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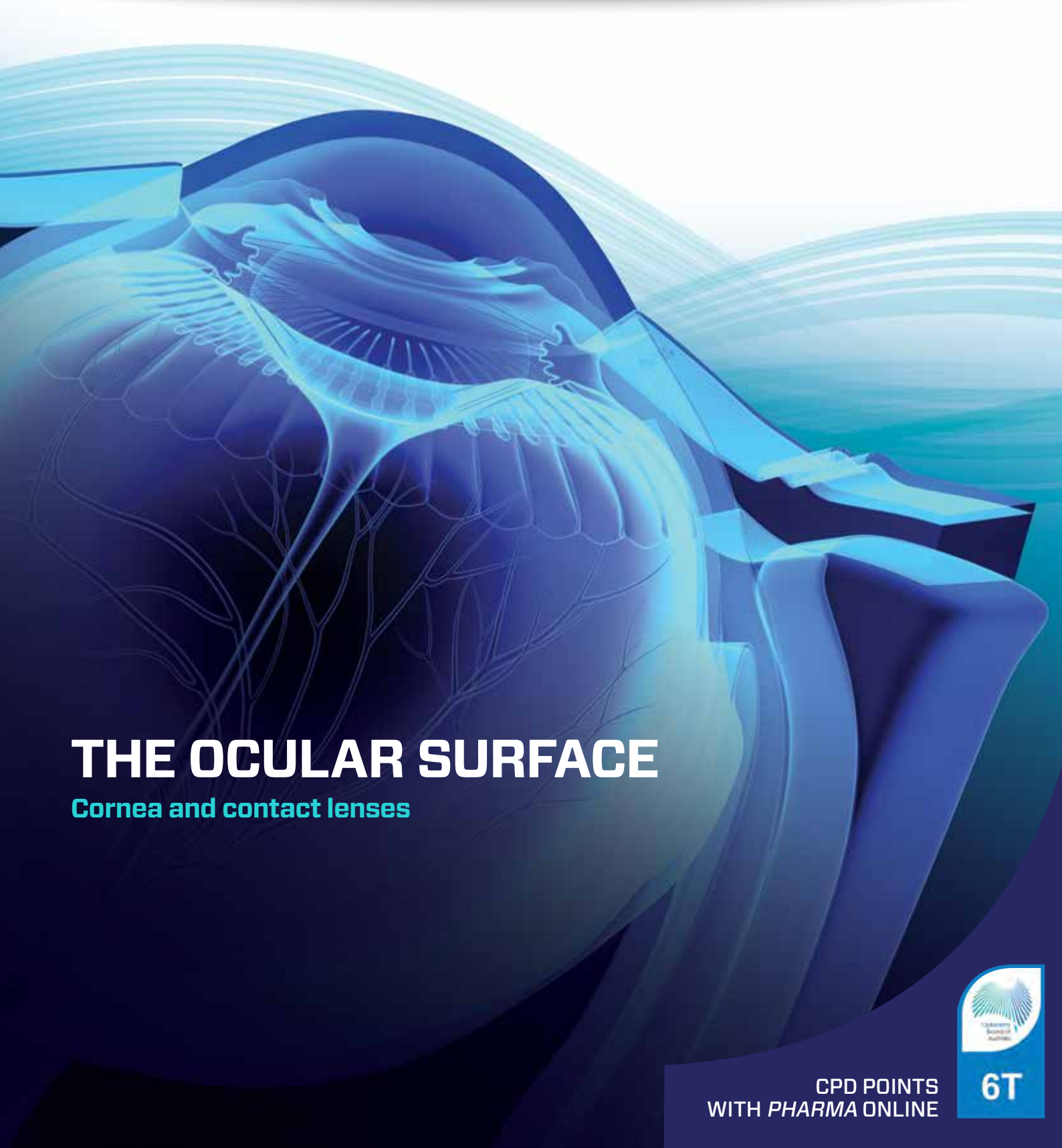

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Cornea and contact lenses

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See product instructions for wear, care and safety information.

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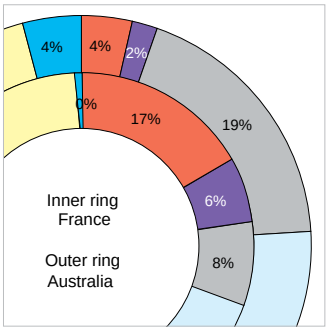

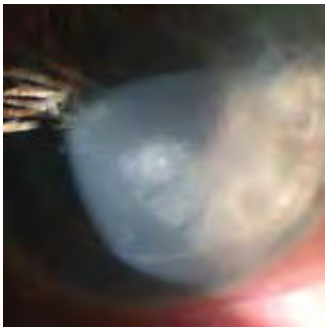

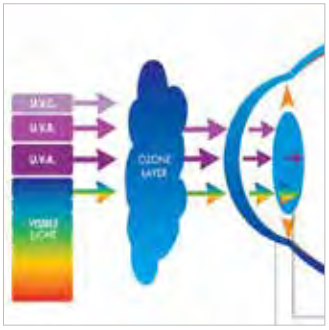

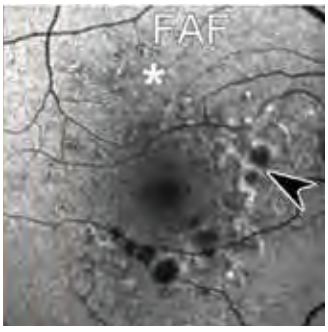

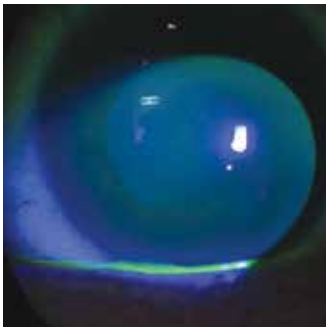

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Contact lens prescribing trends 2016

Efron, Morgan and Woods deliver their 17th annual survey of Australian prescribing habits

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THE 17TH ANNUAL survey of Australian contact lens prescribing was conducted from January to April 2016. The same format as in previous years was employed. An email was sent to all members of Optometry Australia with a link to a downloadable questionnaire, and a request that this be accessed, printed and completed to provide details of the first 10 patients fitted with contact lenses after receipt of the questionnaire.

The survey was specifically designed to be straightforward to complete while capturing key information about their patients. Practitioners were asked general questions about themselves. For each contact lens fitting, they were requested to complete the following details: date of fitting, new fitting or refitting, age and sex of patient, lens material, lens design, frequency of replacement, times per week of wear, modality (daily- or extended-wear) and care system. Practitioners were asked to return the questionnaire by fax, post or email.

Completed questionnaires relating to 320 contact lens fittings were returned, which provides a sound basis for a meaningful analysis. Each fitting was given a weighting based on the number of lenses fitted per year by the practitioner, based on the date information on the form. This means that data generated by practitioners who have a higher frequency of fitting contact lenses were afforded a higher weighting than those taking longer to fit the 10 patients with lenses.

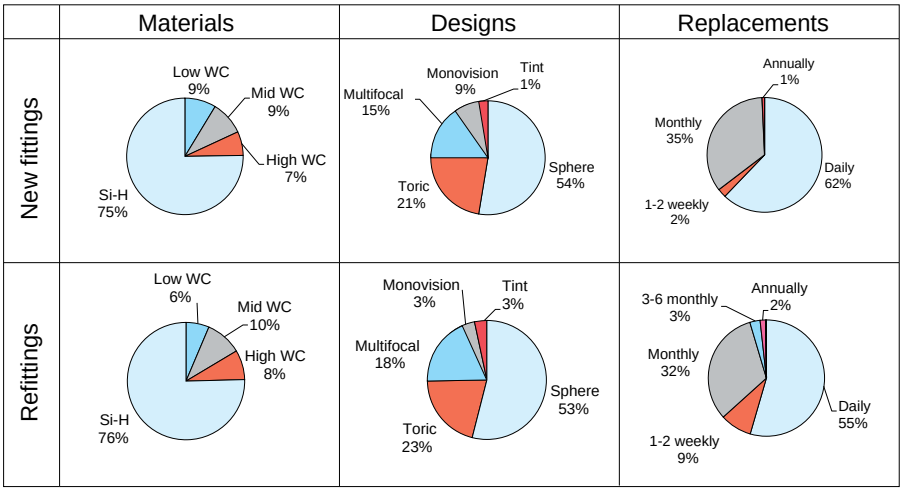
The discussion concentrates primarily on data relating to new lens fittings as opposed to refittings. We believe that new fittings are a more sensitive barometer of current patterns and future trends, whereas refittings are more indicative of previous fitting behaviours.

In keeping with other markets around the world,[1] a majority of lenses (61 per cent) was fitted to females. The average age of contact lens wearers at the time of fitting was 36.3 ± 16.5 years. The age at fitting ranged from eight to 75 years.

Soft lens materials and designs

Soft lenses are still the main type of contact lens fitted and as was the case last year,[2] accounted for 95 per cent of new fittings. Figure 1 is a composite of pie charts detailing the key findings of the 2016 survey in relation to soft lenses. Silicone hydrogels are still the dominant material, representing 75 and 76 per cent of materials prescribed as new fittings and refittings, respectively. This is identical to 2015 data.[2] The balance is more or less evenly split between low-water, mid-water and high-water content hydrogel materials. The reason for the apparent increase in the use of low-water content hydrogels over the past two years (nine per cent of new fittings), compared to zero per cent of new fittings in 2014,[3] is unclear.

The key categories of lens designs are spherical, toric, multifocal, monovision, coloured (tinted) and anti-myopia. Spherical designs represent a small majority of new fittings (54 per cent). About one-quarter of soft lenses prescribed are in toric form (21 per



▲ Figure 1. Detailed results for soft contact lens prescribing in the 2016 Australian survey
Si-H: silicone hydrogel, WC: water content

cent of new fittings and 23 per cent of refittings). Figure 2 shows trends in toric lens prescribing between 2000 and 2016.

The level of prescribing in Australia has consistently fallen short of that which would be expected if all lens wearers with ≥ 0.75 D of astigmatism were fitted with toric lenses.[4,5] The slight decline in toric lens prescribing over the past three years is possibly attributed to accelerated prescribing of silicone hydrogel daily disposable lenses (see below), with the availability of toric designs lagging behind spherical designs for this lens type.

Continuing improvements in soft multifocal lens designs over the past decade have resulted in strong prescribing figures for these lenses. This year, multifocal lenses represent 15 per cent and 18 per cent of new fittings and refittings, respectively, which is a level of prescribing similar to 2015 data.[2] It is evident that for new fittings, multifocals (15 per cent) are preferred over monovision lens wear (nine per cent) for correcting presbyopia. Coloured (tinted) lenses represented one per cent of new fittings and three per cent of refittings, which is broadly similar to last year's result.[2]

Anti-myopia lenses incorporate special designs for arresting the rate of progression of myopia.[6] No anti-myopia lens fittings were recorded, which may not be surprising because these lenses are still in the experimental and development phase. It indicates minimal off-label fitting as the single product now available in some markets (MiSight, CooperVision) is not yet commercially available in Australia.

Soft lens replacement and wearing modality

Of the 34 nations we surveyed in 2015, Australia was the leading nation for daily disposable lens prescribing, which accounted for 62 per cent of new fittings. This level of prescribing has remained the same for 2016. Figure 3 demonstrates how the proportion of all soft lenses (new fittings and refittings combined) prescribed for daily replacement has risen relentlessly in the Australian market since 2000. It is evident that the increased prescribing of daily disposable lenses has largely been at the expense of reusable lenses and to a lesser extent, extended-wear lenses.

The balance of new fittings comprises largely monthly replacement lenses (35 per cent), with the fitting of one to two weeks replacement lenses having declined in recent years, from 21 per cent in 2012[7] to only two per cent over the past two years. Only one per cent of lenses were being replaced less frequently than monthly.

Multi-purpose solutions remain the lens care option of choice for those wearing reusable lenses, representing 91 per cent of prescribed care regimens. The balance comprises almost exclusively peroxide systems.

Extended-wear lenses represented three per cent of new soft lens fittings in 2016, so single-use lenses, that is, extended-wear and daily-disposable lenses combined, represented 64 per cent of new soft lens fittings this year. The prescribing of single-use lenses is likely to continue to rise.

Rigid lenses

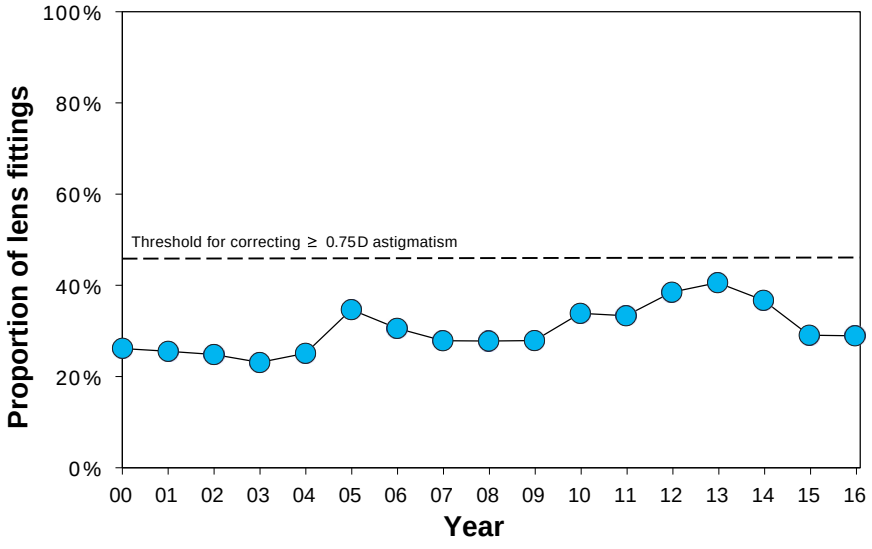
Conventional and orthokeratology rigid contact lenses represented four per cent and two per cent of all contact lens fittings, respectively. Because of the low level of rigid lens fitting in Australia, a valid statistical analysis of subcategories of materials, designs and replacement frequencies cannot be undertaken. The limited extent of orthokeratology fitting in Australia is probably due to the specialist nature and complexities of this fitting activity.

Australia versus France

We have conducted annual contact lens fitting surveys in about 40 countries over the past few years.[1] This provides an opportunity to benchmark against international colleagues and this year we compare contact lens prescribing in Australian with that in France. The comparison is interesting because contact lenses are almost exclusively fitted by optometrists in Australia and by ophthalmologists in France. The differences in patterns of prescribing between Australia and France can be largely attributed to type of practitioner.

The current pattern of contact lens fitting in Australia compared with that in France is displayed in Figure 4. Seven key categories of lens type are represented. The outer and inner rings display the Australian and French data,[1] respectively.

Overall, Figure 4 reveals striking differences in contact lens prescribing patterns between Australia and France. Perhaps the starkest difference is seen in the prescribing of daily disposable lenses, which are represented by the combined grey (daily disposable hydrogel) and pale blue (daily



▲ Figure 2. Percentage of all soft lenses prescribed as toric lenses in Australia between 2000 and 2016. The dotted line represents the expected prescribing rate if all lens wearers with ≥ 0.75 D of astigmatism were fitted with toric lenses.[4]

Continued page 4

Prescribing trends 2016

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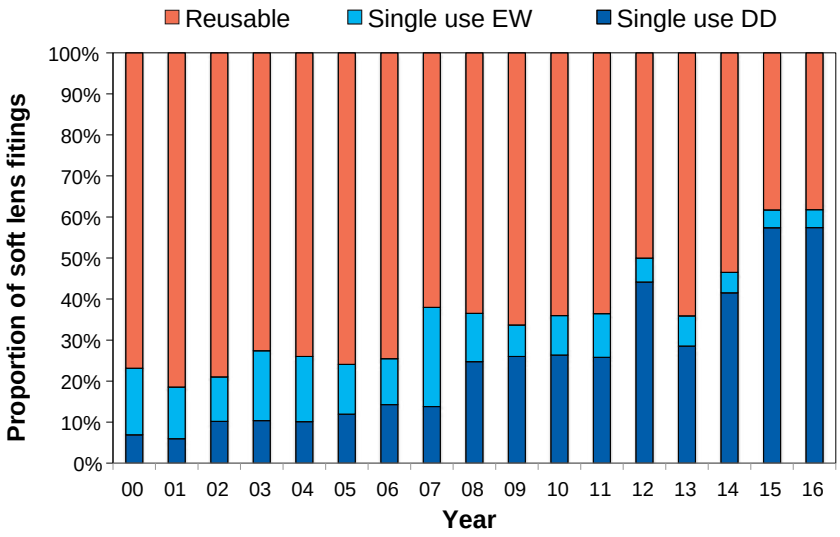
disposable silicone hydrogel) arcs. The overall prescribing of daily disposable lenses in Australia is about three times greater than in France, which may reflect a highly conservative approach to contact lens prescribing by ophthalmologists compared with that of optometrists.

The higher rate in France of rigid lens prescribing, of both orthokeratology and non-orthokeratology designs, also reflects a traditional approach to contact lens prescribing.

Extended-wear lenses represent four per cent of all contact lens fittings in Australia, whereas extended-wear lenses are not fitted in France.[1] The lack of extended-wear prescribing in France may reflect a cautious approach of French ophthalmologists relating to the known greater risk of developing microbial keratitis during overnight lens wear.

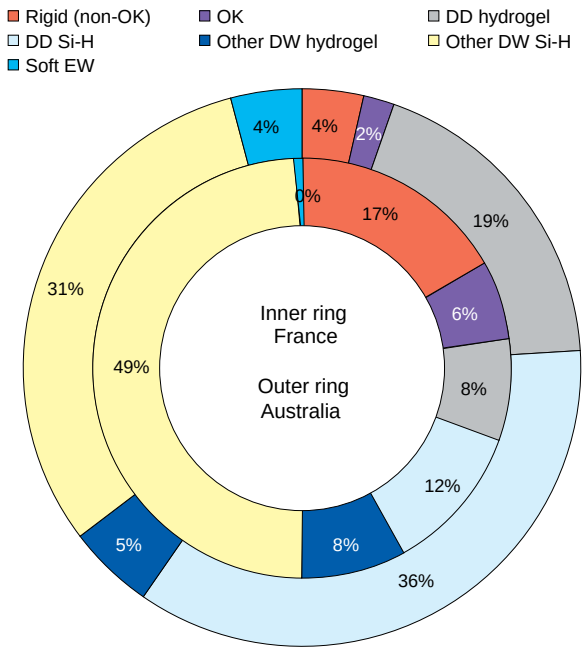


▲ Figure 3. Percentage soft lenses categorised as reusable, single use extended-wear and single-use daily disposable, prescribed in Australia between 2000 and 2016. EW: extended wear, DD: daily disposable

Conclusions

The results of our 2016 survey confirm the high rate of prescribing daily disposable lenses in Australia. The soft lens market overall is dominated by two lens replacement modalities: daily and monthly. Silicone hydrogels are firmly entrenched as the material of choice, representing three-quarters of all soft lens fittings.

We note continuing strong use of multifocals which now outnumber monovision fittings 2:1. The contact lens industry is continuing efforts to develop improved designs to cater for the vision needs of this growing demographic cohort. Toric contact lens fitting continues at high levels, despite falling a little short of the 'optimal' level of prescribing. ▲



▲ Figure 4. Percentage of all contact lenses prescribed in Australia (outer ring) compared with France (inner ring). DD: daily disposable, DW: daily wear, EW: extended wear, OK: orthokeratology, Si-H: silicone hydrogel

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Ultraviolet and blue light damage

Dr Stuart Richer

OD PhD FAAO

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There is potential for biological damage, from cornea to retina

OUR SUN, modern indoors LED lighting, and mobile phone, tablet and computer displays all emit damaging short wavelength high-energy visible blue radiation, defined as a wavelength band of approximately 445 nm +/- 30 nm. The amount of time spent under artificial blue dominant lighting, in particular, is increasing rapidly in all age groups.[1] There is a need to assess the potential damage of not only UV but also this high-energy visible blue radiation band that impacts all ocular tissues. These include the anterior exposed cornea, through the internal tissues of the lens, vitreous and back to the retina.

Ocular exposure to sunlight, UVA and short blue light-emitting sources directed at the human eye results in an immediate inflammatory assault or, over a longer time-frame, combined inflammatory/photo-oxidative induced damage.[2] The induction of cataracts and retinal degeneration typically results from long-term exposure.

Chronic exposure is particularly hazardous to infants, youngsters, teens and young adults with clear ocular lenses, but also after the age of 40 years as a result of lessened protective antioxidant systems combined with an increase in UV and visible light-absorbing endogenous chromophores. The latter efficiently produce singlet oxygen and other secondary reactive oxygen species. That is, during photo-oxidation reactions, phototoxic chromophores in the eye absorb light, are excited to a singlet state and then on to a triplet state. The triplet highly energetic state in turn produces free radicals and reactive oxygen species, which in turn damage the delicate DNA and macromolecular structures comprising ocular tissues.[2]

Cornea and conjunctiva impact

The vulnerable fully-exposed ocular surface tissues (cornea) and surrounding white semi-opaque tissue (conjunctiva) bear the major brunt of radiometric assault by both energetic UV and blue visible radiation.

The shorter the wavelength, the greater the energy, and the potential for biological damage is based on both intensity and duration of that exposure. Most of the UVC (220-280 nm) and some short wavelength UVB (280-320 nm) are filtered by an intact ozone layer. Ultraviolet A (320-420 nm) and visible light (400-700 nm) are typically the most damaging radiation reaching the conjunctiva, and contributing to the development of pinqueculas, a pathological overgrowth of conjunctival epithelium. Radiation not only impacts the cornea but also transverses this tissue itself.

Even on a cloudy day, 60 to 80 per cent of the sun's rays can pass through the clouds and 50 per cent of the UVA received by the eye comes from reflected surfaces.[3] In addition, radiometric data compiled by Wysecki and Stiles suggest that sunlight (6500 K colour temperature) contains as much as 25 per cent blue light. The atmospheric phenomenon of 'blue haze' comprises an even greater 40-50 per cent blue light.[4]

Lens impact

The primary function of the lens is to transmit undistorted light to the retina. With the exception of aphakic patients and pseudophakic patients, all UV radiation below 295 nm is effectively filtered by the cornea. The human lens absorbs most UVA light, with the exact wavelength absorbed dependent on age. That is, very young lenses transmit UV to the vitreous/retina while ageing biologic lens 'chromophores' filter out most of the blue visible spectrum (400-500 nm). These yellow chromophores are 3-hydroxy kynurenine and its glucoside. However, as the lens ages, chromophores that were once protective are biochemically modified, producing phototoxic singlet oxygen.

The lens contains only two types of cells, an anterior lens epithelial layer and lens protein crystalline(s) making up the bulk of lens tissue. With age, there is a decrease in production of antioxidants and antioxidant enzymes combined with a defective ubiquitin lenticular protein repair system. As there is little lens cellular turnover, the process is insidious and cumulative, affecting not only retinal image quality but also the circadian rhythm or 'biological clock' serving sleep and endocrine competence. Regardless, the end result is a change in transparency of the lens, with clouding (cataract).

Cataract is the major cause of world-wide legal blindness. UVA and blue radiation induced oxidative stress on the human lens epithelial cells is the most important factor in cataract formation. LED lights having a colour

Continued page 7

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Essilor Crizal® Prevencia® lenses are Class I medical devices intended for the correction of ametropias and presbyopia and offering selective protection from harmful blue light and UV rays. Essilor informs you that the above information is general information given as prevention and public awareness. For more information Essilor invites you to consult a healthcare professional.

(1) E-SPF® is an index rating the overall UV protection of a lens. E-SPF® was developed by Essilor International and endorsed by third party experts. The E-SPF® index relates to lens performance only and excludes direct eye exposure that depends on external factors (wearer's morphology, frame shape, position of wear). E-SPF® 25 index for all Crizal® Prevencia®

(2) The Blue-Violet light cut may slightly differ depending on lens material. In vitro experiments conducted by Essilor and Paris Vision Institute, Retinal Pigment Epithelium cells were exposed to Blue-Violet light, reproducing the physiological exposure to sunlight of the 40-year-old human eye.

UV and blue light

From page 5

temperature of 7378 K have been shown to cause overproduction of reactive oxygen species of human epithelial lens cells causing severe DNA damage, which triggers G2/M cell mitosis arrest and apoptotic cell arrest.[5]

Vitreous impact

The vitreous is an extracellular matrix comprising primarily water but also collagens and hyaluronan organised into a homogeneously transparent gel. The naturally ageing human lens limits short wavelength radiation to this tissue, by virtue of chromophores. However, blue light and stray UVA photons are able to reach back into the vitreous cavity. Scientists have not yet explored the role of light in the degeneration of the vitreous and formation of vitreal floaters, but the potential for UV and blue light damage is real.

Retinal impact

Visible light can cause retinal damage by photothermal but especially photochemical mechanisms. Blue light traversing the cornea and lens is able to reach the retina.

This 'bad blue' is particularly damaging to young children who do not have the natural build-up of crystalline lens pigment that comes with age to help protect them. As well, most post-operative pseudophakic patients are also vulnerable. Since children do not have the natural build-up of crystalline lens pigment, they need to be wearing blue light lenses and intake vegetables and fruits that are high in antioxidants and dietary carotenoids.

On the macro level, there are three levels of experimental cellular mechanistic evidence in murine (mice) retinal models that blue light is capable of damaging the retina. These include:

- histological retinal thinning
- visual electroretinogram degradation
- immuno-histiochemical probes of increased double strand DNA breaks.[6]

There is now also putative biochemical and subcellular level evidence for the detrimental effect of blue light. Murine cells irradiated with blue light induce reactive oxygen species (ROS) production, ultimately resulting in apoptosis and photoreceptor cell death by both oxidative and endoplasmic reticulum stress using four distinct processes:[7]

- subcellular mitochondrial damage and up-regulation of cytochrome C
- photoreceptor S-opsin aggregation and endoplasmic reticulum stress
- cell signaling MAPK activation and nuclear translocation of NF-kappa beta induces caspase activation
- NF-kappa beta over-stimulation induced autophagy leading to cell death.

Ultimately, another chromophore called lipofuscin builds up in the retina with age and environmental assault. Lipofuscin is formed from incompletely digested photoreceptor outer segments and includes a molecule called A2e, which inhibits subcellular phago-lysosomal degradation of photoreceptor phospholipids. Blue light excites lipofuscin, producing damaging phototoxic singlet oxygen and secondary lipid peroxy radical chain reactions leading to photoreceptor death.

These lines of basic evidence are consistent with the epidemiologic results of The Beaver Dam Study, a longitudinal, population-based study of subjects age 43-86 years examined at baseline and five years later, for risk of age-related macular degeneration (AMD) against sunlight exposure, using questionnaires.[8]

Leisure time spent outdoors during teenage and 30-39 years was significantly associated with the risk

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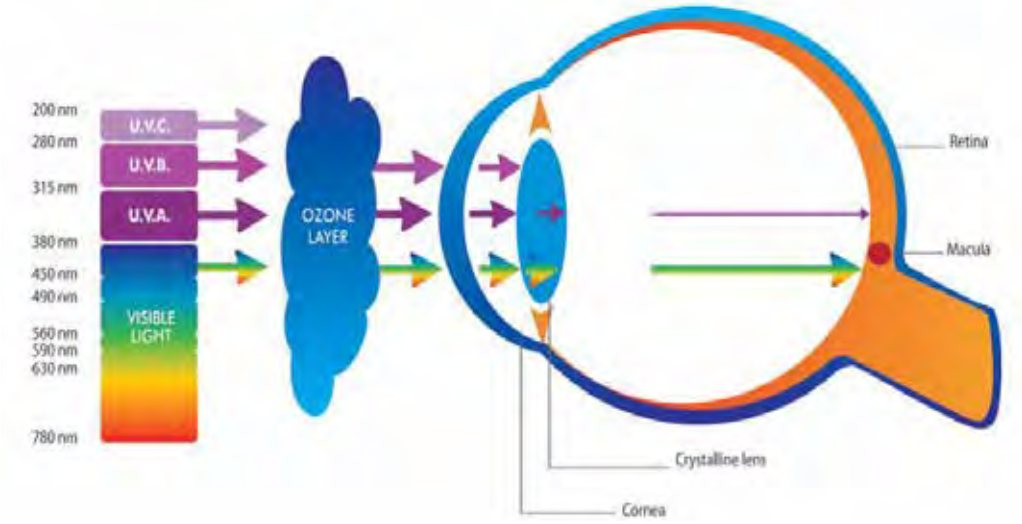

▲ Figure 1. Absorption and transmission of solar radiation of the eye. The cornea and crystalline lens filter out UVB and most UVA, so that the most energetic light reaching the retina is short wavelength blue-violet light. Image: Blue Light Hazard: report of a roundtable, March 16 2013. NY, NY, USA

Solutions for myopia control

RECENT AND PROMISING ideas for preventing myopia include pharmaceutical intervention, increased outdoor activity and multifocal contact lenses, a meeting in Europe of cataract and refractive surgeons has been told.

Dr Nina Jacobsen said that low-dose atropine was one of the most promising treatments, and that the concern had always been the side-effects, but it seemed with the low-dose atropine the side-effect was very small and insignificant.

Dr Jacobsen pointed to the considerable amount of evidence that children who spend more time outdoors during daylight hours were less likely to be or become myopic. Studies in Taiwan and China had shown reductions in incident myopia of 25 per cent to 50 per cent when these children spent an additional 40 to 80 minutes a day outdoors, she said.

'Recent studies have shown that the use of multifocal contact lenses in children with myopia may slow the progression of myopia, possibly by addressing the regulation of eye growth at the peripheral retina, which is now believed to play a role,' Dr Jacobsen said.

The prevalence of myopia in children five to 15 years old is increasing worldwide, even as researchers are seeking solutions to slow the trend. The complications related to myopia include increased risk of glaucoma, cataract, retinal detachment and in the higher degrees, maculopathy.

Jacobsen N. Recent developments in myopia control. Presented at: 34th Congress of the European Society of Cataract and Refractive Surgeons; 10-14 Sept 2016; Copenhagen, Denmark.

UV and blue light

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of early AMD (OR 2.09; 95% CI, 1.19-3.65). There was a protective effect for use of hats and sunglasses during teenage and 30-39 years (OR 0.72; 95% CI, 0.50-1.03). As well, lightly pigmented red-haired or blond-haired subjects were slightly more likely to develop early AMD than people with darker hair (OR 1.33; 95% CI, 0.97-1.83).

Uveal melanoma is the second-most common primary malignancy of the eye world-wide next to childhood retinoblastoma and is the principal fatal intraocular disease in adults. Cumulative epidemiological and experimental evidence now indicates that blue light is a credible risk factor for the development of this cancer.[9]

Conclusion

Ophthalmic lens manufacturers have embraced blue lens protection with a spectrum of new spectacle products. Those that block both UV and blue light are most desirable. The following population groups deserve protection:

- infants and toddlers younger than three years with virtually transparent tissue
- young children and teenagers exposed to blue light displays
- older patients who have lost their natural ocular lens
- persons with a family history of AMD or other retinal disease
- persons on photo-sensitising medications
- persons in occupations in manufacturing, lighting installation and outdoors work; surgeons, dentists, athletes
- persons with prolonged exposure to blue light just before bed or 'iPad insomnia'.

While the damage to the human eye from UV and blue light radiation is going to depend on intensity, chronicity and wavelength, as well as the health and age of the subject, there is an obvious need for ophthalmic radiation-blocking technology. Such protection should begin as early in life as possible. Beginning in middle age, antioxidant protection is depleted, leading to the formation of age-related cataracts and macular degeneration.

Another important consideration is the clinical measurement of macula pigment optical density. The spectrophotometric curve for the dietary (lutein and zeaxanthin) carotenoids align precisely with the action spectra of damaging 'bad blue'. Denser macula pigment improves visual performance, and provides superior driving vision and emerging cognitive benefits while protecting against blue light.[10,11] Thousands of practices in the United States and Europe are now offering macula pigment measurement to their patients.[12]

The use of UV and blue light blocking lenses, as well as dietary carotenoid supplementation, can have a dramatic effect on the protection of vulnerable ocular human tissues, and make a positive contribution to public health and society. ▲

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The ocular surface in glaucoma patients

PATIENTS with ocular surface disease and open-angle glaucoma (OAG) taking anti-glaucoma medication showed greater reductions in tear lipid layer thickness than those without OAG, according to researchers in *Optometry and Vision Science*.

The researchers also found that the total duration of anti-glaucoma medication use was significantly correlated with tear lipid layer thickness.

A total of 85 eyes in 85 patients were included in the study. There were 34 eyes in the control group, who were ocular surface disease patients without OAG, and 51 eyes in the group of ocular surface disease patients with OAG and using topical anti-glaucoma medication. Ocular surface disease was evaluated with lipid layer thickness using an interferometer, tear break up time, total cornea and conjunctival staining, and ocular surface disease index (OSDI).

The researchers found several correlations only in the OAG group: OSDI yielded significant correlations with number of medications and daily number of drops, and mean deviation also showed a significant relation with OSDI. For ocular surface parameters, only average interferometric colour units showed significantly lower values in the OAG patients.

The authors concluded that greater tear lipid layer damage represented as a thinning of the lipid layer thickness can be characteristic of ocular surface disease associated with topical anti-glaucoma medication use.

Lee SY, et al. *Optom Vis Sci* 2016; doi: 10.1097.

Topical cyclosporine for dry eye

CsA has a role in controlling ocular inflammation and co-pathologies of the ocular surface

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DRY EYE disease (DED) or keratoconjunctivitis sicca (KCS) as defined by the 2007 International Dry Eye Workshop is a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance and tear film instability with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface.[1]

Significant improvements to evaporative DED treatment have emerged recently with BlephEx, IPL, Lipiflow and other products; however, treatment of other vital components of this multifactorial disease have either not progressed or are unavailable or inaccessible in Australia.

Aqueous deficient dry eye is separated into Sjögren's-syndrome dry eye and non-Sjögren's-syndrome dry eye. The mainstay treatment for both aqueous deficient dry eye diseases is artificial tear lubricants. Significant developments in artificial tear lubricants have aided treatment but severe aqueous deficiency particularly in Sjögren's syndrome is usually a long, uncomfortable battle for the patient.

DED promotes inflammation of the ocular surface. The inflammatory cascade is activated and inflammatory mediators are released into the tears. The result is tear film instability and increased tear osmolarity: a cycle of activity.[1] Topical steroids are highly effective at treating inflammation and improving ocular symptoms but long-term usage is not advised due to the well-known side-effects and immunosuppression.

Topical cyclosporine A (CsA) was approved by the US FDA in 2002 and released to market in 2003. It is currently unavailable in commercial form in Australia but as it is on the Optometry Board of Australia drug list for optometrists, it can easily be compounded via appropriate pharmacies.*

Topical CsA is an immunomodulator which improves tear secretion by reducing the suppression of tear production caused by inflammation.[2] Indications for usage are aqueous deficient dry eye and ocular surface inflammation treatment.[2] A recent systematic review and meta-analysis revealed twice-daily topical CsA resulted in significant improvements to: reflex tear production (Schirmer test without anaesthesia), tear break up time, goblet cell density, corneal fluorescein staining, and ocular surface disease index scores.

Interestingly, the findings of this review found topical CsA to be effective independent of the type of DED (aqueous deficient, evaporative, Sjögren's syndrome and non-Sjögren's syndrome). The understanding is that topical CsA reduces T lymphocyte-mediated inflammation which is related to both aqueous deficient and evaporative DED.[3] The increase in goblet cell density may improve ocular surface health and tear film stability.

Long-term topical CsA use has been found to be well-tolerated for up to three years.[3] Mild to moderate adverse effects have been found to be more common in CsA compared to control groups. The main concern regarding cyclosporine use is systemic absorption due to the side-effects of oral cyclosporine, notably nephrotoxicity and immunosuppression; however, topical CsA emulsion has been found to have no systemic blood levels after nine to 12 months of use with 0.05% twice daily.[4]

CASE REPORT

A long-standing now 67-year-old female patient presented on 25 March 2014 with another episode of sore uncomfortable eyes, particularly with near fine work and air conditioning. She had a long history of dry eye disease, Salzmann's nodular degeneration and nasal pterygia. Her left pterygium and Salzmann's nodules had been removed three years prior to the appointment. Her systemic medications included hormone replacement therapy and antihypertensive medication. Previous treatment was intensive non-preserved Viscotears and pulsed topical steroid treatments (FML) during symptomatic inflammatory episodes.

Examination showed only trace corneal epithelial staining and meibomian gland dysfunction, but there was conjunctival redness and inflammation of the ocular surface. As these episodes were becoming increasingly regular, the decision to start compounded topical CsA 0.05% twice daily with her existing Viscotears artificial tear lubricants was made rather than another pulsed steroid treatment.

At one month review, symptoms had eased with the patient having 'bad days' only once or twice a week and she had reduced use of Viscotears to twice daily herself. At two months review, she reported only occasional sore eyes and examination showed reduced redness and ocular surface

inflammation. At three month review, she reported her eyes to be 'behaving well' and continued both topical CsA 0.05% and Viscotears twice daily. The patient continued using topical CsA for another year and discontinued only due to compounding supply problems. In 2016, her right pterygium had progressed from the previous year and required removal.

Discussion

This case shows the successful role topical CsA plays in controlling ocular inflammation in dry eye disease and co-pathologies of the ocular surface. In this case, a patient was symptomatic daily prior to treatment and was asymptomatic after three months of treatment.

Inflammation has involvement in DED as previously discussed, but also in Salzmann's nodular degeneration and pterygium.[5,6] Post-pterygium surgery, topical CsA has been shown to reduce recurrence and to inhibit the proliferation of pterygia and normal Tenon's capsule fibroblasts.[7,8] Salzmann's nodular degeneration most often occurs following chronic ocular surface disease involving the corneal epithelium. This results in high metabolic and proliferating activity of the epithelium so controlling chronic ocular surface disease should help prevent Salzmann's nodular degeneration occurrence.[5]

In this case, using topical CsA to treat the primary dry eye condition also benefitted her Salzmann's nodular degeneration and pterygium, or prevented pterygium recurrence. Inflammation of the ocular surface is a key factor in dry eye pathology. Topical CsA has been shown to effectively reduce T lymphocyte-mediated inflammation to manage DED. Importantly, CsA is effective not just for aqueous deficiency and inflammation, but also for evaporative DED as well. ▲

* In every state except Queensland.

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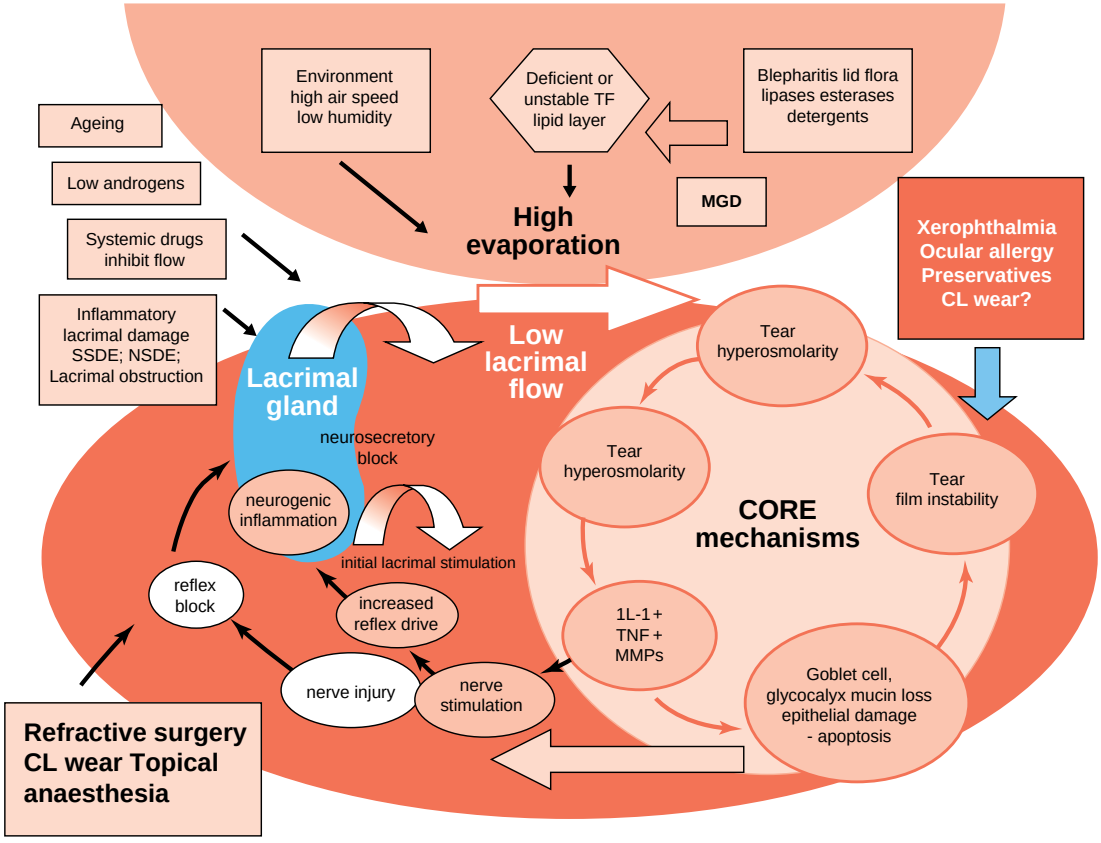

▲ Figure 1. Mechanisms of dry eye[9]

Anterior ocular pathology and ocular surface disease

Management with contact lenses

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THE 2007 REPORT of the International Dry Eye Work Shop (DEWS) discussed the role of contact lenses to protect and hydrate the corneal surface in severe dry eye conditions. Several different contact lens materials and designs have been evaluated, including the use of silicone lenses and scleral contact lenses.[1,2,3,4,5,6]

The advantages include resolving corneal desiccation, improved visual acuity and comfort, decreased corneal epitheliopathy, and healing of persistent corneal epithelial defects.[7,8,9,10,11]

In some circumstances, for very extensive corneal epitheliopathy, the use of an appropriate high dK lens for overnight extended wear may be appropriate to allow the corneal surface to heal optimally and the fragile epithelium to best recover from mild to severe long-standing anterior ocular pathology, including superficial punctate keratitis, bullous keratopathy and many other conditions, as long as the risk of adverse infectious events is minimised through appropriate precautions around hygiene and contact lens care.

There is acknowledgement that these are corneas at higher risk for contact lens-related adverse events such as inflammatory and infectious events and ocular complications such as corneal neovascularisation. However, the symptoms of pain and visual impairment due to severe dry eye signs and symptoms can warrant the use of a bandage contact lens. This allows a considerable degree of improvement to the patient's quality of life.

CASE REPORT 1

Bandage contact lens use for superficial punctate keratitis and reduction in visual acuity and ocular surface disease management of dry eye with Dailies Total1

Triska* is a 30-year-old female and has been a patient in our practice since 2011. Her chief concern is her constant blurry vision, her RE much more than LE. She has no dry eye symptoms.

She was initially wearing Acuvue 1-Day TruEye occasionally, one to two days a week. Triska had no Roaccutane history, no autoimmune conditions and no ocular rosacea history.

Refraction: RE -1.75 6/9+2, LE -2.75 6/6.

Slitlamp signs: considerable central corneal superficial punctate keratitis staining. Moderate to marked conjunctival staining.

Tear break up time was four seconds both eyes.

There were no signs of meibomian gland disease, no ocular rosacea and no teleangiectasia. However, the patient did have low tear volume and evaporative dry eye signs with vertical breaks in tear film.

Diagnosis: dry eye disease with constant, moderate to marked effect on vision, DEWS Category 3.

Strangely, the patient reported no pain or soreness. The incongruity of ocular signs and symptoms was noted in dry eye ocular surface disease.

Management

Triska was prescribed TheraTears nutrition twice a day and Systane Hydration as needed but minimum four times a day.

The patient was given Alcon Dailies Total1 as a bandage contact lens and advised to wear predominantly contact lenses instead of glasses.

Daily wear only of Dailies Total1 for two weeks showed considerable improvement in superficial punctate keratitis.

RE 8.5/14.1/-1.75; improved from 6/9+2 to 6/6 with considerable improvement in corneal epithelial surface staining.

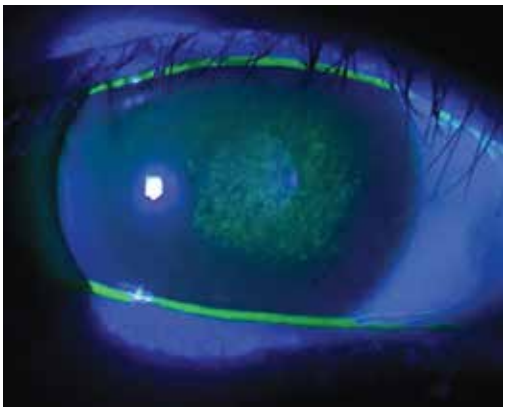
LE 8.5/14.1/-2.75; maintained 6/6 vision with full resolution of corneal epithelial staining.

Results on two-week follow-up showed considerable reduction in superficial punctate keratitis and subsequent improvement in visual acuity from considerable ocular surface resolution with RE improving from 6/9+2 to 6/6.

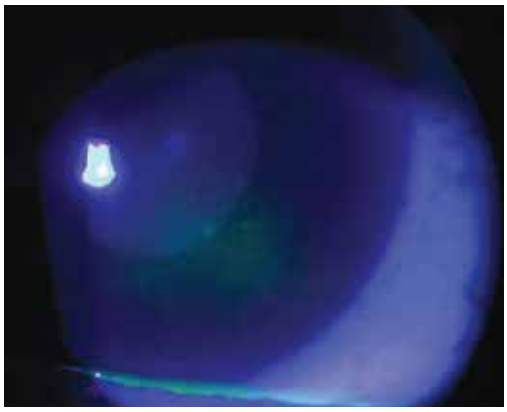
CASE REPORT 2

Bullous keratopathy, advanced bilateral keratoconus, infectious crystalline keratopathy, bandage contact lens RE and speciality scleral contact lenses

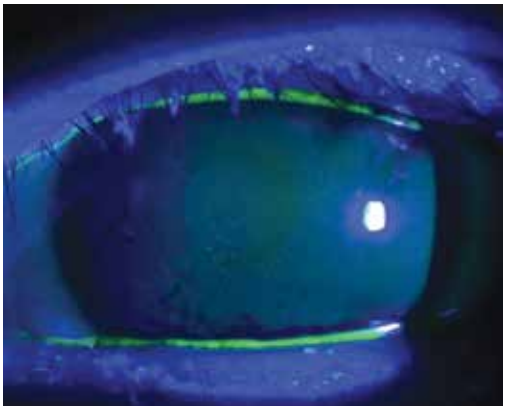
Barry* is a 70-year-old male who presented with bilateral keratoconus, and bullous keratopathy secondary to a long-standing full-thickness penetrating keratoplasty corneal graft starting to fail. His condition had been managed by bandage contact lens; however, the condition had escalated



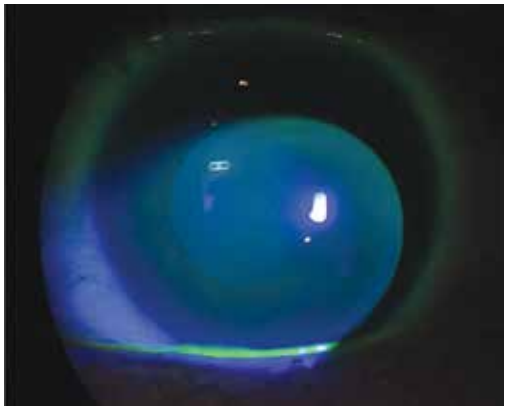
▲ Figure 1. CASE REPORT 1. BEFORE: Triska was prescribed TheraTears nutrition twice a day and Systane Hydration as needed but minimum four times a day.



▲ Figure 2. CASE REPORT 1. AFTER: Triska was prescribed TheraTears nutrition twice a day and because she had no dryness symptoms was advised to apply Systane Hydration when noticing blurriness but also minimum four times a day.



▲ Figure 3. CASE REPORT 1. BEFORE: The patient was given Alcon Dailies Total1 as a bandage contact lens and advised to wear predominantly contact lenses instead of glasses.



▲ Figure 4. CASE REPORT 1. AFTER: The patient was given Alcon Dailies Total1 as a bandage contact lens and advised to wear predominantly contact lenses instead of glasses.

into infectious crystalline keratopathy, which meant that he would require another penetrating keratoplasty graft soon.

Signs in the RE were consistent with bullous keratopathy, which included corneal stromal haze secondary to corneal endothelial compromise in an ageing (approximately 15-year-old) full-thickness corneal penetrating keratoplasty graft, and subsequent corneal oedema and painful erupting bullae. As a temporary fix, he was wearing an extended-wear, high-dK monthly bandage contact lens, and co-managed by an ophthalmologist. His bullous keratopathy was consistent with signs of graft failure.

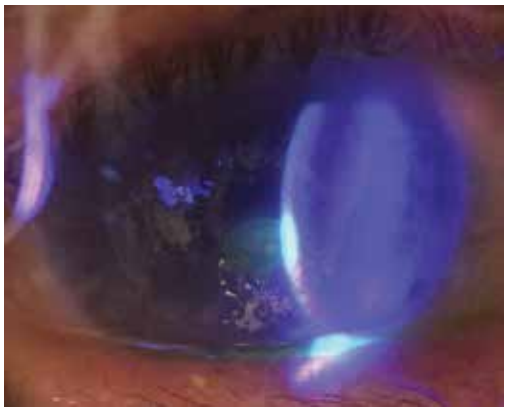
The patient's RE started to develop infectious crystalline keratopathy, which was very challenging to differentially diagnose against the painful, subepithelial erupting bullae on the right cornea coming from the bullous keratopathy. The subepithelial fluid-filled bullae that erupt on the surface cause symptoms of pain similar to those caused by the new infectious crystalline keratopathy.

Infectious crystalline keratopathy as defined by Sharma and colleagues is a corneal infection in which thin, branching crystalline opacities are seen within the corneal stroma, yet no corneal or anterior segment inflammation occurs.[12] In most cases, it occurs as a secondary complication of corneal surgery and keratitis, where streptococcus is a highly suspected microbial causative pathogen.[12]

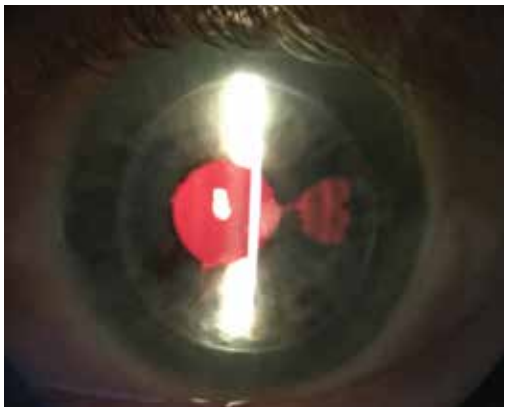
Management can be aggressive antibiotic therapy and adjunctive discontinuation of topical steroids, but in more serious cases where there is continued infection (corneal vascularisation or scar formation that adversely affects visual acuity) it requires treatment by penetrating keratoplasty.[12]

The patient was referred for a speciality contact lens fitting on his LE. He had advanced bilateral keratoconus, a longstanding penetrating keratoplasty corneal graft, and LE iris atrophy. In addition, he had had a LE retinal detachment and subsequent retinal surgery. The current LE rigid contact lens was more than four years old and the lens was displaying apical bearing on the corneal graft. This raised concerns of future apical scarring on the LE. The lens also displaced frequently off the eye due to a suboptimal fitting. Barry was prescribed a custom scleral contact lens for his LE.

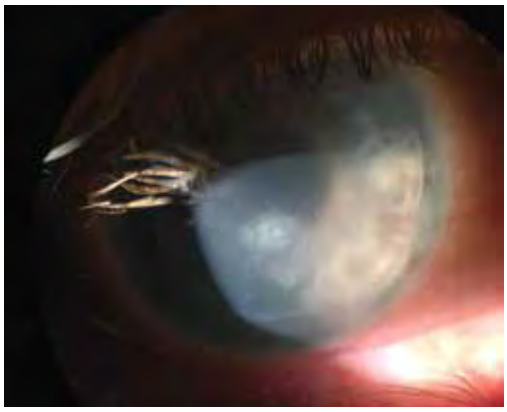
Continued page 14



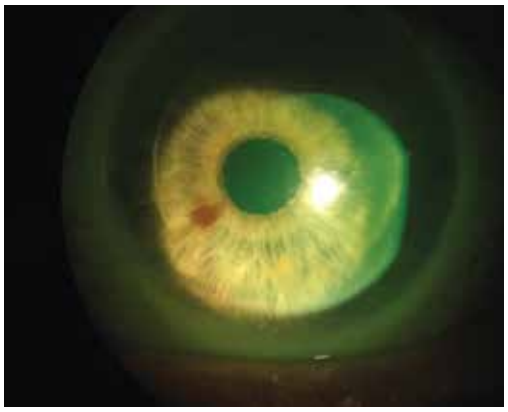
▲ Figure 5. CASE REPORT 2. RE bullous keratopathy, infectious crystalline keratopathy



▲ Figure 6. CASE REPORT 2. LE iris atrophy, full thickness penetrating keratoplasty corneal graft and advanced keratoconus



▲ Figure 7. CASE REPORT 2. RE escalating crystalline keratopathy and early signs of graft rejection



▲ Figure 8. CASE REPORT 2. LE post graft speciality scleral contact lens fitting

CL management

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Entering visual acuity RE was counting fingers at 30 cm, LE 6/9- but the lens needed changing due to heavy apical bearing and risk of future apical scarring.

Management

RE: full thickness penetrating keratoplasty corneal transplant was prioritised on surgical list at Sydney Eye Hospital due to extensive bullous keratopathy as well as recent development of infectious crystalline keratopathy. LE: prescribed Innovative Contacts scleral contact lens.

Habitual visual acuity RE counting fingers at 30 cm, LE 6/9-

Innovative Contacts Custom scleral contact lens prescribed LE 8.40/3900/Increased Flat/16.5/+1.75

Outcome: Post scleral contact lens, VA LE 6/7.5+2.

Lens displaying no conjunctival indentation or subconjunctival vessel blanching and nicely centred (as per figure 8).

LE lens: the patient commented after the two-week follow-up: 'The left eye lens is comfortable. I am really enjoying the crisp vision and the lens is no longer falling off easily, unlike the previous lens.'

*Names have been changed for patient anonymity

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FAF makes the invisible visible

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THE PAST TWO decades have witnessed an unprecedented explosion of innovation in eye imaging. These technologies have transformed the humble, routine eye examination into a plethora of tests integrating new technologies, such as optical coherence tomography (OCT), scanning laser ophthalmoscopy and fundus autofluorescence.

These techniques complement traditional methods of detecting structural signs of early disease and consequently may provide earlier and more accurate disease detection; still, the full utility of these tools may be under-appreciated.

Fundus autofluorescence (FAF) is an advanced imaging modality that provides insight into the health of the retinal pigment epithelium (RPE). The FAF signal derives predominantly from lipofuscin granules in the RPE, which accumulate as by-products of incomplete degradation of photoreceptor outer segments.[1,2] Consequently, abnormal patterns can be interpreted as signs of current or future oxidative stress.

Excessive accumulation of lipofuscin is also recognised as a common pathogenetic pathway in various retinal diseases, including age-related macular degeneration (AMD), which precedes photoreceptor degeneration.

Alternative locations for FAF in the outer retina,[3,4] photoreceptor outer segment[5] and subretinal space[4] and sources, such as macrophages in the new vessel complex,[5] choroidal components[3] and the sclera, have also been suggested. Consequently, FAF imaging provides complementary and at times, unique information compared to other modalities such as colour fundus photography).[3,6-8] As in the case that follows, FAF may often reveal signs of marked retinal dysfunction in areas that appear normal on funduscopy.

CASE REPORT

A 72-year-old Caucasian female was referred to Centre for Eye Health for a macular assessment based on symptoms of an intermittent 'dark blob' in her central vision, most noticeable at night. She had been diagnosed with dry AMD about one year previously and had a medical history of chronic obstructive pulmonary disorder, hypertension, sleep apnoea and was a former smoker. She took the medications Cartia (aspirin) and Betaloc (metoprolol) regularly. Her mother had glaucoma.

Aided visual acuities were 6/9.5+1 OD and 6/15+2 OS and pinhole acuities were 6/9.5 OD and 6/9.5-2 OS. Contrast sensitivities were within normal limits in each eye at 1.60 log units OD and 1.64 log units OS using the MARS test (normal range 1.52-1.76 log units for patients over the age of 60 years). Amsler grid testing revealed the appearance of 'kinked' and mildly jagged lines inferior to fixation and in the upper left corner OD; a small area of displaced lines temporal to fixation were also perceived OS. Anterior eye examination was unremarkable.

Funduscopy, retinal photography and OCT revealed small to large, calcified (crystalline and glistening) macular drusen OU. Geographic atrophy and pigment clumping were also present in the parafoveal region OU. FAF imaging using the Spectralis HRA2 revealed distinct multilobular, extrafoveal areas of hypo-autofluorescence representing geographic atrophy OU, more numerous OD than OS, and a surrounding pattern of 'diffuse trickling' abnormal autofluorescence (Figures 1 and 2, top row).

The patient was diagnosed with bilateral advanced (atrophic) AMD. A review consultation with subsequent imaging was recommended in six months. Other pertinent management considerations included interim daily self-monitoring with an Amsler grid, and the role of AREDS 2 supplements and a healthy diet in reducing the risk of progression.

Follow-up testing one year later revealed no significant change in visual acuities (6/9.5+1 OD and 6/12 OS that improved to 6/9.5+1 OD and 6/12+2 OS with pinhole). Contrast sensitivity was apparently reduced in each eye compared to baseline at 1.44 units OD and 1.40 units OS with the MARS test.

The patient denied noticing any changes on the Amsler grid through self-monitoring. Imaging showed progression of the areas of geographic atrophy including enlargement of existing areas OU, confluence of adjacent areas OD and the development of new areas OU. The progression was best visualised using FAF (Figures 1 and 2, bottom row).

Discussion

Our case study highlights several applications of FAF imaging. FAF images can be obtained clinically using a scanning laser ophthalmoscope[1] such as the Spectralis Heidelberg Retina Angiograph HRA classic or HRA2 (Heidelberg Engineering, Heidelberg, Germany) or Optomap ultra-widefield imaging (Optos, Dunfermline, Scotland, UK).

Alternatively, clinical FAF images may be obtained more inexpensively using a commercially-available fundus camera and standardised filters.[9] However, FAF images obtained using a fundus camera may be inferior, of lower contrast, with higher background noise and more susceptible to degradation by media opacities, compared to a scanning laser ophthalmoscope[10,11] owing to indirect light captured from structures outside of the plane of focus, which is unavoidable in a flash photography system.

Continued page 16

FAF imaging

From page 15

Wavelength variations between the systems are also likely to cause subtle differences in the final FAF image and associated appearance of lesions; however, the clinical findings overall are still likely to be comparable[11,12] and images from different systems can be interpreted using the same framework of evidence.

The final output of FAF imaging appears as an en face, 'front on', image of naturally or pathologically occurring fluorophores, which allows the clinician to visualise the spatial distribution and intensity of autofluorescence across the fundus. The field of view may vary between 30 degrees and 200 degrees, depending on the instrument employed. At present, clinical evaluation of case images is primarily performed qualitatively and may require post-acquisition adjustments in brightness and contrast.[13]

The imaging results of this case correlated consistently with other functional and structural findings,[14] such as the patient's chief complaint and OCT. In particular, FAF allowed for a refined diagnosis and provided a better indication of the extent of retinal dysfunction than OCT or other modalities.

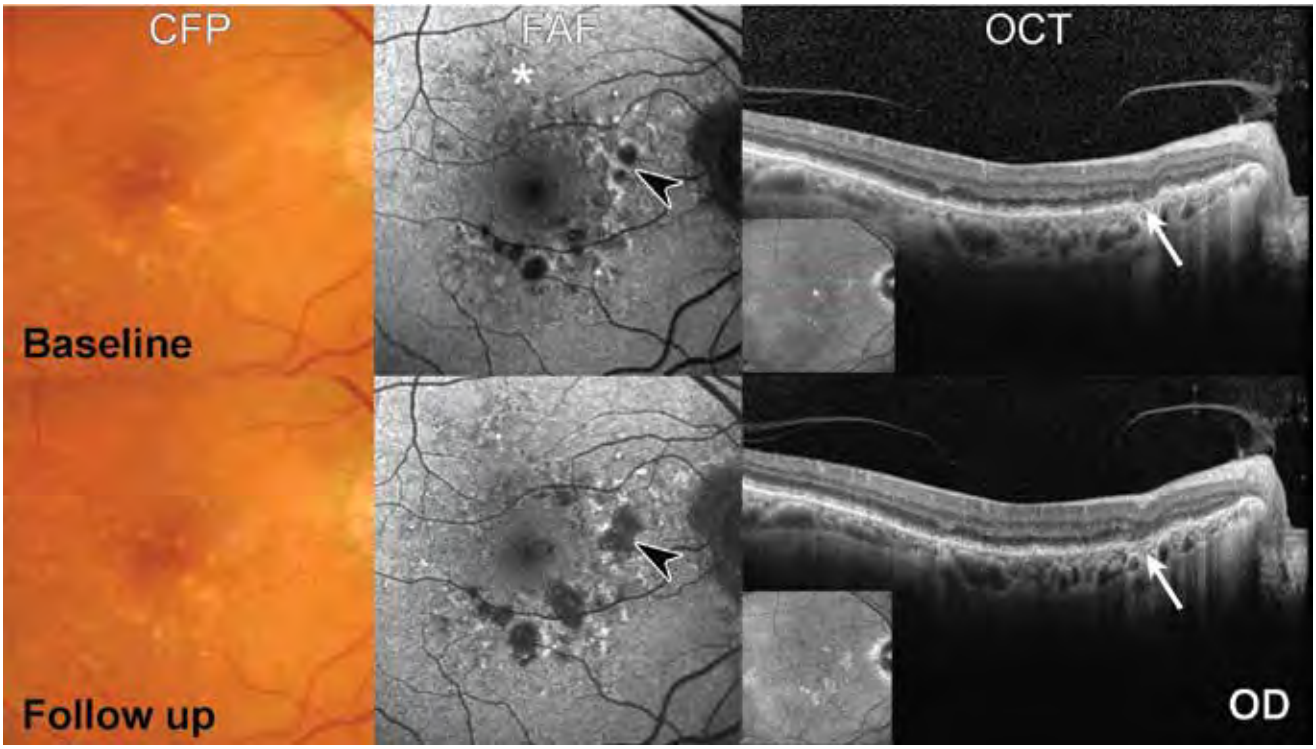
In AMD, areas of geographic atrophy may be difficult to identify using funduscopy or retinal photography alone due to variations in fundus pigmentation and the presence of small, satellite lesions. Had the extent of these areas been underestimated, the true diagnosis of advanced AMD may have been misclassified as intermediate.

In this case, FAF also enabled the identification of a high-risk phenotype, characterised by the 'diffuse trickling'[8] signature in the junctional zone surrounding the atrophic area. The clinical implication of this finding is that the patient needs to be more closely monitored for the potential development of choroidal neovascularisation.

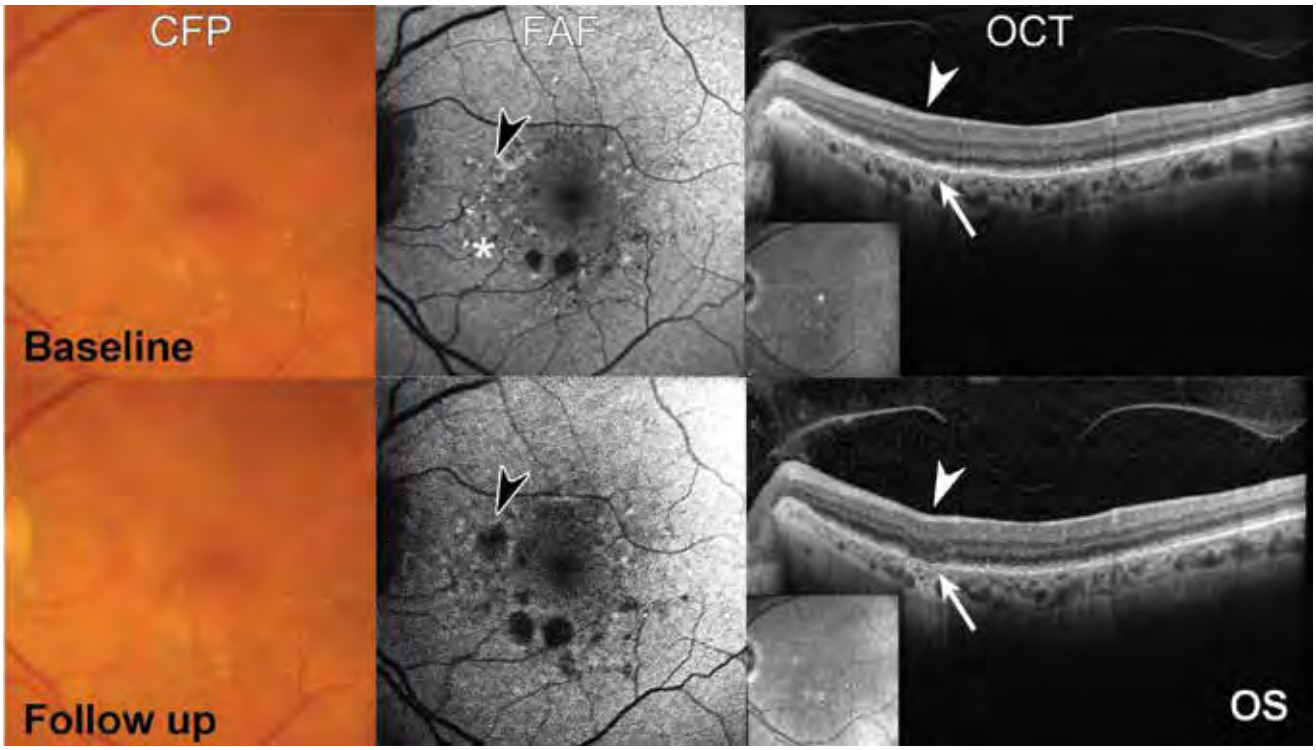
Finally, follow-up FAF imaging allowed for disease progression to be more easily recognised and documented. This information is directly relevant clinically and may be applied to better educate the patient about their increased risk of progression to the exudative form of advanced AMD and to encourage compliance with clinical recommendations, for example, to reinforce the importance of daily self-monitoring with an Amsler grid.

Accordingly, the reasons for using advanced ocular imaging (including FAF) can be summarised as follows:

- to detect ocular disease at an earlier stage, particularly when the results correlate with other structural or functional tests
- to aid in the differential diagnosis of ocular diseases and to specifically stratify phenotypes or stages of disease in order to identify patients at risk of visual impairment
- for closer follow-up of cases or to measure the response to treatment
- for better photo-documentation, patient education and management.



▲ Figure 1. Imaging results from the patient's right eye, obtained at baseline (top) and 12 months later (bottom), showing progression in the areas of geographic atrophy including confluence of adjacent areas (arrowheads) and enlargement of existing areas (arrows). The changes were best visualised using FAF. The baseline FAF image also shows a 'diffuse trickling' or seeping pattern (asterisk) surrounding the areas of geographic atrophy, which has been associated with an eight-fold higher rate of enlargement than other presentations.
CFP: colour fundus photography; FAF: fundus autofluorescence; OCT: optical coherence tomography



▲ Figure 2. Imaging results from the patient's left eye. The black arrowheads show development of a new hypo-autofluorescent area of geographic atrophy, which was confirmed using OCT (white arrows). The follow-up OCT line scan shows the area of geographic atrophy as focal drop-out of the ellipsoid zone and external limiting membrane, and thinning of the outer nuclear layer and RPE. There is increased signal penetration posteriorly and associated collapse of the overlying retinal layers (white arrowhead). As with the right eye, the baseline FAF image also shows a 'diffuse trickling' pattern (asterisk) surrounding the areas of geographic atrophy.
CFP: colour fundus photography; FAF: fundus autofluorescence; OCT: optical coherence tomography

The principles of FAF imaging described in this article using a case of atrophic AMD can also be extrapolated to eye diseases other than AMD.[15]

There has been a clinical paradigm shift toward the inclusion of advanced imaging into routine primary eye care. In the future, we can expect to see a refined understanding and a growth in the collective wisdom regarding the utility of instruments such as FAF in disease detection, stratification and differential diagnosis.

As eye-care professionals, we face the challenge of keeping pace with the evidence base surrounding these new technologies in order to optimally manage our patients. FAF imaging represents a rapid, effective, non-invasive imaging method with significant and often under-estimated applicability.

Acknowledgement

The author thanks Paula Katalinic, Professor Michael Kalloniatis and Michael Yapp for reviewing the article. ▲

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Therapeutic NEWS of note

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Contact lenses for glaucoma? Latanoprost-eluting lenses help glaucomatous monkeys

A preclinical animal efficacy study indicates that contact lens drug delivery may be a future option for the treatment of glaucoma and a platform for prostaglandin drug delivery.

Researchers assessed the IOP-lowering effect of latanoprost-eluting low-dose contact lenses (CLLO), high-dose contact lenses (CLHI) or daily latanoprost ophthalmic solution in glaucomatous eyes of cynomolgus monkeys.

Each monkey consecutively received one week of continuous-wear CLLO, three weeks without treatment, five days of latanoprost drops, three weeks without treatment, and one week of continuous-wear CLHI.

Latanoprost ophthalmic solution resulted in IOP reduction of 5.4 ± 1.0 mmHg on day three and peak IOP reduction of 6.6 ± 1.3 mmHg on day five. The CLLO reduced IOP by 6.3 ± 1.0, 6.7 ± 0.3, and 6.7 ± 0.3 mmHg on days three, five and eight, respectively. The CLHI lowered IOP by 10.5 ± 1.4, 11.1 ± 4.0, and 10.0 ± 2.5 mmHg on days three, five and eight, respectively. For the CLLO and CLHI, the IOP was statistically significantly reduced compared with the untreated baseline at most time points measured. The CLHI demonstrated greater IOP reduction than latanoprost ophthalmic solution on day three ($p = 0.001$) and day five ($p = 0.015$), and at several time points on day eight ($p < 0.05$).

Researchers concluded that sustained delivery of latanoprost by contact lenses is at least as effective as delivery with daily latanoprost ophthalmic solution. They suggested that more research is needed to determine the optimal continuous-release dose that would be well-tolerated and maximally effective.

Ophthalmology 2016; 123: 10: 2085-2092. doi: 10.1016/j.ophtha.2016.06.038.[Epub 2016 Aug 29]

Does keratoconus progress beyond age 30?

According to a study published in the *British Journal of Ophthalmology*, a significant percentage of people with keratoconus exhibit progression beyond age 30.

To determine if significant progression of disease occurs in older, non-contact lens wearing subjects with keratoconus and to identify potential predictive factors, researchers retrospectively analysed clinical and computerised corneal topography records of 449 subjects with keratoconus.

Of those assessed, 43 eyes of 27 patients (6.01 per cent) met inclusion criteria, with median age 38.45 (12.86) years at baseline and median follow-up 4.36 (8.68) years. Topographic parameters assessed included maximum keratometry (Kmax), steep keratometry (Ksteep), flat keratometry (Kflat), inferior-superior (I-S) ratio and the surface asymmetry and regularity (surface asymmetry index and surface regularity index) indices.

The study found that there was a significant increase in Kmax (0.30 [1.21] D), Ksteep (0.27 [0.90] D), Kflat (0.34 [1.12] D) and I-S (0.26 [0.82] D) between baseline and final review, $p < 0.05$.

Notably, 18.6 per cent to 25.6 per cent of eyes demonstrated ≥ 1.00 D increase in one or more of four principal topographic parameters (Kmax, Ksteep, Kflat, I-S ratio), while 18.5 per cent to 37.0 per cent of subjects had ≥ 1.00 D increase in these parameters in at least one eye over the study period. However, less than 10 per cent of eyes exhibited ≥ 1.00 D increase per year in all topographic parameters. The only significant predictor of progression was follow-up time.

The study authors recommended that older subjects with keratoconus should be monitored for progression, particularly with respect to possible corneal collagen cross-linking or astigmatic correction in cataract surgery.

Br J Ophthalmol 2016 Oct 11. doi: 10.1136/bjophthalmol-2016-308682. [Epub ahead of print]

Is purchasing contacts from the prescriber associated with better patient habits?

Researchers conducted an online survey to compare the habits of soft contact lens (SCL) wearers who made their purchases from an eye-care practitioner (ECP), on the internet or over the phone, or at a retail place unaffiliated with the eye-care practitioner who conducted their eye examination. Their results raise questions about the assumed link between patient safety and professional oversight at the time of purchase.

In the survey, 1,057 adult SCL wearers were asked about risk factors for SCL-related complications. A total of 646 SCL wearers (age 44 ± 12 years, 17 per cent male) bought lenses at their ECP; 104 at retail (age 45 ± 13 years, 28 per cent male), and 218 on the internet (45 ± 12 years, 18 per cent male). More males bought at retail ($p = 0.021$). Internet purchases were more likely to be of hydrogel SCLs (45 per cent internet, 34 per cent ECP, 29 per cent retail, $p = 0.0034$).

Internet purchasers were more likely to have more than one year between eye examinations (34 per cent internet, 21 per cent ECP, 17 per cent retail, p =

0.007) and less likely to pay for SCLs with insurance (19 per cent internet, 39 per cent ECP, 29 per cent retail, p < 0.0001). Overnight wear, 'topping up', use of reusable SCLs, and tap water exposures were similar across groups, as were knowledge and attitudes on SCL safety.

The authors concluded that the purchase location of SCL wearers showed limited association with known risk factors for SCL-related inflammation. Wearers who purchased lenses on the internet reported less frequent eye examinations and were more likely to be wearing hydrogel SCLs. Males were more likely to purchase SCLs at retail, not where they had eye examinations.

In this sample, closer access to the eye-care practitioner by in-office SCL purchase did not improve the patient's habits or reduce the prevalence of risk behaviours, leaving the relationship between proximity to eye-care practitioners and the risk of complications unclear.

Cont Lens Anterior Eye 2016 Aug 12. doi: 10.1016/j.clae.2016.08.003. [Epub ahead of print]

Dual focus soft contacts match orthoK in countering myopic progression

According to a study reported in Optometry & Vision Science, both orthokeratology and dual focus soft contact lenses are effective strategies for targeting myopia progression in the clinic.

Researchers reviewed the clinical outcomes for patients attending a specialist myopia control clinic at The University of Auckland Optometry School, New Zealand and presented a comparative case series of 110 patients (aged 4–33 years, mean: 12.13 ± 4.58 years, 62 per cent female) who attended the clinic between 2010 and 2014.

A total of 56 patients were prescribed orthokeratology (ortho-K), 32 wore dual focus soft contact lenses, and 22 received advice only. Mean follow-up time for ortho-K and for dual focus soft contact lenses was the about the same at 1.3 years and 1.33 years, respectively.

There was a significant reduction in the annualised myopia progression in both groups (ortho-K: -1.17 ± 0.55 to -0.09 ± 017 D/yr, p < 0.001; dual focus soft contact lens: -1.15 ± 0.46 to -0.10 ± 0.23 D/yr, p < 0.001). There was no difference between ortho-K and dual focus lens treatment efficacy (p = 0.763), nor in axial or vitreous chamber length changes after treatment (p = 0.184).

Researchers concluded that both ortho-K and dual focus soft contact lenses are effective strategies for targeting myopia progression in the clinic. 'We saw no significant difference in the efficacy of the two methods,' they wrote. 'We believe there are very few barriers for any contact lens practitioner to be actively promoting myopia control treatment to at-risk patients.'

Optom Vis Sci 2016; 93: 9: 1120-1126. doi: 10.1097/OPX.0000000000000957.

Which multipurpose solution is best for protein deposits?

The ability of lens care solutions to remove protein from lenses varies depending on the care solution composition and the polymeric make-up of the contact lens material, according to a report of a study in *Optometry and Vision Science*.

To evaluate the effect of four contemporary lens care solutions on total protein, total lysozyme, and active lysozyme extracted from three contact lens materials, researchers recruited contact lens wearers and randomly assigned them to daily wear of variously etafilcon A, galyfilcon A, or senofilcon A for two weeks.

Four lens care solutions (Biotrue, OPTI-FREE PureMoist, RevitaLens OcuTec, and ClearCare) were used by each subject in random order with a new pair of lenses after an interval between solutions of at least four days. After two weeks of daily wear, contact lenses were collected for analysis.

Higher levels of total protein were extracted from etafilcon A when used with Biotrue compared to other solutions (p = 0.0001). There were higher levels of total lysozyme extracted from galyfilcon A lenses when used with PureMoist than with Biotrue or ClearCare (p < 0.006). Higher total lysozyme was extracted from senofilcon A when used with RevitaLens OcuTec compared to Biotrue (p = 0.002). Lower lysozyme activity was recovered from senofilcon A lenses with RevitaLens OcuTec when compared to all other care solutions (all p < 0.004). When Biotrue, PureMoist, or RevitaLens OcuTec were used, higher total lysozyme was extracted from galyfilcon A compared to senofilcon A (p < 0.01). When RevitaLens OcuTec was used, higher levels of active lysozyme were extracted from galyfilcon A compared to senofilcon A (p = 0.02).

Optom Vis Sci. 2016; 93: 8: 963-72. doi: 10.1097/OPX.0000000000000928.

More evidence for IPL

A new Canadian study suggests that intense pulsed light (IPL) therapy is an effective treatment for dry eye disease.

In a multicentre cohort study, clinical data were reviewed from 100 patients with diagnosis of meibomian gland dysfunction (MGD) and dry eye disease who underwent IPL therapy from September 2012 to December 2014.

On average, patients underwent four IPL sessions. In their review, the researchers found that there was a significant decrease in scoring of lid margin oedema (mean = -0.3; range -1.5 to 0), facial telangiectasia (mean = -0.7; range -2.5 to 0), lid margin vascularity (mean = -1.2; range -2.5 to 0), meibum viscosity (mean = -1.1; range -3 to 0) and OSDI score (mean = -9.6), all with p < 0.001.

There was also a significant increase in oil flow score (mean = 0.9, range -0.5 to 2) and tear break up time (mean = 3.4 seconds, range -2 to 7), both p < 0.001. No significant changes in intraocular pressure or acuity were noted and there were no cases of adverse ocular effects.

The authors conclude that the change in objective signs and OSDI scores show IPL to be a safe and effective treatment for patients with evaporative dry eye disease.'

Can J Ophthalmol 2016; 51: 4: 249-253. doi: 10.1016/j.jcjo.2016.01.005.[Epub 2016 Jun 22]

Management of watery eyes

Underlying causes and treatment of epiphora vary

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WATERY EYE is a common presentation that varies from constant debilitating epiphora to intermittent, wet-feeling eyes.

Ordinarily, tears are swept across the eye by lid closure and the slight lateral to medial slant of the lower lid. The tears are then pumped through the canaliculi, into the lacrimal sac and finally down the nasolacrimal duct to emerge in the inferior part of the lateral nose, under the inferior turbinate. While there is still debate about the precise nature of this 'lacrimal pump,' what is clear is that reasonable tone in the eyelids is important for the efficient movement of tears.

Epiphora results when the capacity of the drainage system to clear tears is overwhelmed by the amount of tears produced. As such, epiphora can be caused by increased tear production, decreased tear clearance or a combination of the two.

The assessment of patients with epiphora begins with a focused history. Constant, severe watering points to an obstruction somewhere in the tear drainage pathway. Intermittent watering in the setting of ocular irritation is more likely to respond to conservative measures.

As well as a general history, I ask specifically about sinus and rhinitis symptoms such as nasal obstruction and facial pressure or fullness. Facial trauma alters the surgical anatomy of the nasolacrimal system. A rare but serious symptom is bloody epiphora which is associated with lacrimal sac tumours.

Clinical examination begins with inspection. Both ectropion and entropion will cause watering. If there is an ectropion, look for any skin lesions that are rolling the eyelid out (Figure 1). The laxity of the lower eyelid is subjectively assessed by distracting the eyelid away from the globe, medially and laterally.

I examine the patient under the slitlamp with a drop of fluorescein in each eye. I assess the tear film height and symmetry, the punctal size and position, corneal and conjunctival staining, tear film break up time and any tarsal papillae consistent with allergic eye disease. Significant conjunctivochalasis occasionally lies over the inferior puncta.

Special tests I perform routinely include the dye disappearance test (DDT) and lacrimal syringing.

The DDT involves placing a drop of 2% fluorescein in the eye and assessing the patient five minutes later. At five minutes, if there is any more than a tiny slither of fluorescein, the test is positive. This is a specific but not particularly sensitive test for outflow obstruction.[1]

I then syringe the lacrimal system. I use 26 gauge cannula on a 3 ml syringe. The canaliculus classically has a 2 mm vertical component and an 8 mm horizontal component. It is important to have the eyelid stretched laterally and to follow this path with the cannula to avoid damaging this sensitive structure. Syringing tells me whether the lacrimal system is completely obstructed and gives some information on where the obstruction is. However, this is not a physiologic test and patients with functional nasolacrimal duct obstruction (NLDO) often still have epiphora despite a patent syringing.

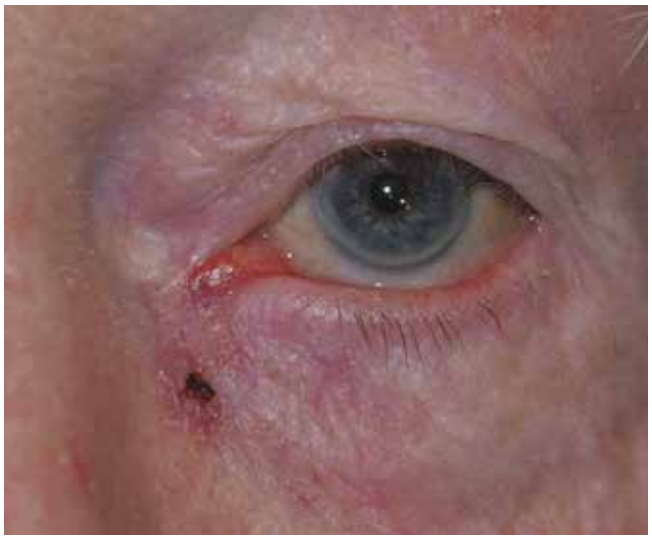
The management of epiphora depends on the underlying cause. Commonly, simple treatments for ocular surface irritation such as warm compresses, artificial tears and anti-allergy drops will relieve the epiphora sufficiently. Punctal stenosis can be treated effectively in the outpatient setting with punctoplasty.[2] Significant conjunctivochalasis can be resected.

In a patient with complete NLDO, the treatment is a dacryocystorhinostomy (DCR) which involves anastomosing the lacrimal sac to the nasal mucosa by removing the bone of the anterio-medial orbit. This can be done either externally or endonasally. Both techniques have advantages and disadvantages as described in Table 1.[3]

Dacryocystitis is a serious complication of NLDO and presents with painful swelling of the lacrimal sac. This often requires intravenous antibiotics to resolve followed by a DCR procedure. It is important to note that dacryocystitis causes a mass below the medial canthal

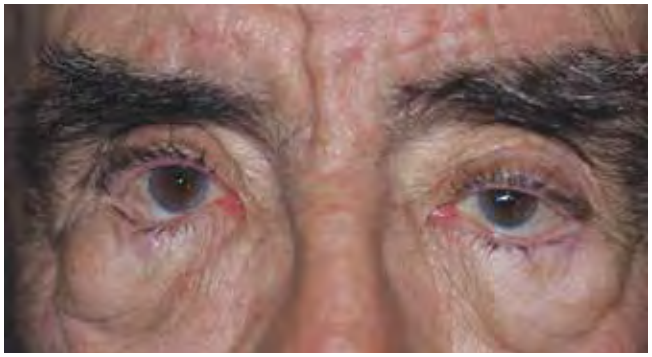
External	Endonasal
90% success	90% success
Small scar	No external scar
Less equipment required	Requires endoscope
Single surgeon procedure	Often ENT and Oculoplastic
Periorbital bruising	No periorbital bruising
Similar complication rates	Similar complication rates
Pump action disrupted	MCT and orbicularis kept intact

▲ Table 1. External and endonasal DCR[3]



▲ Figure 1. A basal cell carcinoma of the left lower lid causing a medial ectropion

▲ Figure 2. A mass above the medial canthal tendon caused by a squamous cell carcinoma



▲ Figures 3A and 3B. Pre-operative (left) and post-operative (right) images following bilateral lower lid tightening procedures to treat epiphora

tendon. If there is swelling above this structure, then a tumour needs to be excluded with imaging (Figure 2).

Lacrimal pump failure requires tightening of the lower eyelid. This can be performed with either a lateral tarsal strip procedure or sutured canthoplasty (Figures 3A and 3B). This procedure can be combined with a punctoplasty or a DCR procedure.

Canaliculitis is a rare but often missed cause of epiphora.[4] Be aware of the patient with chronic, unilateral discharge and epiphora. Look for an erythematous, pouting punctum with granules able to be expressed with compression. This condition requires dilation of the punctum with or without canaliculotomy, expression of the granules and topical antibiotic drops.

Canalicular obstruction is the most challenging condition to treat. This occurs in the setting of inflammation to the canaliculus such as infection, chemotherapeutic agents, allergy, radiation or chronic drop use. If there is sufficient length of patent canaliculus, this can be anastomosed to the lacrimal sac during an external DCR. If there is little proximal patent canaliculus, then various options are available including retrograde intubation of the canaliculus or Jones tube insertion.

The Jones tube is a glass tube which runs directly from the medial conjunctiva to the nose. These require significant maintenance and not infrequently block or become loose; however, they are the current technique of choice when other surgical options have been exhausted. A recently-described technique involves using botox to the lacrimal gland. This has been used for some time in patients with aberrant regeneration of the 7th nerve causing gustatory lacrimation; however, there are recent reports of this technique being used for complete outflow obstruction, not amenable to surgery.[5] ▲

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PBS list of medicines prescribed by optometrists

Revised November 2016

By writing a PBS prescription for an antiglaucoma agent, the prescriber is certifying that the criteria set out in the PBS guidelines are satisfied

	Product	Max qty	Repeats
ANTI-GLAUCOMA PREPARATIONS			
Betaxolol eye-drops, suspension, 2.5 mg (as hydrochloride)/mL, (0.25%), 5 mL	Betoptic S	1	5
Betaxolol eye-drops, solution, 5 mg (as hydrochloride)/mL, (0.5%), 5 mL	Betoptic, BetoQuin	1	5
Bimatoprost eye-drops 300 mcg/mL (0.03%), 3 mL	Lumigan	1	5
Bimatoprost eye-drops 300 mcg/mL (0.03%) 30 x 0.4 mL unit doses	Lumigan	1	5
Bimatoprost with timolol eye-drops containing bimatoprost 300 mcg/mL (0.03%) with timolol 5 mg (as maleate)/mL (0.5%), 3 mL	Ganfort 0.3/5	1	5
Bimatoprost with timolol eye-drops containing bimatoprost 300 mcg/mL (0.03%) with timolol 5 mg (as maleate)/mL (0.5%), 30 x 0.4 mL unit doses	Ganfort PF 0.3/5	1	5
Brimonidine tartrate eye-drops 1.5 mg/mL (0.15%), 5 mL	Alphagan P 1.5	1	5
Brimonidine tartrate eye-drops 2.0 mg/mL (0.2%), 5 mL	Alphagan P 2.0	1	5
Brinzolamide 10 mg/mL (1%) eye-drops containing brimonidine tartrate 2 mg/mL (0.2%), 5 mL	Alphagan, Enidin	1	5
Brimonidine with timolol eye-drops containing brimonidine tartrate 2 mg/mL (0.2%) with timolol 5 mg (as maleate)/mL (0.5%), 5 mL	Combigan	1	5
Brinzolamide eye-drops 10 mg/mL (1%), 5 mL	Azopt, BrinzoQuin	1	5
Brinzolamide with timolol eye-drops containing brinzolamide 10 mg/mL (1%) with timolol 5 mg (as maleate)/mL (0.5%) 5 mL	Azarga	1	5
Dorzolamide eye-drops 20 mg (as hydrochloride)/mL (2%), 5 mL	Trusopt, Trusamide	1	5
Dorzolamide with timolol eye-drops containing dorzolamide 20 mg (as hydrochloride)/mL (2%) with timolol 5 mg (as maleate)/mL (0.5%), 5 mL	Cosopt, Cosdor, Dorzolamide/ Timolol Sandoz 20/5	1	5
Latanoprost eye-drops 50 mcg/mL (0.005%), 2.5 mL	Latanaprost, Xalaprost, Xalatan	1	5
Latanoprost with timolol eye-drops containing latanoprost 50 mcg/mL (0.005%) with timolol 5 mg (as maleate)/mL (0.5%), 2.5 mL	Xalacom, Latanocom, Xalamol 50/5, Latanaprost/Timolol Sandoz 50/5	1	5
Pilocarpine eye-drops containing pilocarpine hydrochloride 10 mg/mL (1%), 15 mL	Isopto Carpine	1	5
Pilocarpine eye-drops containing pilocarpine hydrochloride 20 mg/mL (2%), 15 mL	Isopto Carpine	1	5
Pilocarpine eye-drops containing pilocarpine hydrochloride 40 mg/mL (4%), 15 mL	Isopto Carpine	1	5
Timolol eye-drops 5 mg (as maleate)/mL (0.5%), 5 mL	Tenopt, Timoptol	1	5
Timolol eye-drops (gellan gum solution) 2.5 mg (as maleate)/mL (0.25%), 2.5 mL	Timoptol XE	1	5
Timolol eye-drops (gellan gum solution) 5 mg (as maleate)/mL (0.5%), 2.5 mL	Timoptol XE	1	5
Timolol eye gel 1 mg (as maleate)/g (0.1%), 5 g	Nyogel	1	5
Travoprost eye-drops 40 mcg/mL (0.004%), 2.5 mL	Travatan	1	5
Travoprost with timolol eye-drops containing travoprost 40 mcg/mL (0.004%) with timolol 5 mg (as maleate)/mL (0.5%), 2.5 mL	Duotrav	1	5

NOTE: Antiglaucoma preparation Tafluprost 15 mcg/mL (0.0015%) eye-drops is PBS listed for optometric prescribing. At this time it is not included on the Optometry Board of Australia approved list of drugs that optometrists are authorised to prescribe. As a result, optometrists cannot currently prescribe Tafluprost eye-drops.

	Product	Restriction	Max qty	Repeats
ANTI-VIRAL EYE PREPARATIONS				
		Restricted:		
Aciclovir eye ointment 30 mg/g (3%), 4.5 g	Zovirax	Herpes simplex keratitis	1	0

PBS list of medicines prescribed by optometrists (continued)

	Product	Restriction	Max qty	Repeats
ANTIBIOTICS		Unrestricted		
Ciprofloxacin eye-drops 3 mg /mL (0.3%), 5 mL	CiloQuin, Ciloxan	Authority required: bacterial keratitis	2	0
Framycetin sulfate eye-drops 5 mg/mL (0.5%), 8 mL	Soframycin		1	2
Gentamicin sulfate eye-drops 3 mg/mL (0.3%), 5 mL	Genoptic	Restricted: Suspected pseudomonal eye infection	1	2
Ofloxacin eye-drops 3 mg/mL (0.3%), 5 mL	Ocuflox	Authority required: bacterial keratitis	2	0
Tobramycin eye-drops 3 mg/mL (0.3%), 5 mL	Tobrex	Restricted: Suspected pseudomonal eye infection	1	2
Tobramycin eye ointment 3 mg/g (0.3%), 3.5 g	Tobrex	Restricted: Suspected pseudomonal eye infection	1	0
ANTI-INFLAMMATORY AGENTS				
Dexamethasone eye-drops 1 mg /mL (0.1%), 5 mL	Maxidex	Applications for increased maximum quantities and/or repeats will not be authorised.	1	0
Fluorometholone eye-drops 1 mg/mL (0.1%), 5 mL	Flucon, FML Liquifilm		1	0
Fluorometholone acetate eye-drops 1 mg/mL (0.1%), 5 mL	Flarex		1	0
Hydrocortisone acetate eye ointment 10 mg/g (1%), 5 g	Hycor		1	0
Prednisolone acetate with phenylephrine hydrochloride eye-drops 10 mg-1.2 mg/mL (1%-0.12%), 10 mL	Prednefrin Forte	Restriction: Uveitis. Applications for increased maximum quantities and/or repeats will not be authorised.	1	0
ANTI-ALLERGY AGENTS		Restricted:		
Sodium cromoglycate eye-drops 20 mg/mL (2%), 10 mL	Opticrom	Vernal keratoconjunctivitis	1	5
MYDRIATICS/CYCLOPLEGICS				
Homatropine hydrobromide 20 mg/mL (2%) eye-drops, 15 mL	Isopto Homatropine		1	2
TEAR SUPPLEMENTS		Restricted: Severe dry eye including Sjögren's syndrome		
Carbomer 980 eye gel 2 mg/g (0.2%), 10 g	Optifresh eye gel	As above	1	5
	PAA	As above	1	5
	Viscotears	As above	1	5
Carmellose sodium 5mg/mL (0.5%) with glycerol 9 mg/mL (0.9%) eye-drops, 15 mL	Optive	As above	1	3
Carmellose sodium eye-drops 10 mg/mL (1%), 15 mL	Refresh Liquigel	As above	1	5
Carmellose sodium eye-drops 5 mg/mL (0.5%), 15 ml	Refresh Tears plus	As above	1	5
Hypromellose eye-drops 3 mg/mL (0.3%), 15 mL (contains sodium perborate)	In a Wink Moist'ing Genteal	As above	1	5
Hypromellose eye-drops 5 mg/mL (0.5%), 15 mL	Methopt	As above	1	5
Hypromellose 3 mg/mL (0.3%) with carbomer 980 2 mg/g (0.2%) ocular lubricating gel, 10 g	HPMC PAA Genteal gel	As above	1	5

PBS list of medicines prescribed by optometrists (continued)

	Product	Restriction	Max qty	Repeats
TEAR SUPPLEMENTS		Restricted: Severe dry eye including Sjögren's syndrome		
Hypromellose 3 mg/mL (0.3%) with dextran eye-drops 1 mg/mL (0.1%), 15 mL	Poly-Tears, Tears Naturale	As above	1	5
Polyethylene glycol 400 mg/mL (0.4%) with propylene glycol 3 mg/mL (0.3%) eye-drops, 15 mL	Systane	As above	1	5
Polyvinyl alcohol eye-drops 14 mg/mL (1.4%), 15 mL	PVA Tears, Liquifilm Tears	As above	1	5
Polyvinyl alcohol eye-drops 30 mg/mL (3%), 15 mL	PVA Forte, Liquifilm Forte	As above	1	5
Polyvinyl alcohol eye-drops 14 mg/mL (1.4%), 15 mL (contains sodium chorite/hydrogen peroxide as preservative)	Vistil	As above	1	5
Polyvinyl alcohol eye-drops 30 mg/mL (3%), 15 mL (contains sodium chorite/hydrogen peroxide as preservative)	Vistil Forte	As above	1	5
UNPRESERVED TEAR SUPPLEMENTS		Authority required:		
Carbomer 974 ocular lubricating gel 3 mg/g (0.3%), single dose units 0.5 g x 30	Poly Gel	Severe dry eye syndrome in patients sensitive to preservatives in multi-dose eye-drops	3	5
Carbomer 980 eye gel 2 mg/g (0.2%), single dose units 0.6 mL x 30	Viscotears Gel PF	As above	3	5
Carmellose sodium eye-drops 5 mg/mL (0.5%), single dose units 0.4 mL x 30	Cellufresh Optifresh Tears	As above	3	5
Carmellose sodium eye-drops 10 mg/mL (1%) , single dose units 0.4 mL x 30	Celluvisc Optifresh Plus	As above	3	5
Carmellose sodium eye-drops 2.5 mg/mL (0.25%), single dose units, 0.6 mL x 24	TheraTears	As above	4	5
Carmellose sodium ocular lubricating gel 10 mg/mL (1%), single dose units 0.6 mL x 28	TheraTears	As above	3	5
Hypromellose 3 mg/ mL (0.3%) with dextran eye-drops 1 mg/mL (0.1%), single dose units 0.4 mL x 28	Bion Tears	As above	3	5
Polyethylene glycol 400, 4 mg/mL (0.4%) with propylene glycol 3 mg/mL (0.3%) eye-drops, single dose units 0.8 mL x 28	Systane	As above	2	5
Sodium Hyaluronate sodium hyaluronate eye-drops 1 mg/mL (0.1%), 10 mL	Hylo-Fresh	As above	1	5
Sodium Hyaluronate sodium hyaluronate eye-drops 2 mg/mL (0.2%), 10 mL	Hylo-Forte	As above	1	5
Soy lecithin eye spray 10 mg/mL (1%), 10 mL	Tears again	As above	2	5
TOPICAL OCULAR LUBRICANT OINTMENTS				
Paraffin 1 g/g compound eye ointment 3.5 g	Polyvisc, Duratears		2	5
Paraffin 1 g/g pack containing 2 tubes compound eye ointment 3.5 g	Polyvisc (2 pack), Ircal (2 pack), Refresh Night Time (2 pack)		1	5
Pariffin paraffin + retinyl palmitate 138 mcg/g (0.0138%) (equivalent to 250 units/g vitamin A) eye ointment, 5 g	VitA-POS		2	5

HYLO® EYE CARE

For severe or chronic dry eye

For mild or moderate dry eye

The systematic approach to eye lubrication

for **Dry Eyes**

- Preservative-free and phosphate-free
- Can be used for 6 months after opening
- Compatible with all types of contact lenses
- At least 300 measured drops per pack, or 150 treatments (both eyes)
- Delivered through the unique COMOD® multi-dose application system

STREAMLINED AUTHORITY CODE 4105

PBS Information (Medical/Nurse Practitioner): Authority Required (STREAMLINED): Severe dry eye syndrome in patients who are sensitive to preservatives in multi-dose eye drops.
PBS Information (Optometrist): Authority Required: Severe dry eye syndrome in patients who are sensitive to preservatives in multi-dose eye drops.

Available from:

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START STRONG STAY STRONG[1-6]

EYLEA®
aflibercept (rch)
solution for intravitreal injection

PBS Information: Authority required for the treatment of wet age-related macular degeneration, diabetic macular oedema and central retinal vein occlusion. Refer to PBS schedule for full Authority Required information.
EYLEA is not listed on the PBS for branch retinal vein occlusion and myopic choroidal neovascularisation.

Please review the full Product Information before prescribing.

MINIMUM PRODUCT INFORMATION EYLEA® [aflibercept (rch)] INDICATIONS: EYLEA (aflibercept) is indicated in adults for the treatment of neovascular (wet) age-related macular degeneration (wet AMD); visual impairment due to macular oedema secondary to central retinal vein occlusion (CRVO); visual impairment due to macular oedema secondary to branch retinal vein occlusion (BRVO)*; diabetic macular oedema (DME), visual impairment due to myopic choroidal neovascularisation (myopic CNV)*. **CONTRAINDICATIONS:** Known hypersensitivity to aflibercept or excipients; ocular or periocular infection; active severe intraocular inflammation. **PRECAUTIONS:** Endophthalmitis, increase in intraocular pressure; immunogenicity; arterial thromboembolic events; bilateral treatment; risk factors for retinal pigment epithelial tears; treatment should be withheld in case of rhegmatogenous retinal detachment, stage 3 or 4 macular holes, retinal break, decrease in best-corrected visual acuity of ≥ 30 letters, subretinal haemorrhage or intraocular surgery; treatment not recommended in patients with irreversible ischemic visual function loss; population with limited data (diabetic macular oedema due to type 1 diabetes, diabetic patients with HbA1c > 12 %, proliferative diabetic retinopathy, active systemic infections, concurrent eye conditions, uncontrolled hypertension, myopic CNV: no experience in the treatment of non-Asian patients, previous treatment for myopic CNV and extrafoveal lesions*); see full PI for effects on fertility, pregnancy, lactation, effects on ability to drive or use machines. **ADVERSE EFFECTS:** Very common: visual acuity reduced*, conjunctival haemorrhage, eye pain. Common: retinal pigment epithelial tear, detachment of retinal pigment epithelium, retinal degeneration, vitreous haemorrhage, cataract, cataract cortical, cataract nuclear, cataract subcapsular, corneal erosion, corneal abrasion, intraocular pressure increased, vision blurred, vitreous floaters, vitreous detachment, injection site pain, foreign body sensation in eyes, lacrimation increased, eyelid oedema, injection site haemorrhage, punctate keratitis, conjunctival hyperaemia, ocular hyperaemia. Others: see full PI. **DOSAGE AND ADMINISTRATION*:** 2 mg aflibercept (equivalent to injection volume of 50 µL). EYLEA is for intravitreal injection only. The interval between doses injected into the same eye should not be shorter than one month. Advice on treatment initiation and maintenance of therapy specific to each patient population is described in the section below. Once optimal visual acuity is achieved and/or there are no signs of disease activity, treatment may then be continued with a treat-and-extend regimen with gradually increased treatment intervals to maintain stable visual and/or anatomic outcomes. If disease activity persists or recurs, the treatment interval may be shortened accordingly. Monitoring should be done at injection visits. There is limited information on the optimal dosing interval and monitoring interval especially for long-term (e.g. > 12 months) treatment. The monitoring and treatment schedule should be determined by the treating ophthalmologist based on the individual patient's response. If visual and anatomic outcomes indicate that the patient is not benefiting from continued treatment, EYLEA should be discontinued. *For wet AMD:* Treatment is initiated with one injection per month for three consecutive months, followed by one injection every two months. Long term, it is recommended to continue EYLEA every 2 months. Generally, once optimal visual acuity is achieved and/or there are no signs of disease activity, the treatment interval may be adjusted based on visual and/or anatomic outcomes. The dosing interval can be extended up to every 3 months. *For CRVO:* Treatment is initiated with one injection per month for three consecutive months. After the first three monthly injections, the treatment interval may be adjusted based on visual and/or anatomic outcomes. *For BRVO:* Treatment is initiated with one injection per month for three consecutive months. After the first three monthly injections, the treatment interval may be adjusted based on visual and/or anatomic outcomes. *For DME:* Treatment is initiated with one injection per month for five consecutive months followed by one injection every two months. After the first 12 months, the treatment interval may be adjusted based on visual and/or anatomic outcomes. *For myopic CNV:* EYLEA treatment is initiated with one injection of 2 mg aflibercept (equivalent to 50 µL). Additional doses should be administered only if visual and/or anatomic outcomes indicate that the disease persists. Recurrences are treated like a new manifestation of the disease. **DATE OF PREPARATION:** Based on PI dated July 2016. Approved PI available at http://www.bayerresources.com.au/resources/uploads/PI/file10294.pdf or upon request from Bayer Australia Ltd, ABN 22 000 138 714, 875 Pacific Highway, Pymble NSW 2073.

**Please note changes in Product Information.*

References: 1. Eylea Product Information, July 2016. **2.** Schmidt-Erfurth, U. *et al.* (2014) Intravitreal aflibercept injection for neovascular age-related macular degeneration. *Ophthalmology.* 121:193-201. **3.** Brown, D.M. *et al.* (2015) Intravitreal Aflibercept for Diabetic Macular Edema - 100-Week Results From the VISTA and VIVID Studies. *Ophthalmology.* 122(10):2044-52. **4.** Ogura, Y. *et al.* (2014) Intravitreal Aflibercept for Macular Edema Secondary to Central Retinal Vein Occlusion: 18-Month Results of the Phase 3 GALILEO Study. *Am J Ophthalmol.* 158:1032–1038. **5.** Heier, J.S. *et al.* (2014) Intravitreal aflibercept injection for macular edema due to central retinal vein occlusion: Two-year results from the COPERNICUS study. *Ophthalmology.* 121(7):1414-1420. **6.** Clark, W. L. *et al.* (2016) Intravitreal Aflibercept for Macular Edema Following Branch Retinal Vein Occlusion: 52-Week Results of the VIBRANT Study. *Ophthalmology.* 123:330-336.