

Nutrition and the eye

Dry eye and the role of nutrition



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Module 6 Part 6
Nutrition and the Eye series

About the author

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Dry eye is one of the most common complaints seen by optometrists. Epidemiological studies have found a prevalence of about 15% in patients over the age of 50, with this prevalence increasing with age¹⁻³. As a result, with the changing demographics of Western civilisations, we are seeing increasing numbers of dry eye patients in our practices.

Our understanding of dry eye disorders has improved dramatically in the past several years. This increased understanding has enhanced our ability to diagnose and treat patients who have these traditionally challenging conditions. This article looks at the natural history, diagnosis, and treatment of dry eye disorders – beginning with the underlying mechanisms.

Classifying dry eye disorders

Central to virtually all dry eye disorders is a loss of water from the tear film, which increases its osmolarity (concentration) above the normal limit of 311mOsm/L⁴. Tear film osmolarity increases when water is lost from the tear film, while solutes, such as sodium and potassium, are not. This loss of water and increase in osmolarity may result from any condition

which either decreases tear production or increases tear evaporation (Figure 1).

Increased tear osmolarity is the link between changes in the lacrimal glands and lids, and disease of the ocular surface. Studies of pre-clinical models of lacrimal gland disease and meibomian gland dysfunction show that the ocular surface changes of dry eye disease are dependent upon and proportional to increases in tear film osmolarity⁵⁻¹⁰. Clinical studies corroborate these findings^{11,12}.

Decreased tear secretion may result from any condition which damages the lacrimal gland or its excretory ducts. Autoimmune disease with inflammation of the tear gland is the most common cause. Less common causes include cicatricial ocular surface conditions. Tear secretion also may be decreased by any condition that decreases

Figure 1
Decreased tear secretion or increased tear film evaporation increase tear film osmolarity, causing the progressive ocular surface changes observed in dry eye disease (modified from W.B. Saunders Company, Philadelphia, In Albert DM, Jakobiec FA eds: Principles and Practice of Ophthalmology, 1994; 257-76).

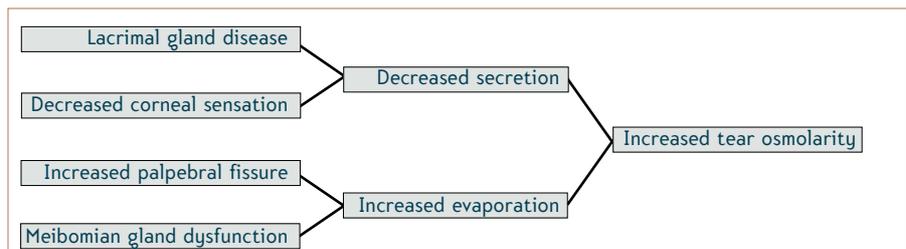


Table 1
Four milestones of dry eye disease

The natural history of dry eye disease dictates the sensitivity of diagnostic tests and the efficacy of treatment. The most efficacious treatment now addresses all four milestones of dry eye disease.

Milestones	Diagnostic tests	Eye drop type
1. Increased tear osmolarity	Patient history Tear osmolarity	Sufficiently/optimally hypotonic
2. Decreased goblet cell density Decreased corneal glycogen	Conjunctival staining Impression cytology	Tear-matched electrolyte balance
3. Increased corneal epithelial desquamation	Corneal staining	Preservative-free lubricants of various viscosities
4. Decreased corneal cell surface glycoproteins	Tear film break-up time	Preserved lubricants of various viscosities

corneal sensation¹³, including diabetes, herpes zoster, long-term contact lens wear and surgery which involves corneal incisions or ablates corneal nerves.

Increased tear evaporation may occur in one of two ways:

1. Long-standing posterior blepharitis causing meibomian gland dysfunction. When these glands function properly, they produce an oil layer which coats the tear film and retards evaporation.
2. A large palpebral fissure width, occurring either naturally, secondary to cosmetic surgery or with thyroid eye disease¹⁴, places evaporative stress on the tear film. Evaporation is proportional to the palpebral-fissure surface area. Increased evaporation also explains why symptoms become worse with exposure to air conditioning, dry heat, low humidity or wind.

Ageing tends to result in a gradual decline in tear secretion secondary to the associated decline in corneal sensation and meibomian gland function¹⁵. In most patients, physiologic reserve, along with a bit of ptosis, is adequate to prevent the development of symptoms and disease.

Milestones of dry eye

While studies of human disease have shown the ocular surface changes which occur with dry eye, the study of pre-clinical models of keratoconjunctivitis sicca (KCS) helps us delineate the natural history of these changes. We now know that dry eye disease evolves through a sequence of four milestones:

- Loss of water from the tear film with an increase in tear osmolarity
- Decreased conjunctival goblet-cell density and decreased corneal glycogen
- Increased corneal epithelial desquamation
- Destabilisation of the cornea-tear interface

Decreased tear production or increased tear evaporation is rapidly reflected by an increase in tear osmolarity, and soon thereafter by a decrease in goblet cell density. The loss of goblet cells is significant because they produce mucus, the major lubricant in the tear film, and serve in the defence of the ocular surface (mucus fired from goblet cells helps trap foreign matter and expel it from the eye).

The increase in the osmotic gradient between the tear film and the ocular surface, in addition to decreasing goblet cells, pulls water between conjunctival epithelial cells. This action breaks the delicate attachments between these cells and increases conjunctival cell desquamation. In unison with the decrease in goblet cells is a decrease in corneal glycogen. This loss of glycogen is clinically important because glycogen is the energy source for the sliding step of corneal

wound healing.

However, the cornea does not stay unaffected forever. Much later in the natural history of the disease, after resisting changes in the tear film, the attachments between corneal cells finally loosen. The result is an increase in corneal desquamation with a resultant decrease in corneal barrier function. Even later in the natural history of the disease, changes in the corneal epithelial cell surface become severe, resulting in a loss of corneal surface glycoproteins and destabilisation of the cornea-tear interface (the attachment between the cornea and the tears).

Understanding the natural history of the disease is crucial for interpreting and evaluating diagnostic tests and appreciating treatment advances (Table 1).

Why dry eye?

In most cases, the diagnosis of dry eye can be made based upon the patient history. The purpose of the examination is to determine why the patient has dry eye.

Patients with dry eye, either from decreased tear production or increased evaporation, most frequently complain of chronic sandy-gritty irritation or dryness in their eyes which gets worse as the day goes

» Table 2

What causes chronic eye irritation?

Causes other than dry eye and meibomitis may explain a patient's chronic eye irritation. Consider these other possible causes and their symptoms.

Anterior blepharitis

Patients have crusting and irritation at the base of lashes without diurnal variation. Onset is insidious.

Medicamentosa

Patients complain of burning and irritation without diurnal variation. Symptoms are equivalent throughout the day because overuse of topical medications promotes damage. You should suspect this condition in all patients who use traditional artificial tears more than four times a day. Patients generally have a history of escalating tear use.

Lacrimal drainage obstruction

Patients often have symptoms of tearing with actual and demonstrable tear overflow. Patients with meibomian gland dysfunction may feel like their eyes are tearing, but these patients have frank epiphora.

Allergic conjunctivitis

The primary symptom for this condition is itchy eyes. Patients' eyes may also exhibit increased mucus production. Onset of this condition is commonly seasonal, and it may be associated with hay fever, asthma and eczema.

Nocturnal lagophthalmos

Patients' eyes may burn upon awakening. Patients frequently have a history of lid surgery or thyroid eye disease.

Superior limbic keratoconjunctivitis

Symptoms include burning and irritation without diurnal variation. Abrupt onset and remissions characterise this condition. Patients often have a history of thyroid dysfunction.

Superficial punctate keratitis (Thygeson's)

Patients with this condition experience insidious onset of photophobia, eye irritation and decreased vision. The condition is episodic and recurring.

Dry eyelid skin

Patients complain of 'dry eyes'. This condition underscores the importance of accurate localisation of symptoms.

Tarsal foreign body

Patients experience a chronic sensation of having a foreign body in their eye. This sensation results from exogenous material or an exposed meibomian gland-derived conjunctival concretion.

Mucus fishing syndrome

Symptoms include chronic eye irritation and increased mucus production. Patients who reach into their conjunctival cul-de-sac to remove mucus strands caused by conjunctival trauma initiate the condition. A vicious cycle can develop.

Blepharospasm

Patients may complain that their eyes feel 'tired'. Careful questioning reveals that patients are experiencing an involuntary closure of the eyes, rather than eye irritation. Driving, reading and exposure to sunlight worsen symptoms.

Non-specific ocular irritation

Normal eyes, abnormal environment. Eye irritation in response to smoke would be a typical example.

Normal eyes with hypochondriasis

This condition is uncommon. A careful history which fails to mesh with the examination can provide the first clue to its presence.

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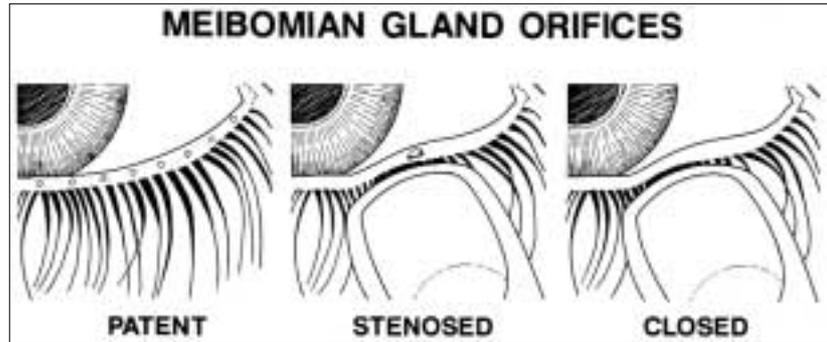


Figure 2

Natural history of meibomitis. Meibomian gland inflammation leads first to stenosis and then closure of the meibomian gland orifice (by courtesy of *International Ophthalmology Clinics* 1994; 34: 27-36)

on¹⁶. This is because eye closure during sleep forms a watertight seal over the tear film and gives the ocular surface a chance to recover. When the eyes open, evaporation begins, which increases tear film osmolarity as the day goes on¹⁷. It is difficult to overstate the usefulness of this history in diagnosing dry eye. If there are symptoms for more than three months and if the onset was gradual, the patient has dry eye until proven otherwise.

Keep in mind that patients with active meibomitis (also known as posterior blepharitis) also complain of chronic sandy-gritty eye irritation. But in these patients, the irritation is worse upon awakening. This is because tear production decreases during sleep, and eye closure brings the inflamed lids right up against the eye where the release of inflammatory mediators act on the cornea all night. When these patients awaken, tear flow increases, the lids pull away from the cornea, and their symptoms improve as the day goes on.

Eventually, the chronic meibomian gland inflammation leads to meibomian gland dysfunction. When that happens, these patients develop a second peak in symptoms from dryness toward the end of the day. Finally, when the meibomian gland inflammation and secondary healing obliterate the meibomian glands, the morning symptoms resolve and patients are left with symptoms from dryness alone, with sandy-gritty irritation and dryness which gets worse as the day goes on.

Eye irritation has many other causes, which should be considered when approaching a patient with chronic symptoms (Table 2).

The most sensitive and specific test for dry eye is osmolarity measurement of nanolitre tear samples collected from the inferior marginal tear strip. This is consistent with expectations, given that loss of water from the tear film defines the disease. The effort of performing measurements is the only limiting factor to this test's usefulness. Unfortunately, at the current time, there is no way to do this test in the clinical setting.

On examination, look first for facial

telangiectasias, which may presage meibomitis or meibomian gland dysfunction associated with rosacea. Because evaporation is proportional to surface area, measure palpebral fissure width. Palpebral fissure widths greater than 10mm place significant evaporative stress on the tear film. Study the meibomian gland orifices with a slit lamp. As the natural history of meibomitis advances, the meibomian gland orifices progress from open to stenosed to closed (Figure 2).

Next, touch a wet fluorescein strip to the patient's inferior tarsal conjunctiva and examine the tear film. Lack of spontaneous fluorescence indicates decreased tear volume. In patients with more markedly decreased tear volume, debris will be visible in the tear film as well as, possibly, dehydrated mucus which has precipitated in the inferior fornix.

The appearance of the tear film of patients with meibomian gland dysfunction has a watery quality. The tears tend to 'splash' around more because meibomian oils, in addition to decreasing tear evaporation, also lower the surface tension of the tear film, which holds the tear film 'tight' to the eye.

There is a common misconception that dry eye patients can experience 'tearing'. In fact, patients with dry eye from meibomian gland dysfunction may report that it "feels like" their eyes are tearing. This sensation results from their tears 'splashing' around more, and because the oil barrier created by the secretion of oil onto the lid margin is missing. As a result, tear fluid can touch the cutaneous portion of the muco-cutaneous junction, making it feel as if the eyes are tearing. It is important to note that these patients will not have tear overflow. Patients who have tear overflow (frank epiphora) have nasolacrimal drainage obstruction until proven otherwise.

Evaluation options

The two methods for clinically evaluating the conjunctival changes which occur relatively early in disease are impression cytology and rose bengal staining. Impression cytology requires setting up a small laboratory. This takes serious

commitment and will not be covered in this article. In contrast, rose bengal staining is clinically practical. Another dye, called lissamine green (not available in the UK yet), appears to stain the ocular surface equivalently to rose bengal, but with fewer irritating side effects. In light of the natural history of dry eye, either rose bengal or lissamine green stain the conjunctiva some time after tear osmolarity increases and once goblet cell loss has become quite significant. Recent evidence suggests that staining occurs when surface cell glycoproteins are altered to an extent that cells have less capacity to retain mucus^{17,18}.

The pattern of rose bengal staining is more useful than merely the presence or absence of stain or even the amount of stain. With dry eye, the nasal conjunctiva stains more than the temporal conjunctiva, and the resilient cornea stains less than the conjunctiva and later in the disease process. Corneal staining usually begins with the loss of corneal cell surface glycoproteins – the last of the four milestones in the natural history of dry eye disease (Table 1).

The rapid development of randomly located dark spots in the pre-corneal tear film (evident after the instillation of fluorescein dye) reflects tear film instability. This finding has been used diagnostically as the tear film break-up time measurement, yet as many as half of patients with dry eye will have normal tear film stability. We now understand that this is because dry spots are a result of, not a cause of, dry eye disease. The corneal epithelial changes required to cause tear film instability – loss of corneal cell surface glycoproteins – occur late in the natural history of dry eye disease. Although not a sensitive test (it is not highly positive in the presence of disease), break-up time is probably highly specific in that it is negative when disease is absent.

Given that decreased tear production or increased evaporation can cause dry eye, it is understandable why, in controlled studies, the value obtained through the Schirmer test is not the best means of diagnosis. This poor sensitivity, specificity and predictive value pertains whether or not dry eye patients are selected based on symptoms, increased osmolarity or ocular surface disease. In other words, no matter how you diagnose dry eye, whether by history, increased osmolarity, or rose bengal staining, the Schirmer test has poor sensitivity, specificity and predictive value.

While lacrimal gland disease decreases Schirmer measurements, meibomian gland dysfunction increases these measurements. With decreased oil on the lid margin, the Schirmer strip wets more easily. Indeed, in a rabbit model of dry eye with meibomian dysfunction, elevated tear film osmolarity, decreased conjunctival goblet-cell density, decreased corneal glycogen, and rose bengal staining, Schirmer test strips wet more than those used in control eyes⁸.

Targeted treatment

Early treatments for dry eye disorders targeted the late milestones in dry eye disease. In part, this was because the late milestones were easier to spot. For example, dry spot formation was more obvious than increased tear osmolarity and loss of conjunctival goblet cells. But as our knowledge and understanding of dry eye have improved, treatment has begun to target earlier milestones in the disease progression.

Many years ago, demulcents (polymers) were added to artificial tear solutions to improve their lubricant properties and change their viscosity. In 1975, a classic study demonstrated that artificial tear solutions (all containing a preservative at the time) transiently increased tear film stability in normal subjects¹⁹. These solutions, whether of high or low viscosities, act by temporarily mimicking cell surface glycoproteins, which are lost late in the disease. Solutions of higher viscosity remain in the eye longer. Whatever relief preserved artificial tear solutions provide hinges on their ability to temporarily stabilise the cornea-tear interface.

The next treatment advance – preservative-free artificial tear solutions – occurred about 15 years ago, shortly after researchers recognised that preservatives increased corneal desquamation²⁰. A recent study showed that traditional preservative-free artificial tear solutions improved, but did not normalise, corneal barrier function in dry eye patients. Improved corneal barrier function reflects decreased corneal epithelial desquamation and improved corneal cell junctions. Treatment with a preserved artificial tear solution, while briefly increasing tear film stability, actually diminished corneal barrier function²¹. Preservative-free solutions, by eliminating corneal 'peeling' due to preserved artificial tear solutions, established a new benchmark in artificial tear solution treatment, yet still did not address the desquamation caused by dry eye itself.

Since then, researchers have tried to improve the effect of these preservative-free solutions on corneal barrier function by adding various ions. The electrolyte balances of these preservative-free solutions were the best which could be designed, while focusing only on issues related to corneal morphology²². From the natural history of dry eye disease, we know that decreases in conjunctival goblet-cell density and corneal glycogen are much more sensitive indicators of ocular surface health than changes in corneal morphology.

Knowing what we know now about the mechanism and natural history of dry eye, we would expect that the next advance in treatment would address decreased conjunctival goblet cells, decreased corneal glycogen and elevated tear film osmolarity. A recently introduced artificial tear is sufficiently hypotonic to lower elevated tear

film osmolarity, while its tear-matched electrolyte balance permits the restoration of mucus-containing conjunctival goblet cells and corneal glycogen²³.

Punctal occlusion also helps to lower elevated tear film osmolarity, reduce rose bengal staining and improve symptoms²⁴. However, controlled studies indicate that punctal occlusion does not have any effect on goblet-cell density²⁵. Why? In our studies of KCS patients with lacrimal gland disease, we found an increase in tear osmolarity and all measured tear electrolytes. There was, however, a significantly disproportionate increase in tear sodium levels in these patients¹⁵. Disproportionately high sodium levels deplete conjunctival goblet-cell density²⁶. So, while punctal occlusion can add water to the tear film, it cannot correct the disproportionate increase in tear sodium seen in KCS which depletes goblet cells.

Nutrition and dry eyes

Until now the approach to dry eye has been a topical one, and by lowering elevated tear film osmolarity and providing a tear-matched electrolyte-balanced tear solution, effective treatment has been achieved. Now we are seeing a new approach to dry eye treatment, a revolutionary change, with the introduction of oral treatments.

Oral supplementation for dry eye began with the introduction of a product called HydroEye. The problem with this supplement, however, is that it adds to the already excessive amount of omega-6s in the average Western diet. Omega-6s increase serum levels of arachidonic acid (AA) and promote heart disease, stroke and other degenerative diseases.

New research, presented for the first time at the 2003 annual meeting of the Association for Research in Vision and Ophthalmology, has found that high dietary intake of omega-3 essential fatty acids decrease the risk of dry eye²⁷. Using the Women's Health Database at the Harvard School of Public Health, the investigators examined the dietary intake of essential fatty acids in 32,470 female health professionals. They found that the higher the dietary ratio of omega-3 to omega-6 essential fatty acids, the lower the likelihood of dry eye, and the higher the dietary omega-3 intake, the lower the likelihood of dry eye. Conversely, they found that the lower the ratio of omega-3s to omega-6s, the higher the likelihood of dry eye.

Omega-3s are essential fatty acids. 'Essential' means that, because they cannot be produced by the body, their inclusion in the diet is essential for good health. The two best sources of omega-3s are dark, oily, cold water fish, and flaxseed. They are known to have a multitude of health benefits yet, as a population, Westerners are omega-3 deficient (Figure 3).

Omega-6s are another group of essential

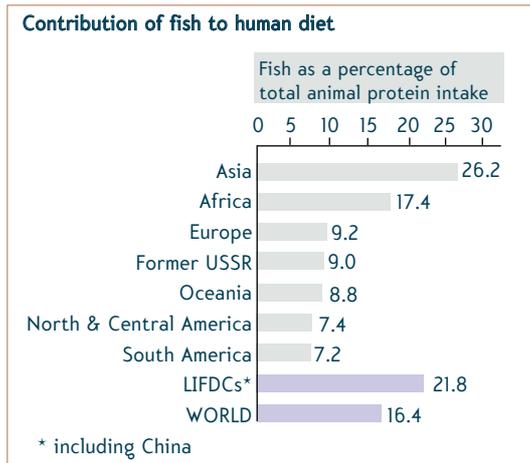


Figure 3

Westerners, in general are omega-3 deficient

fatty acids. Omega-6s are consumed in beef, dairy and vegetable shortening and cooking oils (i.e. hamburgers, cheese burgers, pizza, ice-cream, potato chips etc). Unfortunately, while the recommended ratio of omega-3s to omega-6s is 1:2.3, the existing ratio of omega-3s to omega-6s consumption has been estimated to be as low as 1:10²⁸.

This, and other research, contributed to a switch to using omega-3s, with flaxseed oil, fish oil and vitamin E, in supplements. Such formulations address both meibomitis and the underlying causes of dry eye.

Omega-3s decrease inflammation

Omega-3s in the diet, once consumed, are elongated by enzymes to produce anti-inflammatory prostaglandin E3 (PGE3) and anti-inflammatory leukotriene B5 (LTB5) (Figure 4). Even more importantly, eicosapentaenoic acid (EPA), a long-chain omega-3 provided directly by fish oils, blocks the gene expression of the pro-inflammatory cytokines tumour necrosis factor alpha (TNF- α), interleukin-1 α (IL-1 α), interleukin-1 β (IL-1 β), proteoglycan degrading enzymes (aggrecanases) and cyclooxygenase (COX-2) (Figure 5).

These anti-inflammatory effects go a long way to explain why omega-3s have been useful in treating patients with posterior blepharitis or meibomitis. The results are so positive that it may result in the displacement of systemic tetracyclines as treatment for the early morning eye irritation suffered by meibomitis patients. However, the effects of omega-3s only begin with their effects on meibomitis.

Omega-3s decrease apoptosis

Suppressing TNF- α is also important because in Sjögren's syndrome and in lacrimal gland-based dry eye, increased TNF- α in the lacrimal glands increases lacrimal gland apoptosis (programmed cell death). Increased apoptosis contributes to the decrease in tear production, and

increase in tear film osmolarity which drives dry eye ocular surface disease.

In addition, TNF- α induces apoptosis on the ocular surface in dry eye. Specifically, Luo and co-workers found that increasing tear film osmolarity in animal models increases the expression of TNF- α and the associated cell regulators that increase apoptosis on the ocular surface¹⁰. There has been a lot of interest recently in ocular surface inflammation in dry eye. This important study shows that it is elevated tear film osmolarity which induces the increased expression of pro-inflammatory cytokines in dry eye, just as elevated tear film osmolarity has been shown to produce all the morphological ocular surface changes described in dry eye.

While EPA decreases the gene expression

of TNF- α , DHA, another long-chain omega-3 provided directly by fish oils, protects cells from TNF- α -induced apoptosis. Yano and co-workers have demonstrated that vitamin E works synergistically with DHA to protect cells from TNF- α -induced apoptosis²⁹. So EPA and DHA work together to protect the lacrimal gland and ocular surface from apoptosis.

Omega-3s stimulate tear secretion

The effects of suppressing pro-inflammatory cytokines do not stop here. It is known that the pro-inflammatory cytokines, TNF- α , IL-1 α , and IL-1 β , impair tear secretion in lacrimal gland disease-based dry eye by inhibiting the release of neurotransmitters from neural synapses, and interfering with the secretory response of lacrimal gland acinar cells to stimulation^{30,31}. This is probably the main mechanism by which tear secretion decreases in dry eye.

The profound importance of this has been illustrated in recent work which shows that when TNF- α gene expression is blocked by gene therapy in an animal model, autoimmune lacrimal gland disease can be reversed, and tear secretion restored³². The relevance of this animal model is supported by the epidemiological data cited above, as well as an additional study which reported that Sjögren's patients had a lower dietary intake of omega-3s, including EPA and DHA, than age-matched controls³³.

While EPA is central in blocking the gene expression of pro-inflammatory cytokines, DHA may help in a complementary way. Neural synapses

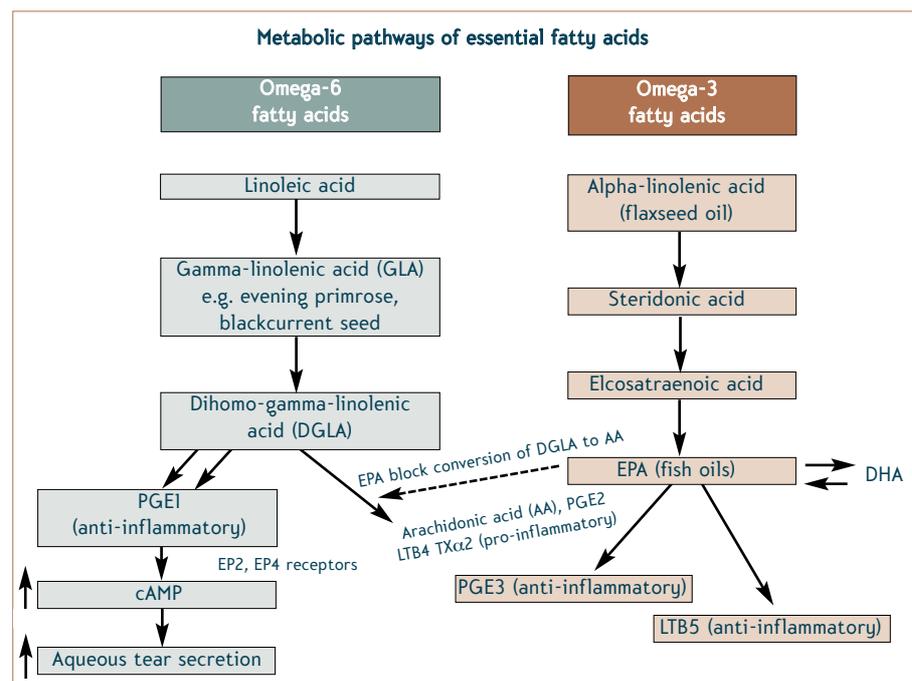
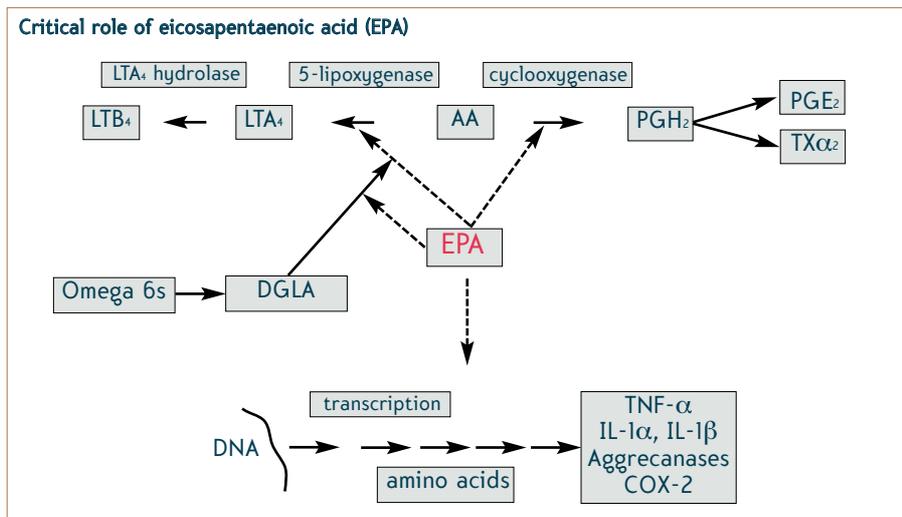


Figure 4

Flaxseed and fish oils to generate anti-inflammatory PGE3 and LTB5 and suppress blepharitis, while increasing PGE1 to promote tear secretion (see text for other effects)

Figure 5

EPA supplied by fish oils, blocks the gene expression on TNF- α and other pro-inflammatory cytokines



development of xerophthalmia – a condition characterised by squamous metaplasia of the ocular surface, with increased keratinisation and loss of conjunctival goblet cells. Experimental studies have found that deficiencies of vitamin A and zinc can reduce corneal and conjunctival microvilli and conjunctival goblet cells, and increase ocular surface keratinisation. It has also been shown that supplementation with these antioxidants can reverse these changes⁴⁰.

In one study, antioxidant supplementation has been tried as a therapy in patients with mild dry eye, and shown to increase conjunctival goblet cell density and non-invasive tear film break-up time. These changes were not associated with an alteration in tear production or an improvement in patient symptoms⁴¹. It can be hypothesised, therefore, that while the antioxidant supplementation promoted normal ocular surface differentiation, it did not address the underlying elevation in tear film osmolarity which is responsible for patient symptoms.

contain among the highest concentration of DHA in the body and research has shown that dietary supplementation with DHA restores neural DHA levels and improves age-related declines in synapse function³⁴. DHA may reduce the ability of pro-inflammatory cytokines in the lacrimal gland to inhibit signal transduction at the synapse. Lending credence to this hypothesis is the finding that severity of dry eye in Sjögren's patients has been found to be inversely proportional to membrane and serum levels of DHA³⁵.

Omega-3s affect the lacrimal gland in another way. EPA and DHA and alpha-linolenic acid (ALA) from flaxseed oil competitively inhibit the conversion of omega-6s to arachidonic acid (AA) thereby promoting the conversion of dihomo-gamma-linolenic (DGLA) to prostaglandin E1 (PGE1) (Figure 4). PGE1 also has anti-inflammatory properties and, in addition, acts on the E-prostanoid receptors EP2 and

EP4 to activate adenylate cyclase, increasing cyclic AMP (cAMP). PGE1 and cAMP have been shown to stimulate aqueous tear secretion^{36,37}.

Omega 3s, the meibomian gland oils
Meibomian glands use essential fatty acids to synthesise oil (meibum). Dietary intake of omega-3s in general, and EPA and DHA in particular, has recently been shown to affect the polar lipid profiles of meibum as observed by HPLC³⁸. Indeed, Boerner has observed the clearing and thinning of meibomian gland secretions with omega-3 supplementation along with an improvement of patient symptoms³⁹. Further studies are needed to determine whether these effects are sufficient to bolster the oil layer and retard evaporation.

Oral antioxidants

In areas of the world where nutrition is poor, vitamin A deficiency leads to the

Conclusion

With these findings and insights, it is possible to approach patients who have chronic eye irritation in a more systematic and effective way. Understanding the natural history of dry eye disease improves our ability to diagnose the condition and to appreciate the meaning of our examination and testing. With this information, we can better differentiate between various treatments at our disposal and offer patients the best care possible.

References

For a full set of references, email nicky@optometry.co.uk or visit http://www.dryeyeinfo.org/Dry_Eye_In_Depth.htm#Nutrition_and_the_eye.

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MCQs

Module 6 Part 6 of the Nutrition and the Eye series**Dry eye and the role of nutrition**

Please note there is only ONE correct answer

1. Which one of the following does NOT decrease tear production?
 - a. Autoimmune lacrimal gland disease
 - b. Herpes zoster
 - c. Small palpebral fissure
 - d. Long-term contact lens wear
2. Which one of the following is NOT a milestone in the natural history of dry eye surface disease?
 - a. Increased tear film osmolarity
 - b. Increased corneal glycogen
 - c. Decreased goblet cell density
 - d. Increased corneal epithelial desquamation
3. Which one of the following statements about goblet cells is INCORRECT?
 - a. They decrease in the presence of increased tear osmolarity
 - b. They help lubricate the ocular surface
 - c. Simultaneously with the decrease in goblet cells, corneal glycogen levels increase
 - d. They produce mucus
4. Which one of the following statements describes patients with dry eye?
 - a. They are most symptomatic in the morning
 - b. They have two symptom peaks: one in the morning upon awakening, and again later in the day
 - c. They have no diurnal variation and have symptoms all day long
 - d. They typically become more symptomatic as the day goes on
5. Which one of the following statements best describes patients with active meibomitis?
 - a. They are most symptomatic when they first wake up
 - b. They never develop two symptom peaks
 - c. They have no diurnal variation and have equal symptoms all day long
 - d. Their symptoms typically become worse as the day goes on
6. Which one of the following is INCORRECT about rose bengal staining?
 - a. It is clinically practical
 - b. It can help evaluate conjunctival changes which occur early in disease
 - c. The pattern of rose bengal staining offers valuable clues about a patient's condition
 - d. Rose bengal stains the conjunctiva some time before tear osmolarity increases and goblet cell loss has become significant
7. Up to what percentage of patients with dry eye have normal tear break-up time?
 - a. 20%
 - b. 50%
 - c. 15%
 - d. 75%
8. Which one of the following is CORRECT about omega-6 supplements?
 - a. They reduce the risk of stroke
 - b. They add to the excess of omega-6 in the American diet
 - c. They reduce the risk of heart disease
 - d. They decrease arachidonic acid levels
9. Punctal occlusion does all of the following except which one?
 - a. Lower elevated tear film osmolarity
 - b. Improves symptoms
 - c. Reduces rose bengal staining
 - d. Restores conjunctival goblet cells
10. Which one of the following does not need to be addressed by a dry eye treatment?
 - a. Decreased conjunctival goblet cells
 - b. Decreased corneal glycogen
 - c. Elevated tear film osmolarity
 - d. The blood supply of the living cells that comprise the ocular surface
11. EPA, available directly from fish oils, does all of the following except which one?
 - a. Blocks the gene expression of TNF- α
 - b. Competitively inhibits the conversion of omega-6s to arachidonic acid
 - c. Promotes the conversion of DGLA acid to PGE1
 - d. Improves age-related declines in synapse function
12. Increased dietary intake of omega-3s does all of the following except which one?
 - a. Reduces the risk of dry eye
 - b. Has been observed to reserved to result in a thinning and clearing of meibomian gland oils
 - c. Addresses both meibomitis and the underlying causes of dry eye
 - d. Increases the risk of sudden death from heart disease and stroke

An answer return form is included in this issue. It should be completed and returned to: CPD initiatives (c4750f), OT, Victoria House, 178-180 Fleet Road, Fleet, Hampshire, GU51 4DA by July 2, 2004.

Under no circumstances will forms received after this date be marked – the answers to the module will have appeared in our July 4 issue and scores sent electronically to the accrediting bodies.

Answers

Nutrition and the eye: Nutrition and glaucoma: Do supplements reduce IOP?

Here are the correct answers to Module 6, Part 5, which appeared in our May 7, 2004 issue.

1. The proportion of glaucoma in the total population over the age of 40 years is:
 - a. 0.1-0.2%
 - b. 1-2%
 - c. 3-4%
 - d. 5-6%
2. The proportion of POAG at the age of 80 years is highest in which one of the following populations?
 - a. Scandinavians
 - b. Caucasians
 - c. Asian races
 - d. Afro Caribbean races
3. In 1996, according to the RNIB, glaucoma accounted for approximately how many people registered as blind and partially sighted?
 - a. One in 10
 - b. One in 20
 - c. One in 30
 - d. One in five

b is correct

The overall incidence of glaucoma is 1-2% of the total population over the age of 40 years, with approximately 25% of these cases going undiagnosed in the community.

d is correct

Around 1% of Caucasians have POAG at the age of 50 years¹, rising to around 4% at the age of 80 years. Estimates for Afro-Caribbeans are 3% and 13% at the equivalent ages.

a is correct

According to the Royal National Institute of the Blind, glaucoma is the primary cause for 11.7% of those registered as blind or partially sighted.

Answers

4. Which one of the following types of glaucoma is the most common in the UK?

- a. Chronic angle-closure
- b. Acute angle-closure
- c. Primary open-angle
- d. Low tension

c is correct

Primary open-angle glaucoma (POAG) is the most common form of glaucoma in the UK, where it accounts for over 60% of diagnosed cases.

5. A safe level of IOP for glaucoma is:

- a. 15mmHg
- b. 25mmHg
- c. 19mmHg
- d. none of the above

d is correct

The 'safe' level is very patient dependent and is reliant on factors such as age, presenting IOP, visual fields, corneal thickness and optic nerve damage and analysis. Different optimal IOP levels need to be set for each case and there is no one, single optimal IOP which can be prescribed for all.

6. The current mainstay of first-line current glaucoma treatment is:

- a. YAG laser treatment
- b. 5FU trabeculectomy
- c. Laser trabeculoplasty
- d. Beta-blockers and/or prostaglandin analogue medication

d is correct

Topical beta-blockers and prostaglandin analogues are first-line treatments.

7. Which one of the following foods give a definite causal link to glaucoma?

- a. Bilberry extract
- b. Vitamin C
- c. Vitamin A
- d. No food stuffs or vitamins

d is correct

No studies have proved conclusively a connection between specific foods and glaucoma.

8. Which one of the following is correct regarding ReVision Formula?

- a. It contains vitamin A
- b. It contains ginkgo biloba
- c. It is an eye drop
- d. It is best taken at midday

b is correct

ReVision Formula includes bupleurem, dong guai, white peony, poria, atractylodes and licorice, plus additional herbs tree peony, ginger, gardenia, coleus, ginkgo biloba, milk thistle, dandelion, eyebright, bilberry and hoelen.

9. Which one of the following is correct regarding the effect of marijuana?

- a. IOP increases
- b. IOP decreases
- c. The anterior chamber angle opens
- d. The anterior chamber angle closes

b is correct

Although it is well known that smoking marijuana can reduce pressure within the eye, the drug may also reduce the blood supply to the optic nerve head.

10. Which one of the following is correct regarding ginkgo biloba?

- a. Blood flow in NTG is enhanced
- b. The trabecular meshwork opens
- c. The trabecular meshwork closes
- d. The action of prostaglandin analogues is enhanced

a is correct

The nutritional extract of ginkgo biloba enhances blood flow and appears to improve pre-existing visual field damage in some patients with NTG.

11. Which one of the following is correct regarding large doses of vitamin C?

- a. The oxidative stress in the eye is increased
- b. It causes bowel intolerance
- c. It causes constipation
- d. It interacts with beta-blocking agents

b is correct

Alternative medical practitioners often suggest that people with glaucoma take enough vitamin C to cause loose stools, and then reduce this amount slightly, to an amount termed 'bowel tolerance'.

12. Salvia miltiorrhiza is:

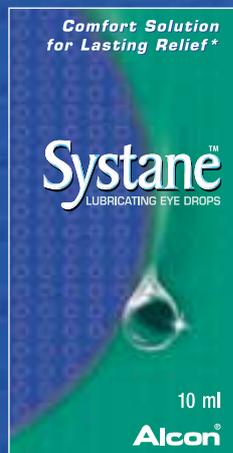
- a. a fungus
- b. a bacteria
- c. a botanical used in Chinese medicine
- d. found in the retinal ganglion cells

c is correct

Salvia miltiorrhiza is a common botanical used in Chinese medicine.

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