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What's in this issue

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Cover: Infection control - the new normal
Now is the time to set the 'new normal' for infection control in optometry.

Collaboration is the key to preventing diabetes-related blindness

A note from Professor Greg Johnson, Chief Executive Officer, Diabetes Australia

Diabetes is one of the most significant health challenges of the 21st century. There are currently around 1.8 million Australians living with diabetes, and the number of people diagnosed with the condition continues to increase.[1] In fact, in the time it takes to read this foreword another person has probably been diagnosed with the condition.

It is a particularly insidious challenge because, on top of the sheer number of people affected, it impacts every part of the body. This means that all health professionals, including optometrists, have important roles to play in Australia's response to the diabetes epidemic.

Diabetes is the leading cause of kidney failure, heart disease, limb amputation and preventable blindness in Australia. On top of the human toll, diabetes also significantly impacts on the economy, with estimates suggesting it costs our country $14.6 billion every year.[2]

The good news is many diabetes-related complications are preventable with early intervention and treatment. That is why we work across the health sector to ensure all health professionals have the information and systems they need to support people with diabetes.

This includes programs like KeepSight, Australia's diabetes eye check reminder system. In just two years more than 150,000 people with diabetes have registered to receive a reminder when a diabetes eye examination is due.

All Australians with diabetes are at risk of developing diabetic retinopathy (DR) and there are currently about 100,000 Australians with vision-threatening DR.[3] Even more concerning is the fact that the number of people with DR is expected to double by 2030.[3] But the good news is that nearly all diabetes-related vision loss is preventable if detected and treated early. That's why regular eye examinations are so important.

We are encouraging every person with diabetes and eye health professionals to register with KeepSight. The program has already demonstrated that a coordinated and systematic approach to preventative and timely health care can have a real impact.

Similar programs have been introduced in other countries with enormous success. In the UK a recall and reminder system was established in 2003, and by 2010 DR was no longer the leading cause of preventable blindness, reversing a 50-year trend of diabetes being the leading cause of vision loss in working aged adults.[4]

We are confident that with the support of the eye-health sector Australia will see similar results in the years to come. Optometrists play an important role in encouraging people with diabetes to register with the program. We want every optometrist and all other health-care professionals performing DR screening to participate in KeepSight. Throughout 2021 we will be releasing software integrations that will make it easier to ensure KeepSight is accessible to the entire eye-care sector.

We believe KeepSight is the missing link in Australia's diabetes care system helping people with diabetes become more engaged in their eye health care. By working together, we can make diabetes-related blindness a thing of the past.

Professor Greg Johnson
Chief Executive Officer
Diabetes Australia

1. NDSS [Internet]. Diabetes data snapshots c 2021. [Cited 2021 May 24]. Available from: https://www.ndss.com.au/about-the-ndss/diabetes-facts-and-figures/
2. Lee C, Colagiuri R, Magliano D, Cameron A et al. The cost of diabetes in adults in Australia. Diabetes Res Clin Prac 2013; 99: 385-390.
3. Diabetes Australia. [Internet] Diabetes in Australia. [Cited 2021 May 24]. Available from: https://www.diabetesaustralia.com.au/about-diabetes/diabetes-in-australia/
4. Liew G, Michaelides M, Bunce C. A comparison of the causes of blindness certifications in England and Wales in working age adults (16–64 years), 1999–2000 with 2009–2010. BMJ Open 2014; 4: e004015.

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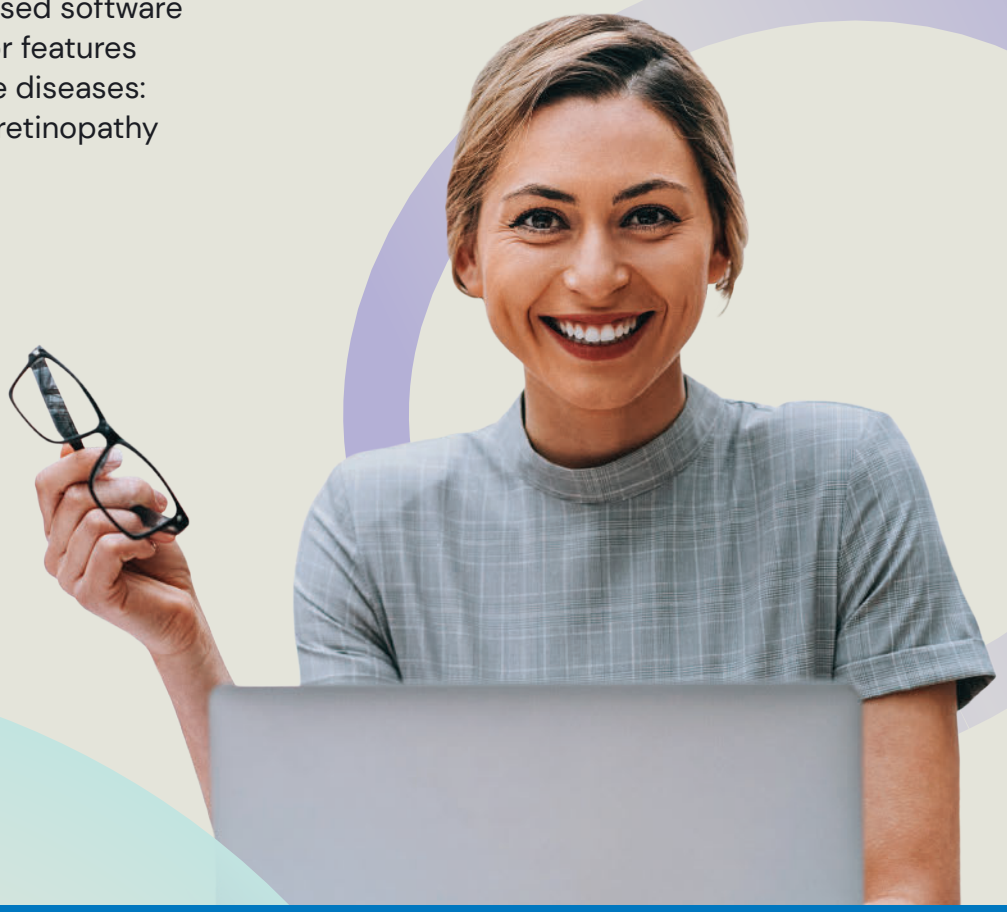

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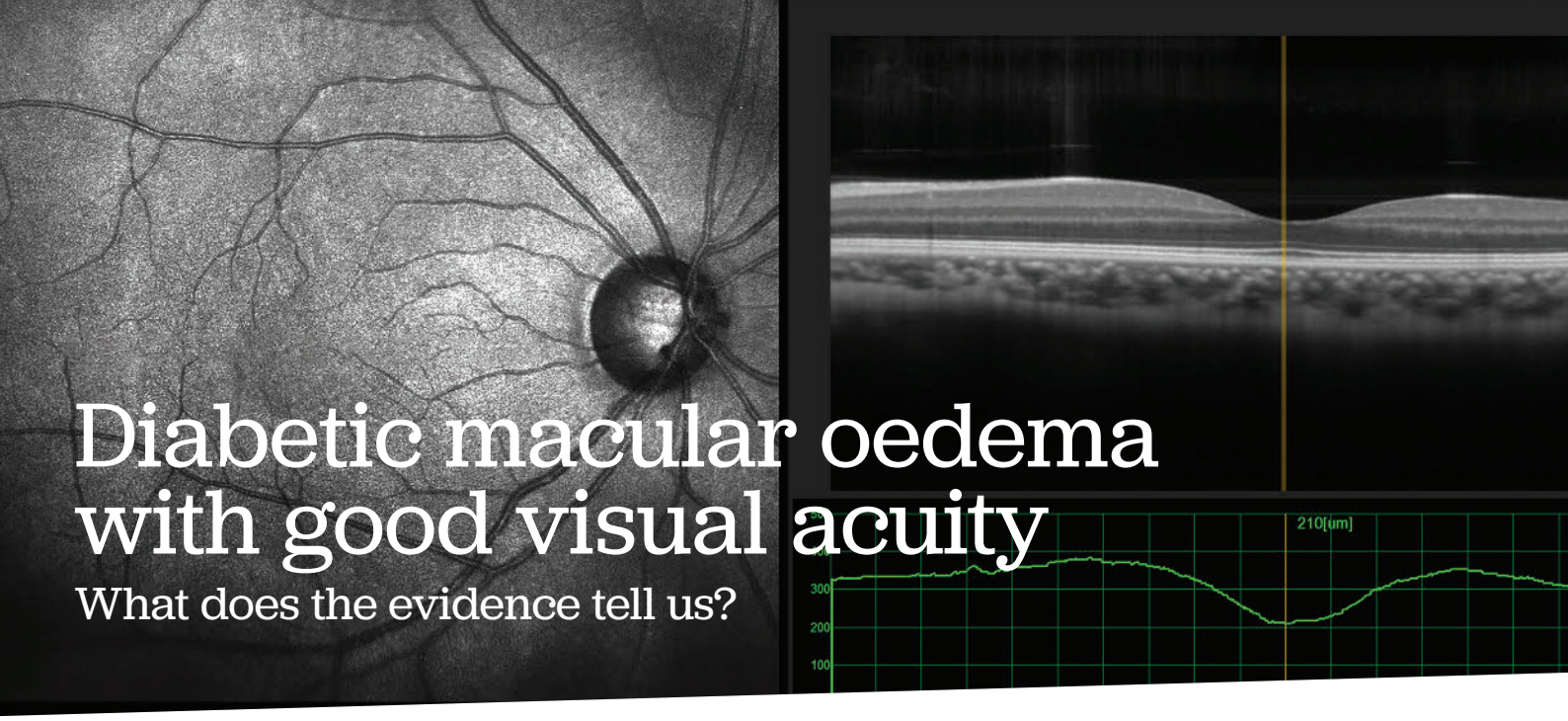

Diabetic macular oedema with good visual acuity

What does the evidence tell us?

FEATURE ARTICLE

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Lead Clinician (Diabetes and Vascular Disease),
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With the increased uptake of OCT imaging in Australian optometry practices, subtle signs of early diabetic macular oedema (DMO) are a common finding in patients presenting for diabetic retinopathy assessments. Centre-involved DMO is defined as retinal thickening within the central subfield zone that is 1 mm in diameter i.e., within 500 micrometres from the centre of the fovea.[1]

In the context of good visual acuity, evidence-based management of these patients is guided by two recently published studies: The Diabetic Retinopathy Clinical Research Retina Network (DRCR.net) Protocol V study,[2] a multi-centre randomised clinical trial, and the OBTAIN retrospective, observational cohort study.[3,4] Following a case study which illustrates the management of a patient with early macular oedema at the Centre for Eye Health (CFEH), Sydney, the results of these two clinically relevant studies are discussed below.

Case study

A 56-year-old male with type 2 diabetes diagnosed 19 years earlier was referred to the CFEH for a diabetic retinopathy (DR) assessment. At his baseline visit, he reported poor blood glucose control but could not recall his most recent HbA1c. His medications comprised treatments for diabetes, systemic hypertension, hyperlipidaemia and diabetic neuropathy, and included Janumet, Diamicron, Avapro, Crestor, Lyrica and Novomix insulin. Insulin had been prescribed by his endocrinologist three months earlier in response to poor glycaemic control.

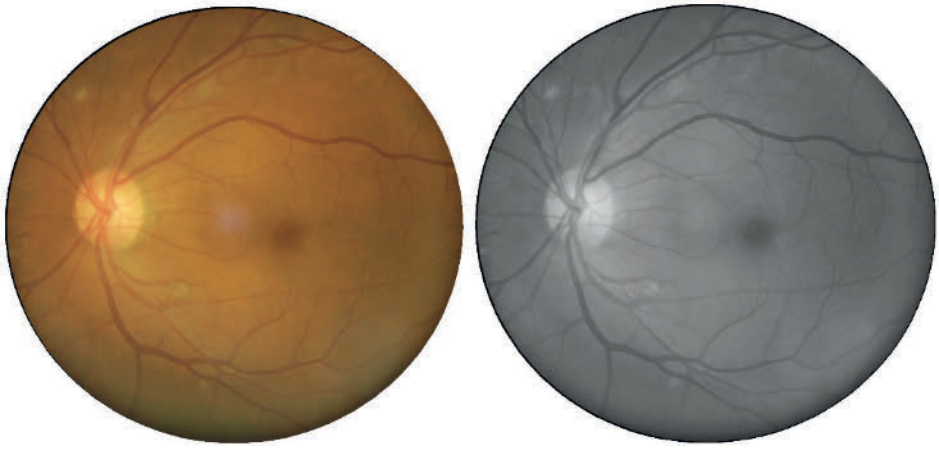

Figure 1.
At the baseline visit (July 2019), posterior pole colour and red-free image of the left eye revealed cotton wool spots superior to the optic disc and in the inferotemporal arcades, scattered microaneurysms and dot/blot haemorrhages, and a patch of hard exudates within the temporal fovea.

Best-corrected visual acuities were R: 6/6 and L: 6/6-1 and funduscopic examination of the right eye revealed scattered intraretinal haemorrhages and cotton wool spots without any signs of macular oedema. The left eye showed numerous cotton wool spots superior to the optic disc and in the inferotemporal arcades, dot and blot haemorrhages, and a patch of hard exudates within the temporal fovea (**Figure 1**). Spectralis OCT imaging of the left eye (**Figure 2**) demonstrated cystoid oedema and hard exudates within 500 µm of the centre of the macula. The level of DR was graded as moderate non-proliferative DR in each eye, with centre-involved diabetic macular oedema (CI-DMO) in the left eye and a possible hypertensive retinopathy component.

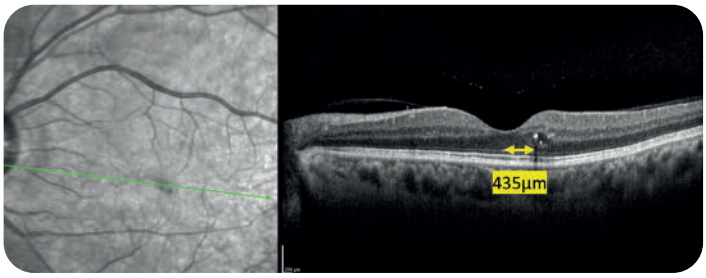

Figure 2.
Baseline Spectralis OCT imaging showed cystoid oedema and hard exudates within the temporal fovea, within 500 micrometres of the centre of the macula.

In co-management (via telehealth) with the centre's consultant retinal specialist ophthalmologist, a three-month optometric review at the CFEH was arranged. The importance of optimising control of blood glucose levels, blood pressure and other risk factors for diabetic retinopathy progression was discussed with the patient. In addition, the assessment results and the potential role of oral fenofibrate in reducing the risk of diabetic retinopathy progression, were communicated to his endocrinologist and general practitioner.

At three-month follow-up with CFEH optometrists, the DR and CI-DMO persisted but with no signs of worsening and stable VA. The patient reported some improvement in his glycaemic control and he was encouraged to continue improving his blood glucose levels and blood pressure in collaboration with his endocrinologist.

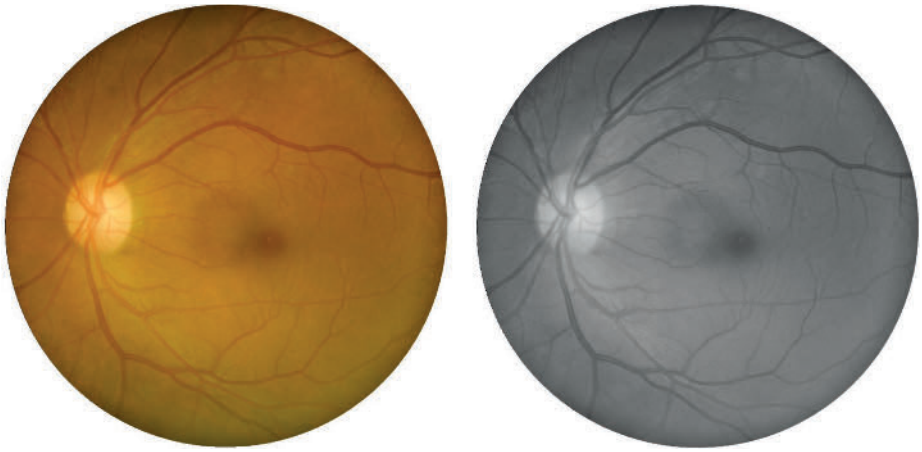

Figure 3.
At the most recent follow-up (March 2021), posterior pole colour and red-free image of the left eye showed resolution of the hard exudates and majority of the cotton wool spots, with persistent, scattered microaneurysms and dot/blot haemorrhages.

The patient returned four months later and was now taking Coversyl for added control of his blood pressure, and his glycaemic control had improved further. Best-corrected VA was 6/7.5+2 in the left eye, and there were definite signs of improvement in both the level of diabetic retinopathy and macular oedema, suggesting that poorly controlled blood pressure may have been contributing to the retinopathy presentation.

Given the improvement, a longer follow-up period of six-months was scheduled. At this most recent visit, there was a marked reduction in the number of cotton wool spots and complete resolution of the macular oedema and hard exudates (**Figure 3**) on clinical examination, Cirrus OCT macular thickness maps (**Figure 4**) and Spectralis OCT line scans (**Figure 5**).

In this particular case study, management involved close monitoring of the DR and macular oedema in conjunction with collaborative care with the patient's endocrinologist and GP to optimise systemic risk factors for DR progression (glycaemic control, blood pressure and blood lipid levels). No anti-VEGF injections or laser treatment were required prior to resolution of the macular oedema. →

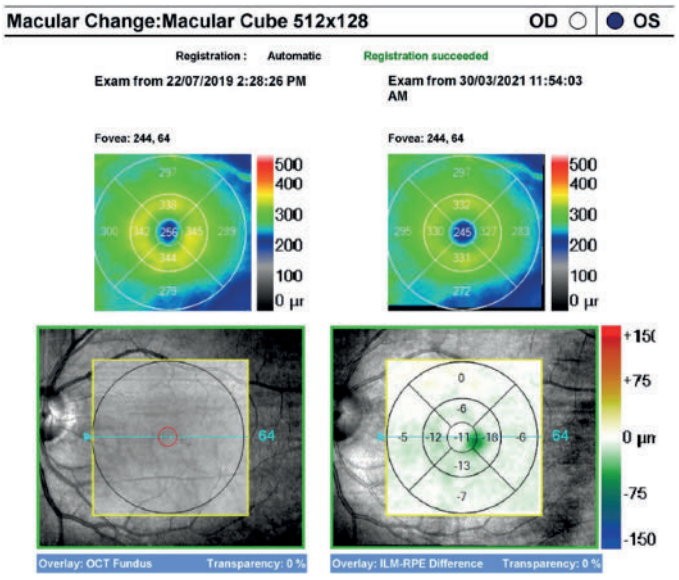

Figure 4.
At 20-month follow-up, Cirrus OCT macular change analysis demonstrated reduced thickening of the central subfield (by 11 µm) and parafoveal subfields (by 6-8 µm), associated with resolution of the diabetic macular oedema, as compared to the baseline visit in 2019.

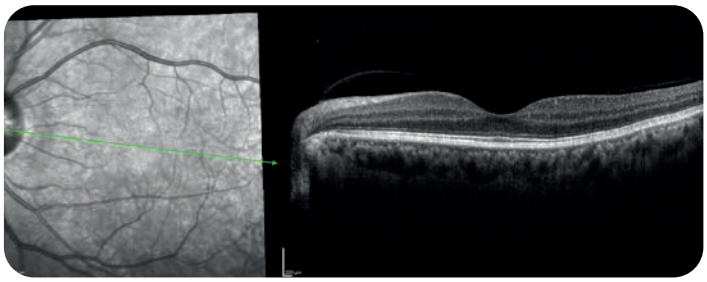

Figure 5.
At 20-month follow-up, there was no evidence of diffuse or cystoid oedema, or hard exudates, in the Spectralis OCT line scan.

So, what does the evidence tell us?

The DRCR.net Protocol V study

Conducted at 91 sites in the US and Canada, the Protocol V study compared visual acuity loss at two years in eyes with CI-DMO and VA of 6/7.5 or better at baseline. All eyes had clinical signs of macular oedema on ophthalmoscopic examination and repeatable thickening of the central OCT subfield. Study participants had either type 1 or type 2 diabetes, were at least 18 years of age, and one eye per participant was randomised to treatment with aflibercept, focal/grid laser photocoagulation or observation.

With regards to treatment randomisation, 236/702 eyes were assigned to observation and no treatment was initially provided to this group. A further 226/702 were randomised to aflibercept treatment. This group received an injection at baseline and were evaluated for repeat injections up to every four weeks as per study protocols. The remainder were randomised to receive laser photocoagulation with treatment at baseline and retreatment at 13-week intervals if indicated.

During the study, the observation group underwent follow-up visits at eight weeks, at 16 weeks then every 16 weeks unless VA or central subfield thickness (CST) worsened. If the VA decreased by 10 or more letters at one visit, or 5-9 letters at two consecutive visits, aflibercept was initiated.

At study completion, the proportion of patients whose VA had reduced by five or more letters was comparable between the three treatment groups: 16% of eyes treated with aflibercept, 17% of eyes treated with laser photocoagulation and 19% of the observation group. Furthermore, 66% of the observation group maintained a VA of 6/6 or better at two years (compared to 77% in the aflibercept group) and the mean change in the central macular thickness was -42 µm in the observation group (compared to -48 µm in the aflibercept group).

Overall, 66% of the observation group and 75% of the laser treatment group did not receive aflibercept during two years of follow-up, suggesting that close observation without treatment may be a reasonable strategy. Of equal importance, the Protocol V study suggested that delaying treatment until VA decreased (by 10 or more letters at one visit, or 5-9 letters at two consecutive visits) resulted in similar visual outcomes for treated patients.

The OBTAIN study

This 12-month retrospective, observational cohort study evaluated patients with visual acuity of 6/7.5 or better at baseline, a thickened central macula, and evidence of intraretinal and/or subretinal fluid on OCT imaging. Of the 249 eyes of 210 patients enrolled, no treatment was initiated for 147 eyes at baseline and 94 eyes continued observation at 12-month follow-up. The study found that, in a real-world setting, the majority of patients with DMO and very good VA maintained vision at 12 months, whether or not they were treated. Several baseline predictors for vision loss were identified in the OCT images including (1) the presence of hyperreflective foci (HF), seen as bright dots within the retinal layers, and (2) disorganisation of the inner retinal layers (DRIL). Eyes with both signs plus disruption of the ellipsoid zone were at further risk of VA loss. The authors concluded that earlier anti-VEGF treatment in this subset of patients may potentially decrease the risk of vision loss at 12 months.

What does this mean for optometrists in clinical practice?

Optometrists make clinical decisions about their patients with diabetes on a daily basis. OCT imaging enhances the care of our patients, often revealing subtle retinopathy signs that would not be evident with either fundoscopy or retinal photography.

The Protocol V and OBTAIN studies support the monitoring of patients with CI-DMO and good visual acuity, however it is crucial that evidence-based protocols are followed and the importance of collaborative care is recognised. Availability of OCT imaging and an individual optometrist's skill level and experience should also be considered in monitoring early signs of DMO.

Optometrists should recognise their important role in the diabetes team: close communication with our patient's GP and/or endocrinologist ensures that risk factors for diabetic retinopathy onset and progression are optimised. Close collaboration with a local ophthalmologist is also critical to ensure that patients are referred for treatment appropriately.

1. International Council of Ophthalmology. ICO Guidelines for Diabetic Eye Care, Updated 2017. Accessed 18/04/2021 http://www.icoph.org/downloads/ICOGuidelinesforDiabeticEyeCare.pdf

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These original clinical images were submitted by Optometry Australia member Geraldine Bendell in response to our call for images

Geraldine Bendell
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Stuart Macfarlane Optometrist, Brisbane, QLD

Tuberous Sclerosis

Corneal and retinal involvement

Tuberous Sclerosis Complex (TSC) is one of the Phakomatoses. It is a rare multi-system genetic disease that most commonly causes benign tumours in the brain, lungs, skin, renal and cardiac structures.[1] Ocular signs are present in about 50% of people with TSC and include optic nerve harmatomas, elevated intracranial pressure, cranial nerve palsies, cortical visual impairment, and visual field defects.[2] The most common ocular signs are retinal harmatomas which are also found with neurofibromatosis. The lesions can be smooth surfaced noncalcified harmatomas or the classical calcified mulberry harmatoma. Hypopigmented retinal areas are sometimes present. Generally, these retinal lesions are stable and rarely affect vision, with about 50% of retinal signs unilateral.[1]

A 51-year-old patient had been under biannual neurological and annual ocular review since 1997. Acuity was RE 6/6= and LE 6/7.5=. She had a superior field defect in the left eye which had been stable. Perimetry was sometimes difficult as it was a triggering factor for her photosensitive epilepsy. The right fundus was clear. In the left fundus she had an obvious superior harmatoma along with a parafoveal harmatoma which appeared as a hazy area (**see Figure 1 and 2**). These lesions had been stable since initial diagnosis in 1997. She also had a rare finding of a primary corneal harmatoma[3] in the left eye which extended over the inferior third of the cornea to just below the visual axis. The corneal lesion has been slowly extending the last two decades and may warrant a keratoplasty if her visual axis is invaded.

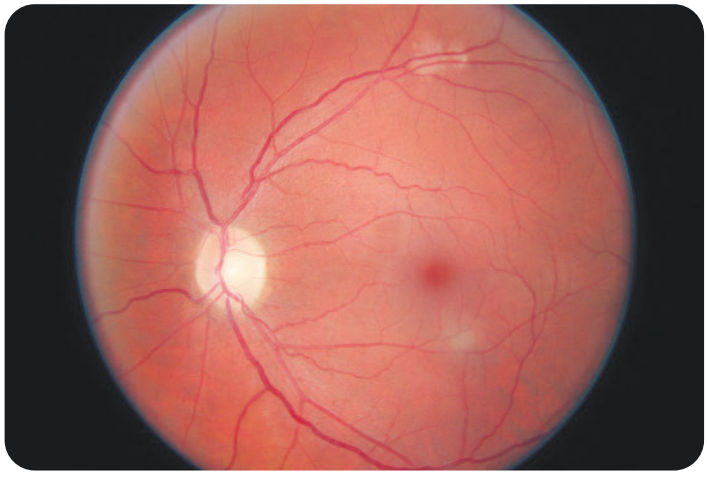

Figure 1.
Fundus image of the LE revealing a retinal harmatoma inferior to the macula and superior harmatoma.

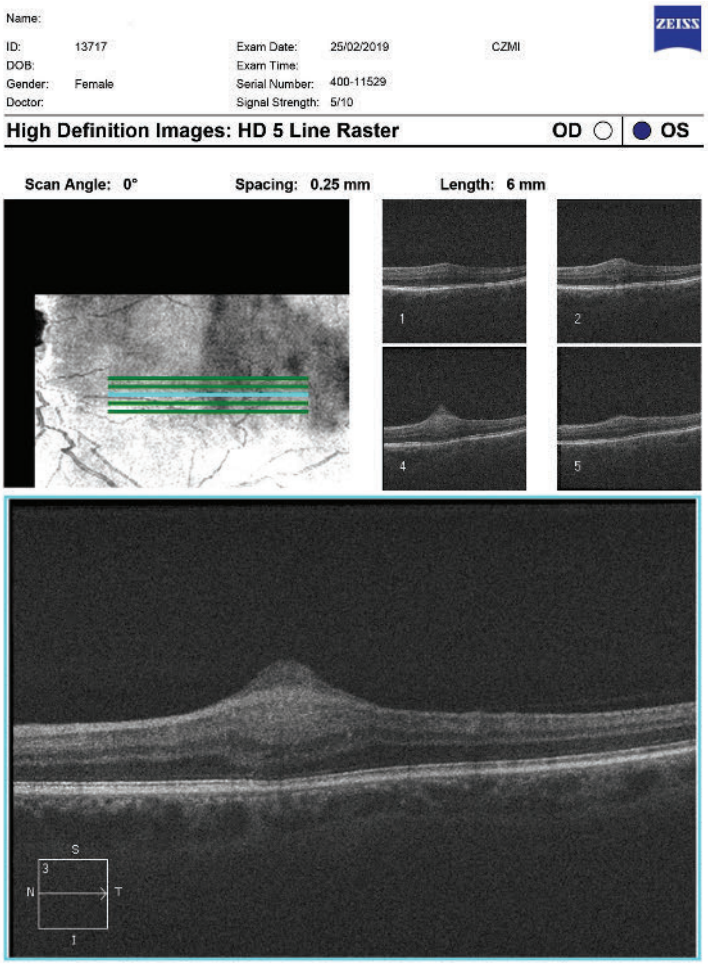

Figure 2.
OCT image of the LE retinal harmatoma inferior to the macula.

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3. El Houda HA, Belghmaidi S, Bouda R et al Corneal hamartoma: a rare case report. Poster presented at: 24th European Society of Cataract & Refractive Surgeons Winter Meeting; 2020 Feb 21-23; Marrakech.

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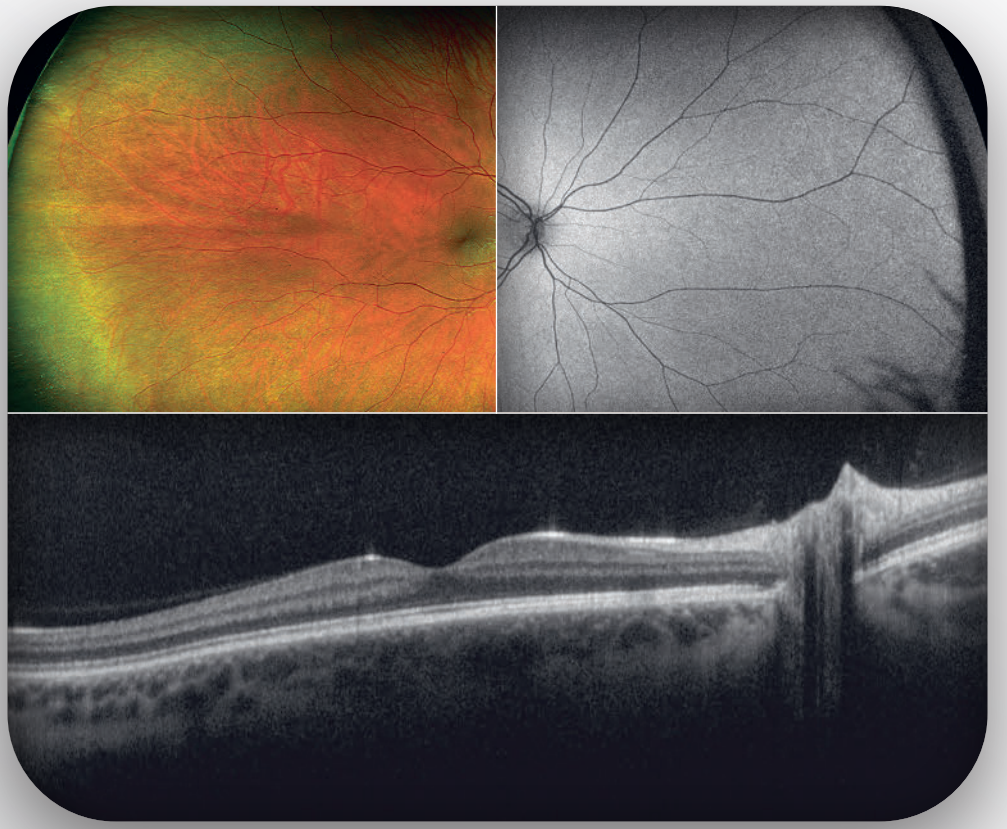

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Dr David Ng
MBBS MPH FRANZCO
Ophthalmologist, Vision Eye Institute

Managing the diabetic eye

Diabetes mellitus is a multi-organ disease responsible for much morbidity and mortality. According to self-reported data, an estimated 1.2 million Australians had diabetes in 2017–2018.[1] Since only 70% of Australians with diabetes are diagnosed,[2] the true prevalence is probably at least 25% higher. In addition, diabetes contributed to over 10% of all deaths in 2018.[1]

Diabetes damages the blood vessels of the body. It affects the larger vessels of the heart, brain and legs and the smaller vessels of the kidneys, eye and nerves of the legs. Based on survey data from 2017–2018, it was estimated that 89,500 adults had vision loss due to their diabetes.[3]

Since multiple organs and systems may be affected, diabetic patients benefit greatly from a multidisciplinary team consisting of an endocrinologist, ophthalmologist, nephrologist, diabetes nurse and optometrist, for example. Collaboration and communication of clinical information within the diabetes health-care team has been shown to improve patient care and metabolic control, and reduce cardiovascular risk factors.[4] In the community, this is best coordinated by a GP. Optometrists play a critical role in detecting ocular diabetic complications and referring patients for treatment.

As early detection and timely appropriate treatment can prevent more than 98% of severe vision loss associated with diabetes,[5] the optometrist's contribution can be significant.

Diabetes classification

Type 1 diabetes is usually diagnosed in people under the age of 30 and accounts for 5–10% of all diabetes cases.[6] It is thought to be autoimmune-mediated and typically presents acutely with an absolute lack of insulin production by the beta cells of the pancreas. The majority of type 1 diabetic patients will develop diabetic retinopathy within 20 years of diagnosis.[7]

Type 2 diabetes is the most common form of diabetes and accounts for 90% of all cases.[6] It is usually diagnosed in patients over 30, although it is increasingly being identified in children. Although insulin is still produced by the pancreas, the body's cells are resistant to its effects. Diabetic retinopathy will affect over 60% of type 2 diabetic patients within 20 years of diagnosis.[7]

Lifestyle modification can prevent type 2 diabetes and reduce the risk of complications. This is significant given the high prevalence of type 2 diabetes within the diabetic population. Such interventions address obesity (the biggest risk factor), low physical activity, hypertension and poor diet. In fact, the DiRECT study found that 86.1% of patients with type 2 diabetes who lost 15 kg or more went into diabetes remission.[8] The addition of a dietitian to the team can be important, and optometrists can reinforce patients' understanding of the role of diet and lifestyle change in diabetes management.

Gestational diabetes is high blood sugar recognised during pregnancy, which returns to normal levels post-partum. It is diagnosed in about one in six pregnancies.[1] Lifetime risk of type 2 diabetes is increased in this population and ongoing screening is recommended.[9]

Diabetic retinopathy (DR)

Diabetes is the most common cause of blindness amongst working-age people and can affect most parts of the eye (**Table 1**). Approximately one-third of diabetic patients will develop DR, and a further one-third of these will have sight-threatening eye disease.[10] Factors that increase the risk of DR are outlined in **Table 2**.

DR involves the microvasculature of the retina. Normal retinal capillaries consist of endothelial cells surrounded by pericytes. In diabetic eyes, there is a loss of these supportive pericytes. This loss allows the formation of microaneurysms and increased vascular permeability, leading to haemorrhage and retinal oedema. Hard exudates may form at the junction of relatively normal retina and oedematous retina.

Lacrimal Gland	Dry eye
Cranial nerves	III, IV, VI ophthalmoplegia
Cornea	Neurotrophic Endothelial cell dysfunction
Iris	Rubeosis iridis
Lens	Cataract
Vitreous	Haemorrhage
Retina	Retinopathy Maculopathy Epiretinal membrane Retinal detachment
Optic Disc	Ischaemic optic neuropathy

Table 1.
Parts of the eye affected by diabetes

Risk Factors
• Duration of disease
• Poor glycaemic control
• Hypertension
• Renal disease
• Anaemia
• Pregnancy

Table 2.
Risk factors for development of diabetic retinopathy

DR causes the closure of blood vessels, resulting in retinal ischaemia, and increases their leakiness, leading to retinal oedema. The retinal ischaemia particularly affects the midperipheral retina. Intraretinal microvascular abnormalities (IRMA) are formed, which are abnormal shunts connecting the arterioles to venules. Cotton wool spots are areas of ganglion cell infarction. →

Retinal ischaemia then leads to the production of vascular endothelial growth factor (VEGF), amongst other factors, encouraging the formation of new vessels (neovascularisation). Unfortunately, unlike the native vasculature, these new vessels are not arranged in an orderly manner and leak, are fragile and tear easily, resulting in vitreous haemorrhage. These vessels are also associated with a connective tissue framework that can contract and cause a tractional retinal detachment.

Screening and staging DR

All patients diagnosed with type 2 diabetes should be screened for diabetic eye disease at diagnosis. If no eye disease is detected, then repeat screening should be performed every 1–2 years. Although more labour-intensive, in my professional experience annual screening is likely to result in greater compliance from the patient – it is harder to forget than biennial screening. A reliable system to recall patients is necessary to limit loss of patients. The number of patients requiring screening is such that it should be performed by ophthalmologists, optometrists and interested GPs.

DR is usually asymptomatic until its late stages. Good vision can still be experienced despite the presence of advanced diabetic damage, but can result in sudden loss of vision overnight.

Optometrists are well-placed to educate patients about the importance of regular screening in detecting asymptomatic ocular damage.

DR is diagnosed by the typical constellation of retinal signs and exclusion of differential diagnoses, including retinal vein occlusion, hypertensive retinopathy, anaemia, leukaemia and radiation retinopathy. Once diagnosed, it should be staged, as this is a measure of the severity of the disease and risk of severe visual loss (**see Table 3**[11]). The stage of disease helps determine how quickly the patient needs to be seen by an ophthalmologist.

Diabetic macular oedema (DMO)

Maculopathy can occur at any stage of diabetic retinopathy. It is defined as microaneurysms, haemorrhages, hard exudates or retinal thickening within 2 disc diameters of the foveal centre. This should be further classified into centre-involving (vision-affecting) and non-centre-involving (vision-threatening) DMO.

Centre-involving DMO is defined as microaneurysms, haemorrhage, hard exudates and retinal thickening within one-third of a disc diameter (500 microns) of the foveal centre. Patients should be referred to an ophthalmologist within four weeks.

In the case of non-centre-involving DMO, patients should be referred within 12 weeks.

About the author

Dr David Ng is a general ophthalmologist with a particular interest in glaucoma and medical diseases of the retina, such as diabetic retinopathy, age-related macular degeneration and retinal vessel disease. He has considerable experience in performing laser cataract surgery and regularly lectures on this area. Dr Ng practises at Vision Eye Institute's Chatswood, Drummoyne and Mosman clinics.

Management of diabetic eye disease

Treatment for DR and DMO consists of intravitreal injections of anti-VEGF, or corticosteroids, laser or surgery. Intravitreal injections are the mainstay of treatment and an average of nine injections are required for the first year. At one year, visual acuity in eyes affected by diabetic maculopathy improves by an average of one to two lines.[12] The number of injections required decreases substantially for the second and third years, with the majority of patients not requiring injections after three years.

Macular laser for diabetic maculopathy has been shown to be less effective than intravitreal injections and is not routinely performed.

Panretinal photocoagulation remains the principal treatment for proliferative diabetic retinopathy. This is usually carried out over several visits. Intravitreal injections of ranibizumab have been shown to be equally effective[13] but require more ongoing visits.

Severity scale and recommended referral timeframes	
Mild non-proliferative diabetic retinopathy (NPDR) – a few microaneurysms.	Review in one year.
Moderate NPDR – more signs than microaneurysms but less than that defined by severe NPDR.	Refer to an ophthalmologist within 12 weeks.
Severe NPDR – 4 quadrants of intraretinal haemorrhages, or 2 quadrants of venous beading or one with IRMA.	Refer to an ophthalmologist within four weeks.
Proliferative diabetic retinopathy – presence of neovascularisation at the disc (NVD), elsewhere on the retina (NVE), on the iris (NVI), or vitreous or preretinal haemorrhage.	Refer to an ophthalmologist within one week.

Table 3.
Staging and referring diabetic retinopathy[11]

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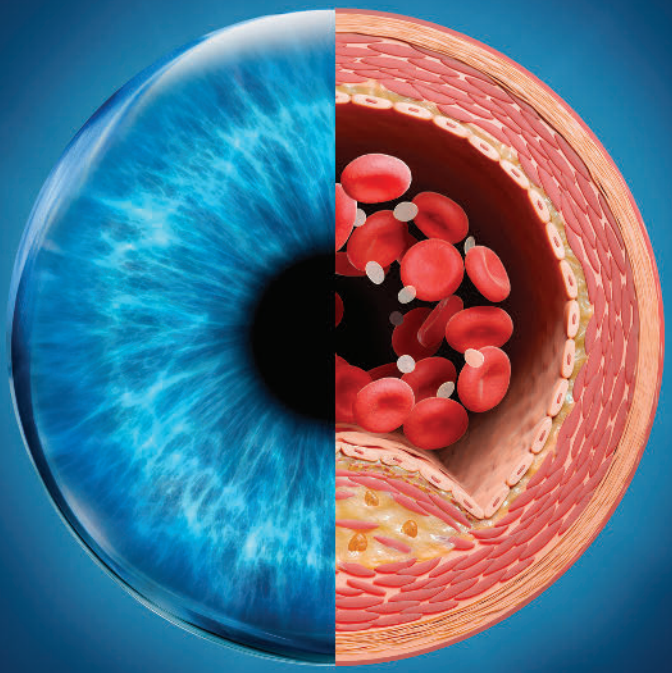

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**Please note changes in PI.*

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VIATRIS

Taryn Black
KeepSight Program Director, Diabetes Australia

KeepSight two years on

An update from Diabetes Australia

Vision loss is one of the health complications people living with diabetes most fear, a recent survey conducted by Juvenile Diabetes Research Foundation Australia found.[1] Fear of losing eyesight was greater than heart disease, kidney problems and even lower limb amputation.

But people shouldn't have to be afraid. While diabetes is currently the leading cause of blindness among working age Australians, almost all diabetes-related blindness is preventable with early identification and treatment. Regular eye checks can help ensure eye complications are detected early when treatment is most effective, however around half of all Australians with diabetes don't undergo eye examinations within the recommended timeframes.[2]

Diabetes is a complex condition that requires significant management, particularly on the part of the person with diabetes. By some estimates, the average person with diabetes has to make 180 health-related decisions every day.[3] Amid the myriad of appointments people are required to make, some, understandably, may get overlooked. This can be particularly true of eye examinations, given diabetes-related eye disease is often asymptomatic.

This is why Diabetes Australia, along with the Australian Government, Vision 2020 Australia, Centre for Eye Research Australia, Bayer, Novartis, Mylan, Specsavers and Luxottica Retail ANZ have come together to support KeepSight. By providing people with timely reminders to book in a regular appointment, the program represents a once-in-a-generation opportunity to dramatically increase the rates of eye examinations and reduce diabetes-related vision loss and blindness in Australia.

Success in the UK

Programs similar to KeepSight, that reduce the burden on people with diabetes having to remember to book in their own appointments, are not without precedent.

Since commencing in 2003, the English NHS Diabetic Eye Screening Programme (DESP) has had a significant positive effect on both screening rates and health outcomes among people with diabetes at risk of vision loss.[4] Using the individual identification number every NHS patient is assigned, people with diabetes above the age of 12 are sent an annual letter inviting them to an eye screening appointment. Patients who attend these appointments undergo mydriatic photography using a non-mydriatic camera, with all images graded by trained assessors.[5]

The results speak for themselves. In 2015-16 uptake of the program was over 80%, with 2.14 million patients out of the 2.59 million who were offered screening attending an appointment.[6] If this result is not significant enough, in 2010, for the first time in at least 50 years, diabetic retinopathy/maculopathy was no longer the leading cause of certifiable blindness among working age adults in England and Wales.[7]

Speaking in Australia in 2019, Professor Peter Scanlon, program director of the DESP, said a similar scheme is feasible in Australia but requires a unified approach.

"Everyone would agree that reducing blindness from diabetic retinopathy is a worthwhile cause. What's required is the sector working together across patient and professional groups, speaking with one voice, and being focussed on achieving the task. This is perfectly feasible within any healthcare system.

"It is particularly achievable in Australia where there is a public and private system that has significant investment and so the main barriers are organisational and not financial."[8]

KeepSight and DESP are fundamentally very similar. When people with diabetes register with KeepSight, they receive reminders and prompts when their regular eye examinations are due.

In the two years since KeepSight has commenced it has already transformed Australia's eye-care sector, with more than 150,000 people with diabetes having registered with the program and more than 40,000 reminders sent.[9]

Most of these people have been registered with KeepSight by their optometrist, so that Diabetes Australia can follow-up with the person if they do not respond to optometry recall messages for their follow-up appointment.

The centralised structure of the NHS means that keeping in contact with patients is reasonably straightforward. To achieve similar results in Australia, KeepSight requires a high level of ongoing support from the local practitioner community. Australia's National Diabetes Service Scheme offers a database of people with diabetes, and practitioners registering people who are having eye checks with KeepSight providing the other half of the equation. With the support of local practitioners, a level of success similar to the UK could be achieved here in Australia.

Optometry Australia members who would like to find out more about KeepSight can visit www.keepsight.org.au

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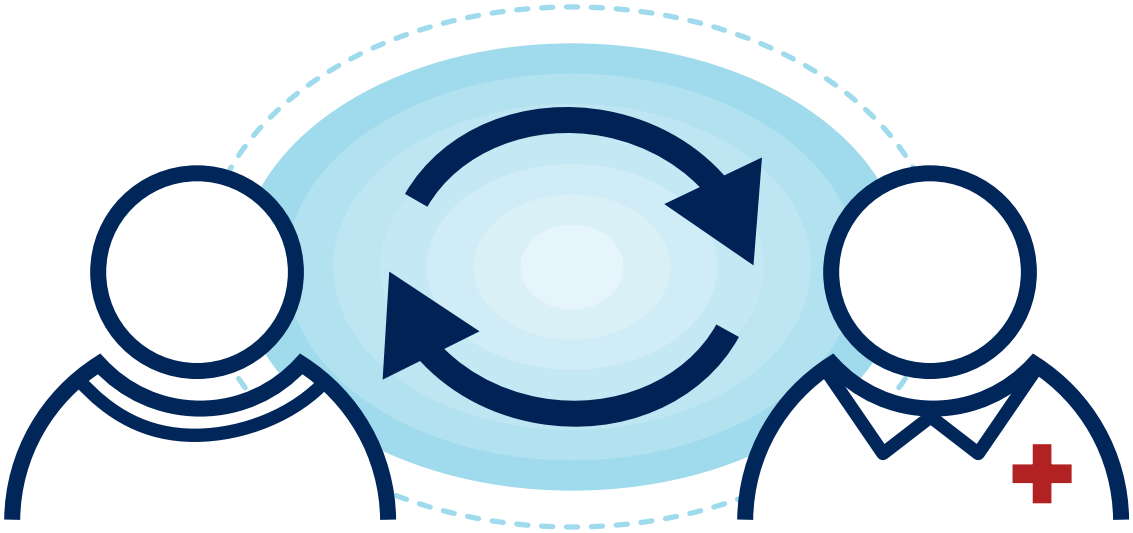
A rote approach to diabetic photo interpretation



The best way to prevent visual impairment in diabetes is to detect, refer, and treat vision-threatening diabetic retinopathy (DR) as soon as possible, and communicate DR severity to primary-care physicians to help guide medical management. Recent advances in diagnostic imaging technology have allowed for better visualisation of the retina. Fundus photography is superior to clinical fundoscopy examination alone for DR screening,[1] and spectral domain optical coherence tomography (SD-OCT) imaging has become the gold standard for clinical detection of diabetic macular oedema (DME), as it is objective, quantifiable, non-invasive, and repeatable.[2]

While reading a diabetic photo or macular OCT may seem easy to a well-trained eye, the devil is in the details for the novice. When reading a fundus photograph, it is human nature to immediately gravitate attention towards the largest retinal lesion visible – for example a large haemorrhage or cotton wool spot. However, this natural inclination may impair the clinician's ability to detect the smaller, less apparent and yet more important microvascular changes that are characteristic of vision-threatening DR. Developing a systematic approach to reading a fundus photo may reduce this human error and improve detection of DR (**Figure 1**). →

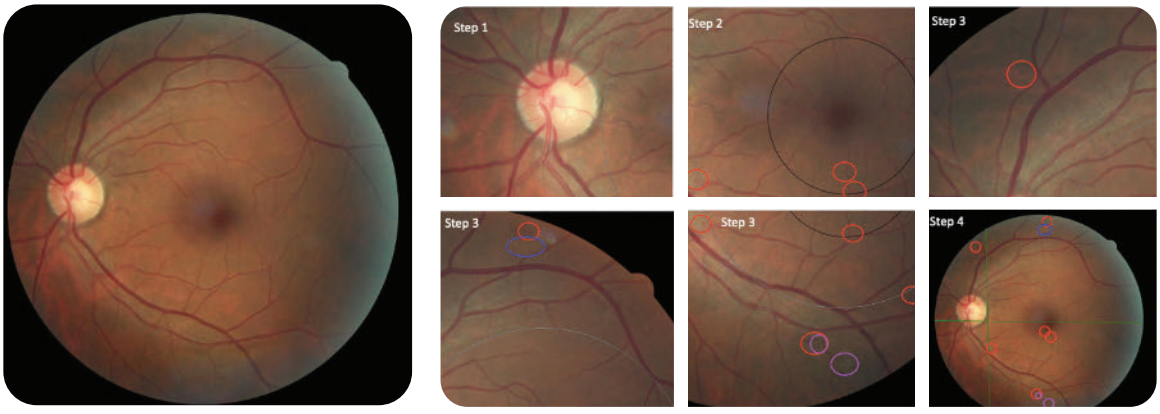


Figure 1 .
Left: Single-field view of the posterior pole of the left eye. On initial glance minimal retinopathy is appreciated. Right: With enhanced imaging, microaneurysms (red circles), hard exudates (blue circles), and cotton wool spots (purple circles) are identified. This eye has moderate diabetic retinopathy.

DR is caused by microvascular damage to the retinal capillary networks. While there are numerous clinical findings like microaneurysms, intraretinal haemorrhages, cotton wool spots, venous beading, and intraretinal microvascular anomalies (IRMA) in non-proliferative DR (NPDR), these findings in isolation do not cause visual impairment. The conditions that cause vision loss in DR include intraretinal complications of NPDR like macular oedema and ischemic maculopathy, as well as extraretinal complications of proliferative DR (PDR) like vitreous haemorrhage, tractional retinal detachment, and neovascular glaucoma.[3] Thus, the most important findings include hard exudates, macular intraretinal haemorrhages, vitreous haemorrhages, neovascularisation, retinal fibrosis, and optic nerve head appearance.

There are a few problems with visualising DR. Foremost, the lesions are tiny. For example, a microaneurysm is considered to be any round, red dot that is ≤ 125 µm and has sharp margins. 125 µm is roughly the size of the width of the average major vein on the optic disc margin[4] – and that is the largest microaneurysm you will see. Mild NPDR can be undetected if these microaneurysms are missed. Further, a frond of neovascularisation grows slowly into an ever-expanding net of vessels on the surface of the retina and penetrating into the vitreous cavity. Due to the superficial location of retinal neovascularisation, small fronds are easily camouflaged by background intraretinal retinopathy. It is imperative that the clinician zoom in on fundus photographs to identify and differentiate these microvascular findings.

It is clinician preference where to start. The important thing is to always start at the same place and develop a rote, step-by-step pattern of evaluation. Personal preference - start at the optic disc.

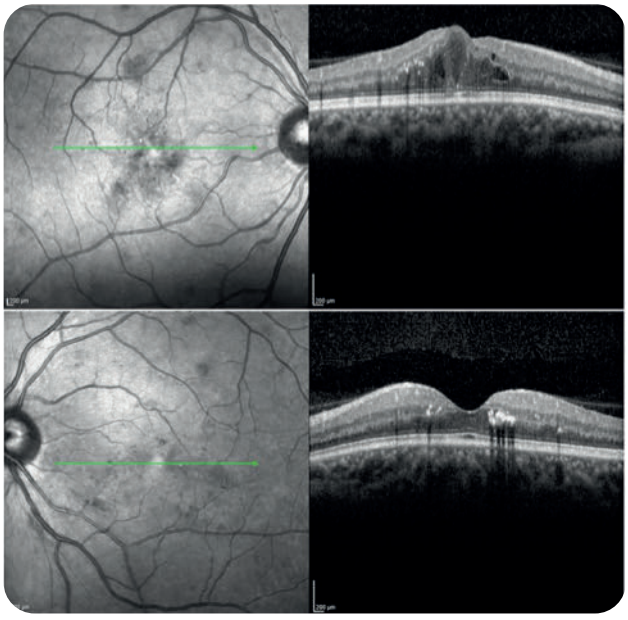


Figure 2.
Macular OCT images of two eyes with (top) and without (bottom) diabetic macular oedema. Notice that both images have intraretinal hard exudates (hyper-reflective lesions) near the fovea, that would have been observed on photography, however, the level of adjacent intraretinal oedema would not have been appreciated.

Step 1: Evaluate the optic nerve

Look for signs of neovascularisation at or surrounding the optic nerve head. Assess the outer disc margin for areas of possible optic nerve head oedema. Many patients with diabetes have co-morbidities like hypertension that may make them susceptible to ischemic optic neuropathy, and diabetes itself can also cause inflammation of the optic nerve, known as diabetic papillitis. Next, assess the integrity of the rim tissue and cup. Look for signs of glaucoma as patients with diabetes are at risk for neovascular and open-angle glaucoma.[5]

- **Clinical pearl:** Due to the distribution of the retinal nerve fibres entering the optic disc, haemorrhages on the optic nerve are often superficial and flame shaped. Odd-shaped haemorrhages or small round, red lesions on the optic nerve should be suspicious for developing fronds of neovascularisation, especially in an eye that has moderate or severe NPDR or PDR.

Step 2: Evaluate the macula

Current guidelines for treatment of DME pertain to the location of oedema relative to the centre of the fovea, as well other qualitative and quantitative features of the maculopathy.[5,6] Therefore it is imperative to identify signs of intraretinal oedema like hard exudates, retinal thickening, and clusters of haemorrhages, and their location relative to the fovea **(Figure 2)**.

- **Clinical pearl:** Hard exudates are hard lipoprotein deposits that remain after the retina has reabsorbed surrounding fluid. Hard exudates are a sign of current or previous intraretinal oedema. When hard exudates are seen at or within 1,500 µm (approximately one optic disc diameter) from the fovea, the clinician must assume the presence of intraretinal oedema, and confirm (or reject) this assumption with a macular OCT.

Hard exudates are small, white or yellowish-white waxy deposits with sharp, irregular margins. They are usually located in the outer or middle layers of the retina, often near a microaneurysm, haemorrhage or at the edge of intraretinal oedema. These lesions need to be differentiated from drusen. Drusen are hard or soft lipoprotein deposits at or anterior to Bruch's membrane. They are also yellow, but often duller in appearance, round, and may have a halo of pigmentation. While it may be easy to differentiate a large drusen from a small hard exudate, it can be difficult for even a seasoned clinician to subjectively differentiate between a small hard exudate and a small drusen. Thus, macular OCT may be necessary to objectively diagnose lesions in the macula.

- **Clinical pearl:** Assume any yellow lesion in or near the macula is a hard exudate until proven otherwise on OCT

New areas of fluid leakage may not have associated hard exudates, and patients with chronic retinal ischemia may have areas of capillary drop out, where the retina is dysfunctional but appears clinically "retinopathy-free" on dilated fundoscopy and fundus photography. Visual acuity can guide clinicians in these cases on the necessity of further ancillary testing beyond photography. Chronic and extensive ischemic maculopathy can be seen as intraretinal thinning on macular OCT. However, retinal ischemia and blood flow dynamics are best visualised on fluorescein angiography and/or OCT angiography.

- **Clinical pearl:** Always perform a macular OCT on a patient with diabetes who is unable to attain 6/6 visual acuity on refraction/pinhole testing, as they may have DME without visible hard exudates.

Step 3: Evaluate the superior/inferior temporal and nasal arcades

When viewing the retinal arcades, look for ischemic changes to the vessels. Assess for localised increases and decreases in venous diameter, venous beading, and venous loops (**Figure 3**). This is also a great opportunity to evaluate for signs of co-morbid disease manifestations of hypertension, atherosclerosis, or heart disease including arteriovenous impingement, arterial narrowing, or localised, dense areas of intraretinal haemorrhages along the distribution of a retinal vein, that may be indicative of a retinal vein occlusion and not diabetic retinopathy. Be observant for areas of retinal whitening and retinal emboli that may be a sign of retinal artery occlusions, carotid artery, and/or heart disease.

Look carefully for areas of IRMA and venous beading as they can be difficult to see, and are important for classifying severity.[4] IRMA are abnormalities of innate intraretinal capillary vessels and are large, tortuous vessels at the level of the superficial or deep capillary plexus. IRMA are pathophysiologically different from retinal neovascular vessels as they are non-fenestrated, less brittle/leaky, and lie within the retina. It may be difficult to differentiate an area of IRMA from a frond of neovascularisation on fundus photography. In these cases an additional diagnostic imaging modality like fluorescein angiography and/or OCT angiography may be needed.

- **Clinical pearls:**
  - → A patient diagnosed with retinal neovascularisation needs to have anterior segment evaluation to rule out neovascularisation of the angle and iris.
  - → Patients who have undergone laser and/or anti-VEGF injections for PDR still need to be monitored closely, as active proliferative lesions can recur.

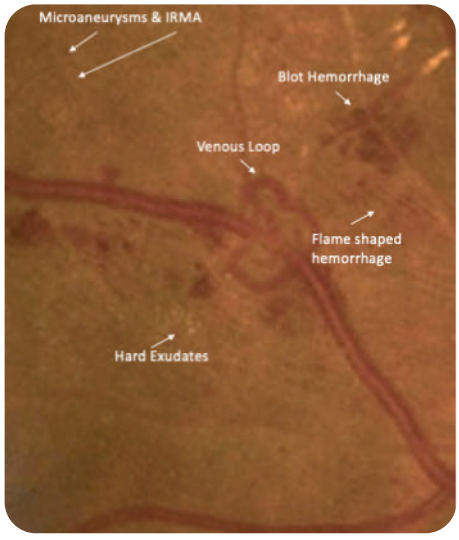


Figure 3.
Fundus photograph of intraretinal vascular complications of non-proliferative diabetic retinopathy. Image Courtesy EyePacs and UC Berkeley School of Optometry

Step 4: Take a global view and evaluate the peripheral retina

By viewing the entirety of the fundus at the end of the evaluation, the clinician has already noted the pertinent microvascular retinal anomalies. In this last view, the clinician can appreciate the overall amount of NPDR to stage severity,[4,6] as well as observe for extraretinal findings like preretinal and vitreous haemorrhage

- **Clinical pearl:** Diffuse vitreous haemorrhages may be difficult to see. They may appear as small, focal areas of pink-tinged blur on the photo. These can look similar to and be confused with other ocular media opacities like senile vitreous floaters, cataracts and corneal defects.

Up to 50% of microaneurysms, IRMA, and neovascularisation can occur more peripherally than the posterior retina normally visualised by ETDRS photography.[7] Studies suggest that patients with peripheral lesions have a 3.2-fold higher risk of progression in their DR severity and a 4.7-fold higher risk of developing PDR.[7]

Additionally, studies have shown that 10% of eyes are classified as having more severe DR than originally graded when considering retina outside of the standard ETDRS fields.[8]

Due to the increased distribution and utility of diagnostic retinal imaging, it is important for practitioners to increase their comfort level in accurately and efficiently reading diagnostic imaging. Consistently applying a systematic approach to reading photos, and increasing the volume of photos read, can help improve pattern recognition in DR diagnosis: The more you see the better you get.

For more information on the examination and management of patients with diabetes, see the Optometry Australia Clinical Guidelines.[9]

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Light transmission and protection

When considering an optical solution, the effects it will have on the different wavelengths of the light spectrum must be taken into account.

The light spectrum can be split into non-visible and visible (**Figure 1**).

Whilst it is not the intention of this article to discuss in depth the possible damage or health benefits of various wavelengths in the electromagnetic spectrum, there is evidence that shorter wavelengths, specifically Ultra Violet, (UV) are damaging. In a paper on UV radiation[1] Coroneo listed over 40 ophthalmic conditions in which sunlight has been implicated in pathogenesis over the eyelid, anterior and posterior eye. UV light wavelengths are from 100 nm to 380 nm. In Australia, wavelengths to 400 nm are included in the UV umbrella.

Blue light problems and solutions have been publicised to such an extent that patients are often asking what kind of protection is available. No matter what stance we have on this subject it is still important to understand the solutions that are available. Blue light takes up wavelengths to 500 nm. The shorter the wavelength the higher the energy and the more hazardous it could be.

Visible light is taken to be of wavelengths 380 nm to 760 nm, which can be split into the various colours. Visible light is essential for vision, colour perception and some aspects of health. It should be understood that the eye does not respond equally to all wavelengths and has a maximum response to the green/yellow part of the spectrum (555 nm) in photopic conditions that moves steadily towards the blue end of the spectrum in photopic and scotopic light conditions (**Figure 2**).[2] This is important when considering what type of glare protection should be dispensed for use when driving at night.

As the visible spectrum ends the longer wavelengths of Infra-Red (IR) occur. IR can be categorised into zones according to the biological effects, but it is rare, if ever, for natural solar radiation to exceed the maximum permissible exposure level for each category.[3]

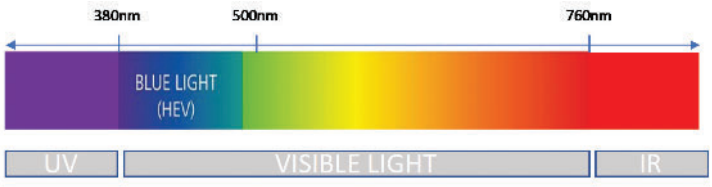

Figure 1.
The light spectrum

There are two ways that light transmission can be prevented:

- Absorption by the material or tint
- Reflection of light from lens surfaces.

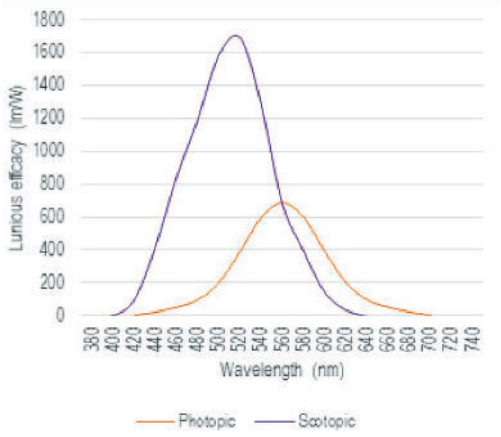

Figure 2.
Visual response in different light conditions

Absorption

Materials can be made to absorb different wavelengths of light by the addition of specific molecules that prevent energy being transmitted by the material, known as absorbers. The most common of these types stop UV transmission, but other wavelengths can be blocked in the same way and we are seeing materials that block to 410 nm or 420 nm.

Generally, indexes above 1.5 will give 100% UV protection, and manufacturers offer UV400 materials. Polarised and photochromic materials also generally provide 100% UV protection. The problem occurs with 1.5 index clear materials that normally absorb only to 350 nm. Some manufacturers add absorbers, but if they don't, and sunglasses are being dispensed, the material needs to be dipped in a UV dye before the tinting process. This is not always automatic and when ordering it is up to the practice to know what level of UV protection they are supplying the patient. Australian standards for UV only apply to ready-made sunglasses, but the patient will expect the same protection from their prescription sunglasses.

If using a material absorption based blue light solution, then the implications of picking a specific wavelength to block should be understood. Looking at the transmission curve of a material that blocks 100% of 410 nm, about 50% of 420 nm is also blocked (**Figure 3**). Full transmission does not occur until around 440 nm. The lens will no longer be white, and the subsequent residual tint could impact the night driving vision of an early cataract patient. The patient should also be warned that colour vision will be impacted, especially if essential for their job.

Blue light solutions can also come from a simple tint. Yellow and amber tints will reduce blue light transmission. →

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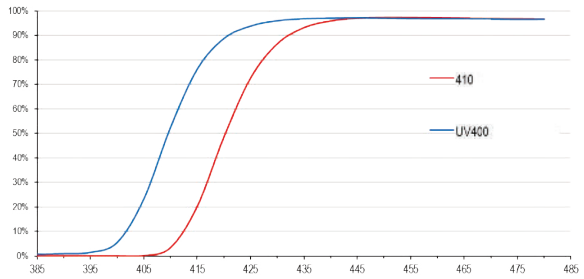

Figure 3.
Transmission curves of blue light absorbing materials

Patients may have seen adverts for more extreme light blocking spectacle solutions, taking out blue through to green wavelengths, to aid the sleep wake cycle of computer users. The implications of wearing any kind of blue blocking solution, especially those that remove wide ranges of wavelengths, has not been assessed and advice should be given accordingly.

Reflection

When light is incident on a lens, light is reflected from both the front and internal back surface. This will cause a reduction in contrast as the transmitted light is reduced. There are distracting internal reflections from both internal and external surfaces of the lens. Depending on the refractive index, transmission of visible light reduces from 92.4% with 1.5 index to 88.2% with 1.67 index. For this reason, high index lenses are generally not available without an antireflection coating.

Blue light problems and solutions have been publicised to such an extent that patients are often asking what kind of protection is available

To reduce reflections over the whole of the visual spectrum, a sequence of layers for different wavelengths is used. These combinations tend to produce coatings that have a residual minimal reflectance around the green area of the spectrum, which gives them a green hue. A coating will pick up the contrast for patients struggling in lower light levels and reduce distracting glare when night driving.

Anti-reflection coatings originally were designed to stop visible wavelengths from reflecting. However, UV can reflect from the back surface of the lens into the eye and so recently an extra layer in the multicoat stack has been developed to stop UV reflection. This extra layer is only necessary on the back surface of the lens.

Most lens manufacturers now offer an anti-reflection coating as a blue light solution. The coat reflects blue light and so has a blue residual hue as a tint or blue blocking material. The colour is indicative of the wavelengths reflected; a more violet hue indicates that the shorter blue wavelengths are reflected. None of these coatings block blue light completely. Indeed, on average they reduce blue light by about 10% to 12%, but individual manufacturers should be consulted for details. As they reduce the transmission of blue light, along with the blue surface hue, the lens will have a residual amber colour.

These coatings are ideal for digital devices as they are designed to reduce specific wavelengths of blue light. They are not as suitable for night driving as they will have back surface and internal reflections.

Generally, a UV solution should always be considered and explained to the patient. UV transmission needs to be blocked by the material or treatment to the material. UV reflectance from the back surface should be addressed by an anti-reflective coating specifically designed to prevent UV reflection. This is just as important with sunglasses as the pupil will be enlarged, reducing the eye's natural defense.

If a high energy visible blue light solution is being offered, materials, tints or coatings can all be offered so long as the patient understands the implications of the amount of blue light being blocked. Consideration should be given to vision in low light levels when the eye's maximum response shifts towards the blue end of the spectrum. Patients who notice distracting reflections caused by a blue anti-reflection coating may be more comfortable using a material or tint solution.

As optometrists we have the power to control light transmission, reflection and absorption, and in doing so enhance our patients' visual experience, comfort and quality of life. It all comes down to choosing the most effective lens solution.

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Infection control guidelines for optometrists

The new normal

Optometry Australia recently published an update to its 2016 Infection Control Guidelines in *Clinical and Experimental Optometry*, now available freely online.

There are two levels of infection control precautions that can be implemented in optometry practices, depending on the transmission of infection risk. During the COVID-19 pandemic, transmission-based precautions were applied, but this paper will focus on standard precautions – those that should be implemented during routine optometric practice. If you were not following the recommended standard precautions prior to COVID-19, perhaps now is the time to set the 'new normal' for infection control in optometry.

So, when should you wash your hands, and for how long?

Hand hygiene is considered the most important measure in preventing the spread of infection in the health care environment.[1,2] Following the 'Five moments for hand hygiene' will minimise the risk of transmission of microorganisms between the health-care worker, patient and environment:[2]

1. Before touching a patient
2. Before a procedure
3. After a procedure or body fluid exposure risk
4. After touching a patient
5. After touching the patient's surroundings

Hand hygiene usually consists of hand washing (with antibacterial soap and water) or hand rubbing (with alcohol-based sanitiser). The hand wash procedure should be used when the hands are visibly soiled and takes approximately 40-60 seconds. The hand rub procedure should be used when hands are visibly clean and takes approximately 20-30 seconds. Optometrists who are fitting contact lenses should use the hand wash procedure.

A small-scale study of optometrists in the United States of America found approximately 68% of respondents did not have a hand hygiene policy in their practice or were unaware of its presence.[3] Therefore, it is recommended that optometrists ensure they have and are familiar with the hand hygiene policy at their practice, and all staff are educated and trained on correct hand hygiene practices. Consider performing a hand hygiene 'audit' and providing feedback if compliance issues are observed.

Do you know when it is necessary to use personal protective equipment (PPE)?

Optometrists should understand the rationale for wearing PPE and its appropriate usage.[4] The PPE items most likely to be needed in optometric practice are gloves, masks and protective eyewear.

Gloves should be used when there is a possibility of contamination with blood or body fluids, or for procedures involving contact with mucous membranes, such as examining an eye with discharge.[5,6] They are single-use items and should not be substituted for hand washing; hands should be washed before and after using gloves.[5]

Masks should be used when either the patient or optometrist has respiratory symptoms, or when the optometrist will be in close contact with a patient who has a known or suspected pathogen that may be transmitted by airborne means, for example talking,

coughing or sneezing.[6] Surgical masks are single-use items that should be removed and discarded if they become soiled or wet.[1]

Protective eyewear will predominantly be used if there is a chance that contaminated fluids (including respiratory droplets) will be splashed into the eyes of the optometrist, or where there is the potential for airborne infection.[1]

A useful clip to review the correct procedure for donning and doffing PPE can be found at: **https://www.youtube.com/watch?v=qk6ai3JUL9U**. It is important to remember that hand hygiene must always be performed prior to donning PPE and immediately after the removal of PPE.[5]

How often do you clean the patient chair?

Environmental cleaning of optometric practices should be carried out regularly. The National Health and Medical Research Council (NHMRC) has produced guidelines for recommended routine cleaning of health-care facilities.[1] Optometry practices are considered low risk, but it is nevertheless recommended that the following items are cleaned daily: consulting room chair, hand washing sink and the doorknob to the consulting room. It is recommended to clean the following items weekly: consulting room computer, keyboard and mouse, benchtops, light switches and the waste receptacle. All of these items should be cleaned if they become visibly soiled. It could be argued that electronic devices and light switches should be cleaned more frequently in optometry practices, but if the correct hand hygiene protocols are being followed this should not be necessary.

Instrument shields, including slitlamp breath shields, should be cleaned and disinfected on a regular basis. Slitlamp breath shields are recommended for ongoing use in the COVID new-normal.[7]

Are you still using alcohol swabs for reprocessing of reusable equipment?

Reprocessing of reusable equipment requires cleaning, followed by disinfection or sterilisation. Any device that will make contact with mucous membranes or non-intact skin is considered semi-critical and must undergo high-level disinfection.[1,8] These devices include tonometer probes, gonioscopy lenses and trial contact lenses. Alcohol wipes are not considered high-level disinfection, but can be used for disinfection of non-critical items such as phoropters, trial frames, head and chin rests and other surfaces. Recent research[9] also suggests alcohol wipes may not effectively kill all viruses on semi-critical instruments.

A comprehensive list of protocols for disinfection or sterilisation of common reusable devices, instruments or equipment in optometry practices can be found in Appendix 2 of the infection control guidelines.[7] Please refer to Table 1 in this document for the protocols for reprocessing of various tonometry devices and PPE.

What do you do if you need to cough or sneeze?

Covering sneezes and coughs prevents infected persons from dispersing respiratory secretions into the air.[1] Ideally, if you need to cough or sneeze, this should be done by: covering your mouth and nose with a tissue, immediately discarding the tissue, and performing hand hygiene with soap and water.[1]

Optometrists should not come to work if they have symptoms of respiratory illness. Patients with a cold or influenza should be asked to reschedule their appointment if their eye examination is not urgent. If they must attend, patients with symptoms of respiratory illness should be kept as far away as possible from others and wear a mask.

What do you do with the bottle cap when you instill drops in the eye?

The aim of a clean technique is to reduce the overall number of microorganisms and lower the risk of transmission to a susceptible patient site.[1,10] One such 'clean' technique should be utilised when instilling multiuse drops into the eye. Best-practice involves:[3,7]

1. Checking that the product has not reached its expiry
2. Performing hand hygiene
3. Ensuring that the bottle cap is held in the hand (without touching the inside of the cap) while the bottle is in use
4. Instilling the drop into the eye, ensuring that the bottle tip does not touch the patient's eye, lids or lashes, the optometrists' hands or environmental surfaces. If this does occur, the bottle should be discarded.
5. Replacing the bottle cap immediately after use
6. Storing the opened product according to manufacturer's recommendations (for example, Mydriacyl – must be stored below 25°C, but not refrigerated).

Optometrists have had to become more diligent and rigorous with their infection control processes during the COVID-19 pandemic. "This presents an opportunity to create a 'new normal' where, amongst others: the appropriate use of PPE is standard; slitlamp breath shields are a permanent fixture; reprocessing of reusable equipment is in line with current recommendations; and environmental cleaning is part of the daily routine."[7]

Infection control survey results

Thank you to all those that participated in the infection control research. We are in the process of analysing the results and aim to publish them later in the year. Preliminary results indicate there are instances where practitioners are not feeling supported in their workplace to implement the recommended infection control protocols. If you feel this applies to you, please contact the Optometry Australia Professional Services helpdesk for support and advice: national@optometry.org.au or (03) 9668 8500

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CLINICAL RESOURCE

Infection control equipment guide

From Optometry Australia's infection control guidelines 2020 published in volume 104, issue 3 of *Clinical and Experimental Optometry*

Item	Storage	Before each use
PPE		
Eye protection – face shield, eye goggles		
Slitlamp breath shield		
Tonometry		
Tonometer reusable probe (Perkins or Goldmann)	Store in clean, closed container when not in use	Inspect for damage before use Wipe with 70% isopropyl alcohol wipe and air dry
Rebound tonometer (e.g. iCare, Finland)	Store in clean, covered case	
Tono-Pen (Reichert, USA)	Store in clean, covered case with fresh Ocu-Film Tip Cover on	Check if patient has allergy to Latex – Ocu-Film Tip Covers contain natural rubber latex[64]
Non-contact Tonometry (NCT)	Cover with dust cover overnight	

- The full list of references for this guide is available in Optometry Australia's infection control guidelines 2020, published in volume 104, issue 3 of *Clinical and Experimental Optometry*.
- For additional information on infection control including videos, factsheets and more, visit optometry.org.au/practice-professional-support/coronavirus-covid-19-what-optometrists-need-to-know/. This webpage is continually updated with the latest advice.
- Don't forget to look out for bonus *Optometry Connection* clinical resources, appearing online in August.

In an update to the 2016 infection control guidelines, authors Hart et al. carefully review and update the key practises and procedures that play a role in everyday optometry practice. The guide provides recommendations on both standard precautions and transmission-based precautions.

The table presented below is an excerpt from the guide, summarising key infection control procedures for commonly used practice equipment.

	After each use
	Low/intermediate-level disinfection required Consider manufacturer's instructions **OR** clean manually with an appropriate surface disinfectant, for example, Clinell Universal Wipes (Gama Healthcare Ltd, United Kingdom) – inside of face shield/eye goggles, followed by the outside.[62]
	Low/intermediate-level disinfection required Consider manufacturer's instructions. **OR** clean manually with a surface disinfectant, for example, diluted household bleach, 70% isopropyl alcohol wipe or an appropriate TGA-listed hospital-grade disinfectant with specific claims.
	High-level disinfection required *Sodium hypochlorite* Clean with mild pH neutral detergent*, then rinse with sterile water/saline before disinfecting. Soak prism on its side fully immersed in sodium hypochlorite (5000ppm) for 5-10 minutes, rinse thoroughly (avoid tap water) and completely dry before use. Note: The appropriate concentration of sodium hypochlorite is 5000 ppm, approximately 0.5%. Household bleach is typically 5-6% sodium hypochlorite, so a 1:10 dilution of bleach (1 part bleach, 9 parts water) equals 5000 ppm, and this solution should be made daily. Please note this is 'off-label' use for bleach products which are typically registered with the TGA for surface disinfection. *Tristel Duo OPH* Clean with mild pH neutral detergent*, then rinse with sterile water/saline before disinfecting. Dispense 2 doses of Tristel Duo onto a Tristel Dry Wipe or directly onto the instrument, leave on surface for 2 minutes, rinse thoroughly (avoid tap water) and completely dry before use .
	Single use only Discard probe after each use in sharps container.[63]
	Single use only Replace Ocu-Film Tip Covers before using the Tono-Pen on another patient.
	Low/intermediate-level disinfection required NCT may result in 'splash back'[40], so it is necessary to wipe with an appropriate disinfectant wipe between patients if infection is suspected.[42] It may be prudent to also perform several air puffs in between patients, particularly if infection is suspected or present.[42]

*The cleaning product should be registered by the TGA as a Class I medical device cleaning product.

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Vision and falls

Implications for optometrists

Falls are common in older people. At least a third of those aged over 65 years report falling at least once per year, with about half reporting multiple falls.[1,2] Older and frailer people have higher rates of falls, and the mortality rate associated with falls increases significantly with age. Falls account for over 80% of injury-related deaths in persons aged over 65 years.[2] Importantly, the number of older Australians who experience falls and fall-related injuries will increase due to our ageing population; in 2018, an estimated 3.9 million Australians (16% of the total population) were aged 65 or over,[3] and this is projected to increase to over 20% by 2066.[4]

Falls have traditionally been viewed as accidents that are unpredictable and potentially unavoidable, however, there is clear evidence that falls in older people are associated with well-defined intrinsic and/or extrinsic factors, and are often multifactorial in nature (**Table 1**).[1, 2] The more risk factors you have, the more likely you are to experience a fall. Furthermore, a number of these risk factors are amenable to interventions to reduce the risk of falls, such as exercise programs, environmental modifications, medication reviews and behavioural interventions.[5] Importantly, visual impairment is a key risk factor for falls.

Intrinsic risk factors	Extrinsic risk factors
Increasing age (65+ years)	Poor lighting
Female sex	Presence of trip hazards such as loose rugs
Gait and balance impairment	Inappropriate footwear
Systemic conditions such as arthritis, postural hypotension, stroke, diabetes and Parkinson's disease	Unsafe stairways (no handrail and steps of variable height)
Sedative use	Irregular floors
Taking multiple medications (greater than four, polypharmacy)	Unsuitable bed and bath designs
A history of falls	
Visual impairment	

Table 1.
Risk factors for falls

Recommendations for older patients

The following recommendations are informed by findings from recent randomised controlled trials (RCTs),[6-9] which represent the best form of evidence for clinical interventions. The following results from four RCTs are particularly relevant to the clinical optometric management of patients regarding falls:

- Older, frailer patients prescribed new spectacles with large (over 0.75 D) changes in correction experienced an increased rate of falls.[6]
- Long-term multifocal wearers who were prescribed distance single-vision spectacles for outdoor wear experienced less falls if they were regularly going outside the home, but increased falls if they were less fit and did not get out much.[7]
- Patients with low vision experienced less falls following home hazard reductions.[8]
- First eye cataract surgery reduced recurrent falls.[9]

Clinical recommendations for optometrists

Understanding the risk factors for falls will increase your awareness of which patients are more at risk of falling **(see Table 1)**.[1, 2] It is difficult to determine a definitive falls risk profile for patients, as some risk factors are more strongly predictive of falls (e.g. diagnosis of Parkinson's disease, history of stroke, history of previous falls), while others vary according to the type and severity of impairment (e.g. vision impairment or gait/balance impairment). Determining the level of falls risk is a clinical judgment based on an understanding of the patient's medical, physical, and cognitive profile, and awareness of their exposure to risk. (so e.g., their activity profile and number of extrinsic risk factors). Many of the following recommendations are for patients who would be considered at moderate to high risk of falling.

Observing the patient

Take note of whether a patient seems frail or unstable on their feet, has difficulty with getting up or down stairs and if a carer or relative helps them move around.[2] Ask practice staff to inform you if they observe any of these difficulties. If a frail, older patient has broken their spectacles or has any obvious bruising/injury, ask if they have fallen recently.

Adaptations to your case history

A history of falls is a strong predictor of your patient's future fall risk, so ask your patient about any history of falls in the previous 12 months during your case history.[2] Take the time to establish their spectacle use, particularly whether patients wear their correction when walking inside and outside the home.[1] It is surprising how many older patients with significant ametropia do not like to wear their spectacles when walking outdoors. Tinted or photochromic prescription distance single vision spectacles may be a useful recommendation if they spend a significant amount of time outdoors.[7]

For multifocal (PALs or bifocals) wearers, determine whether they remove their multifocals when negotiating stairs.[1] This may be a good indication that an additional pair of distance single vision spectacles would be suitable for outdoor walking.[1, 7]

Optometric management of patients at moderate-high risk of falling

Promote regular eye exams for these patients, so that small changes in refractive correction can be made at regular intervals, thus avoiding the need for larger changes in correction, which can increase falls.[6] For these patients at-risk discuss the benefits of earlier referral for first-eye cataract surgery, where appropriate, to improve mobility and reduce the risk of falls.[9]

Advise patients to keep their distance spectacles on when walking outside the home where appropriate. However, emmetropes and low ametropes (particularly low myopes) will have a clear view of the travel pathway, steps and stairs when unaided, so that walking without spectacles (especially if they are multifocal wearers) may be a useful recommendation for them.

Where there are changes to your patient's prescription, warn them of any potential magnification changes with their new spectacles. Myopic shifts will make objects, including steps and stairs, look smaller and further away, while hyperopic shifts will make them look bigger and closer, and astigmatic changes will make stairs and steps slope.[1]

Advise low vision patients to seek home modifications to prevent falls, via occupational therapists and other health professionals.[8]

Prescribing to patients at moderate-high risk of falling

Do not change refractive corrections or spectacle designs unless you must: "If it ain't broke, don't fix it" is particularly relevant to older patients who often cope poorly with optical changes.[10] This includes avoiding changes in lens type (even changing the design of a progressive or bifocal lenses), base curve and optical centres, as well as refractive correction.

If required, any change in refractive correction should be conservative. Be very careful in changing the correction of an 'at-risk' patient by more than 0.75 DS.[6] You should also be very careful when making astigmatic changes, particularly if oblique (axes between 30°–60° and 120°–150°).[11] Make partial changes in cylinder and axis as appropriate and provide appropriate advice to patients regarding these changes.[1]

Do not prescribe multifocals if 'at-risk' patients currently wear single-vision spectacles or if patients are minimally ametropic and are used to walking about without spectacles.[1] Also be wary of using a monovision approach with 'at-risk' patients because of the loss of stereoacuity.[1]

Long-term multifocal wearers with minimal ametropia (and particularly low myopia), can be advised that they are less likely to fall if they remove their spectacles when walking outside their own home. If patients have significant ametropia and participate in frequent outdoor activities, they should use distance single-vision spectacles when outside their own home, other than when driving or shopping.[7]

Long-term multifocal wearers with significant ametropia who participate in minimal outdoor activity should continue to wear multifocals for most activities.[6] For those patients who wish to retain multifocals, provide them with reduced reading power multifocals that provide safer walking but allow adequate short-term reading.[12] This could be combined with full addition multifocals or reading spectacles for near work.

Prescribing to patients following cataract surgery

Ensure that the patient is involved in the decision making regarding their post-operative refractive error. For example, some patients who have been myopic all their life might wish to keep distance spectacles and read without spectacles post-surgery. (so i.e., surgeons target slight myopia), rather than lose their distance spectacles and have to use reading spectacles. This would also reduce the magnitude of change in refractive error following surgery, which may reduce falls rate.[13]

Avoid the use of multifocals in active older patients if possible.[11] Also provide new lenses between first and second eye surgery if the patient intends to wear distance spectacles following second eye surgery.[11]

Optometrists play an important role in fall prevention for their older patients, given their expertise in prescribing appropriate refractive corrections, recognising and diagnosing ocular disease, referring patients for appropriate ophthalmic treatment and educating patients about ocular disease. They also play an active role in community health care as part of a multidisciplinary approach to falls prevention and are well placed to connect older patients with other health services to assist in modifying non-visual falls risk factors.

For further information, please refer to the "Guidelines for optometrists to help prevent falls in older patients" available to download from: optometry.org.au/practice-professional-support/clinical-areas-of-interest/.[14]

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An approach to the assessment and management of patients with IRDs

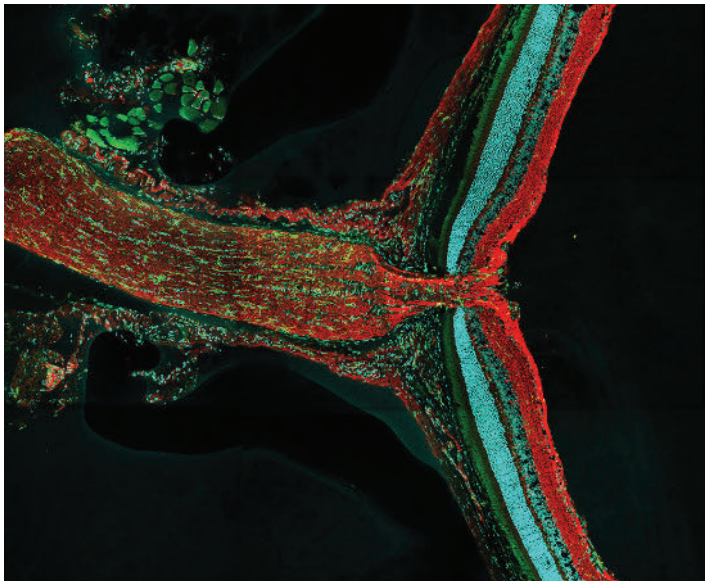
The arrival of gene therapy has led to a re-evaluation of how we care for patients with inherited retinal diseases (IRD). Whilst Luxturna® (voretigene neparvovec) for the treatment of RPE65-associated retinitis pigmentosa (RP)[1] is presently the only TGA-approved retinal gene therapy, a number of other causative genes are being targeted in late phase clinical trials, including choroideremia[2] and X-linked RP.[3] The recent online publication of the Royal Australian and New Zealand College of Ophthalmologists' (RANZCO) guidelines[4] for the assessment and management of patients with IRDs provides a new framework for eye-care professionals to improve and standardise the care we offer patients with IRDs. It formalises an approach that can be implemented for patients suspected of having an IRD and comprises four key components.

1. Establish the clinical diagnosis of an Inherited Retinal Disease

Critical to establishing a clinical diagnosis of an IRD is to consider a genetic cause in the first place. Not uncommonly, macular dystrophies can be misdiagnosed as geographic atrophy secondary to age-related macular degeneration, and early-stage rod dystrophies can be missed if a detailed history and dilated fundus examination are not performed. The diagnostic goal of the clinician is then to clinically categorise the IRD as narrowly as possible. This lowers the false genotype rate, defined as the 'frequency with which one would encounter a plausibly disease-causing recessive or dominant complete genotype when sequencing the coding regions of a specific set of genes in a healthy person'.[5] Narrowing the clinical diagnosis reduces the number of potential causative genes and false positive variants requiring interrogation by the clinical geneticist. The broad categories with which to filter IRDs include:

i) inheritance pattern
ii) affected area or cell type e.g., macular, chorioretinal or photoreceptor disease (the latter sub-divided into rod and rod-cone dystrophy, cone and cone-rod dystrophy)
iii) childhood or adulthood onset, and
iv) syndromic or non-syndromic.

A detailed family history should include drawing a landscape-oriented pedigree[6] in the patient notes to document the inheritance pattern e.g., autosomal dominant, autosomal recessive, X-linked or mitochondrial. Classic clues to look for include male to male transmission to excludes X-linked genes, or the presence of consanguinity, which raises the possibility of an autosomal recessive gene exhibiting pseudodominance.



Patients' earliest visual symptoms can help reveal whether the rod or cone photoreceptors were primarily affected e.g., difficulty seeing at night (rod dysfunction) or poor reading vision at school, photophobia, or dyschromatopsia (cone dysfunction). Progressive symptoms suggest ongoing degeneration whereas a static situation would be more typical of stationary cone or rod impairment e.g., congenital stationary night blindness. Always ask about systemic features, as syndromic RP makes up approximately 15% of all IRDs, and in particular hearing impairment, as Usher syndrome accounts for around one sixth of all RP.

Standard examination techniques are required to effectively assess an IRD patient e.g., slitlamp examination of the anterior and posterior segments. For only a minority of IRDs is it possible to make a genetic diagnosis at the slitlamp due to the limited phenotypic repertoire of the retina. These archetypal fundi include gyrate atrophy (OAT1), choroideremia (CHM) and Bietti crystalline dystrophy (CYP4V2). The most useful imaging modalities include optical coherence tomography (OCT) and fundus autofluorescence (FAF). Progression on OCT can be monitored by measuring the horizontal and vertical length of remaining ellipsoid zone and FAF frequently identifies abnormalities earlier than either clinical examination or conventional colour fundus photography; it is ideally captured using a wide field imaging system.

2. Determining the level of visual function and arrange visual rehabilitation

Further subjective enquiry regarding the level of visual function is best approached from the perspective of activities of daily living e.g., discussing tasks such as driving, reading, mobility, night vision, use of visual and/or mobility aids and degree of independence in familiar or unfamiliar environments. For a more objective assessment, Goldmann semi-automated or manual perimetry may be useful, with binocular roving Esterman field for driving assessments. Microperimetry is also useful but is currently restricted primarily to the research setting. The pivotal clinical trial of Luxturna used an assessment of functional vision called the multi-luminance mobility test (MLMT) performed under standardised luminance to demonstrate efficacy of the treatment.[1] However, the MLMT has demanding set up requirements and is not a practical option for most clinical settings. Extensive resources can be deployed to assist patients following referral to and engagement with nationwide agencies such as Vision Australia (https://www.visionaustralia.org) and Guide Dogs Australia (https://guidedogs.com.au).

3. Establishing the genetic diagnosis and genetic management

Referral to a multidisciplinary specialist ocular genetics clinic is now the gold standard approach when a genetic diagnosis is required. A causative mutation can now be identified in up to 60-80% of patients with IRDs.[5,7] A specialist ocular genetics clinic should include a clinical geneticist, genetic counsellor, ophthalmologist and specialist genetics orthoptist and nurse. Increasingly, multi-disciplinary team (MDT) meetings are required to determine the pathogenicity and significance of some variants, especially the variants of uncertain significance. An experienced genetic counsellor is needed to interpret and communicate results of a genetic report to patients at a follow-up clinic appointment. Through the pre- and post-test clinic visits, the counsellor provides education and physiological support to assist patients' understanding of the significance of the result for both themselves and their family. Patients can be referred to a clinical genetic services throughout Australia. Optometry Australia has published a list on their website for optometrists to refer to.[8]

4. Monitor the disease progression (natural history) and, prepare for therapeutic interventions

The RANZCO guidelines recommend annual ophthalmic follow-up until 18 years old, then every two years from then onwards. Enrolment in natural history studies such as those conducted at the Centre for Eye Research Australia (www.cera.org.au) can be an additional, constructive opportunity for patients to keep up to date with the latest research and for them to make a contribution to advancing our understanding of their particular IRD. Maintenance of IRD research registries also ensures efficient communication with patients regarding recruitment opportunities for upcoming clinical trials.

The most urgent concern expressed by most patients is the extent to which their IRD may have progressed since the last review. Monitoring change over time allows an estimate of the rate of progression. Useful modalities include serial perimetry, measuring the width of the residual ellipsoid zone on OCT, or recording the area of normal FAF area, e.g., in choroideremia,[9] which can together inform patient and clinician alike and usually provides reassurance of the relatively slow rate of progression.

A number of task forces have now assembled guidelines on criteria for treatment with the first approved retinal gene therapy, Luxturna, including the German Ophthalmological Society, German Retina Society and Professional Association of German Ophthalmologists Statement.[10] These include reference to the pivotal phase III clinical trial[1] and in particular the use of the full-field stimulus threshold test (which is typically performed in a specialist centre) and a clinical and anatomical assessment of the viability of remaining retinal photoreceptors. The overall assessment of eligibility for treatment with Luxturna is taken by an MDT including members of the ocular genetics clinic team, treating surgeon and IRD eye specialist(s).

The outlook for most patients with an IRD is still largely one of unmet need for a therapeutic intervention, but there have been encouraging developments led by the arrival of i) Luxturna; and ii) improved access to genetic testing, which will facilitate intervention before loss of viable photoreceptors becomes widespread. Many of the IRDs being targeted in clinical trials have been selected due to their suitability genetically e.g., gene size within the viral vector's payload capacity and a loss-of-function mechanism conducive to a gene augmentation strategy. Numerous other IRD genes present significant challenges for gene therapy researchers to overcome. Encouragingly, however, the eye sector is leading the application of this exciting technology and our clinical practice is evolving to keep pace with new developments by providing comprehensive clinical and molecular diagnoses for our patients.

A partial list of clinical genetic services in Australia is available on the Optometry Australia website: https://www.optometry.org.au/institute-of-excellence/publications/optometry-connection/clinical-resources/

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The diabetes pandemic

How do we fit in?

Optometry is the entryway into the medical system by often being the first contact for diabetic patients. Primary-care physicians often refer their diabetic patients for annual dilated eye examinations to optometrists, however technology – in the form of artificial intelligence (AI) systems – may soon fundamentally interrupt these diabetic retinopathy examinations by optometrists.

Artificial intelligence and optometry

AI is considered to be the fourth industrial revolution in human history. The two medically relevant subsets of AI are machine learning and deep learning. Machine learning converges around the capability of machines to capture a set of data and learn for themselves, adjusting algorithms as they learn more about the data being processed. Deep learning, also known as deep neural networks, is a subset of machine learning.[1] In this case the "machines" are imaging technology that is "trained" in identifying diabetic retinopathy. Currently there are two FDA-approved autonomous AI diagnostic imaging systems for diabetic retinopathy screening. IDx-DR (Digital Diagnostics) and EyeArt (Eyenuk) have been developed to be used in the primary-care physicians', endocrinologists' or diabetologists' office. Staff training is minimal. Photographs are taken using a non-mydriatic digital fundus camera. These autonomous systems identify whether the patient has diabetic retinopathy or diabetic macular oedema and inform the primary-care physician if the patient needs to be referred to a retina specialist. These AI-based diabetic retinopathy screening algorithms have reached, or even outperform, the level of accuracy of clinical retinal experts. IDx-DR has even become part of the American Diabetes Association's (ADA) standards of diabetes care recommendation.[2] These technological advances stand to offload diabetic retinopathy screening examinations from optometrists to computer-based screening programs since they require only lower-compensated and less-skilled operators.[3]

This onrush of technology and other factors should motivate optometrists to "step up their game" by switching gears if they want to continue to grow and evolve within the health care arena. This means getting more involved in a whole patient approach from screening patients for prediabetes, to helping coordinate the patients' care and, finally, to assessing the patients' diabetic status and treatment regimen. Otherwise, your diabetic patients may be outsourced to a diabetic retinopathy screening kiosk soon to be found in every shopping mall, grocery store and general practitioner's office.

It is not that difficult, but it does take some additional assessment and time. Significant impact can be attained just by identifying your patients who may be prediabetic, informing them of your findings and encouraging them to seek appropriate medical evaluation in a timely fashion. Do not be intimidated if you do not know the risk factors for prediabetes. You are not alone. In a recent study of 140 primary care providers (family medicine, internal medicine, nurse practitioners, physician assistants, etc.), only 6% correctly identified the 11 risk factors prompting screening by the ADA guidelines. In addition, 30% of these primary-care providers were not familiar with the ADA guidelines.[4] Prediabetes is the term used for individuals whose glucose levels do not meet the criteria for diabetes but are too high to be considered normal.[5] **Table 1** lists the common risk factors and diagnostic laboratory values for prediabetes. So why do you need to be concerned about prediabetes? The Diabetes Prevention Program reported that diabetic retinopathy has been found in nearly 7% of individuals with prediabetes.[6]

The impact of diabetes

Diabetes affects the eye and the body by two distinct but inter-related processes: macrovascular disease and microvascular disease. Macrovascular disease encompasses cardiovascular events such as myocardial infarction and cerebrovascular events

such as strokes. Microvascular disease causes nephropathy in the form of renal failure, neuropathy which affects the autonomic nervous system, peripheral nerves (cranial neuropathy) and the brain resulting in cognitive decline and, finally, diabetic retinopathy (**Figures 1 and 2**).

This can be extremely overwhelming to the average diabetic patient. There are many patient-related factors that can act as barriers to prevent the patient from not only comprehending the importance of good systemic diabetic control but actually contribute to the patient undermining their own care (**Table 2**).[7-9] There are also system-related factors that can act as barriers for optimal diabetic patient care (**Table 3**).[10,11] Finally, there are clinician-related factors which can act as barriers (**Table 4**).[12] All of these barriers contribute to the concept of clinical inertia. →

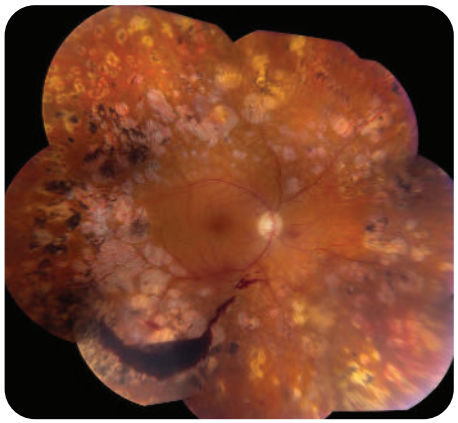

Figure 1.
Recurrence of proliferative diabetic retinopathy with inferiorly located "keel" vitreal hemorrhage. Note extensive panretinal photocoagulation therapy which was performed in the past.

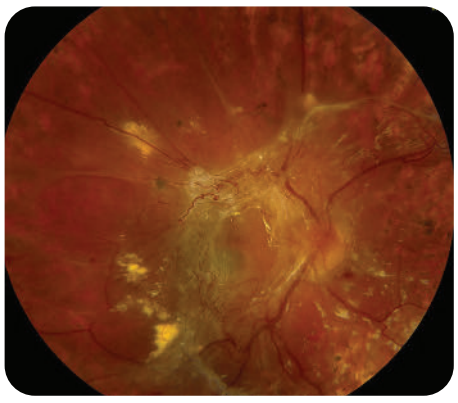

Figure 2.
Extensive fibrotic changes involving the disc and macula in a patient with severe proliferative diabetic retinopathy and treatment which included panretinal photocoagulation and anti-VEGF injections.

Risk Factors
• Age ≥ 45 years
• Body mass index ≥ 25 kg/m2
• Hypertension
• Dyslipidemia
• Cardiac disease
• First degree relative with diabetes
• Physical inactivity
• Black, Latino or Asian
• History of gestational diabetes
Laboratory Values
• Fasting blood glucose: 100-125 mg/dl
• HbA1c: 5.7-6.4%

Table 1.
Prediabetes (adapted from American Diabetes Association guidelines)

• Absence of symptoms leading to denial of disease severity
• Low health literacy
• Cognitive impairment or mental illness
• Poor education about how diabetes affects the eye
• Inability or unwillingness to follow a complex treatment regimen
• Feelings of failure about suboptimal glycemic control

Table 2.
Patient-related factors

• Payer policies and formulary constraints
• Resource constraints limit availability of clinician and staff to educate patients and develop individualised care plan
• Team approach to care is poorly coordinated or nonexistent
• Discordance among clinical practice guidelines
• Inadequate supportive technologies

Table 3.
System-related factors

• Underestimation of patient's need of therapy
• Lack of knowledge, tools or training
• Uncertainty about the need to intervene or the likely outcome
• Poor clinician communication
• Clinician may overrate the quality of the care they already deliver

Table 4.
Clinician-related factors

This concept of clinical inertia was pioneered in the field of diabetology and defined in 2001.[12] The first part of the definition involves the failure of health-care providers to initiate or intensify therapy when indicated. Regarding the treatment of diabetic retinopathy, this may involve hesitating in doing an intravitreal steroid injection in a patient with diabetic macular oedema that is not responding to a series of intravitreal anti-vascular endothelial growth factor (anti-VEGF) medication injections. Additionally, there could be a failure to establish appropriate treatment targets.[13] Diabetic retinopathy examples could include when to add retinal laser to the anti-VEGF therapy and when to do vitrectomy. There is also the failure to make treatment decisions that follow evidence-based guidelines.[13] These may include a type 2 diabetes mellitus patient not having a minimum of four diabetes-focused visits per year with their medical doctor or the diabetic patient not having an annual dilated eye exam. Finally, there is also the failure to de-intensify treatment when appropriate.[14] An example of this is when and how aggressively should the clinician initiate the "treat and extend" protocol in those diabetic patients that are responding to anti-VEGF therapy.

The optometrist's role

So how does an optometrist put all this into practice? A significant part is already being done by optometrists as they provide care in their primary-care practice. The most important aspect of this includes effective communication through patient education. Optometrists should take every opportunity to make sure the diabetic patient is adhering to their treatment and diet regimens. Enquire if the patient's HbA1c is optimised and individualised.[15] Communicate and collaborate with the primary-care physicians and other providers such as endocrinologists, nephrologists, podiatrists and registered dieticians. An Australian study found that diabetic patients treated using a multidisciplinary care approach had improved metabolic control and decreased cardiovascular risk factors.[16] Always remember that the central member of this team is the patient.

Other potential team members include certified diabetes educators who have been shown to improve adherence. These specialists council patients on how they should implement specific lifestyle changes, educate them about diabetes and its causes, create individual self-management plans and make sure that a patient can test and record their blood glucose levels. I know of several optometrists in the United States who have attained certification as diabetes educators* and added this service to their practices.[17] Behavioral health specialists can assist the optometrist by addressing the patient's feelings of failure and diabetes distress regarding their management of the disease and any suboptimal glycemic control.

Optometrists can offer simple, easy to implement suggestions to patients regarding better glucose control. As an example, walking at least 10 minutes after the largest meal of the day lowers blood glucose 22% more than a 30-minute walk before any meal.[18] Finally, optometrists need to embrace new and updated technology. Optical coherence tomography (OCT) and OCT-Angiography show us structural changes that we were never able to see in the past. These advanced technologies help us correctly identify leakage and diabetic macular edema. Electrophysiological techniques can objectively measure retinal function helping the clinician intervene with nutritional supplementation to improve macular edema and visual function.[19] These technologies are a complement of structure and function regarding retinal and macular health. Optometrists should also appreciate AI's potential utility as a prognostic tool. It can be used to help prognosticate the outcome of a given clinical case based on imaging and other data and then it can recommend the best treatment. Once perfected, AI can give us true personalised medicine.

*Optometry is not on the professions list of the Australian Diabetes Educators Association Credentialled Diabetes Educators.

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Normal tension glaucoma

Case study and discussion

Glaucoma has historically been a challenging disease to detect and diagnose as demonstrated by the Blue Mountains Eye Study, where 50% of the estimated 300,000 Australians over the age of 40 with glaucoma were undiagnosed.[1]

Normal tension glaucoma is one of the most common forms of glaucoma in Australia, Japan, Western Europe and the US.[2,3] In addition to other forms of detection such as fundoscopy, enhanced visibility of the retinal architecture enabled by OCT has been hugely beneficial in supporting optometrists to detect early nerve fibre layer and ganglion cell loss. Equipped with this information, optometrists have been more able to make consistent patient management decisions to perform clinically indicated visual field assessment, interpret structural and functional correlations and then refer for ophthalmological intervention appropriately, as recommended in the RANZCO Referral Pathways for Glaucoma Management and Optometry Australia Clinical Practice Guide for the Management of Open Angle Glaucoma.

Case study and examination

An 82-year-old Caucasian female presented for a routine eye test. She had no initial complaints but on questioning mentioned that vision in her right eye wasn't as clear as her left eye, even with optical correction from a year prior. Her medical history included controlled hypertension and migraine headaches, and she was taking multiple medications, but no steroidal medication. Previous ocular history included successful cataract surgery in both eyes six years prior.

Clinical assessment

The patient's best corrected visual acuity was 6/7.5-1 in the right eye and 6/6 in the left, with mild astigmatic correction (R pl/-0.75 x 100, L +0.25/-1.00 x 80). IOP measured with Perkins tonometry at 11:10am, was 11 mmHg in both eyes. Corneal thickness was 474 µm in the right eye and 495 µm in the left.

The patient's angles were open on Van Herick, and there were no signs of pigment dispersion or pseudoexfoliation syndrome. The patient's IOLs were clear and well centred.

The patient was dilated with 0.5% Tropicamide. Internal examination showed flat maculae with no evidence of epiretinal membrane or vitreomacular traction. The right optic nerve showed focal rim thinning inferiorly, with correlating retinal nerve fibre layer (RNFL) wedge defect and thinning of ganglion cell layer (GCL) inferiorly noted in the OCT scan (**Figure 1**). The left optic nerve appeared normal (**Figure 2**). Comparison of the two OCT scans showed marked disc asymmetry (**Figure 3**).

On the basis of the optic nerve findings, a full threshold visual field test was conducted (Medmont M600 Glaucoma module). A repeatable paracentral visual field defect was found in the right eye, in the superior nasal quadrant – this correlated to the inferior temporal thinning of the ganglion cell layer (**Figure 4**).

Diagnosis and management

Several differential diagnoses were considered. With no evidence of pseudoexfoliation or pigment dispersion, secondary causes of open angle glaucoma were ruled out.

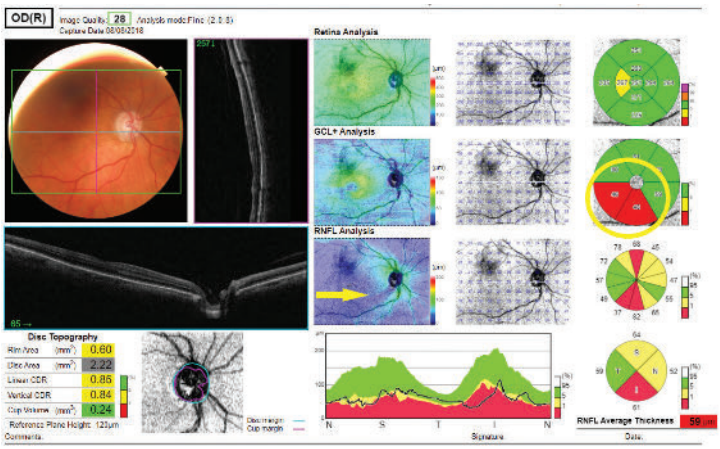

Figure 1.
3D wide report of RE showing RNFL wedge defect (yellow arrow) and GCL thinning (yellow circle)

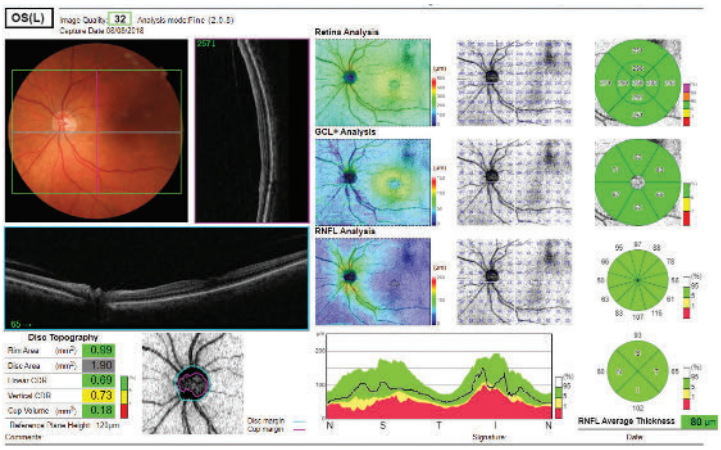

Figure 2.
3D wide report of LE showing RNFL and GCL within normal limit

Physiological disc asymmetry was ruled out by the visual field defect in the right eye, which correlated with the structural changes seen. A diagnosis of normal-tension glaucoma was made, and the patient was referred to a local glaucoma specialist for management.

The patient was also counselled that her right eye vision was affected by the absolute paracentral scotoma, and she was referred to Glaucoma Australia to help facilitate her understanding of her disease. She was also advised to encourage her family members to have their eyes tested.

Discussion

The patient outlined was a classic case of normal tension glaucoma. Compared to other forms of glaucoma, normal tension glaucoma has a relatively slower average rate of progression with reported mean deviation visual field loss of 0.36 dB-0.41 dB/year as opposed to 3.13 dB/year in pseudoexfoliative glaucoma and 1.31 dB/year in high-tension glaucoma.[4,5] However, normal tension glaucoma visual field defects tend to be more localised, dense and closer to fixation.[6-9] In this patient's case, it had resulted in a central visual field defect in her right eye.

Since ophthalmological assessment, the patient's intraocular pressure has been controlled with selective laser trabeculoplasty (SLT) and prostaglandin analogue eyedrops (Xalatan, latanoprost 0.005%) and she is being reviewed on a six-monthly basis.

When we last saw her six months ago, all findings were relatively stable and she was still able to have great quality of life by being able to drive, read and crochet. However, she was still noticing that her right vision was worse than her left and this would sometimes affect her ability to perceive detail. From our discussions and her communications with Glaucoma Australia, she understood that the vision loss was irreversible and whilst it is a shame that we can never grant her that vision back or go back in time to make the diagnosis and referral earlier, we can be thankful that her level of vision has been preserved and with that her quality of life.

Successful glaucoma management is a collaborative effort between optometrist, ophthalmologist and patient advocacy groups such as Glaucoma Australia. Managing the disease is important, but so is managing the patient.

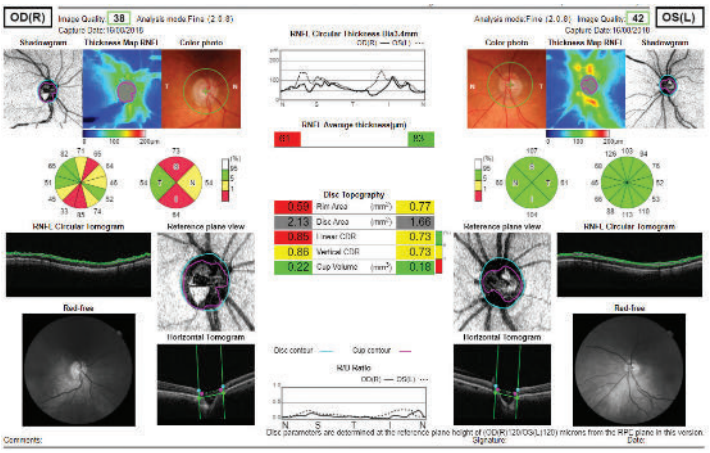

Figure 3.
3D disc report showing disc asymmetry

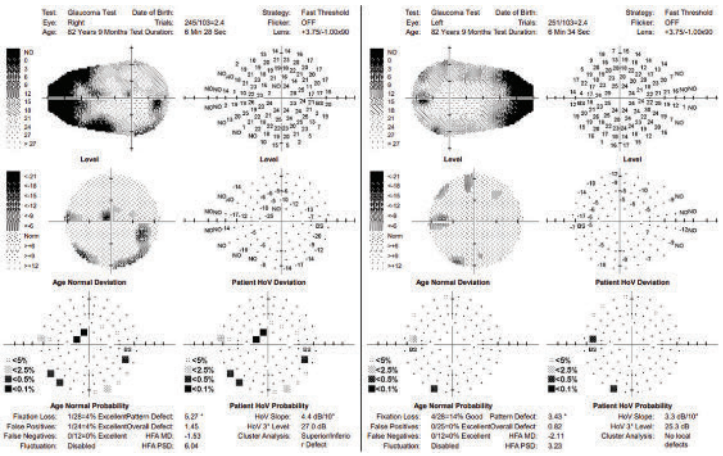

Figure 4.
Visual field from most recent presentation showing a repeatable paracentral defect in the right eye.

About the author

Tyson graduated from UNSW in 2017 and was the recipient of the Andrew Whatham Centre for Eye Health prize, the Leonard Fine Therapeutics prize and a UNSW Summer Research Scholarship. He has had work experience at the Centre for Eye Health, Omni Eye Services in Atlanta and with Lion's Outback Vision as part of the Judy Glover Memorial Scholarship. Since graduating, he has moved to regional NSW to practise and is currently co-chair of the ECONA Rural and Regional Committee.

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Optometry and secure communication

Where are we now and where do we want to be?

Health care in Australia is increasingly moving towards a patient-centric, individually tailored model of care with optometry as a profession aiming to increase its role in this mode of collaborative care.[1] A key enabler for patient-centric care is streamlined and regular communication between associated members of the patient's health care team. This is highlighted in the Optometry Board of Australia's Guideline for the use of scheduled medicines[2] which clearly states that: "communication is the linchpin of effective collaborative care".

Who do we need to communicate with?

To implement communication systems, it is critical to first recognise the key stakeholders that optometrists either currently liaise with or may work with in the future for the benefit of our patients. These can be broadly broken down into several main categories:

- Medical practitioners: the most common optometric patient collaboration is with general practitioners and ophthalmologists in both public and private settings; in a patient-centric model, optometrists will increasingly also be liaising with a wider range of medical practitioners such as rheumatologists, endocrinologists, neurologists, geriatricians, paediatricians, dermatologists, geneticists, haematologists, pathologists, cardiologists, immunologists, psychiatrists, and radiologists.[3]
- Allied health practitioners: pharmacists, genetic counsellors, social workers, psychologists, dieticians, occupational therapists, audiologists, chiropractors, orthoptists, speech pathologists and nurses involved in vision screening programs.[4]
- Community and special interest groups: Glaucoma Australia, Macular Disease Foundation Australia, Diabetes Australia, Guide Dogs, Vision Australia, Keratoconus Australia, Retina Australia etc.
- Public Registries: My Health Record, KeepSight, Save Sight Keratoconus Registry[5] etc.
- Patients, their carers/guardians, school teachers or counsellors, current or future employers
- Regulatory bodies: Roads and Maritime services and the equivalent in other states, Medicare, state and federal courts

What could an ideal medical communication system look like?

1. Data security and encryption for messages to ensure that they cannot be read or received by anyone other than the intended.
2. Cross communication between industry providers such that messages can be sent and received by any practitioner, regardless of the specific vendor to which they subscribe.
3. A constantly updated, centralised directory of practitioners and organisations featuring both practice (location) as well as practitioner specific information, and ideally a method for managing communication from and to locums.
4. Industry-wide standardised referral and report templates which auto populate from all practice electronic medical record (EMR) systems.
5. Synchronised linkages between secure messaging and all currently utilised EMRs to enable reports to be directly assigned to and imported into patient files at the receiver's end.
6. A centralised patient file system to enable reports and results to be accessed efficiently and, with the patient consent where needed by other practitioners involved in the patients care that were not included in the original communication (e.g., in the instance where a patient has changed locations or practitioners).

How close are we to this idealised concept?

There are a large range of providers, systems and technology currently actively working in this space, and this article not designed to be a comprehensive review of available options.

Data security and cross communication

There will always be concerns about data security regardless of the amount of time, money and effort spent on securing and encrypting individual's health records and reports.

What is considered best practice is constantly evolving and can be difficult to establish. In 2015 Optometry Australia released a Practice Note on this topic.[6] The Royal Australian College of General Practitioners is currently reviewing their guidelines following the changes detailed below, as well as with the uptake of new technologies following the COVID-19 pandemic, with an update due in late March 2021.[7]

The Australian Digital Health Agency (ADHA) recently undertook a large project to convert Australian secure messaging providers to an industry-wide standard code known as Fast Healthcare Interoperability Resources (FHIR). This common coding now enables a high level of encryption, while ensuring that all messages sent by different services 'speak the same language'. Following this, it has published a review of the safety and quality benefits of secure messaging.[8]

It is critical to first recognise the key stakeholders that optometrists either currently liaise with or may work with in the future

There are currently 11 listed providers of secure messaging delivery[9] as well as multiple other options including secured emails, encryption software[10] and cloud-based providers typically targeting specific industries such as Oculo. Unfortunately, proprietary and financial interests of individual providers mean that although messages can now be sent to and from multiple platforms, this functionality has had very limited uptake.

Centralised directory of practitioners

Each specific secure communication vendor has a directory of practitioners that use their specific platform for secure messaging. These are also now linked into EMRs such that a practice's EMR system can search for and automatically update contact details within their systems to streamline communication. As mentioned above however, there is limited ability to communicate between different vendors. A potential solution to some degree, (although one which at the time of writing is not an option), would be linking vendor contact lists to the government's Medicare database to enable cross checking of registered provider numbers. Consequently, vendor lists are largely dependent on the individuals to update them with the vendors.

Standardised referral and report templates

Even considering the eye industry alone, obtaining a consensus on what is required in a report or referral template is difficult to achieve. When combined with the plethora of conditions that can be covered in reports and referrals, as well as a wide variety of EMRs and secure messaging platforms, it is unlikely that uniformity will be achieved. Pro forma templates such as in Oculo and in practice groups that have minimum required information are improving standardisation in referrals and a process of building agreed templates, such as the ACI cataract referral form, into EMRs[11] will continue to improve this aspect with time.

Synchronised linkages between secure messaging and all currently utilised EMRs

GP software has had this functionality for many years in combination with secure messaging providers. Unfortunately, due to the variety of providers (both EMR and secure messaging) and the relatively small number of optometrists nationally, there has been little impetus to implement this functionality in the eye industry.

A centralised patient file system

My Health Record statistics currently report that 22.91 million Australians have an active record with over 2.67 billion documents uploaded. This last statistic however is skewed by a large percentage (86%) being Medicare documents (records of billing etc.). EMRs are now rolling out the ability to post reports and images to My Health Record at the same time as sending them through secure messaging to a health-care provider. While this functionality is available in a large number of GP EMRs such as Medical Director, Best Practice etc. as well as products owned by them designed for other health professions such as VIP.net (owned by Best Practice), there does not appear to be a notable push for incorporation within ophthalmic specific software or registries such as KeepSight.

The COVID-19 pandemic has accelerated the pace of change with regards to telehealth and communication. Many of the elements for an ideal communication platform currently exist in some form however, there remains a lot of work to be done to create a cohesive system whereby optometry can and does communicate effectively, efficiently and securely with all related professionals within a patient centric model of care.

The Author would like to thank Dr Angelica Ly for reviewing and valuable input into this article.

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Unveiling the diabetic eye disease epidemic

Diabetes-related vision loss is the leading cause of blindness in working aged Australians. With 1.8 million Australians estimated to have some form of diabetes, this poses an enormous disease burden to not just the individual but the community and nation as a whole.[1] However, if detected early and managed promptly, diabetes-related vision loss is largely preventable.

Diabetes mellitus is defined as a metabolic disorder characterised by the presence of hyperglycaemia secondary to dysfunction in insulin secretion and/or response to insulin. Diabetes can be largely divided into type 1 and type 2: type 1 is principally autoimmune or idiopathic in aetiology and shows earlier onset, while type 2 is predominantly due to acquired insulin resistance thus having onset later in life.[2] Long term, diabetes can lead to both microvascular and macrovascular complications; including retinopathy, neuropathy, nephropathy, as well as cardiac, cerebrovascular and peripheral vascular disease. Diabetic retinopathy is a significant cause in 1.4% of those with visual impairment, and the impact is even greater at 5.2% in the Aboriginal and Torres Strait Islander population.[3]

Screening guidelines

Given the silent nature of the disease, it is of critical importance that diabetic patients are screened and monitored regularly to ensure early detection of diabetic eye disease. It is therefore our role as collaborative health care providers to ensure our patients receive appropriate care. The current Australian National Health and Medical Research Council (NHMRC) guidelines – which are endorsed by Optometry Australia and the Royal Australian and New Zealand College of Ophthalmologists – recommend that all patients receive an initial screening examination at time of diagnosis.[4] For patients with type 1 diabetes, the first screening examination should be conducted when they reach puberty. A recent study found that 6% of patients already had some degree of diabetic retinopathy at time of diagnosis with diabetes.[5] Furthermore, this initial consult is key in establishing a therapeutic relationship with the patient and ensuring the patient understands the imperative of ongoing monitoring. In fact, 75% of Australians who were non-adherent to their screening examinations reported being unaware of the need for regular monitoring, or did not accurately understand →

Severity grading		Clinical features	Recommended ophthalmologist review
Diabetic maculopathy	Non-centre involving	MA, Hb, HEx, thickening within 2DD but not within 500µm of foveal centre	Within 12 weeks
	Centre involving	MA, Hb, HEx, thickening within 500µm of foveal centre	Within 4 weeks
NPDR	**Mild** (1-5% 1-yr risk of PDR)	MA only	Repeat screening in 1 year
	Moderate (12-26% 1-yr risk of PDR)	MA + Hb + VB (less than severe NPDR)	Within 12 weeks (Can monitor every 3 months if low risk)
	Severe (50% 1-yr risk of PDR)	4 : 2 : 1 rule 4 quadrants of MA and Hb + 2 quadrants of VB + 1 quadrant of IRMAs	Within 4 weeks
PDR		NVD, NVE, NVI Vitreous or pre-retinal Hb	Within 1 week
Sudden severe vision loss		4 quadrants of MA and Hb + 2 quadrants of VB +	Same day referral
Vision loss not otherwise explained		1 quadrant of IRMAs	Refer to ophthalmologist (ideally within 4 weeks)

Table 1 .
Summary of diabetic retinopathy severity grading and referral guidelines (grading according to Global Diabetic Retinopathy Project[7]; modified from RANZCO 2020[8] and Optometry Australia 2018[10])

NPDR: non-proliferative diabetic retinopathy, **PDR:** proliferative diabetic retinopathy, **MA:** microaneurysm, **Hb:** haemorrhage, **HEx:** hard exudate, **DD:** disc diameter, **VB:** venous beading, **IRMA:** intraretinal microvascular abnormality, **NVD:** neovascularisation at the disc, **NVE:** neovascularisation elsewhere, **NVI:** neovascularisation at the iris.

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Seeing beyond

the risk of developing diabetic eye disease.[3] Following the initial examination, patients should be reviewed at least biennially unless they are deemed 'high risk', in which case they should be assessed at least every year. High risk features include:

- → Diabetes for ≥ 15 years
- → Poor glycaemic control (HbA1c > 8%)
- → Systemic disease – such as poorly controlled hypertension, blood lipids
- → Other systemic diabetic complications – such as nephropathy, cardiac disease, peripheral vascular disease
- → Of Aboriginal or Torres Strait Island descent

Concerningly, only 50 to 70% of diabetic patients were found to have met their biennial examination recommendation, while only 21 to 28% of patients with diabetes for greater than 15 years followed their annual examination guideline.[5] Furthermore, only half of diabetic patients of Aboriginal or Torres Strait Islander descent received their annual eye check, and less than one in four had any diabetic eye screening test at all.[6]

Diabetic retinopathy is a spectrum of disease that requires individualised management depending on extent and severity. The Global Diabetic Retinopathy Project Group simplified the grading of diabetic retinopathy, and this is still widely used today.[7] Grading is based on the clinical features seen on examination, which dictates the urgency of ophthalmology review and treatment. This is summarised in **Table 1**.

Identifying disease progression

Digital fundus photography can be helpful in easily assessing the posterior segment, particularly with non-mydriatic pupils. With the advance of ultra-wide field high resolution fundus imaging, its utility in screening examinations are becoming increasingly recognised.

Direct clinical signs, however, may be misleading in establishing the degree of underlying retinopathy. A high index of suspicion should be maintained if there is a mismatch between the severity of apparent retinopathy compared to the rest of the patient. For example, if the patient has had diabetes for decades and is known to have advanced diabetic nephropathy and vascular disease, but only appears to have mild retinal changes, there may be more to the picture.

This is where ancillary imaging modalities can provide further information to help identify disease progression that may not be obvious on fundus examination alone. Optical coherence tomography (OCT) has already claimed its place as an integral investigation in diagnosis and management of diabetic maculopathy. In addition to simple OCT, OCT-angiography (OCT-A) can be extremely useful in demonstrating retinal blood flow in various layers of the retina. It is non-invasive and does not require intravenous contrast injection which may be contraindicated in patients with severe renal disease. OCT-A can unveil early vascular changes in diabetic maculopathy that may

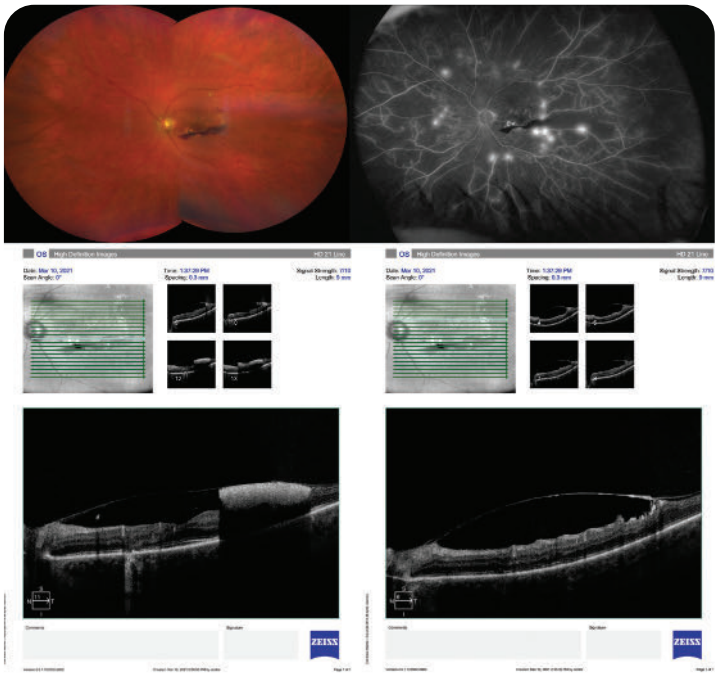

Figure 1.
Ultra-wide field true colour fundus photograph (ZEISS CLARUS 700), fundus fluorescein angiography (FFA - Optos California), and OCT (ZEISS CIRRUS 6000) of a 57-year-old type 2 diabetic patient who presented with sudden onset of a left eye paracentral scotoma. Colour fundus photograph demonstrates pre-macular haemorrhage and peripheral retinal haemorrhages. FFA reveals multiple areas of fluorescein leakage suggestive of neovascularisation as well as peripheral ischaemia, as seen in proliferative diabetic retinopathy. OCT images confirm the location of the haemorrhage at the sub-hyaloid space, as well as vitreoretinal traction.

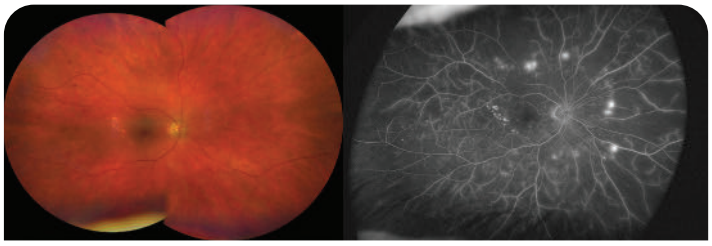

Figure 2.
Ultra-wide field true colour fundus photograph (ZEISS CLARUS 700), and fundus fluorescein angiography (Optos California) of a longstanding type 2 diabetic patient with a history of prior focal macular laser. Though the severity of diabetic retinopathy appears to be non-proliferative on fundus examination, FFA demonstrates extensive peripheral nonperfusion and focal areas of leakage secondary to neovascularisation elsewhere consistent with proliferative diabetic retinopathy. It is important not to be falsely reassured by a benign-appearing retina - always maintain a high index of suspicion if the patient has systemic high-risk features, even though the retinal disease may not appear severe at first glance.

not be evident clinically. Examples of the utility of OCT-A include assessing the size and quality of the foveal avascular zone, detecting microaneurysms or other intraretinal microvascular abnormalities (IRMAs), and assessing neovascularisation at the disc or elsewhere (NVD, NVE).[9]

Alternatively, fundus fluorescein angiography (FFA) can directly capture real-time perfusion and transit time, and remains the gold standard for assessing the severity of diabetic retinopathy. FFA in combination with ultra-wide field imaging allows detection of peripheral areas of nonperfusion, as well as vascular leakage which is not currently examinable on OCT-A. Focal areas of neovascularisation anywhere on the retina can be visualised and can therefore be useful in treatment planning, for example in targeted laser photocoagulation. It is not uncommon for FFA to reveal more advanced retinopathy than what was initially thought, as demonstrated in **Figures 1 to 4**.

With the ongoing advancements of medications and procedures for the treatment of various aspects of diabetic retinopathy, management is becoming increasingly multifaceted and individualised. Early detection and assessment of the severity of diabetic retinopathy is therefore crucial to allow prompt and effective treatment to prevent vision loss. A collaborative approach to managing diabetic eye disease is of utmost importance, and the role of primary care through regular optometrist reviews is fundamental to this process. It is important that the patient is aware of the necessity for screening examinations, and for the health professional to maintain a high index of suspicion particularly when consulting patients with high-risk features.

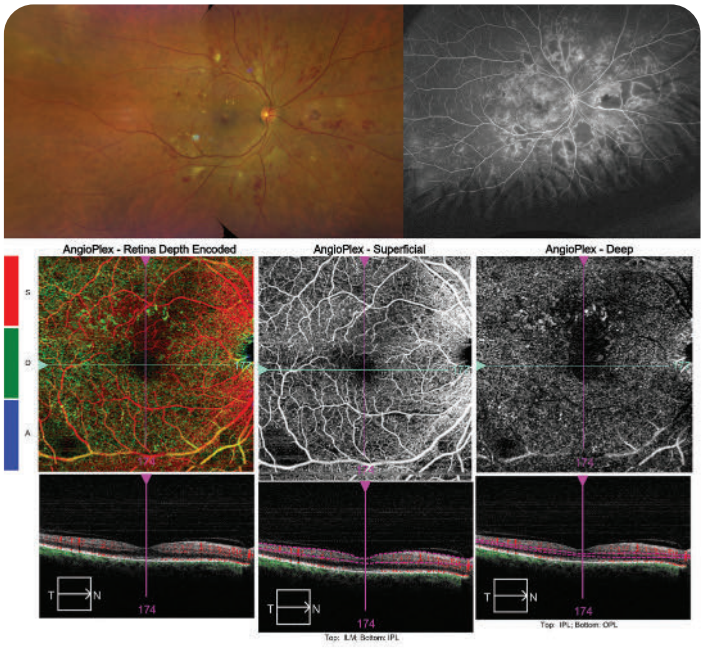

Figure 3.
Ultra-wide field true colour fundus photograph (ZEISS CLARUS 700), fundus fluorescein angiography (Optos California), and 8 x 8 mm OCT-angiography (ZEISS CIRRUS 6000 with AngioPlex) of a 60-year-old type 2 diabetic patient. Colour fundus photograph shows numerous retinal haemorrhages, cotton wool spots and hard exudates. FFA demonstrates extensive areas of peripheral perfusion deficits suggestive of ischaemia. OCT-A of the macula shows an enlarged foveal avascular zone which may not be obvious on fluorescein angiogram.

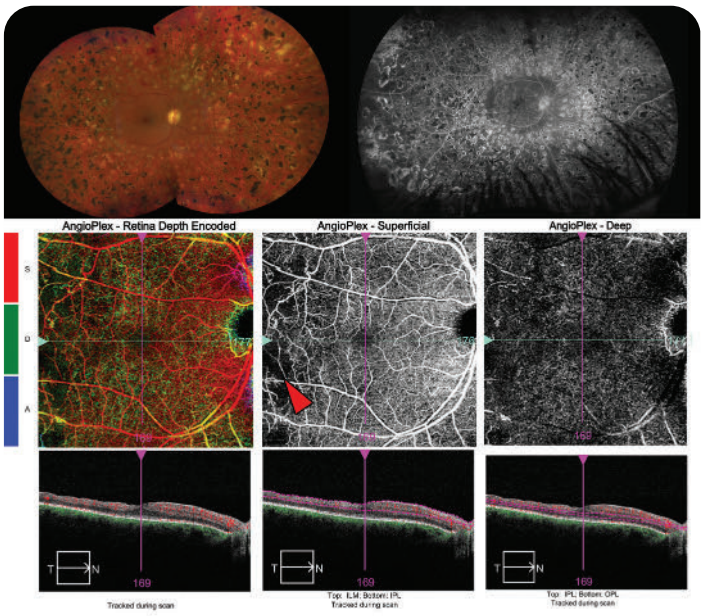

Figure 4.
Ultra-wide field true colour fundus photograph (ZEISS CLARUS 700), fundus fluorescein angiography (Optos California), and 8 x 8 mm OCT-A (ZEISS CIRRUS 6000 with AngioPlex) of a 34-year-old type 1 diabetic patient with prior pan-retinal photocoagulation. FFA demonstrates a crescent of hypoperfusion temporal to the macula; this capillary dropout is further demonstrated on OCT-A (red arrowhead).

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Managing diabetic retinopathy

Assessing the Australian National Health and Medical Research Council's clinical practice guidelines

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Clinical practice guidelines are key to enabling optometrists to provide evidence-based clinical care. For every condition that is diagnosed and managed there are several clinical guidelines, presenting us with the challenge as to which set of guidelines to follow. One of the conditions commonly encountered by optometrists is diabetic retinopathy, with approximately 5.1% of the Australian population having diabetes. Knowing when to monitor and when to refer is key to preventing progression and vision loss. The National Health and Medical Research Council's (NHMRC) clinical practice guidelines on diabetic retinopathy management has been widely used for this purpose since 1997, with them having been updated in 2008. In order for guidelines to be used widely and appropriately, they need to be developed using rigorous strategies and appropriate methodologies. Therefore, a group of authors from the University of New South Wales, led by Mr Rajendra Gyawali, set out to evaluate the quality of these NHMRC guidelines and how they compare to other published guidelines.[1]

Along with the NHMRC guidelines, Gyawali et al. evaluated another five established diabetic retinopathy management international guidelines (Scottish Intercollegiate Guidelines Network, 2017; American Academy of Ophthalmology, 2019; American Optometric Association, 2019; Royal College of Ophthalmologists, UK, 2013, and Canadian Ophthalmologic Society, 2012). To establish the quality of each set of guidelines, they used the Appraisal of Guidelines, Research and Evaluation (AGREE II) instrument. Four independent reviewers were asked to use the AGREE II to score each set of guidelines for scope and purpose, stakeholder involvement, rigour of development, clarity of presentation, applicability, and editorial independence.

The NHMRC guidelines scored 5.3/7, placing it second lowest of all the guidelines evaluated. Although, on the whole, it was comparable to existing guidelines in all areas bar one: editorial independence. Its strength lay in clarity of presentation and rigour of development. Moreover, the NHMRC guidelines were the oldest of the guidelines, with an update recommended by all reviewers. In comparison, two of the other guidelines were revised in 2019. The highest rating belonged to the Scottish Intercollegiate Guidelines Network guidelines, which was also one of the newer guidelines, updated in 2017.

In summary, the authors identified areas of strength of the existing NHMRC guidelines, but also identified that it is well in need of revisions to improve the transparency in its development and applicability to clinical practice. Additional recommendations made by the authors include clearly identifying the target population, including the excluded population; defining the target users of the guidelines; including information on the methods used to formulate the recommendations; inclusion of guidance on how to implement the recommendations; and provision of explicit disclosure of funding support and competing interests of the guideline development group. Given that this set of guidelines is now more than 10 years old, an update is well overdue.

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Anti-aging & eye care

Curious about skincare and cosmeceuticals?

Perhaps out of personal curiosity more than patient concern I recently went down the rabbit hole of learning about cosmetics and cosmeceuticals, and their implications on the ocular adnexa. Cosmetics generally refer to any products which alter appearances without affecting body function or structure, however cosmeceuticals also have therapeutic effects on the skin. Anti-aging eye care extends beyond prescribing wrap-around sunglasses for our patients.

The aging eye

We commonly find and discuss with patients the age-related changes that occur in the eye, such as cataracts and macular degeneration. Rarely we may find an eyelid malignancy or see patients with solar retinopathy. All of these conditions, and more, can be caused by the UV radiation damage that develops with advancing age.[1] We discuss with our patients indicated management options and preventative care. After all that is why they come to see us - to preserve their eyesight, but this conversation can be extended to the care of the skin around their eyes and implications of current skincare regimes.

In general, UV radiation causes aging of the skin in the forms of atrophy of the epidermis, pigmentary degeneration and formation of rhytides (also known as wrinkles).[2] Our patients may complain of dark circles and eyelid wrinkles or bags under their eyes.[2] They also may experience dry or thin skin around the eyes.[2] These signs can be caused by genetic predispositions as well as a range of UV and age-related changes in the skin, from reduction in production of collagen to reduction of orbital fat and other anatomical changes.[2-3] The skin around the eyes is particularly vulnerable to UV radiation and is the earliest facial region to exhibit aging changes, as it is the thinnest skin in the body - approximately 0.2 mm in thickness in some cases.[2-3] Caution should be taken in choosing skincare products to use around the eyes, eyelids and periorbital skin for this reason. Any product, if used too close to the eyes, can seep into the eyes and disrupt the tear film which in turn causes irritation or dry eyes.[4] As with all new products, general advice to patients should be to always test the product on a small area before widely using it on the face.[5]

Anti-aging eye care extends beyond prescribing wrap-around sunglasses for our patients

Prevention

The ultimate first line of defence in anti-aging skincare and eye care is prevention.[2] Pilkington et al. report "UV exposure exponentially accelerates the photoaging process".[2] The advice from Cancer Council Australia is to minimise sun exposure and use protective factors when the UV index is 3 or above.[6] We're familiar with Category 2 or 3 rated sunglasses, with a wrap-around lens fit, and eye-sun protection factor (E-SPF) available in optical lenses.[2,6] However broad-brimmed hats should also be recommended as they alone reduce the UV exposure to the eye by 50%.[6]

Sunscreen prevents UV damage, however it can be difficult to find a sunscreen suitable for use on the sensitive skin around the eyes. Physical blocking sunscreens, rather than chemical sunscreens, are better suited to the skin around the eyes.[2] For optimal protection, sunscreens should have a SPF of at least 30, be broad spectrum - which protects from UVA and UVB radiations - and be water-resistant. The Cancer Council advises to use an appropriate amount (1 teaspoon for the face) and to re-apply regularly.[7] They also recommend using a specific sunscreen rather than make-up with SPF for adequate protection.[7] In March 2021 I was interviewed on this topic on Adore Beauty's Beauty IQ Podcast[8] and although there are many physical blocking sunscreens available, a few of their recommendations safe for use around the eyes are:

- → Ultra Violette Lean Screen SPF50+ (https://www.adorebeauty.com.au/ultra-violette/ultra-violette-lean-screen-spf50-sunscreen.html)
- → La Roche Posay Anthelios XL Anti-shine Dry Touch Facial Sunscreen SPF50+ (https://www.adorebeauty.com.au/la-roche-posay/la-roche-posay-anthelios-dry-touch-untinted-sunscreen-spf50.html)

A few other pharmacy available options I found are:

- → Cancer Council Face Daywear Zinc Moisturiser SPF50 (https://www.cancercouncilshop.org.au/collections/sunscreen/products/face-daywear-zinc-moisturiser-spf50)
- → EGO SunSense Sensitive Invisible SPF50+ (https://www.priceline.com.au/ego-sunsense-sensitive-invisible-spf50-200-g) →

Anti-aging skincare

When it comes to treatment of already UV damaged skin, there is a wide range of skincare options available. Most skincare products have active ingredients that come under the following categories: Antioxidants, Peptides, Growth Acids, Ceramides and Retinoids.[2]

Antioxidants act by neutralising or preventing oxidative damage to the skin which occurs after exposure to UV light, cigarette smoke and pollutants.[2-3] Damage in the skin occurs to the DNA, cell membranes, proteins and collagen.[2-3] Antioxidants in skincare include Vitamin C (Ascorbic Acid), E (Tocopherol), B3 (Niacinamide) and green tea.[2-3] Vitamin C formulations are well researched and tolerated; studies show that it protects skin from UVA and UVB damage as well as improves fine wrinkles, skin laxity and other features.[2,9] Vitamin E has been shown to improve periorbital wrinkling and reduce UVB induced damage.[2] When used in conjunction with Vitamin C, it has also been shown to have a four-fold improvement in protecting skin from redness and sunburn.[2,10] Further research including ferulic acid in the formulation provides stability of the vitamin formulations and doubles the additional protection.[2,10] Vitamin B3 or Niacinamide also has good documentation in its ability to be safely used topically.[2] In addition to antioxidant properties it reduces periorbital hyperpigmentation, and improves skin texture and wrinkles by increasing collagen production.[2] It also improves the skin barrier which in turn reduces transepidermal water loss.[2] Green tea has photoprotective benefits and improves collagen synthesis, but current research suggests its stability and delivery through epidermis is challenging.[2]

Peptides and growth factors in certain formulations have also clinically been shown to improve the appearance of periocular wrinkles, however these are still evolving technologies.[2] Peptides are chains of amino acid sequences, and in some formulations have been found to improve the appearance of facial wrinkles.[2] Growth factors and cytokines have been researched in wound healing but are now being studied in anti-aging.[2] Again, it has been found that growth factors improve periorbital wrinkle appearances however effective skin penetration is still being researched.[2] Backed by research, a potential alternative to human growth factors is the use of snail secretions on periocular wrinkles.[2]

Typical acids used in skincare are Alpha Hydroxy Acids (AHAs) and Hyaluronic Acid (HA). AHAs are exfoliants and include glycolic, lactic, mandelic and benzilic acids.[2] In low concentrations they are particularly useful in treating periorbital hyperpigmentation.[11] Glycolic and lactic acids in particular also improve the synthesis of collagen and elastic fibres in the skin.[2] HA is naturally occurring in the body and is a major component of the skin matrix. It can bind 1,000 times its volume in water and has been shown to maintain tissue elasticity and hydration. However, in cosmeceutical use, HA is only effective at penetrating the dermis at very small molecule sizes.[2]

Ceramides are a component of the skin. Their unique properties act as a biosensor for the skin's barrier.[2] Formulations of ceramides improve the skin barrier function and hydration.[2]

Retinoids, which are synthetic derivatives of Vitamin A, have long been researched and in general improve hyperpigmentation, fine lines and skin texture. However, this varies with preparations.[2-3] When tested on the periorbital skin, they have significant skin reaction potential and can cause conjunctival irritation and corneal toxicity.[2] Retinoic acid has been shown to induce atrophy of the meibomian glands and in turn cause dry eye disease.[4] Based on this, it is recommended to minimise use of retinoids directly around the eye area, however if indicated, formulations with retinaldehyde are most effective with the least potential for irritation.[2]

Patients may not be aware of their skincare's ingredients so websites such as the Environmental Working Group (EWG)'s Skin Deep (https://www.ewg.org/skindeep/) or International Nomenclature of Cosmetic Ingredients (INCI) Decoder (https://incidecoder.com/) can be used to determine if products contain beneficial or cautioned constituents. A few options for products you can recommend with clinically researched skincare ingredients and formulas safe for use around the eye are:

- → Skinceuticals CE Ferulic - Containing Vitamin C, E and Ferulic Acid (https://www.skinceuticals.com.au/vitamin-c-serums/c-e-ferulic/p2755.aspx?gclid=CjwKCAjw9MuCBhBUEiwAbDZ-7q4K92NMTVp9PwrTky6o_VNhr6Yr34jBn1mE3bCLk-7UmwreeYF4TRoCW18QAvD_BwE)
- → Paulas Choice 10% Niacinamide Booster - Containing Vitamin B3 (Niacinamide) (https://www.paulaschoice.com.au/10pct-niacinamide-booster/798.html)
- → Biopelle Tensage Growth Factor - Containing snail derived growth factors (https://biopelle.com/tensage-growth-factor/)
- → Alpha H Liquid Gold - Containing 5% Glycolic Acid (AHA) (https://alpha-h.com/products/liquid-gold-exfoliating-treatment-with-glycolic-acid)
- → NIOD Multi-Molecular Hyaluronic Complex - Containing multiple molecular weight HA (https://niod.deciem.com/au/multi-molecular-hyaluronic-complex-15ml.html)
- → CeraVe Moisturising Lotion - Contains a combination of 3 Ceramides (https://www.cerave.com.au/our-products/moisturisers/facial-moisturising-lotion)

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Eating for eye health

Focus on AMD

Nutrition is an essential component of maintaining a healthy lifestyle. Over the past century evolution towards Western dietary habits, characterised by high amounts of energy-dense, processed foods and high omega-6 to omega-3 ratio, has contributed to the growing burden on healthcare.[1] Despite the well-recognised role that diet plays in the development of chronic disease, healthcare professionals are often not adequately supported to provide high-quality nutrition recommendations in their practice.[2] Application of evidence-based nutrition care is vital for supporting patients in managing certain chronic conditions where poor nutrition is a risk factor.

Optometrists are ideally placed to offer appropriate nutritional advice for managing ocular disease. As most patients highly regard the recommendations provided by their healthcare providers in relation to nutrition, there is opportunity to integrate appropriate dietary recommendations in routine optometric practice. One important ocular condition for which diet is a modifiable risk factor is age-related macular degeneration (AMD), which is the leading cause of irreversible vision impairment in developed countries.[3] The provision of appropriate nutritional care to those with AMD, or who are at risk of AMD, could improve long-term clinical outcomes. This article will provide a summary on the role of diet and nutrition in AMD.

Dietary patterns

Several epidemiology studies have investigated the association between diet and AMD. Diets dominated by foods with high glycaemic index, high consumption of red meat, vegetable oils and animal fats high in omega-6 fatty acids (FAs), and low dairy and calcium, have been associated with an increased risk of AMD progression.[4]

Specific dietary patterns have been evaluated in relation to their potential benefits in AMD. The Mediterranean diet is characterised by a high consumption of fruits and vegetables, legumes, whole grains and nuts, and fish; a low consumption of dairy and red meat; and a moderate consumption of red wine. Olive oil is used as the main source of fats. In ~5,000 people with no or early AMD, adherence to the Mediterranean diet was found to be associated with a 41% reduced risk of developing late AMD.[5]

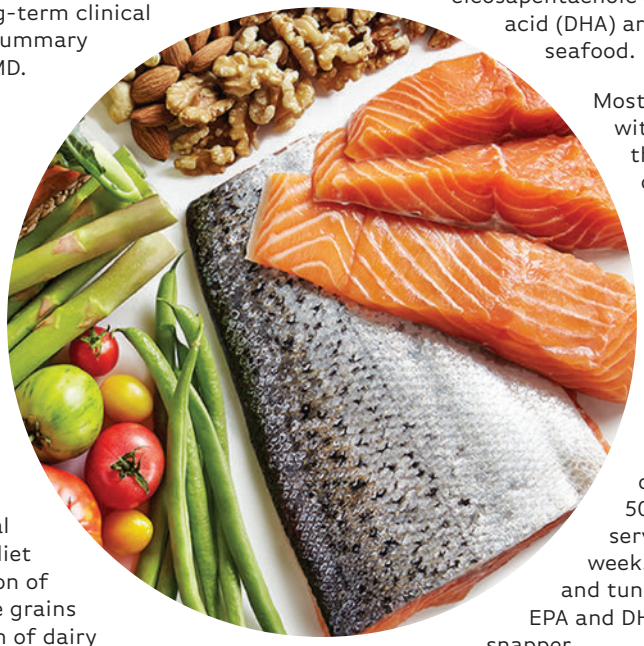
Oriental dietary patterns have higher intakes of vegetables and fruits, legumes, whole grains, and seafood. The Western diet is characterised by a high consumption of processed meat, high-fat dairy products, refined grains and sugars, and a relatively reduced consumption of fruits and vegetables. Implementation of the Oriental dietary pattern is associated with reduced odds for both early and late AMD; closer adherence to the Oriental pattern is thought to gain larger benefit.[6] In contrast, adherence to a Western dietary pattern demonstrated increased odds for early and late AMD. [4,6]

Nutritional components

Several key nutritional components have been evaluated for their association with AMD progression, including omega-3 FAs and macular carotenoids, such as lutein and zeaxanthin.

Omega-3 FAs

Omega-3 polyunsaturated FAs are essential FAs that can only be derived via food or supplementation. There are two main forms of omega-3 FAs. Short-chain omega-3 FA, alpha linolenic acid (ALA), is present in flaxseed and chia seeds, walnuts, and plant oils from canola and soybean. Long-chain omega-3 FAs, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) are predominantly found in fish and seafood.



Most of the health benefits reported with omega-3 FAs are associated with the long-chain forms. The efficiency of conversion from short-chain to long-chain omega-3 FAs is low (5-20%) and varies in different body tissues. It is not clear whether consumption of short-chain ALA is adequate for obtaining systemic long-chain omega-3 FAs levels. Therefore, the best method for obtaining adequate systemic omega-3 levels is through diet. The recommended daily long-chain omega-3 FA intake is approximately 500 mg/day, which equates to two servings (100-150 g) of fatty fish per week. Oily fish, such as salmon, mackerel and tuna, contain higher concentrations of EPA and DHA than lean fish, such as cod and snapper.

The other main polyunsaturated FAs in the body are omega-6 FAs. Long-chain omega-6 FA, arachidonic acid (AA), is found in

Image: Examples of foods that have been associated with benefits in age-related macular degeneration. Image source: www.aarp.org.

most animal products, including meat, eggs and dairy products. Omega-6 FAs are the predominant type of polyunsaturated FA found in Western diets.

In the body, long-chain omega-3 FAs bias the production of anti-inflammatory and anti-thrombotic cytokines, whereas long-chain omega-6 FAs produce mediators with pro-inflammatory and pro-thrombotic effects. The balance of omega-3 and omega-6 FAs plays a key role in modulating systemic inflammation and immunity. Lipid mediators of long-chain omega-3 FA, DHA, can also impart neuroprotection.

Omega-3 rich foods and AMD

Epidemiology studies have associated dietary long-chain omega-3 intake with a lower risk of developing early-stage AMD[7] and progression to late-stage, sight-threatening forms of disease.[8] Fish intake at least twice a week is associated with a reduced risk of both early AMD and late AMD.[9]

Omega-3 oral supplements and AMD

In contrast to the evidence on food-based omega-3 intake, dietary supplementation with omega-3 has not been shown to provide benefit in AMD prevention or slowing disease progression.[10] The reason for this difference may relate to the potential synergistic interactions of FAs with other nutrients and vitamins that are present in whole foods but not supplements.[5] It's important that this subtle, but important, distinction is recognised in making clinical recommendations relating to omega-3 FAs for AMD.

Lutein and zeaxanthin

There are over 700 vitamin carotenoids, classified as either carotenes and xanthophylls.[11] Lutein and zeaxanthin are xanthophylls that are present in high concentrations in the macula. These nutrients cannot be synthesised by the body and must be derived from the diet. Food sources of lutein and zeaxanthin include fruit (especially those yellow-orange in colour, such as oranges and peaches), green vegetables (e.g., spinach, kale, and broccoli), egg yolk, and corn.[12]

Higher dietary intakes of lutein/zeaxanthin is associated with a reduced risk of late, but not early or intermediate, AMD.[13] The average intake of lutein and zeaxanthin is approximately 1-3 mg/day, while a minimum of 6 mg/day is considered to provide benefit in AMD.[14] This is equivalent to approximately 100 grams of spinach, 300 g of broccoli, or 300 g of corn. As long-term use of ß-carotene, retinol, and lutein supplements (individually and without other vitamins) have been associated with an increased risk of lung cancer,[15] increasing consumption via food sources, rather than supplementation, is recommended to minimise potential adverse effects, particularly among smokers.

Oral supplementation with AREDS-based formulations

AREDS and AREDS2 are two large randomised clinical trials that evaluated the efficacy of specific types of nutritional supplementation in AMD. The AREDS trial found that supplementation with vitamins C and E, zinc, copper and ß-carotene reduced risk of progression from intermediate to late AMD by approximately 25% (**Table 1**). Nutritional supplementation is recommended for those with at least intermediate AMD, defined as medium sized drusen (63-125 µm) with pigmentary changes, or drusen > 125 µm.[16] In people with no ageing changes or early AMD, the AREDS study found a low risk of developing late AMD, such that oral supplementation treatment targeting progression is not indicated.[17] →

	AREDS formula*	
Vitamin C	500 mg	500 mg
Vitamin E	400 IU	400 IU
Zinc†	80 mg	80 mg
Copper (cupric oxide)‡	2 mg	2 mg
ß-carotene	15 mg	-
Lutein	-	10 mg
Zeaxanthin	-	2 mg

Table 1.
Nutrients in commercially available formulas based on AREDS/AREDS2

*Not recommended for current or former smokers

†Above the upper level of intake (40 mg/day) recommended by the National Health and Medical Research Council

‡Added to avoid zinc-related copper deficiency

Units: mg = milligrams; IU = international units

In AREDS2, the investigators found no overall additional benefit or harmful effects from adding omega-3 FAs (350 mg/day DHA, 650 mg/day EPA) and/or lutein (10 mg/day) and zeaxanthin (2 mg/day) to the original AREDS formula in AMD progression.[18] However, given the safety concerns in current and former smokers for the potential increase in incident risk of lung cancer, lutein/zeaxanthin may be a safer carotenoid substitute to ß-carotene in AREDS-type supplements.

Summary (Table 2):

- **Epidemiologic studies have found a reduced risk of AMD with high dietary consumption of foods rich in antioxidants (lutein and zeaxanthin) and omega-3 polyunsaturated FAs. However, dietary supplementation with omega-3 FAs have been shown not to provide the same benefit as whole foods for either AMD prevention or slowing disease progression.**
- **Adherence to certain dietary patterns, such as the Mediterranean and Oriental diets, can reduce the risk of AMD progression.**
- **Although there is still little understanding on the effects of diet and nutrition on the initial development of changes associated with AMD, health-care professionals can still proactively promote healthy food choices to those at risk of AMD.**

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Dietary factor	Key points
Fruits and vegetables	• Vegetables: > 200 g per day; fruits 2 large pieces (~200 g) per day.[9] • ↑ Green leafy vegetables and yellow-orange coloured fruits containing lutein and zeaxanthin, e.g., kale, spinach, watercress, basil, peas, lettuce, zucchini, broccoli, tomatoes, corn, brussels sprouts, spring onions, pumpkin and leeks.[4] • Other foods containing lutein and zeaxanthin, e.g., egg yolk.
Polyunsaturated FAs	• Fish two servings per week (each serving 100-150 g), or approximately 32 g per day.[9] • ↑ Oily fish containing omega-3 FAs, e.g., salmon, anchovy, mackerel, tuna, sardines, and swordfish. • ↓ Vegetable oils and animal fats containing high omega-6 FA concentrations.
Other fats	• Avoid trans-fats. • Use olive oil instead of butter.
Carbohydrates	• ↑ Low GI foods (slow-release carbohydrates). • ↓ High GI foods (highly refined carbohydrates).
Meats	• ↓ Red meat consumption. • ↓ Salami or continental sausage; limit to once per month. • Consumption of non-fried chicken is preferable to red meat.
Calcium	• High calcium dietary intake is preferable to low calcium dietary intake. • Caution is required with increasing dairy intake, as dairy products have both pro-inflammatory and anti-inflammatory effects • Other non-dairy based sources of dietary calcium: kale, bok choy, collard green, broccoli, okra, sesame and chia seeds, fresh and canned fish, beans and soya bean products.[4]
Alcohol	• Limit to less than two standard drinks per day.[4]
Oral supplements	• Supplementation with AREDS-based formulations, containing vitamins C and E, zinc, copper, and either ß-carotene (AREDS) or lutein + zeaxanthin (AREDS2 formula), in individuals with intermediate AMD* or worse • AREDS2 formula is recommended for individuals who currently smoke, or have previously smoked • Omega-3 supplementation, either by itself or in addition to AREDS-based formulations, has not demonstrated benefits for reducing AMD progression

Table 2.
Summary of dietary recommendations in AMD

↑ Increase intake.

↓ Reduce intake.

AREDS = Age-related eye disease study.

*Using the Beckman Classification,[19] intermediate AMD is defined as the presence of medium sized drusen (63-125 µm) with pigmentary changes, or drusen > 125 µm.

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WEBINAR FIVE

MONDAY 18 OCTOBER 2021 6.30 pm – 8.00 pm (AEDT)

MONITORING AND (CO-)MANAGING GLAUCOMA

This webinar will discuss glaucoma medication in collaborative care and management of raised IOP after retinal injections. Features fellowship-trained glaucoma specialist and surgeon Dr Jason Cheng, ophthalmic surgeon with sub-specialist training in glaucoma surgery Dr Justin Sherwin, and ophthalmologist with interests in retinal conditions and glaucoma Dr Aaron Yeung. Moderated by A/Prof Tim Roberts.

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