein Antioxidant Supplementation Trial (LAST), which showed improvement in several
symptoms accompanying AMD (Richer 2004).

Low Vitamin B Intake

Several studies show that low levels of certain B vitamins are associated with an increased risk for AMD. The Women's Antioxidant and Folic Acid Cardiovascular Study
(WAFACS) in 5,442 female health professionals showed that daily supplementation with folic
acid, B6 and B12 resulted in significantly fewer AMD diagnoses compared to placebo (Christen
2009).

High Fat Intake

Higher intake of specific types of fat, rather than total fat, may be associated
with a greater risk of advanced AMD. Diets high in omega-3 fatty acids, fish and nuts were
inversely associated with AMD risk when intakes of linoleic acid (an omega-6 fatty acid) was low
(Tan 2009).
A French study found that high total fat, saturated fat and monounsaturated fat intake were all
associated with an increased risk of developing AMD (Delcourt

2007). Eating red meat 10 or
more times per week appears to increase risk for developing early AMD, while eating chicken
more than 3 times per week may confer protection against the disease (Chong 2009a).
High trans fat consumption has been linked to an increased prevalence of late (more advanced)
AMD in a study of 6,734 individuals. In the same study, olive oil consumption offered a
protective effect (Chong 2009b).
Ethnicity. Studies in the USA indicate that a higher percentage of Caucasian-Americans get
macular degeneration compared to African-Americans (Klein 2011).

Conventional AMD Treatments

Dry type macular degeneration develops gradually.
Supplementation with antioxidants, lutein
and zeaxanthin has been suggested by the National Eye Institute and others to slow the
progression of dry macular degeneration and, in some patients, improve visual acuity (Tan AG
2008).

Wet macular degeneration can develop more quickly. Patients require treatment soon after
symptoms appear. There were no effective treatments for wet macular degeneration until
recently. New drugs, called anti-Vascular Endothelial Growth Factor (anti-VEGF) agents, can
promote regression of the abnormal blood vessels and improve

vision when injected directly into
the vitreous humor of the eye (Chakravarthy 2006; Rosenfeld 2006a,b; Anon 2011b).
Photodynamic therapy, a systemic treatment used in oncology to eradicate early-stage cancer
and reduce the tumor size in end-stage cancers, has also been used to treat wet AMD
(Wormald 2007).

Anti-VEGF Medications. Macugen®, Lucentis®, Avastin®, and others are the newest
conventional treatments for wet macular degeneration.

VEGF's main role is to induce new blood vessel formation. It also functions to increase
inflammation and cause fluid to leak out of blood vessels. In wet macular degeneration, VEGF
stimulates the formation of abnormal blood vessels in the macular area of the retina. Bleeding,
leaking, and scarring from these blood vessels eventually causes irreversible damage to the
photoreceptors as well as rapid vision loss if left untreated.

All anti-VEGF medications work in a similar fashion. They bind to and inhibit the biologic activity of VEGF. By preventing VEGF's action, they effectively reduce and prevent the formation of abnormal blood vessels. They also reduce the amount of leakage and therefore reduce swelling in the macula. These actions lead to preservation of vision in patients with wet macular degeneration.

There are three anti-VEGF medications currently being used. Pegaptanib (Macugen®) selectively binds to a specific type of VEGF called VEGF 165, which is one of the most dangerous forms of VEGF (Chakravarthy 2006). Macugen® has been approved by the Food and Drug Administration (FDA) for treatment of wet AMD. It is administered via intraocular injection given every six weeks.

Ranibizumab (Lucentis®) is also FDA-approved to treat wet macular degeneration. Lucentis® inhibits all forms of VEGF. Lucentis® is administered via monthly intraocular injection.
Bevacizumab (Avastin®) is similar to Lucentis® and works to inhibit all forms of VEGF. Avastin® is currently approved by the FDA for metastatic cancer (cancer that has spread to other parts of the body). This drug is commonly used but is not approved by the FDA for wet AMD. The cost of Avastin® is approximately 90% less than the other two agents.

Since VEGF has also been associated with poor prognosis in breast cancer, Avastin® was previously used as treatment. However, the FDA to pulled approval of Avastin® for breast cancer treatment in November 2011after a review of four clinical studies (FDA 2012). These studies concluded that the drug does not prolong breast cancer patients' overall survival or slow disease progression significantly. Rigorous clinical trials for Avastin® are being performed by the National Eye Institute. Lucentis® is available free in the UK as long as patients meet certain criteria related to vision.

Although the mechanisms of action of the anti-VEGF agents are similar, the success rates between the treatments vary. When Macugen® was first approved, seventy percent of patients stabilized with no further severe visual loss

(Gragoudas 2004). Macugen®
has not been found to improve vision. Lucentis® improved on the results of Macugen®.
Ninety-five percent of Lucentis® patients kept their vision, and nearly 40% of Lucentis® patients
completing one year of treatment improved their vision to 20/40 or better (Rosenfeld 2006b).
Because Avastin® is used off-label, and its makers do not plan to seek approval of the drug for
AMD, it has not been as thoroughly investigated as either Lucentis® or Macugen® (Gillies
2006). However, many retina specialists believe that Avastin's® efficacy parallels that of
Lucentis® (Rosenfeld 2006b).

Lucentis®, Macugen®, and Avastin® are all administered via intraocular injection. In other
words, these medications are injected directly into the eye. The injections are given after the
surface of the eye has been cleansed and sterilized. Some doctors will give antibiotic drops
prior to the injection. Some form of anesthesia is usually administered. This can be given in the
form of drops or as a very small injection of anesthetic around the eye. A very fine needle is
used and the actual injection takes only a few seconds.

A fourth intraocular anti-VEGF treatment, the VEGF Trap-Eye, approved in November 2011,
appears to require fewer injections compared to Lucentis®, while still offering the same
improvements in eyesight over a one year period. In trials of more

than 2,400 patients, VEGF
Trap-Eye intraocular injections dosed every two months offered the same benefits as Lucentis®
dosing monthly (Anon 2011b).

Possible complications are retinal detachment and the development of a cataract. High
intraocular pressure usually follows the injection but generally resolves within an hour.
Possible adverse effects of intraocular injections occur in less than 1 percent of every 100
injections (Rosenfeld 2006b). When adverse effects occur, however, they can be very serious
and threatening to eyesight. One possible adverse reaction is a serious eye infection known as
endophthalmitis, an inflammation of the internal tissues of the eyeball, which sometimes leads
to loss of vision or severe damage to the eye.

Photodynamic Therapy (PDT) is a systemic treatment used in oncology by a variety of
specialists to eradicate premalignant and early-stage cancer and reduce the tumor size in
end-stage cancers. PDT involves three key components: a photosensitizer, light, and tissue
oxygen.

Photosensitizing agents are drugs that become active when light of a certain wavelength is
directed onto the anatomical area where they are concentrated. It is an approved treatment for
wet macular degeneration, and is a more widely preferred treatment

that takes advantage of
certain unique properties of subretinal neovascular vessels.

Compared with normal blood vessels, neovascular tissue appears to retain the light-sensitive
medicine used in photodynamic therapy. After the medicine, verteporfin (Visudyne®) for
example, has been injected into a peripheral vein, it can detect abnormal blood vessels in the
macula and attach itself to the proteins in the abnormal blood vessels. Laser light of specific
wavelengths, which activates photosensitive drugs like verteporfin, is focused through the eye
for about one minute.

When verteporfin is activated by the laser, the abnormal blood vessels in
the macula are destroyed. This happens without any damage to surrounding eye tissue.
Because normal retinal vessels retain very little verteprofin, the abnormal subretinal vessels are
selectively destroyed. Blood or fluid cannot leak out and damage the macula any further
(Wormald 2007).

While verteporfin PDT slowed wet AMD progression, newer anti-VEGF therapies have shown
vision improvement in many patients. Combination therapies (PDT + corticosteroid + anti-VEGF)
have shown some promise, particularly in certain classes of disease (Miller 2010).

Laser Photocoagulation

Laser photocoagulation (LP) is an effective treatment for wet type AMD. However, LP is limited to the treatment of well-defined, or “classic” subretinal neovascularization, present in only 25% of those with wet type AMD (Anon 2011a). In eligible patients, LP is effective at preventing future vision loss, but it cannot restore or improve vision.
In addition, choroidal neovascularization can recur after treatment and cause further vision loss (Yanoff 2004). LP has not worked well on atrophic (dry) AMD.

Surgery. Subretinal surgery has been attempted for AMD. Some surgeries were geared toward the removal of blood and the subretinal neovascular membrane. Another type of surgery attempted to physically displace the macula and move it onto a bed of healthier tissue. Overall, research studies show that the results of surgery are disappointing (Bressler 2004). Vision has generally not improved after surgery (Hawkins 2004). Additionally, the frequency and severity of surgical complications were generally thought to be unacceptably high.

In late 2010, the FDA approved a device called the Implantable Miniature Telescope (IMT) to improve vision in some patients with end-stage AMD. The IMT replaces the natural lens through surgery in only one eye and provides 2X magnification. The other

eye is used for peripheral
vision. In the clinical trials upon which FDA approval was based, at 1 and 2 years post-surgery,
75 percent of patients had an improvement in their visual acuity of two lines of more, 60 percent
improved their vision by three lines, and 40 percent had a four-line improvement on the eye
chart (Hudson 2008 and www.accessdata.fda.gov).

Each person may respond differently to the various conventional treatments available for
macular degeneration. From a patient's perspective, it is very important to thoroughly
understand wet macular degeneration and its treatment in order to be able to discuss a
therapeutic plan with his or her doctor. A specific treatment plan should be tailored to each
patient's needs and disease activity.

The advent of anti-VEGF therapies, for example, has been seen as a significant advancement
for patients with wet macular degeneration. It is important to speak with a specialist regarding
the benefits and side effects of anti-VEGF drugs to determine if they are appropriate for your
specific case. It should be noted that there is some speculation, which is not supported by
strong human data, that anti-VEGF macular degeneration treatments may exert systemic effects
and negatively impact vascular health by "leaking" from the eye.

It is, therefore, important to evaluate your cardiovascular health if

you are receiving anti-VEGF treatment for macular degeneration. For instance, a person who recently had a heart attack or has extensive atherosclerosis may opt to avoid anti-VEGF treatments in favor of photodynamic therapy or laser photocoagulation. Individuals receiving anti-VEGF treatments should target an optimal cardiovascular health profile, which includes low-density lipoprotein (LDL) levels below 100 mg/dL, fasting glucose between 70-85 mg/dL, etc. For more tips on supporting your cardiovascular health, read our Atherosclerosis and Cardiovascular Disease Protocol.

Emerging Options: Hormone Therapy DHEA

Research has shown that the hormone dehydroepiandrosterone (DHEA) is abnormally low in patients with AMD (Bucolo 2005). DHEA has been shown to protect the eyes against oxidative damage (Tamer 2007). Because the macula requires hormones to function, an emerging theory hypothesizes that low blood sex hormone levels cause the retinal macula to accumulate cholesterol in an attempt to produce its own hormones (Dzugan 2002).

The accumulation of cholesterol in macula may lead to the production of pathologic drusen and subsequent macular degeneration. An inverse association of female hormone with neovascular AMD was observed with current and former use of hormone replacement therapy among

Caucasian and Latino
women (Edwards 2010). Restoring optimal hormone balance with bioidentical hormones may be
an effective new treatment for both men and women. Clinical studies are underway to test this
hypothesis and possible hormonal treatment options.

Melatonin

Melatonin is a hormone and strong antioxidant that scavenges free radicals. Several
studies have shown that many areas of the eye have melatonin receptors (Rastmanesh 2011;
Lundmark 2006). In a clinical study, 100 patients with dry or wet AMD received 3 mg of
melatonin at bedtime. The treatment prevented further vision loss. After six months, visual acuity
had not diminished and the majority of patients had reduced pathologic macular changes upon
examination (Yi 2005).

Dietary Considerations

Soy. Soy contains the phytonutrient genistein, which has documented antiangiogenesis
properties postulated to be the result of inhibiting VEGF (Yu 2010). This property of inhibiting
blood vessel growth is important in limiting abnormal ingrowth of choroidal blood vessels. In
mice, genistein inhibited retinal neovascularization and expression of VEGF (Wang 2005).
Food rich in Omega-3 fatty acids. Oily fish (e.g., salmon, tuna, and

mackerel) as well as flax
seeds are important sources of omega-3 fatty acids, essential for protection against macular
degeneration and other diseases (Landrum 2001). A meta-analysis found that patients with a
high dietary intake of omega-3 fatty acids had a 38% lower risk of late (more advanced) AMD.
Additionally, an association was observed between eating fish two times a week and having a
reduced risk of both early and late AMD (Chong 2008).

Macular Pigments: Lutein, Zeaxanthin, and Meso-Zeaxanthin

The relationship between the density of macular pigment (MP) and the onset of AMD is well
established. The MP is composed principally of three carotenoids: lutein, zeaxanthin, and
meso-zeaxanthin. They represent roughly 36, 18, and 18 percent, respectively, of the total
carotenoid content of the retina. They are found within the macula and surrounding tissues,
including blood vessels and capillaries which nourish the retina (Rapp 2000).

Lutein, zeaxanthin and meso-zeaxanthin ensure proper functioning of the macula by filtering out
harmful ultraviolet light and acting as antioxidants (Beatty 2000; Kaya 2010). During the aging
process, there is a decrease in levels of lutein and zeaxanthin; low levels of MPs are linked to
AMD (Johnson 2010). An autopsy study on donated eyes found that

levels of all three carotenoids were reduced in those with macular degeneration compared to control subjects. The most significant finding, however, was the sharp decrease in meso-zeaxanthin in the macula of macular degeneration subjects (Bone 2000).

This postmortem study helped confirm other studies indicating the importance of all three carotenoids in maintaining the structural integrity of the macula (Krinsky 2003). These carotenoids protect the macula and the photoreceptor cells beneath via their antioxidant properties and light-filtering capabilities (Landrum 2001).

Intake of lutein and zeaxanthin is an important preventative measure, but may also reverse the degeneration process when it is ongoing (Richer 2004). Because lutein and zeaxanthin have the tissue-specific characteristic of all carotenoids, their natural tendency is to concentrate in the macula and retina. Consumption of foods rich in these substances is especially important, as they have a direct effect on macular pigment density — the denser the pigment, the less likely a retinal tear or degeneration will occur (Stahl 2005). Fruits with a yellow or orange color (e.g., mangoes, kiwis, oranges, and vegetables of the dark green leafy, orange and yellow varieties) are sources of lutein and zeaxanthin (Bone 2000).

Unlike lutein and zeaxanthin, meso-zeaxanthin is not found in the

diet, but is needed to maintain youthful macular density (Bone 2007). Patients with macular degeneration have been shown to have 30% less meso-zeaxanthin in their macula compared to individuals with healthy eyes (Quantum Nutritionals, data on file). When taken as a supplement, meso-zeaxanthin is absorbed into the blood stream and effectively increases macular pigment levels (Bone 2007).

Targeted Nutritional Interventions

Anthocyanidins and Cyanidin-3-Glucoside (C3G). C3Gs are critical components of bilberry as well as being powerful antioxidants (Amorini 2001; Zafra-Stone 2007). Positive results have been noted in many animal studies and some human studies using bilberry for macular degeneration as well as other eye disorders including diabetic retinopathy, retinitis pigmentosa, glaucoma, and cataracts (Fursova 2005; Milbury 2007). C3G has been shown to improve night vision in humans by enabling the rods in the eye responsible for night vision to resume functioning faster (Nakaishi 2000).

In animal cells, C3G regenerated rhodopsin (the retinal complex that absorbs light) (Amorini 2001). The anthocyanidins in bilberry decrease vascular permeability by interacting with blood vessel collagen so as to slow down enzymatic attack on the blood vessel wall. This may prevent the leakage from capillaries

that is prevalent in
neovascular AMD. Studies also show that bilberry increases oxidative stress defense
mechanisms in the eyes (Milbury 2007). There may be additional benefits by adding vitamin E
(Roberts 2007).

C3G, which is highly bioavailable, enhances other functions in the body (Miyazawa 1999; Tsuda
1999; Matsumoto 2001). Its potent antioxidant properties protect tissues against DNA damage,
often the first step in cancer formation and aging of tissues (Acquaviva 2003; Riso 2005).
C3G protects endothelial cells against peroxynitrite-induced endothelial dysfunction and
vascular failure (Serraino 2003).

In addition, C3G fights vascular inflammation by inhibiting inducible nitric oxide synthase (iNOS) (Pergola 2006). At the same time, C3G upregulates
activity of endothelial nitric oxide synthase (eNOS), which helps maintain normal vascular
function (Xu 2004). These effects on blood vessels are especially important in the retina, where
delicate nerve cells depend on the single ophthalmic artery for their sustenance.
In animal models, C3G prevents obesity and ameliorates blood sugar elevations (Tsuda 2003).
One way it does this is by increasing gene expression of the beneficial fat-related cytokine
adiponectin (Tsuda 2004). Diabetics, of course, are predisposed to

severe eye problems
including blindness from elevated blood sugar levels.

C3G helps induce apoptosis (programmed cell death) in a number of human cancer lines, an
important step in cancer prevention (Fimognari 2004; Chen 2005). In a similar fashion (but via a
different mechanism), C3G stimulates rapidly proliferating human cancer cells to differentiate so
they more closely resemble normal tissue (Serafino 2004).

Finally, it was discovered that C3G is neuroprotective in experimental cellular models of brain
function, helping to prevent the negative effects of the Alzheimer's-related protein amyloid beta
on brain cells (Tarozzi 2010).

Grape Seed Extract

Grape seed extract, a bioflavonoid, is a potent antioxidant. Plant-derived
bioflavonoids are readily assimilated into our body when consumed. Bioflavonoids appear to
protect retinal ganglion cells (Majumdar 2010). Studies conducted in fruit flies have revealed
that grape seed extract attenuates the aggregation of pathologic proteins, which suggests a
protective effect against macular degeneration and neurodegenerative disorders. Accordingly,
fruit flies administered grape seed extract exhibited improved eye health (Pfleger 2010). Similar
experiments in diabetic animals indicate that grape seed extract

limits the ocular blood vessel
damage seen in diabetic retinopathy (degradation of the retina), which shares some
pathological characteristics with AMD (Li 2008).

Compelling laboratory evidence demonstrates that grape extracts can inhibit angiogenesis in
human cells (Liu 2010). This suggests that grape seed extract may suppress the aberrant blood
vessel growth observed in wet AMD.

Resveratrol

Resveratrol is a potent polyphenolic antioxidant compound produced by grapes
and other plants for protection against pathogens. In humans, it exerts a broad range of
physiologic effects when ingested orally. Several studies have demonstrated cardioprotective
properties of resveratrol, including endothelial protection and attenuation of oxidized-LDL-induced vascular damage (Rakici 2005; Lin 2010). In addition, emerging evidence
indicates that resveratrol may combat macular degeneration and promote eye health via several
mechanisms. In an animal model, resveratrol was able to stave off diabetes-induced vascular
lesions (Kim 2011).

Moreover, this same study showed that resveratrol was able to dampen
VEGF signaling in mouse retinas, a key pathologic feature of AMD. Another study corroborated
these results by showing that resveratrol inhibited angiogenesis and

suppressed retinal
neovascularization in mice prone to develop macular degeneration due to a genetic mutation
(Hua 2011). Also, several laboratory experiments have suggested additional protective
mechanisms of resveratrol in macular degeneration, including protecting retinal pigment
epithelial cells from hydrogen peroxide-induced oxidative stress and light damage (Kubota
2010; Pintea 2011).

Given these exciting initial findings regarding resveratrol and macular degeneration, along with
its stellar track record in a variety of other conditions, Life Extension believes that individuals
with AMD (especially the “wet” variety) may benefit from supplementation with resveratrol.

Ginkgo Biloba. Ginko biloba improves microcapillary circulation in the eye and slows
deterioration of the macula (Thiagarajan 2002). By inhibiting platelet aggregation and regulating
blood vessel elasticity, ginko biloba improves blood flow through major blood vessels and
capillaries. Ginkgo is also a powerful antioxidant (Mahadevan 2008).

Glutathione and Vitamin C. Glutathione and Vitamin C are antioxidants found in high
concentrations in healthy eyes and in diminished quantities in the eyes of AMD patients. Vitamin
C aids glutathione synthesis in the eye. When combined with cysteine, an amino acid
antioxidant, cysteine remains stable in aqueous solutions and is a

precursor to glutathione
synthesis. Vitamin C is important because it absorbs ultraviolet radiation, which contributes to
cataracts (Tan 2008). Topical Vitamin C inhibited angiogenesis in an animal model of
inflammatory neovascularization (Peyman 2007).

L-Carnosine

L-Carnosine is a naturally occurring antioxidant and anti-glycation agent. Studies
have shown that carnosine inhibits lipid peroxidation and free radical-induced cellular damage
(Guiotto 2005). Topically applied N-acetyl-carnosine prevented light-induced DNA strand breaks
and repaired damaged DNA strands (Specht 2000), as well as improved visual acuity, glare and
lens opacification in animals and humans with advanced cataracts (Williams 2006; Babizhayez
2009).

Selenium

Selenium, an essential trace mineral, is a component of the antioxidant enzyme
glutathione peroxidase, important in slowing the progression of AMD and other eye disorders
including cataracts and glaucoma (Head 2001; King 2008). In mice, increased expression of
glutathione peroxidase protected against oxidative-induced retinal degeneration (Lu 2009).
Coenzyme Q10 (CoQ10). CoQ10 is an important antioxidant that

may protect against free
radical damage within the eye (Blasi 2001). Mitochondrial DNA (mtDNA) instability is an

important factor in mitochondrial impairment culminating in age-related changes and pathology.
In all regions of the eye, mtDNA damage is increased as a consequence of aging and
age-related disease (Jarratt 2010). In one study, a combination of antioxidants including CoQ10,
acetyl-L-carnitine, and omega-3 fatty acids improved the function of mitochondria in retinal
pigment epithelium and subsequently stabilized visual functions in patients affected by early
AMD (Feher 2005).

Riboflavin, Taurine, and Lipoic Acid

Riboflavin (B2), taurine, and R- lipoic acid are other
antioxidants utilized to prevent AMD. Riboflavin is a B complex vitamin that reduces oxidized
glutathione and helps prevent light sensitivity, loss of visual acuity, as well as burning and itching
in the eyes (Lopez 1993). Taurine is an amino acid found in high concentrations in the retina. A
taurine deficiency alters the structure and function of the retina (Hussain 2008). R- lipoic acid is
considered a “universal antioxidant” because it is fat and water soluble. It also reduces choroidal
neovascularization in mice (Dong 2009).

B Vitamins

Recent advances surrounding the causes of AMD have unearthed

shared risk
factors with cardiovascular disease (CVD) as well as similar underlying mechanisms, particularly
elevated biomarkers of inflammation and CVD including C-reactive protein (CRP) and
homocysteine (Vine 2005). Researchers have identified that elevated levels of homocysteine,
and low levels of certain B vitamins (critical to the metabolism of homocysteine), are associated
with an increased risk of AMD and vision loss in older adults (Rochtchina 2007). A strong study
found that supplementing with folic acid, B6, and B12 can significantly reduce the risk of AMD in
adults with cardiovascular risk factors (Christen 2009). The data, along with additional
confirmatory studies, have convinced physicians to recommend B vitamin supplementation in
patients with AMD. A study in more than 5000 women indicates that including folic acid (2.5
mg/day), B6 (50 mg/day) and B12 (1 mg/day) in the diet may prevent and reduce the risk of
AMD (Christen 2009).

Supplement Recommendations from the Age-Related Eye Disease Study (AREDS)

The largest and most important study of nutritional supplements in AMD is the Age-Related Eye
Disease Study (AREDS). The AREDS demonstrated a reduction in the risk of progression to
end-stage AMD when vitamins and zinc supplementation were given

to patients with advanced
forms of the disease. Thousands of patients were followed for over six years. The AREDS
revealed significant improvements for patients with AMD and recommended antioxidants plus
zinc for most patients with AMD, except those with advanced cases in both eyes. The AREDS
formula consists of the following, which is to be taken daily: Vitamin A (Beta Carotene), Vitamin
C, Vitamin E, Zinc and Copper (Fahed 2010).

DHA and EPA

An 8-year trial of 2924 eligible AREDS AMD participants found that independent
of AREDS supplementation, higher intakes of DHA and EPA were associated with a lower risk
for progression to advanced AMD (Chiu 2009).

Zinc. Following the revealing data found from the AREDS, additional research on zinc has
shown significant activity in treating AMD, specifically the dry form of the disease. In a clinical
study, a zinc-monocysteine supplement significantly improved visual acuity and contrast
sensitivity compared to placebo (Newsome 2008).

Summary

There has been limited success within conventional medical treatment protocols to restore lost
eyesight from either form of AMD. Leading researchers are

documenting the benefits of more
holistic approaches to AMD. Patients are encouraged to increase physical fitness, improve
nutrition (including a reduction in saturated fats), abstain from smoking, and protect their eyes
from excessive light. Dietary supplementation with trace elements, carotenoids, antioxidants,
and vitamins is recommended for improving overall metabolic and vascular functioning. Early
screening and patient education offer the most hope for reducing the debilitating effects of the
disease.

Life Extension Suggestions

Lutein: 10 – 20 mg daily
Zeaxanthin: 3 – 8 mg daily
Astaxanthin: 6 – 12 mg daily
Cyanidin-3-glucoside (C3G): 2 – 5 mg daily
Methyltetrahydrolfolate (MTHF): 1000 – 2000 mcg daily
Vitamin B6 (as pyridoxal 5?-phosphate): 100 – 200 mg daily
Vitamin B12 (as methylcobalamin): 1 – 5 mg daily
Beta carotene: 25?000 IU daily
Vitamin C: 1000 – 2000 mg daily
Natural Vitamin E: 100 – 400 IU alpha-tocopherol and 200 mg gamma-tocopherol daily
Zinc: 45 – 60 mg daily
Copper: 2 mg daily
R-Lipoic acid: 300 – 900 mg daily
Selenium: 200 – 400 mcg daily
Taurine: 1000 mg daily

CoQ10 (as ubiquinol): 100 – 300 mg daily
N-acetyl-carnosine eye drops: 1 – 2 drops, 1-4 times daily
Omega-3 fatty acids (from fish): 2000 – 6000 mg daily
Ginkgo biloba (standardized extract): 120 – 240 mg daily
Grape extract: 150 – 300 mg daily
Bilberry (standardized extract): 100 – 200 mg daily
Soy isoflavones: 135 – 270 mg daily
The following blood testing resources may be helpful:
Male and Female Panel
Omega Score®

Coenzyme Q10 (CoQ10)
In addition, the following pharmaceutical options should be discussed with your physician:
Lucentis®
Macugen®
Avastin®

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before starting any diet,
exercise or supplementation program, before taking any medication, or if you have or suspect
you might have a health problem. You should not stop taking any medication without first
consulting your physician.

Life Extension

faim.org
Stop Degenerative Eye Disease with Coconut Oil
Dr. Bruce Fife
Can you avoid age-related degenerative eye disease? Our eyesight naturally diminishes to
some extent as we age, but regardless of our age, our eyes should provide us with good service
for a lifetime. New research reveals that good eyesight can be maintained for life and
age-related eye disorders can be stopped dead cold and possibly even reversed. The key to
maintaining good vision depends on your diet.
The body has an amazing ability for self-repair. We get a cut, a bruise, break a bone, or suffer
some other injury, the body knows exactly what to do to repair the damage. In time, the cut is
completely healed, damaged blood vessels in a bruise are repaired, and broken bones are
fused back together, in many cases the repair is so complete that there is little or no trace that
an injury ever occurred.
Like other tissues, peripheral nerves throughout our bodies have a

high capacity for
regeneration after injury, however injury to nerve cells within the central nervous system do not.

In fact, for many years it was believed that brain tissue, could not be repaired or regenerated.
Once an injury occurred, the neurons, or brain cells, were gone forever. It was thought that the
brain cells we were born with were all that we would ever have. We now know that under the
right conditions brain cells can be regenerated and new brain cells do grow and develop just like
other cells throughout the body.
The brain contains two types of nerve cells: glia and neurons. Glia are the most numerous and
provide the structural support that holds all the brain cells together. They also serve many other
important functions, but they don’t relay signals; that is the function of the neurons. Neurons
transmit signals by means of electrochemical impulses which allow us to think, move, and
function in our environment.
Neurons consist of three basic parts: (1) the cell body (2) the axon, a long cable-like projection
of the cell that carries electrochemical impulses along the length of the cell, and (3) dendrites or
nerve endings that branch out like the branches on a tree.
Messages are passed on by relaying
nerve impulses from the axon of one neuron to the dendrite of other. If you step on a nail, a
message is relayed in this manner almost instantaneously up to the

brain where it is interpreted
as pain and the appropriate reaction can be quickly initiated.
The eyes are extensions of the brain and are also composed of glia and neurons. The light
sensitive portion of the eye is the retina, which lines the inside of the eyeball. When light hits the
retina it sends nerve impulses through the retinal ganglion cells (RGCs) to the brain. RGCs
have long axons that join together like threads in a piece of rope to form the optic nerve.
Any damage that occurs to the RGCs or optic nerve can cause visual impairment and if serious
enough, complete blindness. When injured, retinal ganglion cells generally do not have the
ability for self repair and eventually die, eliminating any chance for regeneration. Injury to the
RGCs or the optic nerve leads to lifelong visual impairment.
The most common degenerative eye diseases that involve damage to the retina and optic nerve
are glaucoma, macular degeneration, and diabetic retinopathy. These three conditions cause
the vast majority of irreversible vision loss in people living in affluent countries.
Glaucoma is believed to be caused, in part, by abnormal pressure within the eye. The eyeball is
filled with a viscous fluid that helps maintain the shape of the eye and circulate nutrients. This
fluid is constantly entering and leaving the eyeball. If it enters faster than it exists, the pressure
within the eye builds up damaging the retina and optic nerve.

Treatment is focused on lowering
fluid pressure with the use of medicated eye drops, drugs, laser therapy, surgery, or some
combination of these. Unfortunately, all these procedures have the potential for producing
unwanted side effects or injury.
Diabetic retinopathy is caused by inadequately controlled diabetes. High blood sugar causes the
blood vessels feeding the retina to degenerate and become leaky. This distorts the retina

leading to permanent damage. Besides trying to get blood sugar under control, treatment may
involve laser surgery to burn or cauterize damaged blood vessels to keep them from leaking.
This permanently scars the retina, but may prevent or slow further vision loss.
Macular degeneration is the slow destruction of the macula – the portion of the retina that is
needed for sharp, central vision. In macular degeneration central vision is lost first and gradually
progresses out affecting side or peripheral vision. Vision loss is permanent. The cause of
macular degeneration is unknown and there is no effective treatment. When macular
degeneration occurs later in life it is usually referred to as age-related macular degeneration to
distinguish it from other forms that may be inherited and appear early in life.
For many years the inability of damaged retinal neurons and optic nerve to regrow was
accepted almost as a “law of nature,” and on the clinical level, retinal

injury was seen as being
irreversible and corresponding vision loss was permanent. Today medical researchers are
starting to unlock the secrets of neuronal regeneration. Under the right conditions, injury to the
retina and optic nerve can be healed. A growing number of studies over the past two decades
have demonstrated that mature RBCs can be transformed into an active regenerative state
allowing these neurons to survive injury and to regenerate axons in the injured optic nerve.
Almost all clinical studies using drugs as a means to protect the retina, optic nerve, and other
components of the eye have failed. However, a special group of naturally occurring proteins
called brain-derived neurotrophic factors (BDNFs) show great promise. BDNFs play a key role in
regulating survival, growth, and maintenance of neurons. They help support the survival of
existing neurons and encourage the growth and differentiation of new neurons.
Normally, injury to the optic nerve induces a rapid die-back of the axons leading to retinal
ganglion cell death. However, when an adequate amount of BDNFs are present, the effects of
injury are diminished and RGCs can be repaired or regenerated.
Animal studies have shown that after severing the optic nerve in adult rats, retinal ganglion cells
progressively degenerate until, after two months, a residual population of only about 5 percent
of these cells survive. When BDNFs are present, however, survival

rate significantly increases.
For example, in one study researchers severed the optic nerves in a group of rats.
The injury to the optic nerve caused a rapid, progressive degeneration of the axons and death
of the RGCs. After 3 weeks, only 10 percent of the RGCs survived. After 5 weeks, the number
dropped to 8 percent, and by the 7th week only 5 percent remained. In a second group of rats
BDNFs was injected into their eyes before the optic nerve was severed. In this group, two to
three times as many RGCs survived compared to untreated controls.1
Studies show that after severing the optic nerve, BDNFs not only protect RGCs from dying, but
promote regrowth of the axons. RGCs sprout new axons, elongate, and form functional
connections with other neurons. In lab animals that have had their optic nerves severed, BDNFs

allowed them to recover the ability of light-dark discrimination.2 In essence, researchers have
been able to restore partial sight to blind mice.
We always have some BDNFs circulating in our central nervous system that helps protect our
brains and eye from degeneration. However, people with diabetic retinopathy, glaucoma, and
macular degeneration have a deficiency of BDNFs, which makes them more susceptible to
neurodegeneration and visual problems.3-4
BDNFs provide the potential for preventing degenerative vision loss

and possibly even restoring
lost vision. In animal studies, BDNFs can be injected directly into the eyeball to boost levels of
these protective proteins. Clinically, this process is not feasible because in order to maintain
therapeutic levels of BDNFs, multiple injections are needed over a period of time to maximize
the benefit.5
Fortunately, BDNFs are produced naturally in our bodies. Using this fact, another approach to
raise BDNFs to therapeutic levels is to boost the body's own production of these protective
proteins. This can be done quite simply through diet.
The production of BDNFs is stimulated by the presence of ketones. Ketones are produced from
fatty acids stored in the body and provide an alternative to glucose as a source of fuel for the
brain. Normally, our cells, including our brain cells, use glucose as their primary source of fuel.
Most the glucose in our bodies comes from carbohydrates in our foods. When we eat a meal,
carbohydrates are converted into glucose and released into our bloodstream. Between meals or
when we don't eat carbohydrates, blood glucose levels fall. Our cells need a continual supply of
energy to function, so when this happens, fat stores in the body are mobilized and fatty acids
are released into the bloodstream. Our cells can use fatty acids for fuel just like they do glucose.
The brain, however, cannot use these fatty acids and must have an

alternative source of fuel.
Some of these fatty acids are converted into ketones, which are readily used as fuel by neurons.
Ketones not only supply the brain with energy, but also trigger the synthesis of BDNFs.
One way to increase the body's levels of BDNFs is to eat a low-carb diet. A low-carb diet keeps
blood glucose levels low, which causes the release of fatty acids and the production of ketones.
A very low-carb or ketogenic diet stimulates greater ketone production and higher BDNF levels.
Theraputic levels of BDNFs can be attained and maintained for an indefinite period of time on a
very low-carb diet.
Another way to raise ketones, and consequently BDNFs, is by eating coconut oil. Coconut oil is
composed predominately of the unique group of fatty acids known as medium chain fatty acids
(MCFAs). When consumed, a significant proportion of these MCFAs are automatically converted
into ketones, regardless of blood glucose levels. You can raise blood ketone and BDNF levels
simply by adding coconut oil into your daily diet.

If you eat enough coconut oil, you can raise blood levels of BDNFs to therapeutic levels. This
would require the daily consumption of 3 to 6 tablespoons of coconut oil daily. A smaller amount
would be needed if you combine coconut oil with a very low-carb diet.
If you want to preserve your vision and protect yourself from

encountering age-related
degenerative eye disorders that affect the retina and optic nerve, your safest and most effective
approach would be to incorporate coconut oil into your daily diet. If you have already
experienced some vision loss due to glaucoma, macular degeneration, or diabetic retinopathy,
combining coconut oil with a low-carb diet can help you prevent any further vision loss and
possibly even regain some of your lost vision.
http://www.faim.org/stop-degenerative-eye-disease-with-coconut-oil
Intravitreal injections of neurotrophic factors
survival of axotimized retinal ganglion cells
reduced levels of brain derived neurotrophic factor
bdnf impairment is associated with age-related
patterns of retinal ganglion cell survival

Vitamin B9 (folate)
High homocysteine levels (perhaps above 8 ?mol/L) appear to increase age-related macular
degeneration. Many people (60% of the population, and 90% of people with depression) can’t
use regular folate well which may contribute to high homocysteine levels. Taking methylfolate
(methyl version of vitamin B9) may increase folate blood levels 700% higher than synthetic
folate and may reduce homocysteine levels much lower. Lowering homocysteine may also
reduce depression, anxiety, dementia, bipolar disorder, schizophrenia, cardiovascular disease,
congestive heart failure, stroke, migraines, and hearing loss.

What else lowers homocysteine-
vitamin B6, vitamin B12 (methylcobalamin may be best), betaine (TMG), vitamin B2, and

magnesium
n-acetyl L-cysteine (NAC)
S-adenosylmethionine (SAMe)

taurine
green vegetables, especially dark green leafy vegetables
oranges
beans
exercise

What to avoid
the prescription drugs cholestyramine, colestipol, fenofibrate, levodopa, metformin,
methotrexate, niacin, nitrous oxide, pemetrexed, phenytoin, sulfasalazine
red meat and dairy products
smoking
coffee
alcohol consumption
advancing age
obesity
Reduced B Vitamin Therapy in MTHFR C677T/A1298C Patients with Major Depressive Disorder
– Clinical Response Correlates with Homocysteine Reduction: A Double-Blind,
Placebo-Controlled Study
Arnie Mech and Andrew Farah
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glaucoma
test 3-5yrs if 40-60yrs old
1 to 2 if over 60
tonometry (eye pressure test) AND ophthalmoscopy
eye pressure may not be enough
generic PGAs work

glaucoma- 3yrs of birth control pill usage doubles
glaucoma risk factors, such as African heritage, diabetes, heart disease, high blood pressure,
hypothyroidism or retinal problems…a history of smoking, early menopause (prior to age 45) or
long-term use of corticosteroids…a family history of glaucoma…and those who are age 60 or
older.

Cataracts

Microstents implanted during cataract surgery lower glaucoma deterioration by 50%.

MSM or DMSO may improve the absorption of medicinal eyedrops.

Cineraria eyedrops may heal cataracts early on, & one drop a day may prevent.

N-acetyl carnosine may also reverse cataracts.

Bilberry & pycnogenol may also help cataracts.

Lecithin liposomal vitamin C & vitC in foods may reduce cataracts.

Onions, garlic, cabbage, broccoli (especially sprouts), cauliflower, greens all may help cataracts by raising glutathione.

N-acetylcysteine (NAC) & Alpha lipoic acid (ALA)

NAC & ALA helps improve glutathione effectiveness for the eye.

only test needed is blood pressure, heart rate, no infection
multifocal replacements up to $4000 out of pocket & increased
repeate surgery, halos, & glare
get toric lens if astigmatism or irregular cornea

on naturally.
3.) Almonds to Improve Your Vision Naturally
Almond is a well known remedy for eyesight. It contains vitamin E, antioxidants and rich omega
3 fatty acids. Almond helps in enhancing concentration and memory. It is an effective and simple
remedy to improve vision naturally. Soak 5-10 almonds in a glass of

water. Leave it overnight
and then in the next morning, peel the skin of almonds and grind them. Make a fine paste and
consume them with a glass of warm milk. Do this remedy on a regular basis for a few months for
best effects.

4.) Indian Gooseberry to Improve Your Vision Naturally

Gooseberry is the most effective home remedy for weaker eyesight. It works as a most effective
herb for low vision. It is packed with a rich source of vitamin C. Vitamin C helps to maintain the
functioning of retinal cells. It contains several nutrients and antioxidants. You can eat
gooseberry in any form such as capsule, jam, tablet, juice or powder. Mix 2-3 teaspoons of
Indian gooseberry juice in a half cup of water. Drink this at least twice in a day for better results.

5.) Eat Healthy to Improve Your Vision Naturally

Healthy diet is an important factor for improving natural vision. If you are not taking healthy and
nutritious diet, it will affect your eyesight. Start consuming a healthy and balanced diet because
it plays an important role to improve your eyesight. You must have these useful fruits and herbs

in your daily diet routine such as eggs, milk, Eat spinach, carrot juice, blueberries, broccoli,
sweet potato, dry fruits, salad, green vegetables, fruits, fish oil and lemon etc. Consult with your
nutrition expert, to set a diet according to your problem.

6.) Bilberry to Improve Your Vision Naturally

Bilberry is the best remedy for weak eyesight and night blindness.

Bilberry contains excellent
antioxidants, which helps to stimulate blood flow of human body. It increases retinal pigments
and strengthens blood vessels. Which protects your eyes from harmful contents. Bilberries
protect from glaucoma and cataracts. Eat about one half cup of ripe bilberry fruits everyday to
improve your vision.

7.) Sunning And Palming to Improve Your Vision Naturally

Sunning and palming is also a good home remedy for weaker eyesights. It is a beneficial
method to use for reactivating your eye lens muscles. Sunning and palming your eyes will
provide benefit to your eyes. This method helps to relax your eyes. For sunning, you have to
allow the sun to heat your eyes directly. Do this for few minutes regularly. For palming, you have
to rub your hands together to generate heat. After that, you have to close your eyes, then cover
your eyes slightly through your hands, it will give you relaxation on your eyes. Do this remedy
regularly.

8.) Carrot to Improve Your Vision Naturally

Carrot is one of the most effective remedy for low vision and weaker eyesight. It contains rich
vitamins and nutrients especially phosphorous, iron, calcium and vitamin A. You can drink one
glass carrot juice regularly or you can also eat carrots as a salad. Eat or drink carrot regularly, it
will help to improve your eyesight.

9.) Barefoot Walking to Improve Your Vision Naturally
Walking is the most important part of life. It helps us to maintain our health. People must include
walking or barefoot walking into their daily routine. Barefoot walking on a green grass in the
morning will help you to get improvement in your eyesight effectively. If you want to gain your
vision power again, then start barefoot walking regularly.
10.) Fennel to Improve Your Vision Naturally
Fennel is also known as the best remedy for low vision or weaker eyes. Fennel is also known as
the herb of eyesight. It contains antioxidants and nutrients which helps to get healthy eyes and
assists to improve our weak eyesight. Take one cup of fennel, one cup of almond and add some
sugar. Then blend these ingredients and make a powder of it. Eat one teaspoon of powder with
a glass of milk before sleep. Use this remedy at least 2-3 months on a regular basis and see the
world around you without the need of glasses.
Goji berries help eyesight.

Astaxanthin
Astaxanthin is an algae extract that is an anti-inflammatory/painkiller, prevents sunburn (taken
internally), and helps eye health. Astaxanthin needs to be taken with a meal with fat or veg oil
to be absorbed. Astaxanthin can help prevent:
Age-related macular degeneration (ARMD)
Cataracts
Inflammatory eye diseases (i.e., retinitis, iritis, keratitis, and scleritis)

Retinal arterial occlusion and venous occlusion
Cystoid macular edema
Diabetic retinopathy
Glaucoma
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https://endalldisease.com/healing-eyesight-vision-loss-with-red-light-therapy/
This review shows that infrared light therapy treats both dry & wet macular degeneration
successfully. It also may help the eye recover from the shots.

It also explains just why refined & heated veg oils that have polyunsatured fat cause AMD
(unheated & unrefined oils are healthy- especially organic extra virgin olive oil from one
country).
And it includes a review finding there may not be an eye disease that near infrared therapy
doesn’t seem to help.
Here’s one of the best bulbs:
https://www.therabulb.com/blogs/test/therabulb-introduces-industry-first-300-watt-near-infrared-i
ncandescent-bulb
12 min 3x a day at 20 inches or 9 min at 15 ininches (unless too hot!)

https://endalldisease.com/healing-eyesight-vision-loss-with-red-light-therapy/
This review shows that infrared light therapy treats both dry & wet macular degeneration successfully. It also may help the eye recover from the shots.
It also explains just why refined & heated veg oils that have

polyunsatured fat cause AMD (unheated & unrefined oils are healthy-
especially organic extra virgin olive oil from one country).

And it includes a review finding there is not an eye disease that near infrared therapy doesn't help.
Here's one of the best bulbs:
https://www.therabulb.com/blogs/test/therabulb-introduces-industry-fir
st-300-watt-near-infrared-incandescent-bulb
12 min 3x a day at 20 inches or 9 min at 15 ininches (unless too hot!)

At the 1:13 minute mark is the information on macular degeneration. At the start is the information on the cause of all heart disease. His research lines up with my research and what I've found is the cause:
https://www.youtube.com/watch?v=Y_09DnSGetE
Here's doctor who's done months of research to find the origins of macular degeneration.

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This review shows that infrared light therapy treats both dry & wet macular degeneration successfully. It also may help the eye recover from the shots.
It also explains just why refined & heated veg oils that have polyunsatured fat cause AMD (unheated & unrefined oils

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He’s found what appears to be the cause of almost all macular degeneration-:
Refined oils (my last email showed they have mercury & formaldehyde from heating) & any oil with polyunsaturated fat that is cooked (formaldehyde) which is all except unrefined coconut,

unrfinede macademia nut, & unrefined MCT oil has a smoke point of 260 degrees so ok for heating but not frying (it also increases metabolism & energy & weight loss).
Peanut oil has the least polyunsaturated of all the oils used for potato
chips.
Refined grains (bleached white wheat flour, white rice) have mercury added
Refined sugar (in contrast organic raw honey causes weight loss).
Transfatty/hydrogenated oils
https://www.youtube.com/watch?v=Y_09DnSGetE

organic extra virgin olive oil from one country).
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Here’soneofthebestbulbs:https://www.therabulb.

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https://endalldisease.com/healing-eyesight-vision-loss-with-red-light-therapy/
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Infrared therapy & the eyes

The only effective treatment for one form of macular degeneration that brings back vision-
https://articles.mercola.com/sites/articles/archive/2017/11/12/photobior
light-therapy.aspx

Infrared & red light together in the red heat lamp is far more effective than the above red light alone because the infrared at 820nm that heals the eye penetrates much better than red light 670nm alone.

The heat lamp may also warm the eye (closed) enough to help kill the bacteria/virus/fungal infection inside.

Avoid blue light except first thing in morning to prevent eye damage https://articles.mercola.com/sites/articles/archive/2020/07/13/light-therapy-can-save-your-eyes.aspx

Prevention macular degeneration https://articles.mercola.com/sites/articles/archive/2018/12/15/is-macular-degeneration-preventable.aspx

What is needed to not have to inject the drugs: https://www.healthline.com/health/eye-health/wt-macular-degeneration-treatment-breakthroughs

Pharmaceutical grade DMSO may take antibiotics throughout the eye to help kill the infection and to prevent it from spreading to the brain. It may also take the anti-VEGF drugs that are normally injected and take them into the eye instead.

Rojas JC, Gonzalez-Lima F. Low-level light therapy of the eye and brain. Eye and Brain.
2011;3:49–67.
J Clin Laser Med Surg. 2001 Dec;19(6):305-14.

https://saunaspace.com/debunk-near-infrared-light-cataract-connection/

http://www.sciencealert.com/near-infrared-light-heals-eyes

Category

1. Uncategorized

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biggs