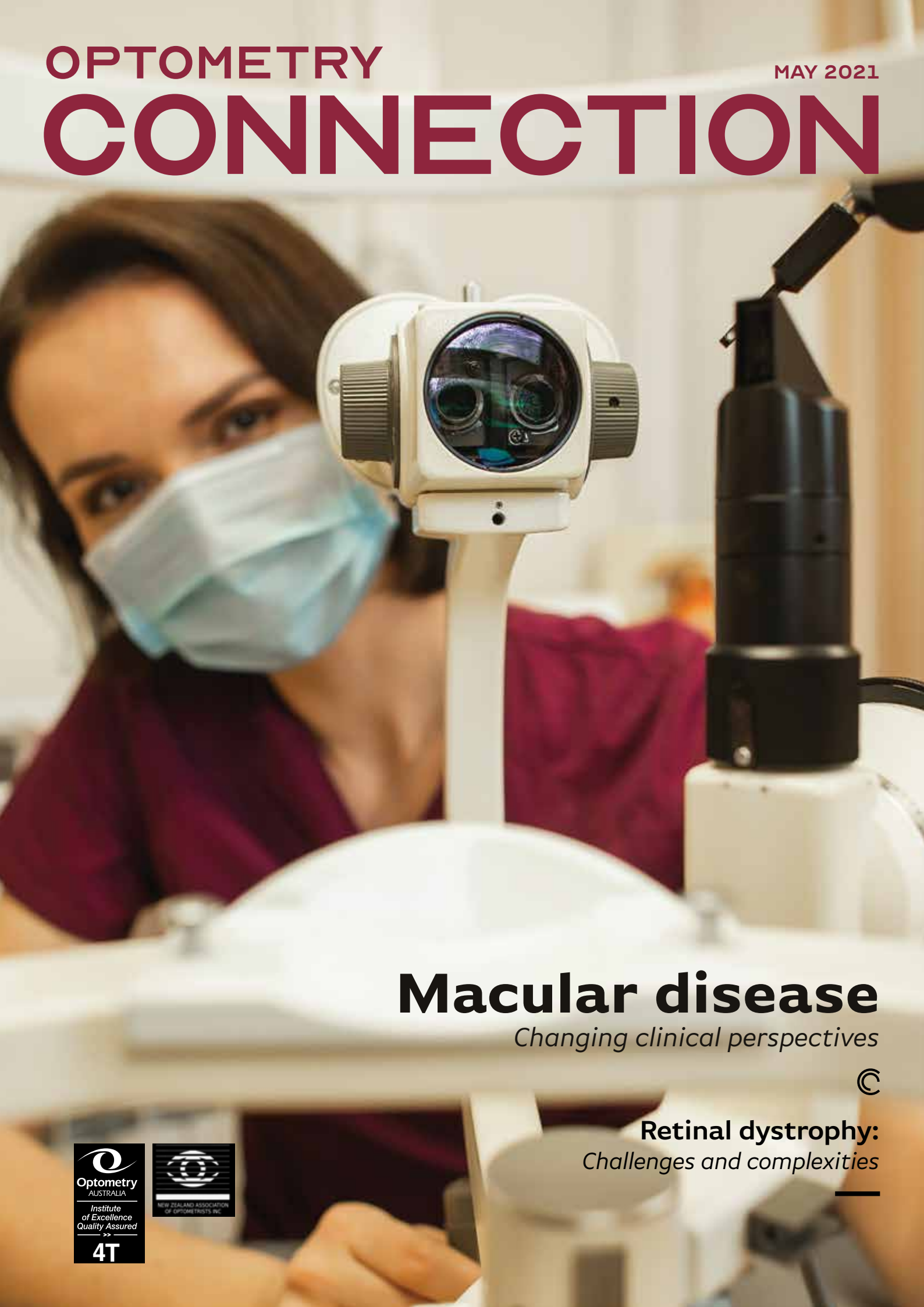
OPTOMETRY
CONNECTION
MAY 2021



Macular disease

Changing clinical perspectives

©

Retinal dystrophy:
Challenges and complexities

GENE THERAPY CAN ADDRESS THE ROOT CAUSE OF MONOGENIC DISEASES

Novartis is at the forefront of gene therapy, an exciting new branch of medicine.

Monogenic diseases are inherited conditions caused by a deletion or mutation in a single gene. Gene therapy delivers a new or working copy of this gene into human cells, making it possible to treat the root cause of the disease and halt their progression.

With the first two of these pioneering therapies now available in Australia, together we have the opportunity to transform the lives of patients.

Discover more at
www.medhub.com.au

NOVARTIS

AU-15620. March 2021

What's in this issue

Editor
Jeff Megahan

Clinical Editor
Kerryn Hart
BOptom GCertOcTher MPH PGDipAdvClinOptom GCertHE FACO

Education Editor
Simon Hanna
BOptom PGDipAdvClinOptom GCertOcTher GCUT

Publications & Digital Manager
Jessica Donald

National Professional Services Advisor
Sophie Koh
BOptom GCertOcTher LmusTCL

Partnership Director
Mark Cushway

Brand Custodian & Multimedia Designer
Lachlan Hessing

National Professional Development Advisor
Andrew Kotsos
BOptom PGCertOcTher

Optometry Australia ABN 17 004 622 431
Suite 101,
68-72 York Street
South Melbourne VIC 3205
Ph 03 9668 8500
www.optometry.org.au

Optometry Connection is distributed in Australia and New Zealand.

Cover: 'Patient perspectives'
The early detection of AMD provides an opportunity to avoid or delay the burden of vision loss on patients and their families. The earlier that AMD is detected, the earlier steps can be taken to help slow its progression.

doctors health fund

Avant mutual

We were made for you

Supporting you, your health and your profession

VALUE. Top Cover Gold is the only hospital cover that pays a medical gap benefit up to the AMA List of Medical Services and Fees, giving you top-level protection against out-of-pocket costs.

CHOICE. Because you know who is best to treat you, we have no restricted provider networks for your extras cover.

SUPPORT. One of Australia's leading funds in providing financial support to members during COVID-19.

SERVICE. Exceptional member service with a Member Satisfaction Rating above 94% for over 10 years.*

Dr Arany Nerminathan,
Doctors' Health Fund Member since 2018

It takes just 5 minutes to switch

1800 226 126

doctorshealthfund.com.au

* Doctors' Health Fund Member Satisfaction Surveys 2010-2020

DHF 306_4/21

Supporting your patients this 'Macula Month'

A note from Dee Hopkins,
Chief Executive Officer, Macular Disease Foundation Australia (MDFA)

I am excited to welcome you to the May edition of *Optometry Connection*, which shines a spotlight on macular disease. May is 'Macula Month,' MDFA's annual campaign drive to encourage people to book a comprehensive eye examination – including a check of the macula – with their local optometrist.

Since 2004, these national awareness campaigns have been instrumental in increasing Australians' awareness of macular conditions, how to detect early symptoms and the importance of regular eye examinations.

This year, Macula Month will showcase 'Check My Macula' – a new online tool encouraging over-50s to see their optometrist for an eye examination. In less than a minute, www.CheckMyMacula.com.au reveals the respondent's individual risk factors, enables them to find their nearest optometrist and book a potentially sight-saving check-up with an optometrist via a back-end connection to existing optometrists' booking systems.

Macula Month relies on the support of optometrists and other health-care practitioners to spread these key messages to the wider community. As always, MDFA will provide practical communications toolkits to help optometrists get the word out. In particular, we can provide in-store materials with a QR code so that your patients can click and do a quiz while they are waiting for their appointment.

Through our National Helpline and other services, MDFA can support your patients, their families and carers from early diagnosis and throughout their journey with macular disease. MDFA gives your patients the right information at the right time, and I urge optometrists to connect their patients with MDFA as the first port of call for expert, independent and free advice and support.

In one of our most exciting projects, MDFA is launching free CPD courses for optometrists this June through funding by the Australian Government as one of the initiatives in the National Strategic Action Plan for Macular Disease. Optometry Australia quality-assured courses on age-related macular degeneration and diabetic eye disease will be available on Optometry Australia's Institute of Excellence from next month.

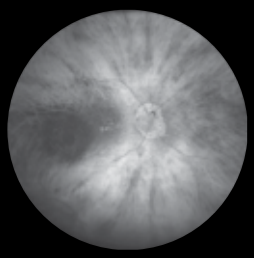
By working collaboratively, optometrists and MDFA can provide a quality, integrated care-team solution to every member of the macular disease community. I look forward to our continued engagement with Optometry Australia and eye-health practitioners across the country. Together, we can make a positive impact on the life of every Australian with a macular condition.

Download your Macula Month toolkit from our website: www.mdfoundation.com.au.

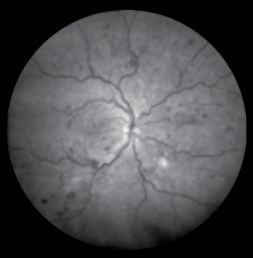
Dee Hopkins
Chief Executive Officer,
Macular Disease Foundation Australia

Imaging ultra-wide without compromise.

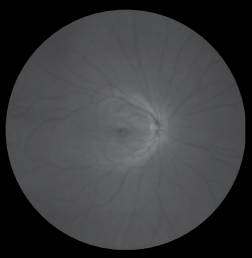
ZEISS HD Ultra-widefield



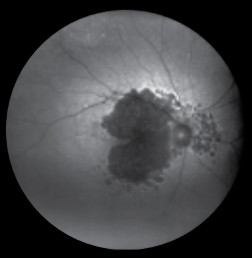
Choroidal Nevus
Red channel separation



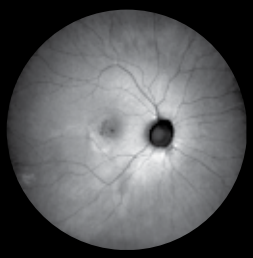
Proliferative Diabetic Retinopathy
Green channel separation



Retinal Nerve Fibre Layer
Blue channel separation



Geographic Atrophy
FAF-Blue



Dry Age-related Macular Degeneration
FAF-Green

ZEISS CLARUS 500

Colour. Clarity. Comfort.

Compromising image quality may leave some pathology unseen. Introducing CLARUS 500, a next generation fundus imaging system from ZEISS that provides true colour and high resolution in a 200 degree ultra-widefield image.

www.zeiss.com/CLARUS

ZEISS

ZEISS
Ph: 1300 365 470
med.au@zeiss.com

Reticular pseudodrusen in AMD

What are they? And why should we care about them?

FEATURE ARTICLE

Dr Zhichao Wu
BAppSc(Optom) PhD FAAO

Dr Carla Abbott
BOptom PhD PGDipOcTher FACO

Professor Robyn Guymer AM
MBBS PhD FRANZCO FAHMS

Centre for Eye Research Australia, Royal Victorian Eye and Ear Hospital
Ophthalmology, Department of Surgery, The University of Melbourne

Age-related macular degeneration (AMD) remains a leading cause of irreversible vision loss, and one in seven Australians have the early signs of AMD.[1] These individuals are at risk of developing vision-threatening, late complications including choroidal neovascularisation (CNV) or geographic atrophy (GA). There are currently no specific interventions that can effectively slow or prevent the development of late AMD in those with the early signs of AMD, apart from general lifestyle advice[2] or nutritional supplements.[3,4] For those who develop GA, there are also no treatments available to slow or prevent the progressive enlargement of atrophy.

Fortunately, treatments are now available for CNV based on intravitreal injections of antiangiogenic agents, but the outcome for each individual is highly dependent on visual acuity at presentation for treatment.[5,6] Early detection of CNV before vision is affected is thus a key part of the management of those with the early stages of AMD by optometrists.[7,8] Higher rates of early detection could be achieved through more frequent clinic monitoring (in addition to a patient's vigilant self-monitoring of vision). Identifying those potentially at a higher risk of progression that require such increased clinic-based monitoring frequency is crucial.

With great advances in our ability to routinely image the retina with exquisite detail, we can now start to identify new features that are associated with a higher risk of disease progression. In August 2020, we presented to the Early Career Optometrists of Victoria and South Australia (ECOVSA) ways of using optical coherence tomography (OCT) imaging to identify potential risk factors for AMD progression. In that presentation, we also discussed how to use OCT imaging to detect potential signs of late AMD, including retinal fluid, and the implications on the management of patients with AMD. In this article, we focus on one of the features seen in the early stages of AMD that optometrists should become familiar with – reticular pseudodrusen.

Reticular pseudodrusen – what are they?

Three decades ago, Mimoun et al.[9] described a peculiar pattern of drusen-like deposits associated with AMD that are characterised by having a faint network of broad interlacing ribbons (forming a 'reticular' pattern), which were seen more clearly with blue light (*les pseudo-drusen visibles en lumière bleue*). This pattern of deposits has thus been termed 'reticular pseudodrusen.' With the advent of modern OCT imaging, it was revealed that this peculiar pattern represented deposits that were localised to the subretinal space (above the retinal pigment epithelium (RPE), rather than below the RPE, where conventional drusen are typically located; Figure 1).[10] Recent histological studies have also revealed that their composition is distinct from conventional drusen;[11,12] reticular pseudodrusen are rich in vitronectin, proteins and immune cells, compared to the lipids predominantly associated with conventional drusen.

These subretinal drusenoid deposits (SDD) have since been observed to be present not simply with eyes with the peculiar faint-interlacing pattern of drusen-like deposits (described as 'reticular') but that they also corresponded with more discrete dots with a pale-yellow appearance (that can resemble small, hard drusen; Figure 2). Nonetheless, these deposits are clinically referred to as 'reticular pseudodrusen' (RPD) or SDD. However, we and others have shown that RPD are missed on examination of colour fundus photographs by experienced graders between approximately 60 per cent[13] to 80 per cent[14] of the time. They are thus present more frequently than might be expected. Our study (and others) utilising multimodal imaging demonstrated that RPD are present in approximately 25-30 per cent[13,14] of individuals with intermediate AMD.

Membership means you are never alone

As the profession's peak body, no other organisation understands optometry like us, or has a more influential voice. Since 1918, we have united, led, engaged and promoted optometry, optometrists and community eye health and vision care.

Representing the largest community of optometrists in Australia, we put our members first in all we do.

As a member we can provide you with the networks, education, support and guidance to help you succeed.

Membership gives you access to our Optometry Advisors for personalised one-on-one advice as well as access to our human resources and legal services. You can tap into our extensive range of clinical and practice guidelines and our vast array of resources created with you in mind.

You will also be provided you with an extensive range of CPD tools and systems so you can concentrate on the things that matter.

We've developed your digital learning plan and we automatically record your CPD hours when completing any Optometry Australia Institute of Excellence quality assured course. You can participate in our complimentary and growing range of webcasts, podcasts and our annual national virtual conference, while also receiving our publications and more.

And as a member, we give you peace of mind with automatic best-in-market professional indemnity insurance.

Our range of member services, along with the extensive work that we do to promote and progress the profession, to evolve optometrists' scope of practice and to secure optometry's future, is extensive.

If you are not already a member, we would welcome your contact and if you are a member, your membership renewal.

For more information, and to explore joining and renewal options, visit optometry.org.au, email us on national@optometry.org.au, call 03 9668 8500 or the state division where you are based.

Reticular pseudodrusen – why are they important?

What is the significance of this often hard-to-detect feature? Here are three key reasons:

1. Risk factor for progression

We (and others) have previously shown that in those with unilateral CNV, the presence of RPD on multimodal imaging (including OCT imaging) was associated with an increased risk of progression to late AMD independent of the conventional features of large drusen and pigmentary abnormalities in the eye without CNV.[15,16] A meta-analysis of several other studies have since confirmed these findings,[17] underscoring the importance of RPD as a risk factor in those with unilateral CNV.

For those with only early signs of AMD, a recent, large study (n = 646) also showed that RPD detected on fundus autofluorescence (FAF; an imaging modality that outperforms colour fundus photography for detecting RPD[13]) was associated with an increased risk of disease progression.[14] However, other studies, including our own (unpublished) have not observed RPD as detected on OCT imaging to be associated with an increased risk of progression to late AMD in those with the early stages of AMD –although these studies include fewer participants than the abovementioned study.[18,19] Further data is therefore needed before evidence-based guidance can be provided in terms of how to best counsel patients with AMD and RPD and if more frequent review is beneficial.

Currently, the RANZCO referral pathway[7] and Optometry Australia's Chairside Reference for AMD management[8] recommend optometry review every six to 12 months for those with the earlier stages of AMD, depending on risk modifiers such as presence of RPD.

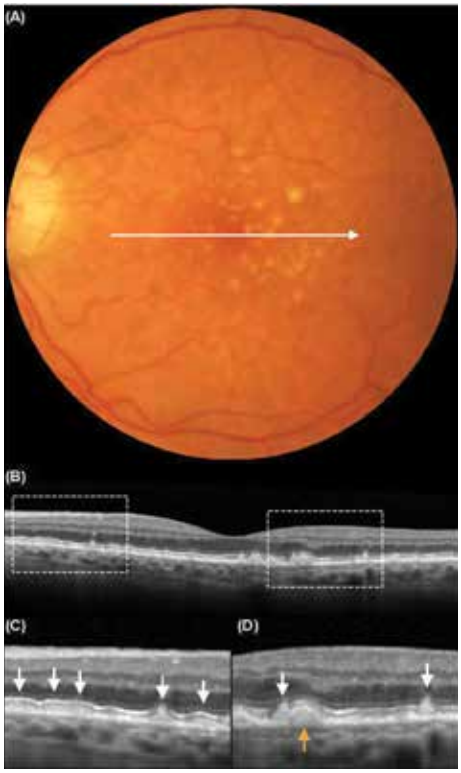


Figure 1.
A: Example of a left eye with large drusen and reticular pseudodrusen (RPD) as seen on a colour fundus photograph; note the faint network of broad interlacing ribbons of drusen-like deposits that represent RPD in the superior arcade (hard to discern). B: An optical coherence tomography (OCT) B-scan was taken through the fovea (indicated by the white horizontal arrow), and it reveals the presence of RPD above the retinal pigment epithelium (RPE); C-D: white vertical arrows on the magnified inserts, corresponding to the white dashed rectangles in B that were distinct from conventional drusen below the RPE (orange vertical arrow).

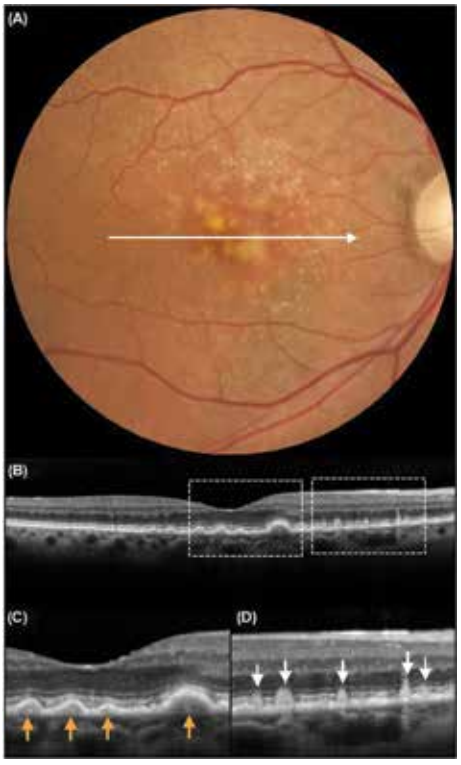


Figure 2.
A: Example of a right eye with large drusen and reticular pseudodrusen (RPD) as seen on a colour fundus photograph; note the presence of pale-yellow, discrete deposits that represent RPD (but could be mistaken for small hard drusen). B: An optical coherence tomography (OCT) B-scan was taken through the fovea (indicated by the white horizontal arrow), and it reveals both conventional drusen below the retinal pigment epithelium (C: RPE; orange vertical arrow on the magnified insert) along with the presence of RPD above the RPE (D: white vertical arrows).

2. Impaired dark adaptation

Our work[20,21] (and others[22]) has recently revealed that those with RPD in particular experience a marked impairment in dark adaptation. Indeed, a recent study revealed that those with RPD had the lowest scores on a Low Luminance Questionnaire,[23] which is consistent with the clinical observations that patients with RPD often report difficulties adjusting to different lighting conditions, especially going from bright light into dim light (for example: going indoors after having been out to hang washing on the line) and taking a considerably longer time to be able to see in the dark (driving into an underground car park). This knowledge can help optometrists explain these symptoms and in their counselling of patients with RPD.

3. Potentially crucial predictor of treatment response

We recently completed a randomised-controlled trial examining the efficacy of a novel subthreshold nanosecond laser (SNL) treatment aimed to prevent or slow late AMD in the early stages of AMD (the Laser Intervention in the Early Stages of AMD [LEAD] study).[24] The LEAD study showed that overall, those randomised to receive SNL treatment did not show a significantly slower rate of progression to late AMD when compared to those who were randomised to a sham treatment.

However, a post-hoc analysis revealed that there was a more than four-fold slowing in disease progression in the SNL compared to the sham group for those who did not have co-existent RPD at baseline, while there was a more than two-fold increased rate of progression in those that did have co-existent RPD.

It is well-recognised that post-hoc analyses in clinical trials should be interpreted with caution and require replication.[25] Nonetheless, these findings highlight the possibility that there are different aetiological pathways involved in these two different AMD phenotypes and that treatments that may be useful for those without RPD may not necessarily be effective for those with RPD. It may therefore be crucial to distinguish between those with and without RPD when considering the choice of interventions in the future.

Future preventative treatment trials may also wish to include, or exclude, those with RPD as part of their eligibility criteria. It is thus imperative that optometrists become familiar with this important feature, so that they can provide a more accurate assessment and counselling when offering patients an opportunity to be involved in clinical trials to find a preventative treatment for AMD.

Reticular pseudodrusen – where to from here?

While we have come a long way in terms of distinguishing RPD from typical drusen and in terms of understanding their clinical implications, substantial efforts continue to be required to understand the disease mechanisms behind their development. This is urgently needed so that we can begin to develop targeted treatments for those with RPD.

Our team at the Centre for Eye Research Australia (CERA), in collaboration with other leading researchers at the University of Melbourne, Walter & Eliza Hall Institute and our collaborators in the UK, Europe and USA, have recently begun one of the world's most comprehensive studies to understand the disease pathways behind the development of RPD. This study, funded by the National Health & Medical Research Council ($5 million over five years), brings together experts in eye health, artificial intelligence, genetics, stem cell research and bioinformatics to tackle RPD. This study has been named the 'Synergy High-Risk AMD Study.'

One of the key goals of this study is to perform in-depth characterisation of a large cohort of individuals with the early stages of AMD, by using different risk factor questionnaires, new retinal imaging, visual function tests and by obtaining biological samples (blood to examine genes and skin biopsies for stem cell research). This will allow potential pathways that drive the development of RPD to be identified, which will then enable us to identify new therapeutic strategies for people with this important AMD phenotype.

We are thus calling on the partnership of eye-care professionals throughout Victoria to help identify people with non-neovascular AMD, and to offer them an opportunity to volunteer to take part in the Synergy High-Risk AMD Study. This involves, at the most basic level, a once-off appointment at the Macular Research Unit, Centre for Eye Research Australia (co-located at the Royal Victorian Eye and Ear Hospital).

Without the help of the optometry profession and without our advocacy for these patients by offering them an opportunity to contribute to the efforts for finding an effective treatment, we will not be able to make headway in improving the lives of those with this potentially devastating condition.

1. Keel S, Xie J, Foreman J et al. Prevalence of Age-Related Macular Degeneration in Australia: The Australian National Eye Health Survey. *JAMA Ophthalmology* 2017; 135: 1242-1249.

2. Meyers KJ, Liu Z, Millen AE et al. Joint associations of diet, lifestyle, and genes with age-related macular degeneration. *Ophthalmology* 2015; 122: 2286-2294.

3. Age-Related Eye Disease Study Research Group. A randomized, placebo-controlled, clinical trial of high-dose supplementation with vitamins C and E, beta carotene, and zinc for age-related macular degeneration and vision loss: AREDS Report No. 8. *Arch Ophthalmol* 2001; 119: 1417-1436.

4. The Age-Related Eye Disease Study 2 Research Group. Lutein + Zeaxanthin and Omega-3 Fatty Acids for Age-Related Macular Degeneration. *JAMA* 2013; 309: 2005-2015.

5. Tufail A, Xing W, Johnston R et al. The Neovascular Age-Related Macular Degeneration Database: Multicenter Study of 92 976 Ranibizumab Injections. *Ophthalmology* 2014; 121: 1092-1101.

6. Gillies MC, Campain A, Barthelmes D et al. Long-Term Outcomes of Treatment of Neovascular Age-Related Macular Degeneration. *Ophthalmology* 2015; 122: 1837-1845.

7. Royal Australian and New Zealand College of Ophthalmologists. RANZCO Referral Pathway for AMD Management. 2018.

8. Hart KM, Abbott C, Ly A et al. Optometry Australia's chairside reference for the diagnosis and management of age- related macular degeneration. *Clin Exp Optom* 2020; 103: 254-264.

9. Mimoun G, Soubrane G, Coscas G. Macular Drusen. *Journal Francais D Ophtalmologie* 1990; 13: 511-530.

10. Zweifel SA, Spaide RF, Curcio CA et al. Reticular Pseudodrusen Are Subretinal Drusenoid Deposits. *Ophthalmology* 2010; 117: 303-312.

11. Greferath U, Guymer RH, Vessey KA et al. Correlation of histologic features with in vivo imaging of reticular pseudodrusen. *Ophthalmology* 2016; 123: 1320-1331.

12. Chen L, Messinger JD, Zhang Y et al. Subretinal drusenoid deposit in age-related macular degeneration: histologic insights into initiation, progression to atrophy, and imaging. *Retina* 2020; 40: 618-631.

13. Wu Z, Ayton LN, Luu CD et al. Reticular Pseudodrusen in Intermediate Age-Related Macular Degeneration: Prevalence, Detection, Clinical, Environmental and Genetic Associations. *Invest Ophthalmol Vis Sci* 2016; 57: 1310-1316.

14. Domalpally A, Agron E, Pak JW et al. Prevalence, Risk and Genetic Association of Reticular Pseudodrusen in Age-related Macular Degeneration. AREDS2 Report 20. *Ophthalmology* 2019; 126: 1659-1666.

15. Finger RP, Wu Z, Luu CD et al. Reticular Pseudodrusen: A Risk Factor for Geographic Atrophy in Fellow Eyes of Individuals with Unilateral Choroidal Neovascularization. *Ophthalmology* 2014; 121: 1252-1256.

16. Hogg RE, Silva R, Staurenghi G et al. Clinical Characteristics of Reticular Pseudodrusen in the Fellow Eye of Patients with Unilateral Neovascular Age-Related Macular Degeneration. *Ophthalmology* 2014; 121: 1748-.

17. Zhou Q, Shaffer J, Ying G-s. Pseudodrusen in the fellow eye of patients with unilateral neovascular age-related macular degeneration: a meta-analysis. *PLoS ONE* 2016;11:e0149030.1755

18. Sleiman K, Veerappan M, Winter KP et al. Optical Coherence Tomography Predictors of Risk for Progression to Non-Neovascular Atrophic Age-Related Macular Degeneration. *Ophthalmology* 2017; 124: 1764-1777.

19. Thiele S, Nadal J, Pfau M et al. Prognostic Value of Retinal Layers in Comparison with Other Risk Factors for Conversion of Intermediate Age-related Macular Degeneration. *Ophthalmology Retina* 2020; 4: 31-40.

20. Luu CD, Tan R, Caruso E et al. Topographic Rod Recovery Profiles after a Prolonged Dark Adaptation in Subjects with Reticular Pseudodrusen. *Ophthalmology Retina* 2018; 2: 1206-1217.

21. Tan R, Guymer RH, Luu CD. Subretinal Drusenoid Deposits and the Loss of Rod Function in Intermediate Age-Related Macular Degeneration. *Invest Ophthalmol Vis Sci* 2018; 59: 4154-4161.

22. Flamendorf J, Agrón E, Wong WT et al. Impairments in dark adaptation are associated with age-related macular degeneration severity and reticular pseudodrusen. *Ophthalmology* 2015; 122: 2053-2062.

23. Yazdanie M, Alvarez J, Agrón E et al. Decreased visual function scores on a low luminance questionnaire is associated with impaired dark adaptation. *Ophthalmology* 2017; 124: 1332-1339.

24. Guymer RH, Wu Z, Hodgson LAB et al. Subthreshold Nanosecond Laser Intervention in Age-Related Macular Degeneration: The LEAD Randomized Controlled Clinical Trial. *Ophthalmology* 2019; 126: 829-838.

25. Rothwell PM. Subgroup analysis in randomised controlled trials: importance, indications, and interpretation. *The Lancet* 2005; 365: 176-186.

For further reference

View the Centre for Eye Research Australia (CERA) website (www.cera.org.au/synergy-high-risk-amd-study/) or contact the research team on: (03) 9929 8113 or amd-studies@cera.org.au. CERA can provide further information on the eligibility criteria, as well as referral pathways (including mail, fax or Oculo) for potential participants.

To learn more about reticular pseudodrusen, as well as the latest evidence-based updates on AMD that will help in the management of patients with this condition, CERA has developed a short course entitled 'AMD for Primary Eyecare Practitioners.' This course can be accessed on Optometry Australia's Institute of Excellence through the following link: https://www.optometry.org.au/institute-of-excellence/cpd-events/e-course-age-related-macular-degeneration-for-primary-eyecare-practitioners/

Introducing Biofinity® toric multifocal

Helping you address the vision and lifestyle needs of more of your patients

Existing and emerging presbyopes represent a major practice growth opportunity and a significant proportion have astigmatism. A combination of three outstanding technologies will help you address the vision and lifestyle needs of more presbyopes.

Optimised Toric Lens Geometry™

Excellent stability and predictable orientation

89% of contact lenses were within 10° of the desired orientation position*[1]

Equivalent to Biofinity® toric for **stability and orientation**[2]

Made with the same **high precision and repeatability** as Biofinity® XR toric

Balanced Progressive® Technology

Remarkable vision performance

Multiple zones of vision correction for clarity at distance, intermediate and near.

Wearers preferred their overall vision with Biofinity® toric multifocal over Proclear® multifocal toric by more than **4:1***[1]

Aquaform® Technology

Creates a comfortable and healthy lens-wearing experience

Wearers rated Biofinity® toric multifocal

8 out of 10 for comfort[3]

CooperVision®

*Assessed approximately 1 hour post-insertion †Rated at 2-week follow-up (p=0.01) **References: 1.** CooperVision Data on file 2018. Non-dispensing, subject masked, randomised, bilateral, cross-over short-term clinical evaluation. 27 astigmatic, presbyopic soft contact lens wearers at 2 sites (UK & US) fitted using CooperVision fitting guide. **2.** CooperVision Data on file 2019. Sub-analysis of lens orientation and stability on settling with Biofinity® toric and Biofinity® toric multifocal. N=89. **3.** CooperVision Data on file 2017. Subject masked, randomised, bilateral, crossover 2-week dispensing study controlled by cross-comparison. 23 astigmatic, presbyopic soft contact lens wearers fitted using CooperVision fitting guide. CooperVision® soft contact lenses for vision correction. Read the instructions for use (https://coopervision.net.au/patient-instruction) and follow the instructions. Biofinity® and Proclear® are registered trademarks of CooperVision Inc and it's subsidiaries.

HYDRATE AND PROTECT YOUR DRY EYES[1]

Systane® #1 DRY EYE BRAND IN AUSTRALIA*

- ✓ PRESERVATIVE-FREE
- ✓ 2x MOISTURE RETENTION[1#]
- ✓ SUITABLE WITH SOFT CONTACT LENSES

Alcon

ALWAYS READ THE LABEL. FOLLOW THE DIRECTIONS FOR USE. IF SYMPTOMS PERSIST, TALK TO YOUR HEALTH PROFESSIONAL.

#Than hyaluronic acid in a laboratory study. References: 1. Rangarajan, R., et al. (2015). J Ocul Pharmacol Ther. 31 (8). p. 491-497 (Sponsored by Alcon)

 *IQVIA 01 Tears S1K Sell-in Value MAT 2019-10.

Megan Zabell
BOptom
Professional Affairs Associate, Vision Care ANZ

Alcon

Perioperative use of ocular lubricants

Study reveals the importance of the use of ocular lubricants before and after cataract surgery

Optometrists and eye-care practitioners in Australia are aware of the importance of managing the tear film and ocular surface of dry eye patients prior to ocular surgery in order to achieve the best possible outcome, particularly in surgeries generally considered more routine such as cataract surgery. A team in Italy have recently completed a retrospective study looking at the effect of using an ocular lubricant perioperatively with patients who had previously not been diagnosed with any dry eye disease, and who underwent routine cataract surgery (50+ years old, standard phacoemulsification process, in-capsule IOL placement, no sutures or limbal relaxing incision).[1]

In the study, patients (excluding those with other ocular pathology) underwent unilateral cataract surgery after which they received a standard post-operative anti-inflammatory and antibiotic combination consisting of a combination dexamethasone + tobramycin drop four times a day for 10 days and the non-steroidal anti-inflammatory (NSAID) drop nepafenac 0.1% three times a day for one month.

Patients were divided into three groups. Group A: those who used a lubricating drop (combined hydroxypropyl guar and hyaluronic acid drop) three times a day for one week immediately before surgery, as well as three times a day for two months post-surgery; Group B: those who used the combination hydroxypropyl guar and hyaluronic acid drop three times a day for two months post-surgery only (no pre-operative use); and Group C: those who didn't add any lubricating drop perioperatively.

Pre-operatively and at each follow-up visit, invasive tear break-up time (TBUT) was measured after the instillation of fluorescein; corneal staining was assessed using the Oxford scale; and the SPEED dry eye questionnaire was administered. Patients also had a pre-operative Schirmer I test (not repeated at subsequent visits). The combination of these tests allowed the researchers to assess both dry eye signs and symptoms in the cohorts.

Disclaimer: This study was conducted overseas, and currently there are no eye drops indicated in ANZ for pre-operative use. The below responses are the views and opinions expressed by the health care provider and do not necessarily reflect those of Alcon.

Pre-operatively there were no statistically significant differences between average patient age, TBUT, SPEED score, Schirmer I test results or corneal staining score. However, at one, four and eight-weeks post-surgery, the two groups of patients using the combination hydroxypropyl guar/hylauronic acid lubricating drop perioperatively (groups A and B) had statistically significantly lower SPEED scores and statistically significantly higher TBUTs on average than those who didn't use a lubricating drop (group C).

While all patients had a reduction in their TBUT after surgery, those who used the hydroxypropyl guar/hyaluronic combination drop both pre- and post-operatively (group A) demonstrated a longet TBUT 4 weeks after surgery than those who only used it post-surgery (group B), and both of these groups had a longer TBUT than those who didn't use a lubricating drop at all (group C).

This study highlights the importance of using a lubricating drop in the perioperative period, even for patients with no prior history of dry eye and who undergo routine, uncomplicated phacoemulsification cataract surgeries. The combined hydroxypropyl guar/hyaluronic acid lubricating drop was effective at reducing post-sugery ocular discomfort and tear instability, particularly if also administrated in the preoperative period.

1. Favuzza E, Cennamo M, Vicchio L et al. Protecting the Ocular Surface in Cataract Surgery: The Efficacy of the Perioperative Use of a Hydroxypropyl Guar and Hyaluronic Acid Ophthalmic Solution. *Clinical Ophthalmol* 2020; 14: 1769–1775.

Megan Zabell BOptom, Alcon Laboratories interviewed Dr Ben LaHood MBChB (dist) PGDipOphth (dist) FRANZCO, who did not participate in the study, for his analysis of the results and their clinical implications.

Was there anything that surprised you about the results of this study?

Despite being a surgeon who places a lot of emphasis on the health of the ocular surface perioperatively, I had not expected that pre-operative lubrication would give such a marked improvement in post-operative dry eye signs and symptoms. Prior to reading this study, my aim of pre-operative lubrication was purely to obtain the best quality biometry to optimise refractive outcomes and did not focus on post-operative patient comfort. This study changes that view and gives even more reason to treat all eyes before cataract surgery.

Do you normally ask your patients to use a lubricant both pre- and post-surgery? Does this depend on any pre-op screening tests?

I do not routinely ask all of my patients to use a lubricant pre-operatively but all of them use a lubricant post-operatively. Pre-operatively, I will ask patients to use a lubricant if I have any concerns about any dry eye signs or symptoms picked up during their consultation or during biometry. If present, I use lubricants as part of my pre-operative ocular surface optimising treatment plan.

Would you find it useful if referring optometrists had already begun the patients on a lubricating drop regimen before you saw them?

Yes, absolutely! I really can't emphasise enough how wonderful it is when the patient feels like their optometrist and ophthalmologist are on the same team, working to give them the outcome they want. Part of this is when their optometrist gets their eyes in optimal condition with lubricants and other dry eye management strategies so that there is minimal delay in obtaining biometry and getting their surgery done. This study will highlight to patients that not only will treatment help get them the best outcome but will also make their experience even more comfortable.

I really can't emphasise enough how wonderful it is when the patient feels like their optometrist and ophthalmologist are on the same team, working to give them the outcome they want.

Do you have any cases that stand out in your mind that highlight the importance of using a lubricating drop pre- and post-surgery?

Yes. Like a lot of ophthalmologists, I was initially uncertain of the real-life importance of perioperative lubricating drops. However, managing perioperative dry eye has, without a doubt, been the biggest factor in improving my surgical outcomes.

I use a lot of presbyopia correcting IOLs and I have had one particular situation that always reminds me of the importance of the ocular surface. I was referred a patient who was unhappy with their trifocal IOL outcome due to glare post-operatively ever since their surgery one month previously. They were referred for a lens exchange surgery. Despite only having mild dry eye symptoms and signs, with an appropriate dry eye management plan and lubricants, they were transformed to a very happy patient with good distance, intermediate and near vision. I believe they could have had a much happier post-op experience if treated with adequate lubricants from the start.

How can an optometrist establish a protocol with the referring ophthalmologist for use of lubricants in patients who will undergo cataract surgery?

The best thing to do is to get in touch and have a chat. I love to hear from my optometrist colleagues and really enjoy talking about our patients to make a plan. Once a standard plan is established, it makes it much easier to tailor things to an individual patient.

This interview was made in collaboration with Alcon Laboratories (Australia).

At Specsavers, we're committed to helping you reach your potential.

As an optometrist we will help you map out a solid career path and support you to achieve your professional goals.

From day one onwards we will provide you with a host of professional development opportunities – from our ophthalmology-led education programs to our clinical conference and 24/7 access to our MyCPD Portal.

And we also provide genuine career progression, from our graduate program all the way to partnership, supported with leadership and training through our Pathway programs.

Whether you're looking for your first job, looking for new opportunities or feel ready to take that next step to partnership, at Specsavers you're only limited by the scale of your own ambition.

Go to **spectrum-anz.com** or contact us:
Partnership enquiries: Marie Stewart +61 408 084 134
Optometry recruitment enquiries: Madeleine Curran +61 437 840 749

Specsavers

These original clinical images were submitted by Optometry Australia member Minh Nguyen in response to our call for images.

Minh Nguyen
BVisSc (Dist), MOptom (Dist)
Brisbane, QLD

Figure 1.
Right eye

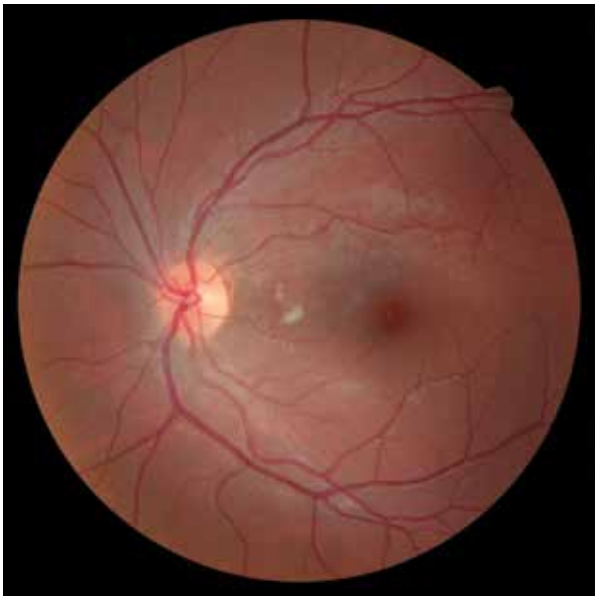


Figure 2.
Left eye

A 28-year-old female (GS) presented with symptoms of painless central scotoma in the right eye of 15-day onset. She had returned from Thailand two days ago after recovering from Dengue fever that she contracted 22 days prior. GS had mild symptoms of sinus congestion and minor fatigue but was otherwise in good health. She was taking fish oil and amoxicillin prescribed by her general practitioner from an unrelated injury.

Best corrected visual acuity (BCVA) was RE -0.25/-0.75x120 V.A. 6/18, and LE plano/-0.50x65 V.A. 6/6. Intra-ocular pressures were 16 mmHg in both eyes. Ocular motility and pupil reactions were normal. Dilated fundus examination with slit lamp indirect ophthalmoscopy revealed retinal nerve fibre layer haemorrhage and cotton wool spot at the macula in the right eye, and para-foveal cotton wool spot in the left eye (Figures 1 and 2). Optical coherence tomography (OCT) scan revealed cystoid macula oedema in the right eye.

Diagnosis: Dengue maculopathy

GS was referred on the same day to Princess Alexandra Eye Casualty and subsequently diagnosed with Dengue maculopathy. Initial treatment was close observation. A one-off intravitreal anti-VEGF (Avastin) was administered to the right eye three months from initial presentation to treat eccentric cystoid macula oedema. Despite some residual scarring at the macula in the right eye, BCVA recovered to 6/6 in the right eye, and 6/4.5 in the left eye. GS is in good spirits and continues to do art.

Dengue fever is a mosquito-borne viral illness most prevalent in tropical areas. Although not endemic in Australia, mosquitos may act as vectors between infected and non-infected persons.[1,2]

Typical symptoms include fever, headache, vomiting, muscle and joint paints, and a characteristic skin rash. Ocular involvement varies and may present with anterior uveitis or pan uveitis, maculopathy, retinal haemorrhages, cotton wool spots, and maculopathy.[1,3,4]

Ocular pathogenesis from dengue fever is poorly understood and management of ophthalmic disease depends on the degree of inflammation.[3,4,5] Ocular inflammation may be treated with ophthalmic steroids or close observation.[5]

In this case, anti-VEGF was used to resolve cystoid macula oedema and GS is under close monitoring by ophthalmologist. There is no specific treatment for the dengue virus, however systemic infection is treated with fluids, pain relievers, and in severe cases hospitalisation.[6]

1. Queensland Health, Dengue: virus, fever and mosquitoes, health.qld.gov.au, Last updated 20/02/2020 [cited 2021 Jan 27]. Available from: https://www.health.qld.gov.au/clinical-practice/guidelines-procedures/diseases-infection/diseases/mosquito-borne/dengue/virus-fever#:~:text=Dengue%20fever%20is%20not%20endemic,East%20Asian%20and%20Pacific%20countries.

2. Queensland Health, Dengue, conditions.health.qld.gov.au, Last updated: 21/02/2020 [cited 2021 Jan 27]. Available from: http://conditions.health.qld.gov.au/HealthCondition/condition/14/217/284/Dengue

3. Tabbara K. Dengue retinochoroiditis. *Ann Saudi Med* 2012; 32: 530-533. doi:10.5144/0256-4947.2012.30.4.1105

4. Juanarita J, Azmi MN, Azhany Y, et al. Dengue related maculopathy and foveolitis. *Asian Pac J Trop Biomed* 2012 2: 755-6. doi: 10.1016/S2221-1691(12)60223-8. PMID: 23570008; PMCID: PMC3609368.

5. Bacsal KE, Chee S, Cheng C, et al. Dengue-Associated Maculopathy. *Arch Ophthalmol* 2007; 125: 501–510. doi: 10.1001/archopht.125.4.501

6. Haritoglou C, Scholz F, Bialasiewicz A et al.. Okulare Manifestation bei Dengue-Fieber [Ocular manifestation in dengue fever]. *Ophthalmologe* 2000; 97: 433-6. German. doi: 10.1007/s003470070094. PMID: 10916388

Nicola Peaper
National Sales and Professional Services Manager

Rodenstock Australia

Sport and safety frames

Attaining clear and comfortable vision

High-wrap sunglass and sports and safety frames are commonly dispensed in general practice, but it may come as a surprise that most lenses ordered with this type of frame are standard spherical grind or even stock. This article will examine the reasons that cause this combination of lens and frame to fail both cosmetically and, more importantly, optically.

Consider a typical order of either a safety spectacle or high-wrap sunglass:

- Single vision 1.5 index, hard coated
- R -4.00 DS
- L -2.75 / -1.00 X 145
- Pupillary distance (PD) 58
- Frame 56 x 18 (Giving a minimum blank size of 72mm)
- Face Form Angle (FFA) 20°
- Corneal vertex distance (CVD) 13mm
- Pantoscopic Tilt (PT) 7°

How many ways will this fail?

1. Base Curve

Base curve choice is generally a compromise between optics and cosmetics. This frame face form angle will require a base curve of minimum 6 D, otherwise the degree of flatness of the lenses will cause the frame to splay out.

Using Vogels formula for base curves,[1] we can see that, for the right lens, the ideal base is equal to 4 D (= half the spherical equivalent power + 6 D). However, if we examine a -4.00 D stock lens, it will be produced on a flatter base curve of around 1.5 to 2.0 D. This flatter base curve will reduce both minimisation, by reducing the back vertex distance a high curved lens will have, and edge thickness. Both of these things will improve the cosmetics of the lens. If a stock lens is used in this example, even with careful bevelling the frame will be unwearable as the sides will be splayed.

Producing this power on a 6 D base curve, it can be bevelled to fit the frame and keep the correct shape. However, the narrow pupillary distance of 58 will significantly add to the temporal thickness and weight of the lens. Figure 1 compares our example pupillary distance (PD) with one of 70 mm.

	Sph -4.00 D	
	PD 29 mm	PD 35 mm
Centre thickness [mm]	1.9	1.8
Minimum edge thickness [mm]	3.0	3.0
Maximum edge thickness [mm]	6.7	5.3
Base curve [D]	6.5	6.5
Weight [g]	6.9	6.4

Figure 1.
Comparison of example PD with one of 70mm

B.I.G. VISION™ FOR ALL

BIOMETRIC INTELLIGENT GLASSES

CONSTRUCTING A COMPLETE INDIVIDUAL BIOMETRIC EYE MODEL

With the combination of our DNEye® Scanner and our patented technologies, we can determine all relevant biometric data.

This data is used to create a unique biometric eye model and, based on this model, we are able to calculate a lens that matches each individual person to the micrometre. We call this our DNEye® PRO Technology and Rodenstock is the only lens manufacturer able to directly transfer all of these measurements into the production of the lens. This means we create unique biometric eye models for both eyes. That data is then digitally transferred to Rodenstock.

RODENSTOCK

Because every eye is different

2. Cut Out

High wrap frames are frequently ordered with narrow PDs. In the example, a 72 mm blank is needed. This may be possible in this minus script but probably would not be possible in plus.

3. Optics

This is the most significant problem. The dispensed lens sits at a different angle than the prescribed lens. This means that light is incident at an oblique angle and our patient is no longer experiencing a -4.00 D lens (Figure 2).

The power the patient is experiencing can be calculated as follows (taking only the face form angle into account):

Ordered power (F) -4.00 DS

FSPH = F (1 + $\sin^2 \theta/2n$) Where θ is the tilt angle and n is refractive index [1]

FSPH = - 4.16

FCYL = FSPH $\tan^2 \theta$ [1]

FCYL = -0.53

Power experienced at vision point

-4.16 / -0.53 x 90

If a spherical stock or grind lens has been dispensed and checked to be correct on a vertometer where the lens sits flat, the patient is now wearing a lens in a position where the power experienced sits outside tolerance. For the patient to experience a -4.00 D lens when presented with this degree of tilt then a compensated lens needs to be calculated. This will have a power weaker than -4.00 D spherically and will have an opposite powered cyl.

There are two ways that this can happen:

- The dispenser takes the angle the lens sits at and calculates a compensated power to be supplied.
- The dispenser gives the lab the frame parameters and asks for a compensated lens.

This is a simplification of the process as when a compensated power is calculated FFA, PT and CVD are all taken into account.

When compensating a lens for wrap, another important consideration is prism caused by the curvature of the lens. The formula for calculating this is: $\Delta = 100 \tan\theta \, t/n \, F1$. Where t is the centre thickness of the lens and F1 is the base curve.[2]

In the example, the prism due to curvature is 0.3Δ base out. This would require the compensated power to include 0.3Δ base in. While this does not seem significant, consider what happens with a +4.00 D lens on the same base. The extra lens thickness now causes a prism of 1.2Δ.

Considering these calculations, at minimum the patient should be supplied with a lens compensated for both power and prism at the ocular centre to experience the refracted power. In the same way compensations should also be included for pantoscopic tilt and corneal vertex distance. This will give good central vision and, as the power at ocular centre is precise, the peripheral aberrations will be less significant.

However, this is not the best that can be done for the patient. In Figure 2, the vision point is taken to be the optical centre of the lens. As the patient scans across the lens to different points of interest, both the angle that light is incident and the corneal vertex distance change. It is possible to produce an aspheric/atoric freeform surface individually designed for the wearer based on face form angle, corneal vertex distance of the frame shape and pantoscopic tilt. A lens produced with these parameters will give clear vision out to the rim of the frame. This quality of lens should always be considered for drivers especially professional truck and car drivers and patients who have sports activities that require good peripheral vision.

While only single vision lenses have been considered, the same calculations are necessary for progressive lenses. As discussed previously[2] (in the March issue of *Optometry Connection*) the face form angle and pantoscopic tilt have a significant effect on the performance of progressive lens, including increased swim and narrowing of corridor. The face form angle will also affect the relative position of the corridor so the pupil will no longer follow the centre of the corridor. In the worst case the corridor will be unusable.

High wrap frames are a necessity for protection both in industry and for sun wear. When dispensing this form of protection, it is equally important to ensure that the patient has clear and comfortable vision and all lenses are dispensed to within tolerance.

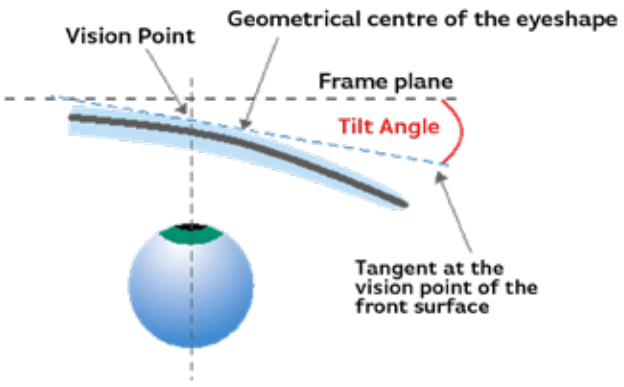
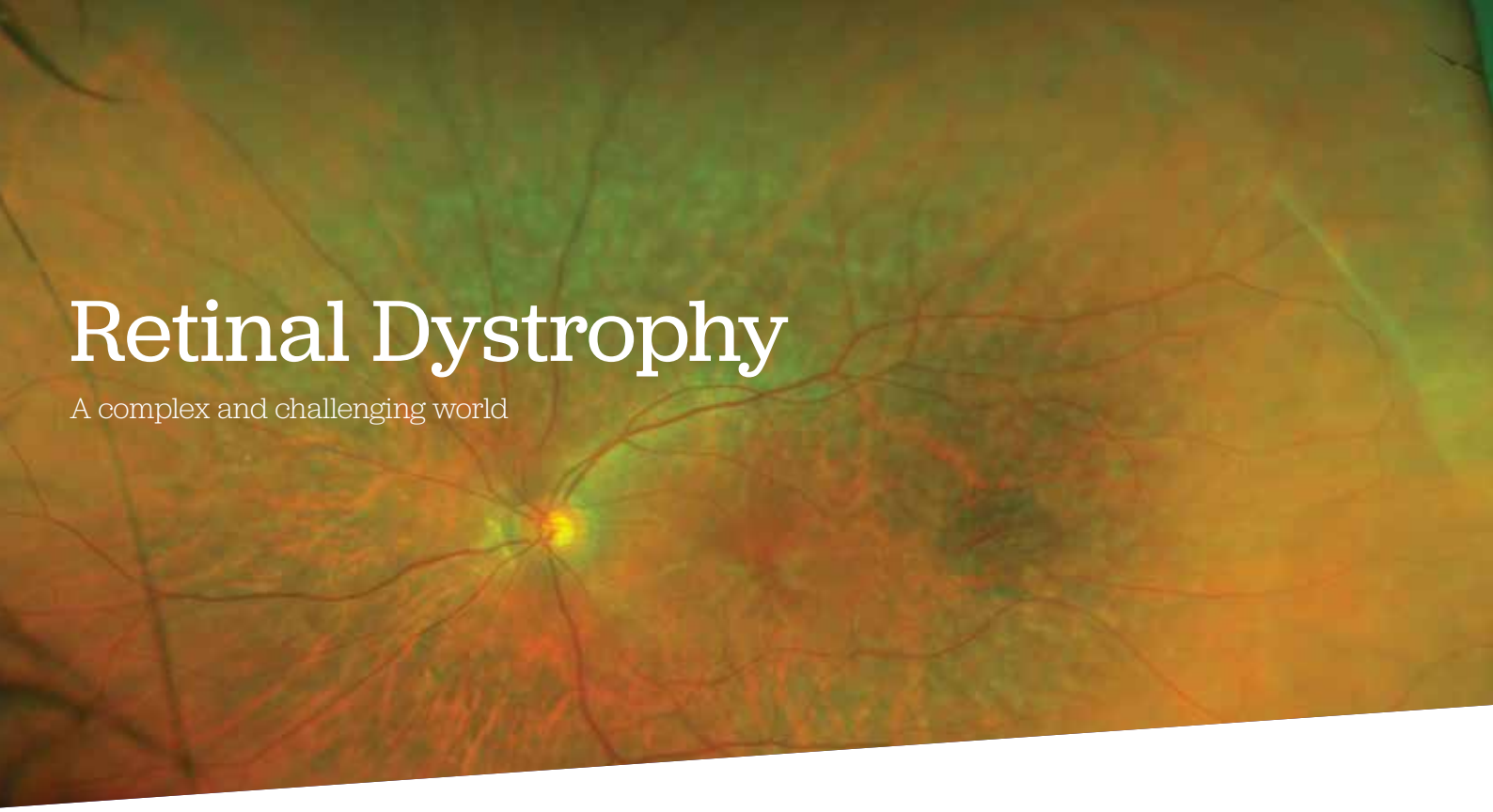


Figure 2.
The difference between the testing (frame) plane and the dispensed lens plane is referred to as the tilt angle.

1. Brooks CW, Borish IM. *System For Ophthalmic Dispensing*, 3rd ed. St Louis, MO: Butterworth Heinemann, 2006: 411-413

2. Peaper N. Optimisation and compensation. *Optometry Connection* 2021; March: 20-21.



Retinal Dystrophy

A complex and challenging world

MULTIDISCIPLINARY MANAGEMENT

Pauline Xu
BOptom (Hons) MOptom GradCertOcTher
Lead Clinician – Retinal Dystrophies

Centre for Eye Health

Inherited retinal dystrophy (IRD) is a complicated topic. The diagnosis is challenging and requires an armamentarium often beyond the reach of standard practice. Management often warrants a multidisciplinary approach combining the skills and expertise of a retinal specialist, geneticist, optometrist, occupational therapist, social worker, general practitioner, psychologist and so on.

Technological advancements in genetic testing and gene therapy including the recent approval of Luxturna, the first gene therapy for the treatment of Leber's congenital amaurosis caused by the RPE65 mutations, marks the dawn of a new era in managing IRD. This article presents a case of IRD and shines a spotlight on the need for a paradigm shift in caring for these patients.

Case study

Jane (name changed to protect the identity of the patient) is a 53-year-old Caucasian female who was referred to the Centre for Eye Health (CFEH) for electrophysiology by a glaucoma specialist, who noted her visual field loss was not explained by glaucoma. Jane has seen several ophthalmologists in the past and was told there was something wrong in the retina at the age of forty. She declined further follow-up as she perceived the consultations as costly and time-consuming with no definitive answers. Her father and brother were both diagnosed with age-related macular degeneration (AMD) and there was no reported consanguinity. Her medical history was unremarkable.

Her best corrected visual acuities were 6/9 in the right and 6/12 in the left. Farnsworth–Munsell 100 hue test showed a tritan defect in each eye. Anterior segment examination revealed bilateral laser peripheral iridotomies and mild nuclear sclerosis (Figure 1). Colour fundus photography (Figures 1A and 1B) and ultra widefield imaging (Figures 1C and 1D) showed bilateral epiretinal membrane (ERM) and pigmentary abnormalities in the perimacular and midperipheral fundus.

Fundus autofluorescence (FAF) (Figures 1E and 1F) displayed a symmetric presentation of speckled hyper- and hypo-autofluorescence associated with the pigmentary abnormalities and peripapillary sparing characterised by a ring of relatively normal autofluorescence surrounding the optic discs (Figure 1F).

Notably, there were several well-demarcated, hypo-autofluorescent patches in the midperiphery representing complete retinal pigment epithelium (RPE) atrophy. Spectral domain optical coherence tomography (SD-OCT) confirmed bilateral ERM with distortion of foveal pit, focal loss of the ellipsoid zone nasal to the fovea in the right (Figure 1G), microcystic spaces and more diffuse loss of the ellipsoid zone, interdigitation zone and RPE in the left eye (Figure 1H).

Visual evoked potentials (VEP), pattern electroretinography (pERG) and full field electroretinography (ffERG) were performed according to the International Society for Clinical Electrophysiology of Vision (ISCEV) standard.[1-3] VEP ruled out optic nerve dysfunction and pERG confirmed macular

dysfunction. ffERG showed normal rod a-waves and abnormal b-waves in the dark-adapted status suggestive of issues in the rod-bipolar cell pathway (Figure 2A). In the light-adapted status, flicker ERG was abnormal as was the photopic b-waves whilst the a-waves were delayed with reduced amplitude suggestive of dysfunctional cone pathway. Whatham et al. offered more detailed explanation on the role of ERG in diagnosing retinal dystrophy.[4]

The clinical assessment showed apparent retinal dystrophy affecting both the photoreceptors and RPE and characteristic peripapillary sparing and the electrophysiology results excluded rod-cone dystrophy (retinitis pigmentosa). A tentative diagnosis of Stargardt disease was made in discussion with the Centre's consultant ophthalmologist.

Discussion

Stargardt disease is a one of the most common juvenile onset macular dystrophies affecting one in 10,000 persons,[5] associated with autosomal recessive inheritance of a mutation in the *ABCA4* gene.[6] *ABCA4* encodes for a transporter protein located in photoreceptor outer segments responsible for cellular transportation of the retinoids from photoreceptors to RPE. Impaired transport can lead to accumulation of lipofuscin and eventual death of the RPE and overlying photoreceptors.[6]

Although Stargardt disease is usually diagnosed within the first two decades of life, adult, and late onset have also been reported.[7-9] The condition features progressive central vision loss with multifocal yellow-white fundus flecks and atrophic macular lesions. A 'dark choroid' sign presents in more than 80 per cent of Stargardt patients and refers to the absence of early choroidal hyper-fluorescence in fluorescein angiography due to the blocking effect of the high-grade lipofuscin accumulation in the RPE.[10] A further characteristic clinical feature of Stargardt disease includes peripapillary sparing where an annulus of normal retina tissue free from flecks and RPE atrophy can be found surrounding the optic nerve head.[11] While common, this sign is neither universal nor pathognomonic of Stargardt disease.[12,13]

The typical work-up for Stargardt disease requires a series of clinical and functional tests including multimodal imaging and visual field assessment to establish a clinical diagnosis followed by genetic confirmation.

Stargardt disease can be differentiated from AMD based on the shape, distribution, and autofluorescent properties of the flecks. Flecks are fishtail-shaped, typically scattered throughout the posterior pole and display intense autofluorescence due to the overload of lipofuscin, while drusen are round-shaped, tend to congregate in the central macula and show increased, normal, or decreased autofluorescence to a modest degree compared to flecks.

However, differential diagnosis can be challenging towards the later stage of Stargardt disease as the flecks resorb and hypo-autofluorescent RPE atrophy develops.

Another mimicking condition named multifocal pattern dystrophy simulating Stargardt disease describes a subtype of pattern dystrophy of the RPE. Flecks are the shared features between these two conditions. Features that distinguish pattern dystrophy from Stargardt disease include autosomal dominant inheritance pattern, adult onset (40-50s), relatively good and stable VA, and absence of 'dark choroid' sign on fluorescein angiography.[14] Detailed phenotypic presentations of pattern dystrophy can be found in the CFEH chairside reference guide.

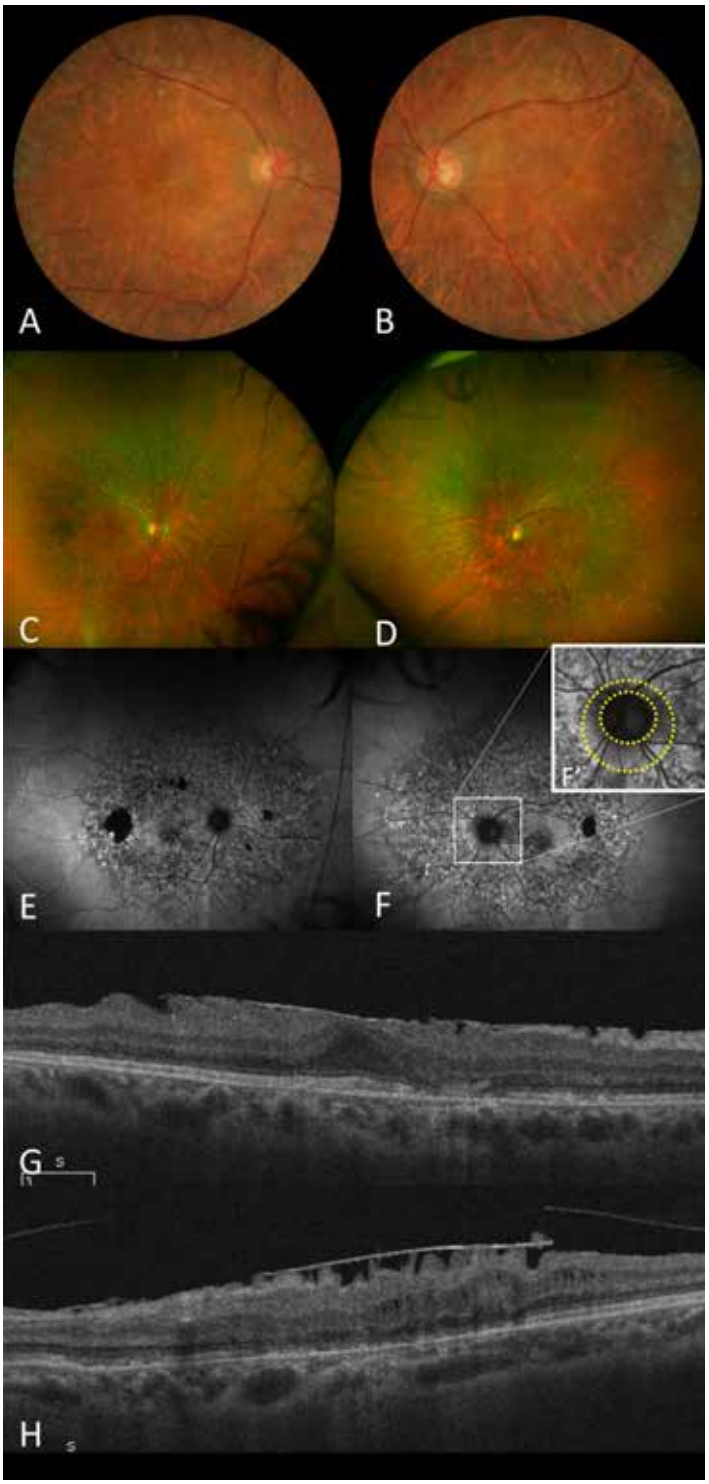


Figure 1.
Multimodal imaging results of the case. A: Right and B: left colour fundus photographs showing pigmentary abnormalities around the vascular arcades and in the macula. C-D: Widefield imaging showing that the pigmentary abnormalities extend beyond the posterior pole. E-F: Fundus autofluorescence (FAF) showing symmetric, speckled hyper and hypo-FAF. F': Magnified image of FAF in the left eye showing an annulus of peripapillary sparing (bordered by the dotted yellow line). G: Spectral-domain optical coherence tomography (SD-OCT) of the right eye showing epiretinal membrane, focal outer retina loss and disturbance of ellipsoid zone. H: SD-OCT of the left fovea showing epiretinal membrane with loss of foveal pit, microcystic spaces and diffuse loss of ellipsoid zone, interdigitation zone and RPE.

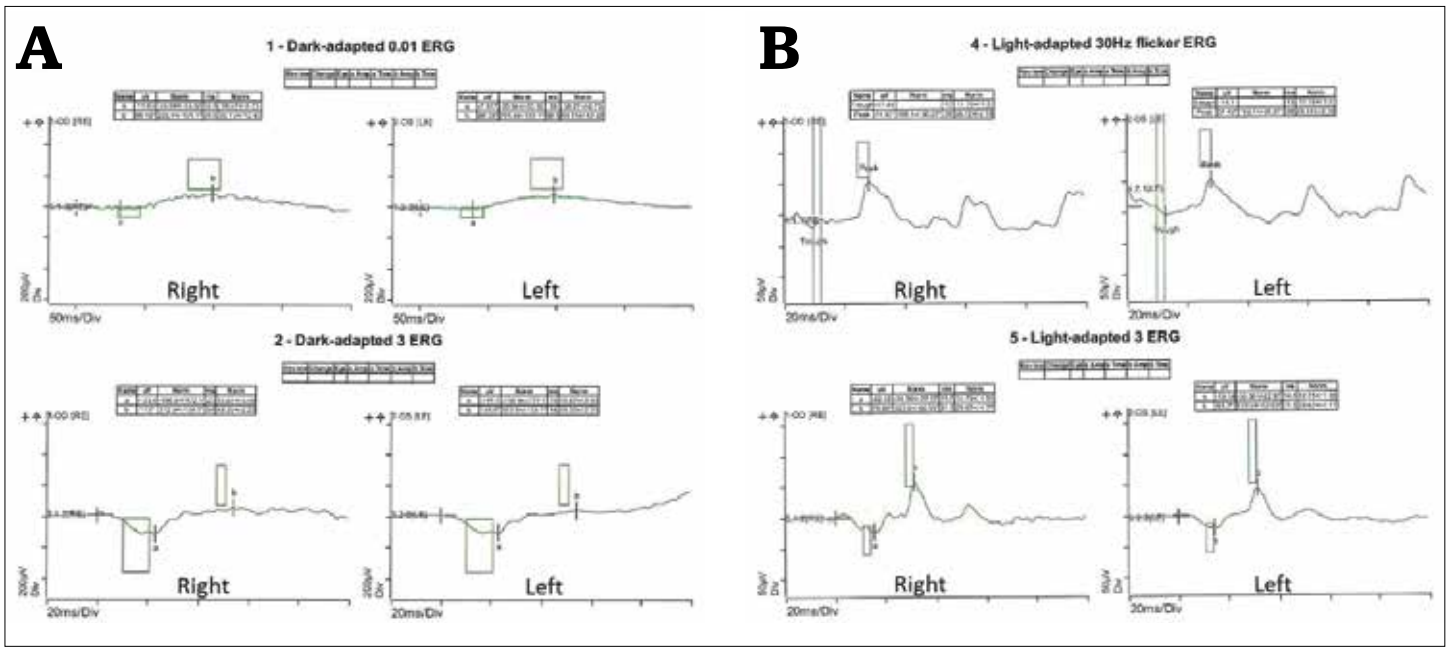


Figure 2.
A: Full field electroretinogram results showing normal a-waves, and abnormal b-waves suggestive of rod-to-bipolar cell pathway dysfunction in the dark-adapted state B: and cone pathway dysfunction in the light-adapted state. Age-matched normal limits are represented by the green boxes.

Electrophysiology is valuable in excluding other IRD. Rod-cone dystrophy is characterised by more prominent rod dysfunction, leading to markedly reduced scotopic responses before the cone involvement. Conversely, cone dystrophy and cone-rod dystrophy feature more severely affected photopic responses than scotopic responses in ffERG, while pattern dystrophy typically does not show any pan-retinal rod or cone involvement. ffERG results in Stargardt disease can vary from normal scotopic and photopic responses (group 1), abnormal photopic response but normal scotopic response (group 2) and abnormal photopic and scotopic responses (group 3), and the group 3 have higher risk of future deterioration than group 1 and 2.[15,16] In this patient, the fact that both rod and cone function was affected indicates a shorter review cycle. As electrophysiology is a specialised area, the interpretation of the results is performed by the trained personnel including the retinal ophthalmologist.

Genetic testing helps to confirm the diagnosis and is often the only way to verify the extra gene mutation as the cause of the condition. Genotyping is imperative to determine the suitability for participating in clinical trials when they become available. There is also implication in family planning based on the inheritance pattern. Genetic testing can be ordered by the patient's retinal ophthalmologist, counselling services can be offered to the patient to make informed decision about the genetic test by the genetic counsellor, and the test results are typically delivered by the team of ophthalmologist and geneticist.

The prognosis of Stargardt disease depends on the age of onset, vision at the initial visit and the status of the fovea. Late onset, better initial VA and foveal sparing from the atrophic changes is associated with a better prognosis.[17,18] Genetic information, ERG results, and intrafamilial presentation should also be considered in the counselling of the patients about prognosis.[9]

Jane's experience resonates with many patients living with IRD: the follow-up was perceived as a burden due to a lack of the treatment options. Eye-care practitioners need to spend significant chair time and develop expertise on this specific topic to educate and engage with their patients.

General advice to patients with Stargardt disease should include sunlight protection to reduce lipofuscin build-up, smoking cessation, and avoid Vitamin A dietary supplements.[19] Examination of their family members is essential to construct a pedigree and to screen for other diseases such as AMD as the prevalence of AMD is higher in families with Stargardt.[20,21] Following up these patients is crucial, although it may prove challenging in real life.

Jane's experience resonates with many patients living with IRD: the follow-up was perceived as a burden due to a lack of the treatment options. Eye-care practitioners need to spend significant chair time and develop expertise on this specific topic to educate and engage with their patients. Referrals

to a low-vision service may be needed when central vision deteriorates further and in more advanced cases where peripheral vision loss occurs, orientation and mobility support would be indicated.

The future

There is currently no cure for Stargardt disease. Two potential therapies that are currently in the phase 2 trial stage include oral tablets that modulate the visual cycle, and intravitreal injections that inhibit complement system and reduce cell death.[22] Gene therapy targeting *ABCA4* gene and stem cell therapy which aims to rejuvenate or replace damaged RPE cells are in the early stage of development.[22] Patients should discuss with their ophthalmologists regarding the current development and their suitability to clinical trials.

Recently, a survey was developed to understand the views of people living with IRD on potential genetic therapies in the Australian context. The results will help guide clinicians on how to advise their patients and offer insight as to the infrastructure needed for more widespread genetic testing and counselling.[23]

CFEH in collaboration with Prince of Wales Hospital Ophthalmology and Guide Dogs NSW/ACT, is expanding the inherited retinal dystrophy clinic that features a multidisciplinary team. It is envisaged that the evolving IRD clinic will offer a 'one-stop-shop' service for most patients like 'Jane' where clinical diagnosis, genetic testing and counselling, future clinical trials, vision rehabilitation, and connection services can all happen under one roof ensuring that patients' needs can be taken care of at all levels.

Acknowledgements

The author thanks Dr Angelica Ly and Professor Michael Kalloniatis for reviewing the manuscript.

Disclosures

The Centre for Eye Health receives primary funding from Guide Dogs NSW/ACT.

1. Odom JV, Bach M, Brigell M et al. ISCEV standard for clinical visual evoked potentials: (2016 update). *Doc Ophthalmol* 2016; 133: 1-9.

2. McCulloch DL, Marmor MF, Brigell MG et al. ISCEV Standard for full-field clinical electroretinography (2015 update). *Doc Ophthalmol* 2015; 130: 1-12.

3. Bach M, Brigell MG, Hawlina M et al. ISCEV standard for clinical pattern electroretinography (PERG): 2012 update. *Doc Ophthalmol* 2013; 126: 1-7.

4. Whatham AR, Nguyen V, Zhu Y et al. The value of clinical electrophysiology in the assessment of the eye and visual system in the era of advanced imaging. *Clin Exp Optom* 2014; 97: 99-115.

5. Stargardt K. Über familiäre, progressive Degeneration in der Maculagegend des Auges. *Albrecht Von Graefes Arch Ophthalmol* 1909; 71: 534-550.

6. Michaelides M, Hunt DM, Moore AT. The genetics of inherited macular dystrophies. *Am J Med Genet* 2003; 40: 641-650.

7. Lambertus S, van Huet RA, Bax NM et al. Early-onset stargardt disease: phenotypic and genotypic characteristics. *Ophthalmology* 2015; 122: 335-344.

8. Westeneng-van Haaften SC, Boon CJ, Cremers FP et al. Clinical and genetic characteristics of late-onset Stargardt's disease. *Ophthalmology* 2012; 119: 1199-1210.

9. Georgiou M, Kane T, Tanna P et al. Prospective Cohort Study of Childhood-Onset Stargardt Disease: Fundus Autofluorescence Imaging, Progression, Comparison with Adult-Onset Disease, and Disease Symmetry. *Am J Ophthalmol* 2020; 211: 159-175.

10. Fishman GA, Farber M, Patel BS et al. Visual acuity loss in patients with Stargardt's macular dystrophy. *Ophthalmology* 1987; 94: 809-814.

11. Cideciyan AV, Swider M, Aleman TS et al. *ABCA4*-associated retinal degenerations spare structure and function of the human parapapillary retina. *Invest Ophthalmol* Vis Sci 2005; 46: 4739-4746.

12. Burke TR, Rhee DW, Smith RT et al. Quantification of peripapillary sparing and macular involvement in Stargardt disease (STGD1). *Invest Ophthalmol Vis Sci* 2011; 52: 8006-8015.

13. Burke TR, Allikmets R, Smith RT et al. Loss of peripapillary sparing in non-group I Stargardt disease. *Exp Eye Res* 2010; 91: 592-600.

14. Saksens NT, Fleckenstein M, Schmitz-Valckenberg S et al. Macular dystrophies mimicking age-related macular degeneration. *Prog Retin Eye Res* 2014; 39: 23-57.

15. Lois N, Holder GE, Bunce C et al. Phenotypic subtypes of Stargardt macular dystrophy-fundus flavimaculatus. *Arch Ophthalmol* 2001; 119: 359-369.

16. Fujinami K, Lois N, Davidson AE et al. A longitudinal study of stargardt disease: clinical and electrophysiologic assessment, progression, and genotype correlations. *Am J Ophthalmol* 2013; 155: 1075-1088 e1013.

17. van Huet RA, Bax NM, Westeneng-Van Haaften SC et al. Foveal sparing in Stargardt disease. *Invest Ophthalmol Vis Sci* 2014; 55: 7467-7478.

18. Rotenstreich Y, Fishman GA, Anderson RJ. Visual acuity loss and clinical observations in a large series of patients with Stargardt disease. *Ophthalmology* 2003; 110: 1151-1158.

19. Federspiel CA, Bertelsen M, Kessel L. Vitamin A in Stargardt disease-an evidence-based update. *Ophthalmic Genet* 2018; 39: 555-559.

20. Allikmets R. Further Evidence for an Association of ABCR Alleles with Age-Related Macular Degeneration. *Am J Hum Genet* 2000; 67: 487-491.

21. Souied EH, Ducroq D, Gerber S et al. Age-related macular degeneration in grandparents of patients with Stargardt disease: genetic study. *Am J Ophthalmol* 1999; 128: 173-178.

22. Cremers FPM, Lee W, Collin RWJ et al. Clinical spectrum, genetic complexity and therapeutic approaches for retinal disease caused by *ABCA4* mutations. *Prog Ret Eye Res* 2020; 79: 100861.

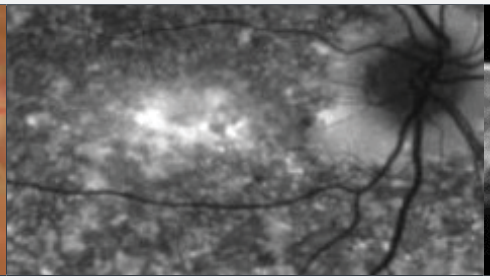
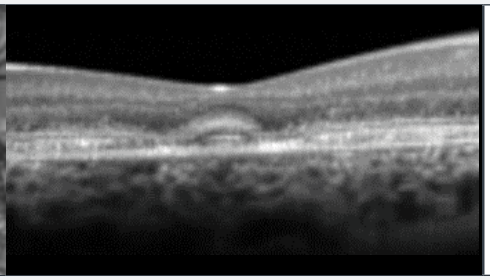
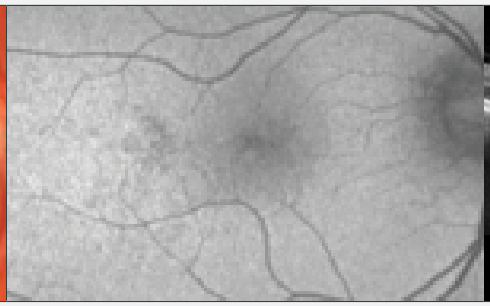
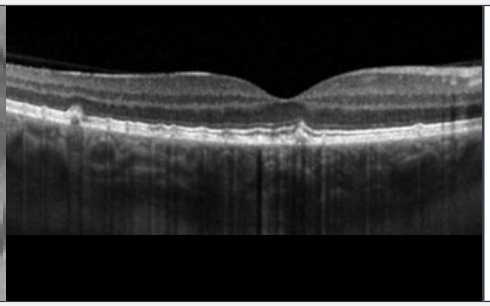
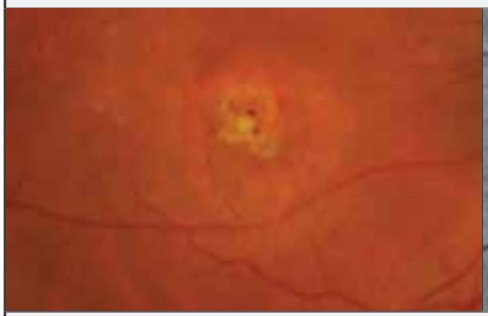
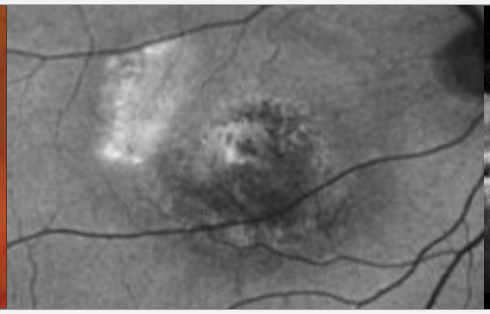
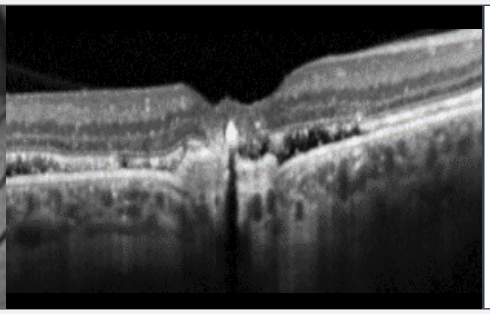
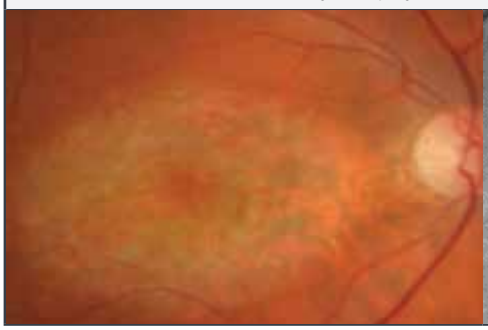
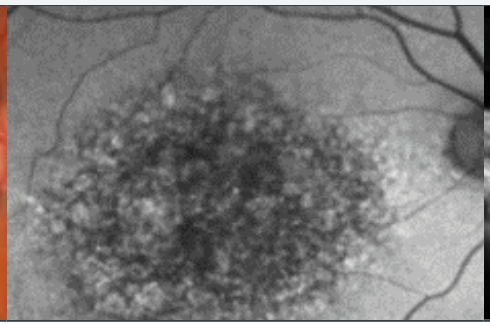
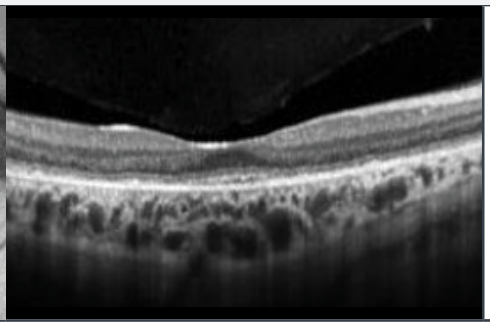
23. Survey: Your thoughts on gene therapy for inherited retinal diseases [Internet]. Centre for Eye Research; 2020 [cited 22 Feb 21]. Available from: https://www.cera.org.au/ird-survey/.

CLINICAL RESOURCE

Chairside reference: macular dystrophies

From the Centre for Eye Health

Macular dystrophies

Retinal Photo	Fundus Autofluorescence	Optical Coherence Tomography (OCT)
Stargardt Disease/Fundus flavimaculatus*		
		
Autosomal dominant drusen /Malattia Leventinese/Doyne honeycomb retinal dystrophy		
		
Best Vitelliform Macular Dystrophy*		
		
Central Areolar Choroidal Dystrophy*		
		

* The specific diagnosis of dystrophies may be uncertain in the absence of electrophysiology results and genetic testing

The material in this reference is current at the time of publication. This reference is designed as a guide to aid diagnosis and management decisions, however, individual cases must be assessed in the context of all available clinical data.

	Description
	➔ Foveal atrophy often surrounded by discrete yellowish round or pisciform flecks scattered throughout the fundus with intense hyper-autofluorescence ➔ Juvenile onset ➔ Gradual and progressive visual decline ranging from 6/15-6/60 ➔ Predominantly autosomal recessive inheritance ➔ Hyper-reflective thickening of the retinal pigment epithelium (RPE) and thinning of the ellipsoid zone (EZ). ➔ May be foveal sparing with regular EZ profile in some cases
	➔ Small, discrete drusen radiate in streaks or lines from the centre of the fovea in the early stage ➔ Drusen progressively become confluent, leading to the honeycomb appearance ➔ Onset in the 3rd to 4th decade of life ➔ Usually asymptomatic before the age of 40, then more rapid progressive central vision loss occurs ➔ Autosomal dominant inheritance ➔ Risk of geographic atrophy and/or choroidal neovascularisation in later stage ➔ A hyper-reflective thickening of the retinal pigment epithelium–Bruch membrane complex, associated with localised dome-shaped elevations
	➔ Initially presents with a yellow, yolk like macular lesion ➔ Progresses to atrophy and/or neovascularisation in later stages ➔ Variable age of onset ranging from 1st to 6th decade ➔ Usually symptomatic before the age of 40 ➔ Autosomal dominant inheritance ➔ Early lesions found between the RPE and sensory retina ➔ Later stage may involve sub-retinal fluid, subretinal fibrosis and oedema ➔ Variable hyper-fluorescence corresponding to vitelliform material, hypo-autofluorescence in atrophic areas
	➔ Initially parafoveal pigmentary RPE changes progressing to enlarged RPE atrophy and eventually confluent chorioretinal atrophy ➔ VA deteriorates at age 30-50 years but may be asymptomatic until later. Usually causes profound vision loss ➔ Occasionally photophobia associated ➔ Autosomal dominant inheritance ➔ Reduced retinal thickness with disruption of the EZ and outer retina ➔ Remaining retinal layers are intact ➔ Speckled pattern of hyper- and hypo-fluorescence confined to the macula region in a round / oval shape

Tim Thurn
B.Optom GCertBus
Essilor ANZ - Professional Services Director

Customised and personalised lenses: the future today

Shifting to personalised products doesn't happen overnight and, by their very nature, the requirements are specific. So why is it so important in the optical industry? Practices are already personalising patient experiences by integrating advanced digital technologies in their consulting rooms. Sophisticated imaging systems, retinal photography, topography etc., have all dramatically changed the approach to clinical patient management. Increasingly this use of technology includes the dispensary with digital tools driving new levels of accuracy, precision, customisation and personalisation. These invested practices are seeing that technology drives patient engagement, increases their acquisition and conversion rates and ultimately, increases patient retention.

Patient benefits

Progressives have become more sophisticated over the last 20 years, to some extent driven by digital surfacing. Aspheric, atoric lenses and double aspheric surfaces (on front and back), all give greater clarity right to the edge of the visual field, regardless of the patient's prescription. To ensure performance, these designs offer 'fit' versions where dispensers take the parameters of pantoscopic tilt, wrap angle and back vertex distance.

This is 'customisation' and ensures the patient achieves their expected vision in their chosen frame. In other words, these lenses optimise vision, matching the power to the way the parameters have altered the position of the lenses in front of the patient's eyes. Customisation is necessary for the patient's vision - it doesn't enhance it. Plus, it allows patients a broad choice of frames while achieving their correct refraction with minimal frame adjustment.

Breaking down barriers

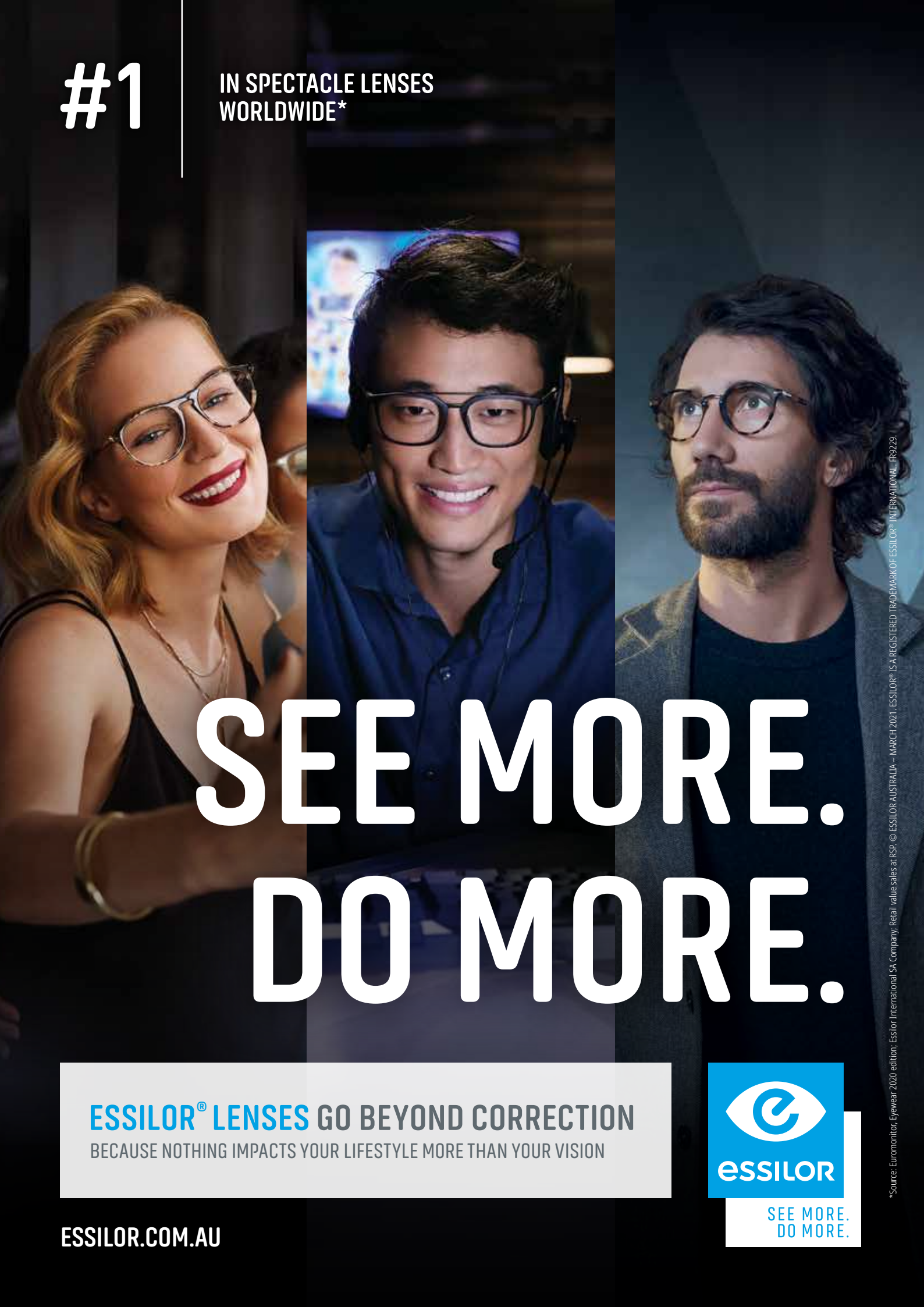
Digital dispensing units were first available around 2004 and had become common enough by 2010 that Dr Wolfgang Wesemann did a comprehensive review of the available models in the February 2010 edition of The Optician Online.[1] These devices helped break down the barriers to systematically using the patient's frame 'fit' parameters and gave easier access to the initial personalised product. The first personalised progressive lens also appeared in 2004. A small unit was used to measure the patient's ratio of head and eye movement, this was then applied to the lens design.

In stepping up to personalised lenses, it is important to note the difference between 'customised' and 'personalised.' A personalised lens design uses a physiological visual characteristic, unique to each wearer, to create a specific lens design for that individual. Some current examples across manufacturers are:

- Real time 3D modelling of the centre of rotation of the patient's eyes to calculate the patient's visual axis
- Use of a pseudo reading task to measure the patient's relative downgaze, working distance, lateral offset and visual behaviour (Figures 1 and 2)
- Sensitivity tolerance testing for astigmatism and power error
- 3D modelling to calculate the axial length of the patient's eyes

Some of these concepts are applied only to progressive lenses while others can also be used for anti-fatigue, extended focus and even single vision.

By assessing a unique visual characteristic, personalisation provides the patient with a lens with greater ease of use and/or visual facility, leading to enhanced performance. Today's consumer, whether we are talking health care or simply retail purchasing, is looking for unique experiences so lens personalisation must result in a 'visual experience' they won't otherwise achieve.


#1
IN SPECTACLE LENSES
WORLDWIDE*
SEE MORE.
DO MORE.
ESSILOR® LENSES GO BEYOND CORRECTION
BECAUSE NOTHING IMPACTS YOUR LIFESTYLE MORE THAN YOUR VISION
ESSILOR.COM.AU
essilor
SEE MORE.
DO MORE.
*Source: Euromonitor, Eyewear 2020 edition; Essilor International SA Company; Retail value sales at RSP. © ESSILOR AUSTRALIA – MARCH 2021. ESSILOR® IS A REGISTERED TRADEMARK OF ESSILOR® INTERNATIONAL. FR9229.

Case study

Px:
Female, 70-years-old has been wearing glasses since her early teens and has been a patient at the same practice since 1990.

Rx:
R -6.00/-3.00 x 178 1.50 base in, 1.75 base up
L -4.00/-1.25 x 2 1.50 base in, 1.75 base down
Add +2.50

VA R&L:
Corrected 6/6

The previous lens type worn by the patient since 2018 (Varilux X), was the same brand but the basic design of the new product (Varilux Xclusive) she is now wearing. The key issue for this patient was a complex Rx and the fact that adjusting to new glasses has always been a difficult and lengthy process, so much so it became an expectation for both herself and the dispenser.

The standard process for this patient would normally involve a week of perseverance to adjust to a new Rx and frame. However, in this case, the product provided was a progressive lens personalised by centre of rotation of the eye and near vision behaviour.

This time, she put her glasses on and was impressed that they felt comfortable instantly. The normal adjustment period that the patient, optometrist and dispenser had come to expect was almost non-existent. Since delivery, she has brought her sister in to get the same brand personalised progressive lenses and both the patient and her sister have ordered an additional pair each.

Personalisation really has no point unless the patient perceives a benefit. In a 136-person study in France:[2]

- 89 per cent of wearers said after wearing their lenses that the visual benefits provided by the modelling of the centre of rotation of the eye were important.
- 93 per cent believed that it made very clear the connection between their visual experience and the product concept

To avoid any bias in the study, the researchers ensured that the subjects were given no information about the product concept until after they had worn the lenses and reported their experience.

Conclusion

While it is common for optometrists to expect some adjustment period for a new Rx or new lens type, for the patient this can be an uncomfortable and difficult process. By using a personalised progressive lens the experience of this long term patient was dramatically altered.

Meeting unmet needs

Just as access to therapeutic drugs has greatly increased the scope of practice for optometrists, the advances in lenses have provided answers to a range of visual needs that were previously unmet. Personalising lenses pushes us to look more closely at the individual physiologic optics of our patients, in doing so, it allows practitioners to answer crucial questions that respond to the patient's expectation that you are their local vision expert:

'Why am I prescribing this for you?'

This is where you relay back to the patient, all their reported visual issues, their answer to your questions plus your observations, along with your measured outcomes and what all of this means for their vision.

'How is it going to resolve a visual issue for you?'

Knowledge and know-how now takes over to link the optical features to solving the patient's vision issue and, finally,

'What are going to be the benefits for your vision?'

Answering these questions, (as with all the visual conditions, medical or optical, that you assess), links your understanding of the patient's vision to your expertise and understanding of how to get the best outcome for them. From this relationship patient loyalty and retention follows.

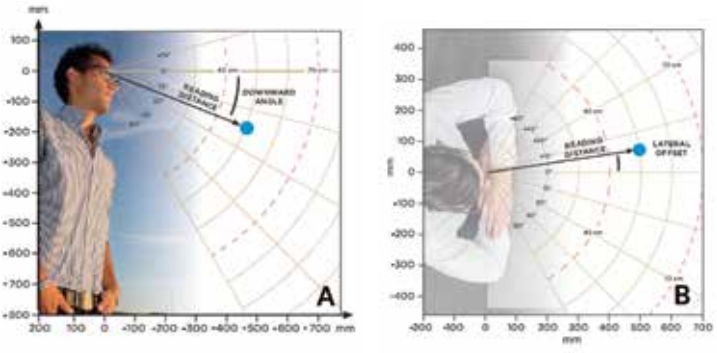


Figure 1.
Factors related to the patient's posture. A: Reading distance and angle of downgaze. B: Lateral Offset

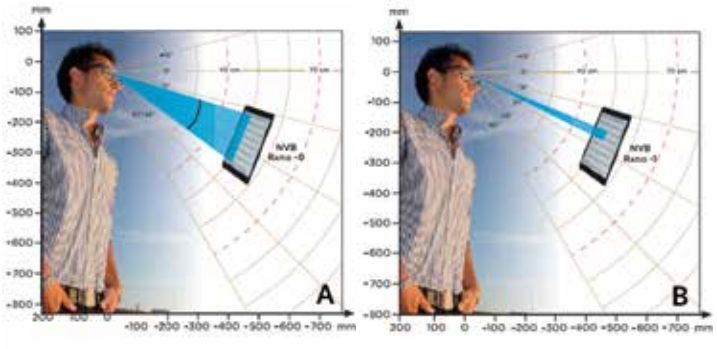


Figure 2.
Factors related to the patient's behaviour. A: Patient with a strong tendency to scan with their eyes. B: Patient whose eyes remain still and they scan with their head

1. Comparison of PD Measuring Devices Part 1 & 2; Dr Wolfgang Wesemann; opticianonline.net; 12 February 2010.

2. Essilor R & D single centre randomised wearer test n = 136, with in-practice measurement and assessment

Looking to *open your own* independent practice or *lift the performance* of your existing business?

Introducing Launch*pro*

Launch*pro* is a complete ***8 step guide*** to opening your own practice or reinvigorating it with ***confidence, support*** and ***success.***

Access your complimentary **100 point checklist** and learn more at ***optom.provision.com.au/launchpro***

ProVision

You support your patients. We support you.

Get in touch with Kate Hall khall@provision.com.au 0448 073 250

Prescription of compounded ophthalmic medications

A pharmacy perspective

Lynn Weekes
BPharm MSc PhD

Iqbal Ramzan
DipPharm MSc PhD

At least once a year, *Clinical and Experimental Optometry* publishes a 'special issue' on a topic of significance to optometrists and researchers, alike. The 2021 special issue is dedicated to 'ocular therapeutics' and brims with cutting-edge research on this topic, dear to the hearts of many practising clinicians.

Although the choice of which article to focus on for this report was difficult, the article by Lynn Weekes and Iqbal Ramzan provided a novel angle that should be at the forefront of the prescribing clinician's mind, and that is the 'Prescription of compounding ophthalmic medications: a pharmacy perspective.' While most medications that optometrists prescribe are commercially available (in fact, only one to two per cent of all prescriptions require compounding), every now and then there is a need to request that the ophthalmic preparation be compounded. This process could present something of a conundrum to the prescribing optometrist, especially if it is not commonly encountered – however, as the authors report, the compounding pharmacist is best positioned to assist with the process.

Compounding refers to the preparation, mixing, assembling, packaging and labelling of a medicinal product based on a prescription order from a licensed practitioner for the individual patient. All pharmacists are legally able to do this for simple prescriptions. There has been an increase in the number of pharmacies specialising in compounding over the last 20 years or so, with 12 such pharmacies in 2002 to approximately 500-600 at present. These pharmacies include the specialised aseptic facilities with appropriate equipment including air handling systems.

Optometrists may request for an ophthalmic preparation to be compounded in the case where the patient has an allergy or sensitivity to an ingredient in the formulation other than the active ingredient; when the dose commercially-available is not suitable to obtain the desired effect; when the commercially-available product is out of stock or unavailable in Australia; or when the product is not very stable and must be prepared fresh each time.

From the pharmacist's perspective, when a prescription for an ophthalmic preparation is received, a systematic process is undertaken to ensure that the prescription is dispensed safely and accurately and that the patient is well-informed. The pharmacist may access the patient's electronic health records and medications to rule out any allergies or potential interactions. The pharmacist will also enquire about previous use of the formulation and the patient's ability to administer the ophthalmic preparation. The pharmacist will also review the prescription and check the known safety and efficacy of the active ingredient, the dose, frequency, route of administration and the duration of treatment. The pharmacist will also check the authenticity of the prescription, as well as verify the patient and prescriber details. In the case of any concerns arising from this systematic process, the pharmacist may contact the prescriber for clarification.

In preparing the formulation, the compounding pharmacist will include 'excipients,' or, non-active components, as well as the active ingredient.* Excipients include ingredients which preserve the product, maintain an acceptable pH, or are necessary for the stability of the formulation. Some eye drops will be prescribed specifically as preservative-free because patients can develop a sensitivity to commonly-used preservatives like benzalkonium chloride, chlorhexidine acetate or chlorobutanol, especially with repeated use; for example, in dry eye disease. All raw materials must be pharmaceutical grade and they must be produced by acceptable (Good Manufacturing Practice - GMP - certified) manufacturers. A compounded prescription medicine will have an expiry date assigned. For eye drops and ointments the expiry date may be up to 28 days depending on the chemical. For products that do not have published stability data, the expiry date will generally be three days or less.

Good communication between the prescriber and the pharmacist is key to an efficient and safe process. It is the duty of care of the pharmacist to provide information and advice to prescribers, patients and care givers. Pharmacists have up-to-date information on factors affecting the prescription of compounded ophthalmic prescriptions, such as information on out-of-stock or discontinued products, safety alerts about active ingredients or excipients, as well as suspected adverse drug reactions which are reported to the Therapeutics Goods Administration in Australia.

Prescribing optometrists would benefit from having a conversation with their local compounding pharmacist to understand their processes and in order to work together when prescribing compounded ophthalmic products to patients. **

* From 1 Feb 2021, prescribers must include the name of the active ingredient on all PBS prescriptions, except for handwritten prescriptions, paper based medication charts in residential aged-care sector and medicinal items with four or more active ingredients.

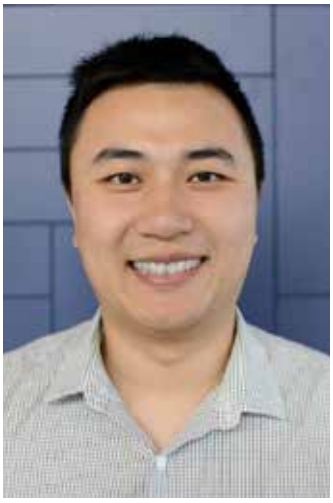
** Optometry Australia has compiled a list of ophthalmic compounding pharmacists, available in the 'Clinical resources' section of the website. If you know a certified ophthalmic compounding pharmacist, please email us at publications@optometry.org.au and we will ensure they are added to future published lists.

Interview with CXO Associate Editors, A/Prof Isabelle Jalbert and Dr Alex Hui

Guest Editors of 2021 CXO Special Issue 'Ocular Therapeutics'

A/Prof Isabelle Jalbert is an award-winning teacher and Associate Professor at UNSW where she teaches therapeutic management of anterior eye diseases. She leads a program of research and education focused on improving the delivery of evidence-based eye care.

Dr Alex Hui is a Senior Lecturer at UNSW where he teaches ocular therapeutics and ocular diseases, and conducts research in the areas of contact lenses, myopia control and drug delivery.



You recently were Guest Editors on the 2021 CXO Special Issue on ocular therapeutics. Which paper or papers would be most beneficial for practising optometrists?

JALBERT: Hart et al.'s update on Optometry Australia's infection control guidelines. I think it should be a go-to resource for practising optometrists both in Australia and elsewhere, considering the current pandemic conditions. The rest of the papers are all very clinically-relevant and useful as resources for the practitioner. The papers by Phu et al. and MacIver et al. discussing open-angle glaucoma management by primary care practitioners and glaucoma therapeutic medications could be seen as complementary in their use for practitioners looking to manage glaucoma, while Ho et al. and Arshad et al. provide timely updates to microbial keratitis and herpetic eye disease management.

Which original research paper has you the most interested to see where the research goes next?

JALBERT: The discussion of sublingual and subcutaneous immunotherapy for ocular allergy by Trivedi et al. is an excellent starting point for eye-care practitioners looking to become further educated in understanding the possibilities of allergy management for their patients, and should provide the necessary background to consider all allergy treatment options. Sandford et al. and Dang et al. also discuss emerging concepts in the management of dry eye and adenovirus, which may continue to become more relevant with time as options for their management continue to increase.

This issue celebrates optometry's role in ocular therapeutics. From your perspective, how has this changed the way optometry is practised around Australia?

HUI: As we discussed in our editorial for the special issue, the main thing is that therapeutics has become part of what the public will expect to receive as part of standard optometric care. The role of optometrists to be the primary eye-care providers to members of the public has thus been enhanced.

What do you think optometry's role in ocular therapeutics will look like in the next decade?

HUI: Oral therapeutics for eye care is likely something on the horizon in the future. Optometrists in New Zealand have already shown the ability of optometrists to prescribe oral medications for the benefits of their patients and this is likely the next step for their colleagues in Australia.

As keynote speakers in Optometry Virtually Connected (18-20 June), Associate Professor Maria Markoulli, Associate Professor Isabelle Jalbert and Dr Alex Hui will delve further into CXO's special issue on ocular therapeutics, highlighting the most clinically-relevant findings and translating them into practical clinical advice.

Simi Sarin
BMedSc(VisSc), MOpt OcTher
OptoMed Norwood, Adelaide

A rare retinal finding

Clinical approach for recognising and managing retinal arteriovenous malformations.

Retinal arteriovenous malformations (AVMs) are rare, congenital vascular anomalies characterised by direct and abnormal blood flow between an artery and vein without an intervening capillary network.[1] The exact aetiology of AVMs is unknown but is believed to involve abnormal embryogenic development of the vascular mesoderm.[2] Females are more commonly affected than males, and in both cases the diagnosis is predominantly made before the age of 30.[2] Three main variants of AVMs have been described and these are distinguished by their differing severity and associated ocular manifestations (Table 1).[1]

AVMs may be an incidental finding on routine examination where they pose no threat to vision, although severe cases have been associated with ocular complications such as central retinal vein occlusion, vitreous haemorrhage, and retinal haemorrhage.[3] They may also be associated with systemic AVMs in the midbrain, mandible, or orbit as part of the Wyburn-Mason syndrome.[3] This case discusses an unusual AVM of the peripheral retina presenting as an incidental finding in a relatively healthy individual.

Case report

A 64-year-old Caucasian female presented for the first time at the practice for her annual eye exam. She did not report any visual concern, however, did note recent dizzy spells when lying in bed. There were no relevant issues identified in ocular, medical, or family history. General health review was unremarkable.

On general inspection she appeared well, and best corrected visual acuity was 6/6 in both eyes. Pupils were normal, motilities were full and smooth and there was no visible proptosis. Anterior eye examination was unremarkable. Fundus examination revealed an unusual vascular lesion in the inferotemporal left retina (Figures 1 and 2).

After dilated fundus examination, a cluster of aneurysmal retinal vessels surrounding an arteriovenous anastomosis was visible. There were exudates surrounding the lesion from the decompensated retinal vessels. An epiretinal membrane was also present in the same eye. No other retinal abnormalities were noted in either eye.

The patient was referred to a retinal specialist for fluorescein angiography and appropriate neuroimaging given the recent dizzy spells. A diagnosis of a type 2 retinal AVM was made. Neuroimaging of the head and orbit did not show any associated pathology. Given the lack of systemic AVMs, yearly observation was recommended for the patient.

Discussion

Retinal AVMs were first described by Magnus in 1874.[4] Since then, reports of retinal AVMs have been frequent and quite varied in presentation, ranging from localised lesions to lesions across the entire retina.[5] Localised AVMs have a predisposition for the superotemporal retina (41 per cent), papillomacular bundle (34 per cent) and nasal retina, in the perpipapillary region (four per cent).[5] The inferotemporal location of the AVM presented in this case seems to be a rare occurrence.[5]

Archer et al.[1] classified congenital AVMs based on their severity and relative prognosis (Table 1).

Both type 1 and 2 follow a stable and benign clinical course, and are rarely associated with systemic complications. Type 3 AVMs tend to involve the entire retina. These patients are at a higher risk of ipsilateral AVMs in the cerebrum and face as part of a

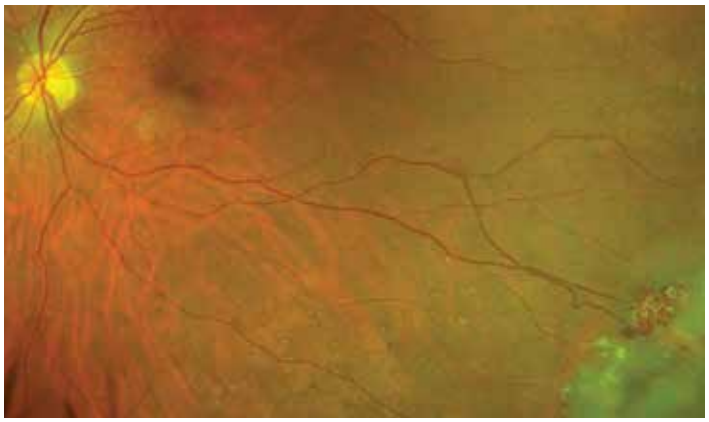


Figure 1.
Ultra-wide imaging of the vascular anomaly

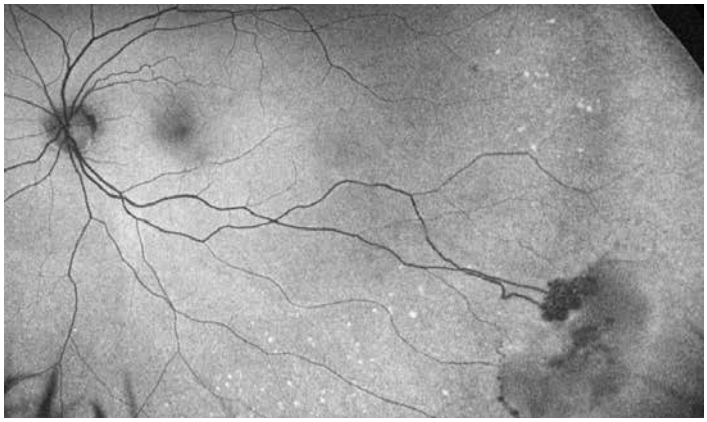


Figure 2.
Fundus autofluorescence imaging of the vascular anomaly

condition known as Wyburn-Mason syndrome.[6]

Wyburn-Mason syndrome is a rare disorder involving AVMs extending from the retina to the brain.[7] Other possible AVM locations include the skin, bones, kidney, and gastrointestinal tract.[7] The AVMs in Wyburn-Mason syndrome have minimal neoplastic potential; however, they can haemorrhage and result in significant morbidity.[7] The incidence of intracranial AVMs in patients with type 3 retinal AVMs is approximately 30 per cent.[7] However, changes in retinal AVMs do not hold any predictive value in the formation or progression of intracranial AVMs.[7]

Retinal AVMs can remain stable for years, slowly regress, or progress to affect other areas of the retina.[8] They are generally diagnosed as incidental findings or as the cause of visual impairment in low vision patients.[2] Severe cases can be associated with symptoms such as flashes, floaters, or sudden vision loss secondary to retinal complications.[2] These complications can include retinal or vitreous haemorrhages, retinal vein occlusion, and rarely neovascular glaucoma.[3]

Retinal complications secondary to AVMs are proposed to involve retinal ischemia stemming from three possible mechanisms. AVMs can result in increased venous flow and decreased retinal perfusion, causing the nearby retina/choroid to become ischemic from a 'steal phenomenon.'[8] In addition, abnormal vascular connections can increase hydrostatic venous pressure and damage vessel walls ultimately leading to thrombus formation.[8] Lastly, direct venous compression can cause an occlusive event.[8]

An optometrist may be the first to diagnose a retinal AVM or Wyburn-Mason syndrome. As per the classification system, not all patients with these lesions harbour cerebrovascular consequences. In severe forms of AVMs, extensive imaging including a CT scan, MRI, and carotid/vertebral angiography is recommended due to the increased risk of systemic complications.[9] Nonetheless, if a patient with a retinal AVM presents with suspicious symptoms (such as dizzy spells in this case), then neuro-radiological imaging is advised - regardless of the type of AVM.[9]

Asymptomatic AVMs do not require any treatment and routine observation should suffice.[9] In cases with associated retinal complications, management can include intravitreal anti-vascular endothelial growth factor (VEGF) agents, periocular steroids or photocoagulation.[9] Severe fundus lesions, as in Wyburn-Mason syndrome, unfortunately do not have any appropriate treatment due to the extent of retina involved.[10]

This case represents a rare presentation of an isolated type 2 retinal AVM. It is important for optometrists to be aware of the varying presentations of this disorder in order to appropriately manage their patients.

Table 1.
Variants of Retinal Arteriovenous Malformations[1]

Type	Characteristics	Retinal Signs	Visual Prognosis	Associations
I	Intervening abnormal capillary or arteriolar plexus between communicating artery and vein	- Involves one sector/quadrant of the retina - Appear as small complexes of dilating retinal vessels - Rarely decompensate nor cause structural abnormalities - Not associated with other retinal abnormalities	- Good - Visual acuity unaffected - No treatment required and can be observed	- Ocular or systemic associations are unusual
II	Direct arteriovenous communication between a branch artery or vein without an intervening capillary plexus	- End-to-end junctions or multiple anastomosis with intermediate (100-200 µm) or large (200-300 µm) channels - Afferent blood vessels may show beading with multiple fusiform dilations and occasional aneurysm formation - Capillary network may be dilated with small microaneurysms - Can lead to breakdown of blood retinal barrier causing leakage, retinal oedema, intraretinal exudates or haemorrhages	- Generally good but visual acuity can be affected if there is macular involvement - Tend to remain stable over time with rare instance of regression and remodeling - Ocular or systemic associations are unusual. No treatment required but photocoagulation can be used to reduce afferent artery calibre	- Usually not associated with intracranial vascular malformation - Carotid or cerebral angiography not necessary unless adequate clinical suspicion exists
III	Arteriovenous connections that are complex and extensive causing severe retinal complications and vision loss	- Cannot separate the arterial/venous components in these AVMs - Anastomoses involves large (500-600 µm) channels involving most of the fundus - Leads to widespread exudation, retinal oedema, and retinal cystic degeneration.	- Poor - Involves loss of vision at an early age - Requires neurosurgical therapy for cerebral complications	- Intracranial involvement very likely - Associated with Wyburn-Mason syndrome

1. Archer DB, Deutman A, Ernest JT et al. Arteriovenous communications of the retina. *Am J Ophthalmol* 1973; 75: 224-241.

2. Laey De J.J., Hanssens M. Vascular Tumors and Malformations of the Ocular Fundus, 1st ed. Ghent, Belgium: Springer Publishers, 1990.

3. Mansour AM, Wells CG, Jampol LM et al. Ocular Complications of Arteriovenous Communications of the Retina. *Arch Ophthalmol* 1989; 107: 232–236.

4. Reck SD, Zacks DN, Eibschitz-Tsimhoni M. Retinal and intracranial arteriovenous malformations: Wyburn-Mason syndrome. *J Neuroophthalmol* 2005; 25: 205-208.

5. Liu W, Sharma S. Ophthaproblem. Retinal arteriovenous malformations (type 2). *Can Fam Physician* 2005; 51: 203–209.

6. Stepanov A, Studnicka J, Dusová J et al. Vitreous hemorrhage due to a retinal arteriovenous malformation – case report. *Wulfenia* 2016; 23: 271-278.

7. Dayani PN, Sadun AA. A case report of Wyburn-Mason syndrome and review of the literature. *Neuroradiology* 2007; 49: 445-456.

8. Accou G, Nerinckx F, Leroy B et al. Vitreous Hemorrhage as Presenting Sign of Retinal Arteriovenous Malformation. *Case Rep Ophthalmol Med* 2020; 4: 1-4.

9. Knutsson KA, De Benedetto U, Querques G et al. Primitive retinal vascular abnormalities: tumors and telangiectasias. *Ophthalmologica* 2012; 228: 67-77.

10. Heimann H, Damato B. Congenital vascular malformations of the retina and choroid. *Eye (Lond)* 2010; 24: 459-467.

Dr Justin Sherwin
BMedSc(Hons) MBBS(Hons) MPhil MChir PhD FRANZCO
Ophthalmologist, Vision Eye Institute

Neovascular AMD:

How optometrists can help shape the patient journey

Over the coming decades, increased population growth and longer life expectancy will see a national rise in the number of patients affected by age-related macular degeneration (AMD). In Australia, this debilitating condition is the most common cause of vision loss in older adults and affects all ethnic backgrounds. The major non-modifiable risk factors for AMD include older age and family history, while smoking is the major modifiable risk factor.[1]

Evidence of AMD is present in around one in seven Australians over the age of 50 years.[2] Most cases are the atrophic subtype, for which no proven pharmacological treatment currently exists. In contrast, safe and efficacious therapies (anti-vascular endothelial growth factor [anti-VEGF] agents) are routinely used to maintain long-term vision in most patients affected by neovascular AMD (nvAMD). Even though treatment is beneficial, it is important that it is started as soon as possible. It is during the earliest stages that neovascular lesions may be the most active and hence the most amenable to intervention with anti-VEGF therapy.

Delayed treatment initiation is one of the most critical parameters impacting the final visual outcome in treatment-naïve nvAMD patients.[3] In general, the earlier a new patient with nvAMD is able to receive intravitreal treatment, the better the final visual outcome.[4] As such, early symptom recognition, clinical assessment and initiation of management with anti-VEGF therapy are all crucial to preserve vision.

Optometrists have been reported as being the greatest facilitator in reducing time to diagnosis of nvAMD.[7]

Diagnostic and treatment delays

There are three main stages at which diagnostic and treatment delays for nvAMD patients are typically seen.

First is the period between recognition of visual symptoms by the patient to actual presentation to an eye-care provider – most commonly optometrists and, to a lesser extent, GPs. Sometimes, an ophthalmologist may already be treating the fellow eye with nvAMD or the fellow/same eye with separate ocular pathologies.

Identification of symptom onset in a new presentation of nvAMD is often delayed if the fellow eye retains good visual acuity and lacks symptoms. Detection of nvAMD in the first eye is usually slower than in the second eye and, correspondingly, worse outcomes are associated with first eyes treated.[5] Improved outcomes in the second affected eye are due to both heightened awareness of associated nvAMD symptoms and systematic follow-up examinations.

Secondly, there is the time between referral from the primary eye-care provider to an ophthalmic clinic or private ophthalmologist. This referral is usually by an optometrist but may also originate from GPs. It is recommended that new patients with nvAMD are seen by an ophthalmologist within one week. Criteria for urgent referral include a history of recent acute/subacute vision loss, acute-onset metamorphopsia, central scotoma or any of the following:

- suspected or definite new-onset choroidal neovascular membrane (CNVM)
- sub- or intra-retinal fluid (clinically or on OCT scan)
- macular haemorrhage (with no other obvious cause)

The third stage in the treatment pathway is the time from ophthalmic assessment to the patient receiving their first intravitreal anti-VEGF injection. Many private ophthalmologists are able to administer treatment immediately after the patient's clinical assessment. However, not all ophthalmologists manage nvAMD and may instead refer the patient to another ophthalmologist who does. Unfortunately, accessing public clinics with the ability to manage nvAMD can often take up to several weeks to obtain an appointment (noting that some clinics have already exceeded their capacity).

The importance of primary eye care

Australian researchers have recognised that a combination of factors (patient, clinician and structural) present significant barriers to timely and optimal AMD care.[6] Disease understanding/denial by the patient were most frequently cited by study participants (optometrists and ophthalmologists) as obstacles. Likewise, a recent Danish study found low population health literacy (for example, lacking knowledge about nvAMD) and delayed patient response to associated symptoms were major risk factors for delayed treatment.[7] Patients often have nvAMD for some months before the condition is diagnosed, during which time they can lose significant vision.[8]

Key challenges are to educate the public about AMD (both subtypes as atrophic can become neovascular), symptoms, risk factors and the importance of regular eye checks. Optometrists have been reported as being the greatest facilitator in reducing time to diagnosis of nvAMD.[7] A British study found optometrists detected signs of AMD in over 50 per cent of cases, while only around a third of patients first noticed symptoms themselves.[9] The same study noted an overlap in symptoms reported by patients with neovascular and atrophic AMD and that it was often only when their activities of daily living were affected that they sought help (with their optometrist the usual first point of contact).

As primary eye care providers, optometrists have frequent contact with the public and (often) the ability to perform OCTs, making them uniquely placed to detect suspected cases of nvAMD.

Facilitating early diagnosis

Increasing health literacy and therefore recognition of macular-specific symptoms by patients is achieved by increasing their health knowledge. Strategies to address this include use of public campaigns via brochures, posters and radio/television/online (examples are available from the Macular Disease Foundation Australia). This information is often readily available at optometry clinics and should be specifically promoted to at-risk patient groups. Shared knowledge through social networks is similarly valuable.

Additionally, system improvements can focus on ensuring regular ocular check-ups in vulnerable age-groups (for example, those over 50 years), increased information sharing by eye-care professionals and facilitating sufficient ophthalmic capacity with the provision of short waiting periods for emergencies such as new-onset nvAMD.

If a patient has possible symptoms of nvAMD (for example, acuity loss, central scotoma, metamorphopsia), semi-urgent optometric assessment should be undertaken. Optometry Australia recently published guidelines to assist practitioners in this regard, while a referral pathway for AMD management is also available from the Royal Australian and New Zealand College of Ophthalmologists.[10,11]

Recommended clinical examination steps include measurement of visual acuity, fundus examination, Amsler Grid, photostress test and assessment of contrast sensitivity. Look for CNVM, which may appear as a well-demarcated grey/green area of the retina, macular fluid (sub- or intra-retinal), lipid or haemorrhage, and pigment epithelial detachment. Figures 1–3 demonstrate CNVM and associated sub-retinal haemorrhage in a patient with nvAMD. Figure 4 is from the same patient following anti-VEGF treatment.

Ideally, auxiliary testing should comprise colour fundus photography and, importantly, OCT.

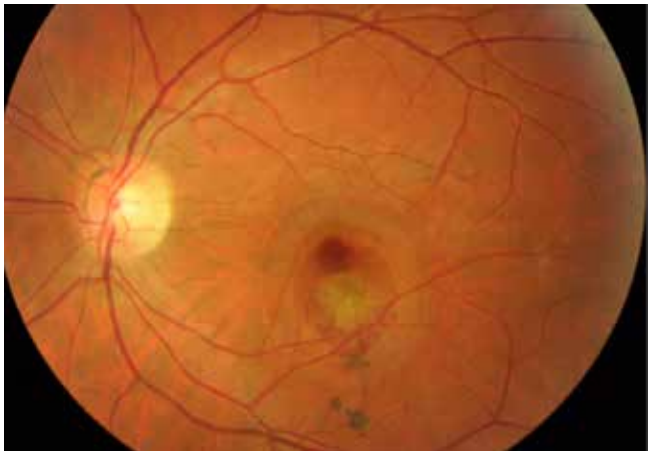


Figure 1.
Colour photograph of sub-retinal haemorrhage due to choroidal neovascular membrane (CNVM)

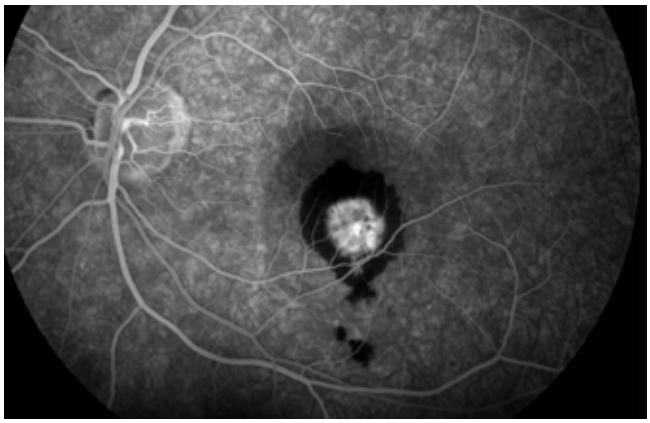


Figure 2.
Late-phase fluoroscein angiogram image of same patient

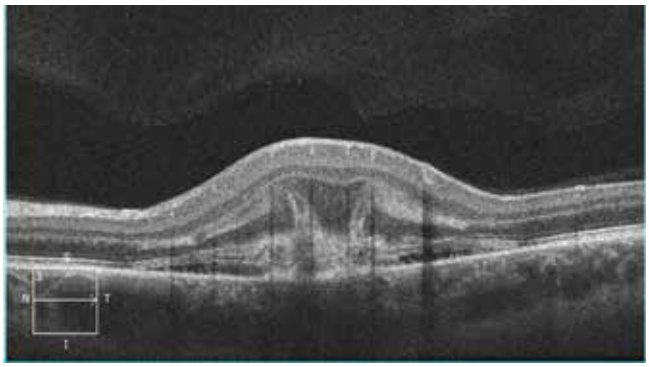


Figure 3.
OCT scan of same patient showing CNVM with associated sub-retinal fluid

The role of OCT

OCT was introduced at a similar time to anti-VEGF therapy and, together, they have revolutionised the diagnosis and treatment of nvAMD. When anti-VEGF treatment was first introduced in Australia, fluorescein angiography (FA) was required to access its use through the pharmaceutical benefits scheme (PBS). More recently, this has been expanded to allow ophthalmologists to make an OCT-supported nvAMD diagnosis, instead of FA.

Use of OCT in optometry practice has increased exponentially in recent years and is pivotal in detecting changes of nvAMD that may have otherwise gone undetected on clinical examination alone. Although this increased sensitivity for detecting macular pathology may give rise to increased false positives, timely ophthalmologist referral will ensure that true nvAMD is accurately diagnosed with treatment commencing soon thereafter (ideally on the same day as the initial ophthalmologist appointment).

Ongoing patient management

Once nvAMD treatment commences, the referring optometrist continues to have an important role in the patient's ongoing management. They are encouraged to take a lead in counselling patients about smoking cessation, dietary modification and supplements (where appropriate). Additionally, they can reinforce the importance of attending eye-care appointments and maintaining treatment with anti-VEGF injections. Optometrists can manage refractive errors as necessary and also monitor the patient for other potential ocular pathologies.

Given the inevitable future increase in nvAMD cases, a robust multidisciplinary approach to patient care is warranted – with optometrists at the forefront now more than ever.

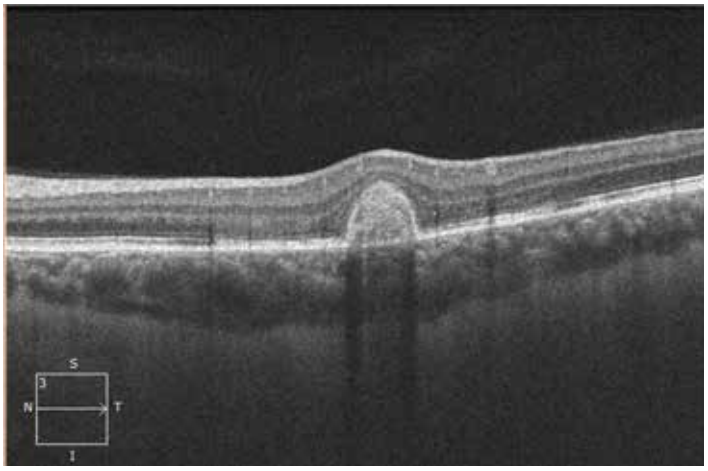


Figure 4.
OCT image following treatment with anti-VEGF therapy with resolution of sub-retinal fluid and reduction in size of CNVM

OCT is pivotal in detecting nvAMD changes that may have otherwise gone undetected on clinical examination alone.

About the author

Dr Justin Sherwin is an ophthalmic surgeon, with special interests in cataract surgery, medical retinal conditions (including performing intravitreal injections) and glaucoma management. Dr Sherwin practises at Vision Eye Institute Camberwell and Footscray and also has a public appointment at the Royal Victorian Eye and Ear Hospital.

1. Chakravarthy U, Wong TY, Fletcher A et al. Clinical risk factors for age-related macular degeneration: a systematic review and meta-analysis. *BMC Ophthalmol* 2010; 10: 31.

2. Keel S, Xie J, Foreman J et al. Prevalence of Age-Related Macular Degeneration in Australia: The Australian National Eye Health Survey. *JAMA Ophthalmol* 2017; 135(11):1242–1249.

3. Arias L, Armadá F, Donate J et al. Delay in treating age-related macular degeneration in Spain is associated with progressive vision loss. *Eye (Lond)* 2009; 23: 326–333.

4. Lim JH, Wickremasinghe SS, Xie J et al. Delay to treatment and visual outcomes in patients treated with anti-vascular endothelial growth factor for age-related macular degeneration. *Am J Ophthalmol* 2012; 153: 678–686.

5. Bressler NM. Early detection and treatment of neovascular age-related macular degeneration. *J Am Board Fam Pract* 2002; 15: 142–152.

6. Jalbert I, Rahardjo D, Yashadhana A et al. A qualitative exploration of Australian eyecare professional perspectives on Age-Related Macular Degeneration (AMD) care. *PLoS One* 2020; 15: e0228858.

7. Wahl AM, Musaeus KD, Sørensen TL et al. Reasons for late diagnosis of neovascular age-related macular degeneration: a mixed-methods study. *Acta Ophthalmol* 2020. doi: 10.1111/aos.14568. Epub ahead of print.

8. Ho AC, Albini TA, Brown DM et al. The Potential Importance of Detection of Neovascular Age-Related Macular Degeneration When Visual Acuity Is Relatively Good. *JAMA Ophthalmol* 2017; 135: 268–273.

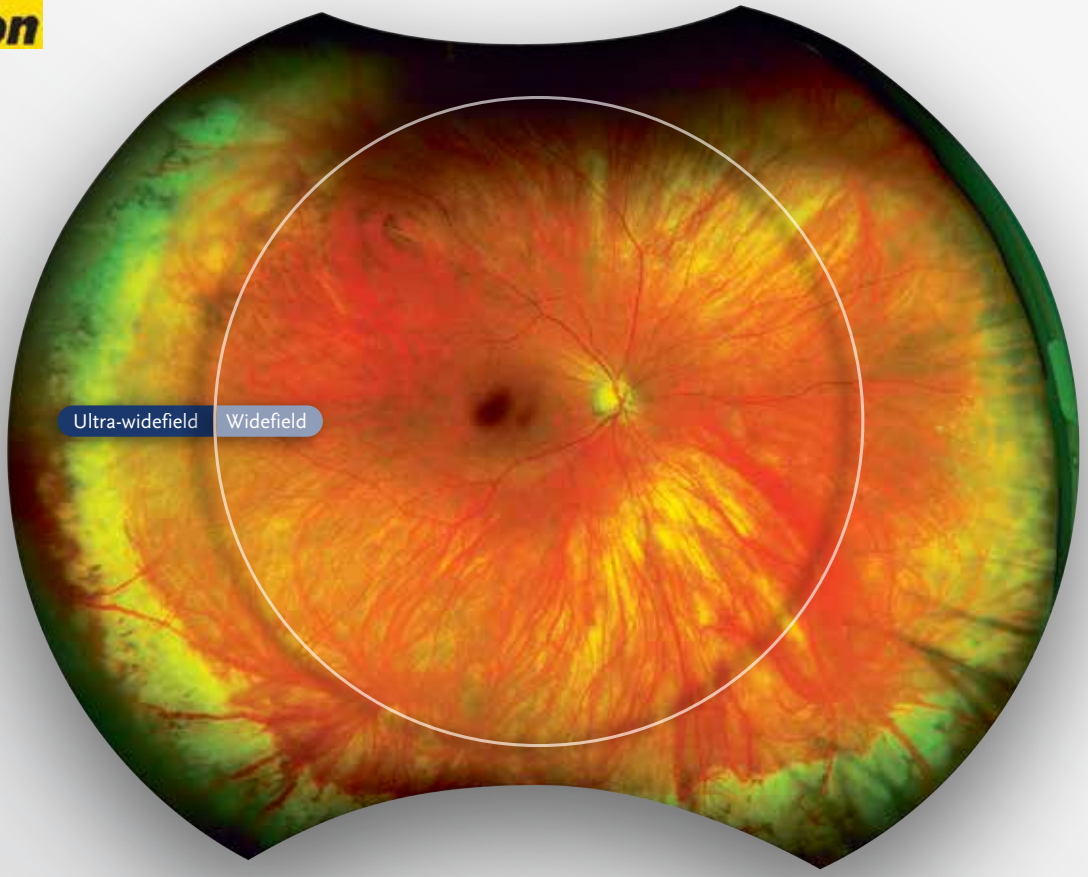
9. Parfitt A, Boxell E, Amoaku WM et al. Patient-reported reasons for delay in diagnosis of age-related macular degeneration: a national survey. *BMJ Open Ophthalmol* 2019 30; 4: e000276.

10. Hart KM, Abbott C, Ly A et al. Optometry Australia's chairside reference for the diagnosis and management of age-related macular degeneration. *Clin Exp Optom* 2020; 103: 254-264.

11. The Royal Australian and New Zealand College of Ophthalmologists. RANZCO: Referral Pathway for AMD Management. Surry Hills, New South Wales: The Royal Australian and New Zealand College of Ophthalmologists; 2020 [cited 5 February 2021]. Available from: https://ranzco.edu/wp-content/uploads/2020/01/080120-RANZCO-Referral-pathway-for-AMD-management-revised.pdf.

Nikon

optos®



See more, treat more.

Optos' latest ultra-widefield retinal imaging technology promotes Technological Innovation, Clinical Outcomes and Practice Efficiency.

- The ONLY single-capture ultra-widefield image takes less than ½ a second.
- Enhance diagnostic capabilities, practice efficiency and patient experience.
- Better diagnose pathology and preventable eye disease.
- Differentiate your practice and increase revenues.

Made even easier before the end of the financial year with the Federal Government's immediate deduction offer* and deferred payment options.** *Conditions Apply. Visit www.ato.gov.au for details. ** Dependant on finance provider.

Image: Cobblestone Degeneration - Daytona Plus

T: (08) 8444 6500
E: auinfo@optos.com

Optos.com

Plaquenil

Rheumatology and optometry/ophthalmology working together

Dr Rob Howie (Optometrist)
BSc BSc (Optom) GCOT GAICD
OPSM Dianella, WA

Plaquenil (Hydroxychloroquine (HCQ) sulphate) is an effective treatment for skin and inflammatory disorders such as lupus, rheumatoid arthritis and Sjögren's syndrome with relatively few side effects compared to alternative medications.[1] It also has a developing role in oncology.[2,3] Plaquenil is a chemical modification of chloroquine. Compared to Plaquenil, chloroquine has a similar but more marked retinal toxicity profile.[1] Plaquenil reduces immune-system-related inflammation and helps to control skin rashes, sores and joint pain.[4] However, it may cause potentially debilitating visual loss in the form of Bull's eye maculopathy. Except for cessation of Plaquenil use, Bull's eye maculopathy is untreatable. Critically, even after Plaquenil use is stopped, Bull's eye maculopathy may progress from minor vision loss to severe loss of visual function.[5] Bull's eye maculopathy can occur after some-to-many years of using Plaquenil depending on dose (as a function of body weight), time taken and other factors such as impaired renal/liver function, tamoxifen use and pre-existing macula conditions.[5]

Bull's eye maculopathy begins as a para-central (2 to 6 degrees from the fixation point and classic Bull's eye)[6] or peri-central (greater than 8 degrees from the fixation point)[6] loss of visual function characterised by damage to the outer retinal layers and retinal pigment epithelium.[2] In most cases losses are para-central but in some cases, especially in Asian races, the losses can be peri-central.[2,6] The visible signs consist of subtle retinal pigment changes near the maculae, extending to the region of the arcades in peri-central loss. Auto-fluorescent retinal imagery changes consist of hypo and hyperfluorescent changes, with spectral-domain OCT (SD-OCT) image changes presenting as damage and thinning of the outer retinal layers, both located in areas consistent with the likely visual field losses (Figures 1-3).[2]

Optometry and ophthalmology's role in preventing Bull's eye maculopathy is to alert rheumatology when there is any reasonable suspicion that Bull's eye maculopathy may possibly be developing.[1] The words 'reasonable' and 'possibly' are used because once the development of Bull's eye maculopathy is certain, the potentially insidious progression of this condition means it may lead to significant functional vision loss.[1] Clinical acumen is used to judge, for example, when a change in visual field is a random variation or a possible early sign of field loss, suggesting validation by more frequent repetitive testing.

Figures 4-5 indicate the risk and prevalence of Bull's eye maculopathy as a function of dose and time. For normal doses (less than 5 mg/kg of body weight) the risk is small within the first five years. The risk increases with dose and time taken.

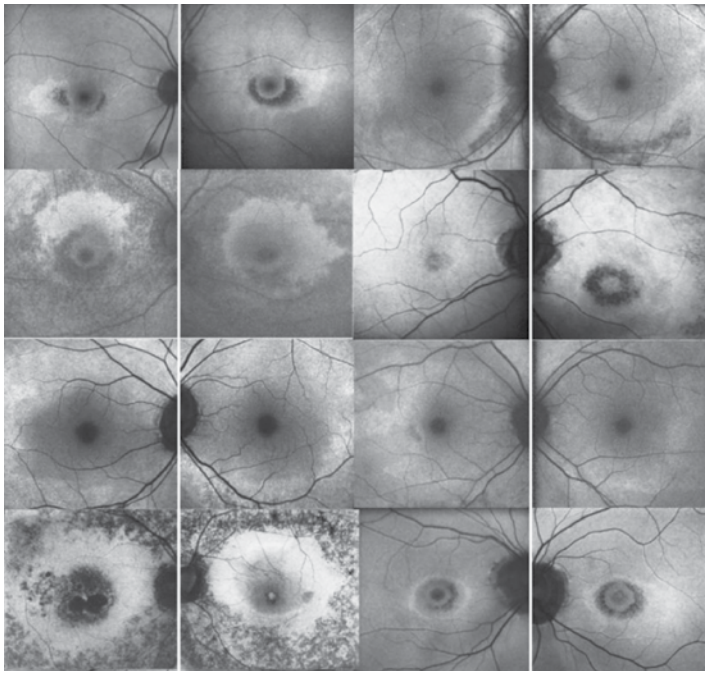


Figure 1.
Various patterns of disease on fundus auto-fluorescence imaging in hydroxychloroquine retinopathy (courtesy of Dr Ronald Melles MD)

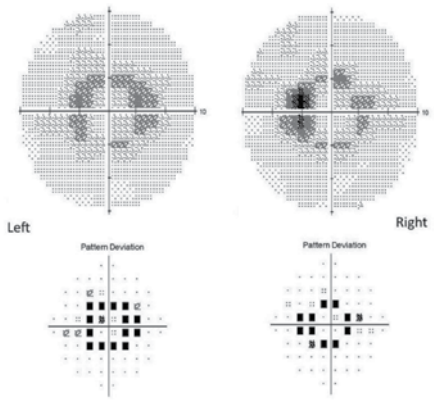


Figure 2.
10-2 Humphrey visual field deficits in hydroxychloroquine retinopathy (courtesy of Dr Ronald Melles MD)

EXCELLENCE IN OPHTHALMIC CARE

- **Comprehensive eye care covering all subspecialties**
- **Urgent and emergency care**
- **State-of-the-art technology and facilities**
- **Dedicated research unit**
- **27 locations** (NSW, QLD, VIC & SA)

Continuity of Care

Throughout the COVID-19 pandemic, Vision Eye Institute has continued to provide necessary eye care services for our patients. Now, with restrictions easing, we are phasing in the return of routine consultations and surgeries. Our commitment to the health and wellbeing of our patients, staff and doctors remains our top priority and we will continue to observe strict infection control protocols and social distancing measures at all times.

DO YOU SHARE OUR VISION?

Then we invite you to join our educational network

At Vision Eye Institute, we're passionate about sharing our knowledge with other healthcare professionals to ensure all Australians receive best-practice ophthalmic care. That's why we created **SHARED VISION** – a network that supports collaboration with our optometric and GP colleagues.

Membership is FREE and includes:

- **24/7 access to our educational materials**
- **A member-only newsletter**
- **A variety of resources for your practice**
- **Earlybird invitations to CPD events, webinars and our annual conferences (now online!)**

For more information, please visit our website
visioneyeinstitute.com.au

vision eye institute | SHARED VISION

VEI29220 | WELLMARK PTY LTD | 09-20

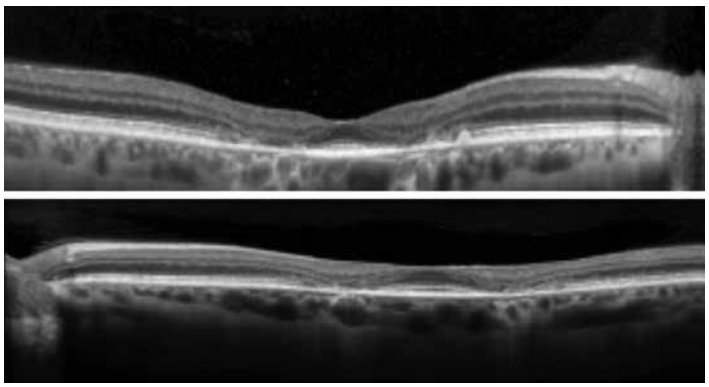


Figure 3.
Parafoveal outer retinal layer disruption in Plaquenil toxicity (courtesy of Ms Pauline Xu BOptom (Hons) MOptom GradCert OcTher)

Baseline tests should be performed within the first year of treatment and screenings at yearly intervals after five years of treatment. If there is an increased risk of toxicity more frequent screenings are indicated.[1,2]

Rheumatologists requests that a full report is provided when optometrists screen patients for Bull's eye maculopathy. The report should include visual field data and the SD-OCT assessment of retinal thickness and retinal layer integrity. This is to provide a baseline or subsequent reference point so change can be assessed at a later date, which may be several decades in the future. Since clients can change location and practitioner over the years, a copy of the report should be sent to the client. Rheumatologists (and/or other medical practitioners) may be unfamiliar with interpreting the results, so an interpretation of the findings should also be included.

The screening report should include an automated central visual field assessment such as the Humphry Visual Field 30-2 test (the 10-2 test is adequate in most cases but may fail with losses of a peri-central nature) or the Medmont central visual field test (with paracentral points added) and an SD-OCT assessment of posterior pole retinal thickness and retinal layer integrity.[2]

In cases where there is suspicion of Bull's eye maculopathy, referral to ophthalmology for multifocal electroretinography (mf-ERG) assessment of macular function is recommended.[2]

As optometrists, what do we do?

- ➔ Assess risk
- ➔ Perform central visual fields and SD-OCT
- ➔ Refer to ophthalmology for mf-ERG if Plaquenil toxicity is suspected
- ➔ Interpret and report to rheumatologist, client and GP
- ➔ Recommend time to reassessment

Assessing the risk

Plaquenil retinal toxicity is more common than previously thought.[1,7] At the recommended doses[3] of Plaquenil, the risk of toxicity is under one per cent up to five years of treatment and under two per cent up to 10 years of treatment. It increases to nearly 20 per cent after 20 years of treatment. After 20 years, the chance of developing toxicity in the next year is four per cent (Figures 4-5).[7]

The risk of Bull's eye maculopathy increases if patients take more than 5 mg/kg/day, also take tamoxifen and/or have compromised renal function or macular disease. Communication with the patient's general practitioner (GP) is required to establish renal function. Patients may inadvertently choose to take more than the prescribed dose and this should be checked for.

Dosage calculations are based on real weight and not ideal weight.[7]

While risk relates to a population, Bull's eye maculopathy affects individuals and this requires use of clinical judgement to decide when the patient may be developing retinal toxicity earlier than expected.

Central visual fields and SD-OCT

Central visual fields

Visual field losses may be para-central, peri-central or both. A study found that genetically European patients are most likely to develop para-central losses (2-6 degrees from the fixation point)

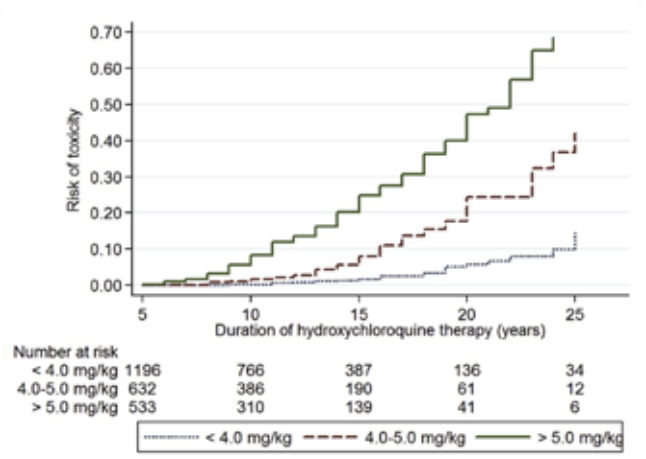


Figure 4.
Risk of hydroxychloroquinine retinopathy by daily dose and duration of drug use (courtesy of Dr Ronald Melles MD)

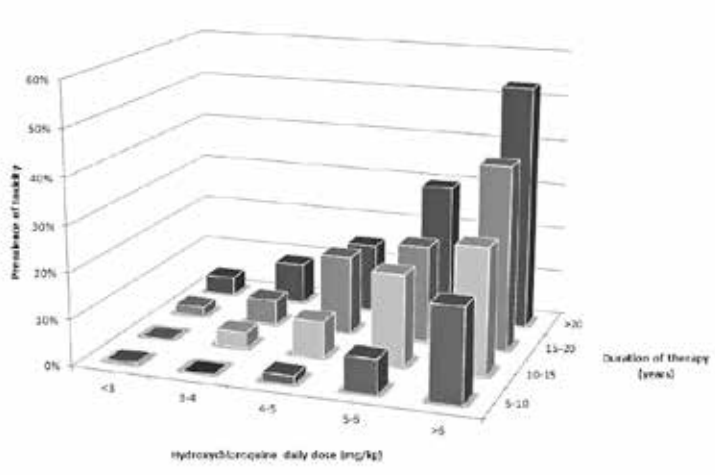


Figure 5.
Prevalence of hydroxychloroquinine retinopathy by daily dose and duration of drug use (courtesy of Dr Ronald Melles MD)

although two per cent developed peri-central losses (more than 8 degrees from the fixation point) only. In the same study, Asian patients also demonstrated para-central losses but a peri-central loss only was found in 50 per cent of cases.

Asian patients require wider field assessment than Caucasian patients with the 30-2 or 24-2 Humphrey visual field tests recommended. Caucasian patients may only need the 10-2 Humphrey test.[1,7] However, a combination of Humphrey 30-2 and 10-2 or similar field tests will cover the low risk of Caucasian patients initially developing peri-central losses and the lesser risk of Asian patients developing para-central losses.

As visual field losses can take some years to develop and can appear initially as a random reduced sensitivity, clinicians need to determine when more frequent testing is required to validate a loss as true.

SD-OCT

SD-OCT is an imaging technique used to capture high resolution two- and three-dimensional images of the retina. Plaquenil retinal toxicity typically results in outer segment abnormalities with focal alterations, especially thinning, of the photoreceptor layer, outer nuclear layer, ellipsoid zone and pigment epithelium. The losses are typically para-foveal but may be peri-macula especially in Asian eyes.[6] Widefield imaging is required in Asian eyes.[1]

Other imaging

Fundus photographs provide an indicator of macular disease but are not an early indicator of Plaquenil toxicity.[5] Fundus photographs using a dilated pupil can be recommended for baseline to documentation to identify pre-existing pathology.[2] Auto-fluorescence fundus imaging detects Plaquenil retinopathy but is less sensitive than mf-ERG,[8] and is also very useful as a baseline.

Referral for Mf-ERG

Mf-ERG assesses retinal sensitivity topographically and can identify reduced retinal responses in early Plaquenil retinopathy. Mf-ERG has been considered the most sensitive test for Plaquenil retinopathy.[8] (Table 1.) Mf-ERG testing can be accessed by referring to ophthalmology or designated tertiary optometry centres.

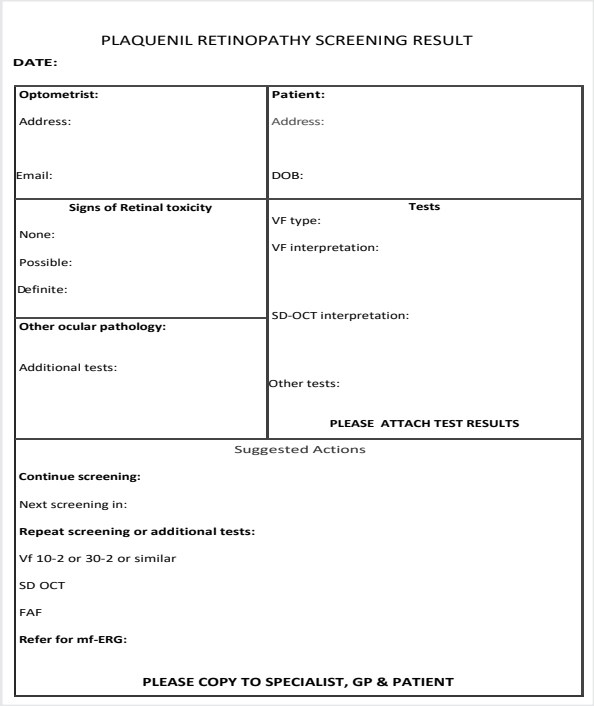
Inform

Figure 6 depicts the Plaquenil retinopathy screening result form which I strongly suggest using, or something similar, to ensure all of the required information is captured. (Also available online in the 'ocular toxicity' drop-down menu on the Optometry Australia 'Clinical areas of interest' page).

The use of Plaquenil is increasing since it is an effective medication with few systemic side effects.[1] Appropriate screening, effective communication with referring specialists, GPs and patients will enable early detection of Plaquenil retinal toxicity and minimise functional vision losses.

Screening test	Sensitivity	Specificity
10-2 VF	85.7%	92.5%
SD-OCT	78.6%	86.9%
Multifocal ERG	92.9%	98.1%
SD-OCT and 10-2 VF	85.7%	92.5%
10-2 VF and mf-ERG	100%	82.2%
SD-OCT and mf-ERG	100%	86.0%

Table 1.
A comparison of screening test efficacy. Sensitivity and specificity of diagnostic tests in HCQ retinopathy[1]


PLAQUENIL RETINOPATHY SCREENING RESULT

DATE:

Optometrist:
Address:
Email:

Patient:
Address:
DOB:

Signs of Retinal toxicity
None:
Possible:
Definite:

Tests
VF type:
VF interpretation:
SD-OCT interpretation:
Other tests:

Other ocular pathology:
Additional tests:

PLEASE ATTACH TEST RESULTS

Suggested Actions

Continue screening:
Next screening in:

Repeat screening or additional tests:
Vf 10-2 or 30-2 or similar
SD OCT
FAF

Refer for mf-ERG:

PLEASE COPY TO SPECIALIST, GP & PATIENT

Figure 6.
The Plaquenil retinopathy screening result form

1. Yusuf IH, Sharma S, Luqmani R, et al. Hydroxychloroquine retinopathy. *Eye (Lond).* 2017; 31 (6): 828–845.

2. The Royal College of Ophthalmologists. Hydroxychloroquine and Chloroquine Retinopathy: NEW screening recommendations February 2018 [Internet]. London: The Royal College of Ophthalmologists; 2018 [cited 2020 Dec 21]. Available from: https://www.rcophth.ac.uk/2018/03/rcophth-guideline-hydroxychloroquine-and-chloroquine-retinopathy-new-screening-recommendations-february-2018/

3. Apotex Pty . Ltd. APO-Hydroxychloroquine [Internet]. NPS MedicineWise. [cited 2021 Jan 10]. Available from: https://www.nps.org.au/medicine-finder/apo-hydroxychloroquine-tablets

4. American Academy of Ophthalmology. What Is Plaquenil? [Internet]. San Francisco: American Academy of Ophthalmology; 2020 [cited 2021 Jan 10]. Available from: https://www.aao.org/eye-health/drugs/what-is-plaquenil

5. American Academy of Ophthalmology. Recommendations on Screening for Chloroquine and Hydroxychloroquine Retinopathy - 2016 [Internet]. San Francisco: American Academy of Ophthalmology; 2016 [cited 2020 Dec 21]. Available from: https://www.aao.org/clinical-statement/revised-recommendations-on-screening-chloroquine-h

6. Melles RB, Marmor MF. Pericentral Retinopathy and Racial Differences in Hydroxychloroquine Toxicity. *Ophthalmology.* 2015; 122: 110–116.

7. Melles RB, Marmor MF. The Risk of Toxic Retinopathy in Patients on Long-term Hydroxychloroquine Therapy. *JAMA Ophthalmol.* 2014; 132:1453–1460.

8. Kellner U, Renner AB, Tillack H. Fundus Autofluorescence and mfERG for Early Detection of Retinal Alterations in Patients Using Chloroquine/Hydroxychloroquine. *Invest Ophthalmol Vis Sci.* 2006; 47: 3531–3538.

When to stop myopia control treatments

Cassandra Haines
B Vis Sci M Optom
Policy and Advocacy Advisor
Optometry Australia

2020 was a year of challenges for the entire globe, but as our attention was rightfully captivated by the COVID-19 pandemic, the ongoing myopia pandemic continued to surge, with rates of progression escalating for children across the world.[1]

Once considered a condition only afflicting the academically-minded young adult in their early twenties, myopia is now truly a disease of childhood as well, with prevalence rates escalating and age of initial onset dropping. It is therefore no surprise that the majority of the landmark myopia studies (BLINK, ATOM, COMET) on how to treat and control the progression of myopia have been focused on younger age groups. It is well established that the younger the initiation of short-sightedness, the worse the outcomes for both visual and pathological risk factors.[2]

Despite these studies, there remains a gap in the literature: when do we consider our younger patients past the 'danger zone'? There remains a significant challenge facing practitioners in providing evidence-based advice to older teenagers and young adults about their myopia control options, and when they can safely stop treatment.

Myopia is not just a disease of paediatric patients, but young adults as well

In a paper produced by the authors of the COMET study, 'The Myopia stabilisation and associated factors among participants of the Correction of Myopia Evaluation Trial,' it was found that 48 per cent of the participants had myopia stabilisation at 15 years of age, 77 per cent at 18 and 90 per cent by age 21.[3] While it is reassuring that half of our young patients may no longer be at risk of progression by 16, it also highlights that 50 per cent still will need myopia management and intervention, and almost a quarter will need it at 18.

A large retrospective study by Bullimore et al.[4] on single vision contact lens wearers aged 20-40 found that 21 per cent of participants progressed by -1.00 D across five years. This was disproportionately skewed to the younger groups; almost half of the 20-25-year-olds progressed -0.75 D, compared to only 25 per cent of those aged 35-40. A later, more-controlled review, including cycloplegia and based at just one practice location, followed Finnish children until adulthood and found similar progression results of an average -0.45D +/- 0.71D across their 20s.[5]

It is also worth considering the individual. Family history of high myopia is one of the strongest predictive factors for onset and progression of short sightedness.[6] McMonnies in 2015[7] discussed a list of possible personal risks for continued myopia progression, including level of parental education and time spent on near-work and electronics. However, there is limited clinical evidence to support these as a basis for treatment protocol.[7]

While this limited data does not provide absolutes and doesn't account for the small number still progressing, it suggests most of our patients in their 20s will have only small refractive jumps which may not warrant intensive intervention. From these studies, we can assume only 20 per cent will experience change of more than -1.00 D, and in many patients that may be in small changes every few years. While every dioptre matters, when progression is less than 0.50 D per year, myopia control interventions may not be warranted.

Current advice and recommendations

Considering that the majority of myopia control evidence is for children, if your patient is out of this age category and you are considering initiating, continuing or ceasing myopia control treatment, it is crucial that you discuss the lack of evidence for older teens and adults. Theoretically, the effectiveness of dual focus and multifocal contact lenses, orthokeratology (orthoK) and atropine should translate across to young adults, but patients need to know that the shortage of age-dedicated studies means it is difficult to make fully-informed choices.

The 'Discontinuation of orthokeratology on eyeball elongation' (DOEE) study looked at transitioning patients out of orthoK treatment after two years of wearing the lenses.[8] They found that in children younger than 14, there is a risk of having a 'rebound effect' where the axial length growth quickly accelerates after cessation of lens wear. The authors concluded that lens wear should ideally continue beyond age 14. However short 'vacations' for illness or holidays can be done safely. Children should be closely monitored for at least six months after discontinuing the use of orthoK lenses.

Klaver et al.[9] used the European growth charts developed by Tideman et al. to develop a policy on atropine cessation.[9] In their Dutch myopia-control practice, children are only removed from atropine treatment if they are at least 15 years of age, and and if their axial length is progressing by no more than 0.10 mm per year.

The three-year randomised clinical trial of MiSight dual focus soft contact lenses by Chamberlain et al.[10] included children up to the age of 13 at the commencement of the study, some of whom were 15 at the conclusion of the trial period.[10] Forty-three per cent of children who completed the study were between 10–12 at baseline, ageing to 13 and above by the end of the three years. While the younger children in control lenses did progress faster, there were similar growth results

between the younger and older age groups in the MiSight lenses, suggesting that while there may theoretically be better control in higher-risk younger children, the lenses still have an evidence base for being effective in slowing myopic growth in children up to the ages of 15.

Things to consider when you do cease treatment

Abrupt cessation of atropine, especially in higher concentrations, is more likely to cause a rebound effect.[11,12] The rebound effect has not been studied as a specific focus, however the ATOM 2 study (with a focus on children aged 6–12) did show rapid refractive growth in the first 12 months after cessation), so it is important to monitor these patients closely.[12] The World Health Organization (WHO) recommends a taper, but does not specify the amount, duration or steps.[13] It then seems sensible to either taper your patients in a similar fashion to the way one would with steroids, or by stepping the concentration down. Patients re-initiated on atropine have been demonstrated to return to effective control,[12] so you can resume therapy if required.

We are now learning that there may be a dose-dependent response to atropine eye drops and their effectiveness in myopia control.[14] However, this also means that as stronger concentrations of atropine are prescribed, there are higher rates of side effects. 0.05% can cause an average accommodative amplitude loss of approximately 2 D which shouldn't affect even particularly studious young adults, but 0.10% causes a marked 10.01 D reduction in amplitude.[12, 14]

While the near demands of an eight-year-old may not be extensive, it is very different for a student completing their final year of high school or in university. Compromised near performance may require a near addition, or a cessation of atropine therapy. Carefully conduct binocular vision assessments on review (as you should do anyway for your student patients) to ensure comfortable, easy learning vision, and prescribe (or withdraw treatment) accordingly.

Myopia control soft contact lenses provide good distance visual acuity, and are effective in controlling myopia progression,[10] but they do decrease other aspects of visual performance.[15,16] While this hasn't been specifically studied, this may be less tolerable in an adolescent or young adult population. Also consider that while young children may have lower rates of contact lens infections than adults, the risk increases in teenagers to levels similar to that in adults.[17] Discuss risks of lens wear and the importance of hygiene with all patients, and the risk-benefit considerations of treatment.

There seems to be limited evidence that continued myopia progression, or resuming axial length growth is correlated with intensive near-work, despite the seemingly clear anecdotal evidence. This could be due to self-reporting of near-work time in most studies compared to objective measures. However when other factors are considered, such as the amount of data consumed, myopic children have been found to have higher rates of smartphone data usage,[18] suggesting we don't yet understand the full picture.

The COMET study found no difference in self-reported near-work rates between the still progressing cohort at 15 and the stabilised group.[3] Despite this, further analysis of the data showed that every extra hour of near-work per week was associated with a two per cent decrease in the chance of stabilisation at 15.[19] Understanding this, and the multiple other benefits of using screens and doing near-work in moderation, practitioners should be confident in reminding patients about visual hygiene and breaks from screen time.

The research indicates that we should monitor young adults well into their twenties. While we have no longitudinal data on typical axial length progression in adult myopia, we know that axial length measurements give an indicator of disease risk. As part of the picture in assessing associated myopic complications, optometrists should tailor the frequency of fundus examination through dilated pupils accordingly.

Reassure young patients that 50 per cent of myopic progressors have stopped changing by 15 years of age, and that seems to be when the discussion of treatment cessation can begin to reasonably occur.

While we are managing a new way of living and a dependence on technology in 2021, we are also facing a new challenge in myopia: a potential for younger onset and progression in our young adult patients. Ultimately, in myopia control there is no easy one-size-fits-all approach; tailor your treatment to each individual's lifestyle and personal risk factors.

Acknowledgements

The author extends acknowledgement and thanks to Myopia Profile for authorship on parts of this source material as previously published on www.myopiaprofile.com

1. Wang J, Li Y, Musch DCet al. Progression of Myopia in School-Aged Children After COVID-19 Home Confinement. *JAMA Ophthalmol* 2021 doi: 10.1001/jamaophthalmol.2020.6239.

2. Hu Y, Ding X, Guo X et al. Association of Age at Myopia Onset With Risk of High Myopia in Adulthood in a 12-Year Follow-up of a Chinese Cohort. *JAMA Ophthalmol* 2020; 138: 1129-1134.

3. Myopia stabilization and associated factors among participants in the Correction of Myopia Evaluation Trial (COMET). *Invest Ophthalmol Vis Sci* 2013; 54 : 7871-7884.

4. Bullimore MA, Jones LA, Moeschberger ML et al. A Retrospective Study of Myopia Progression in Adult Contact Lens Wearers. *Invest Ophthalmol Vis Sci* 2002; 43: 2110-2113.

5. Pärssinen O, Kauppinen M, Viljanen A. The progression of myopia from its onset at age 8–12 to adulthood and the influence of heredity and external factors on myopic progression. A 23-year follow-up study. *Acta Ophthalmol* 2014; 92: 730-739.

6. Liang C-L, Yen E, Su J-Y et al. Impact of Family History of High Myopia on Level and Onset of Myopia. *Invest Ophthalmol Vis Sci* 2004; 45: 3446-3452.

7. McMonnies CW. Clinical prediction of the need for interventions for the control of myopia. *Clin Exp Optom* 2015; 98: 518-526.

8. Cho P, Cheung SW. Discontinuation of orthokeratology on eyeball elongation (DOEE). *Cont Lens Anterior Eye* 2017; 40: 82-87.

9. Klaver C, Polling JR, Group EMR. Myopia management in the Netherlands. *Ophthal Physl Opt* 2020; 40: 230-240.

10. Chamberlain P, Peixoto-de-Matos SC, Logan NS et al. A 3-year Randomized Clinical Trial of MiSight Lenses for Myopia Control. *Optom Vis Sci* 2019; 96: 556-567.

11. Tong L, Huang XL, Koh ALT et al. Atropine for the Treatment of Childhood Myopia: Effect on Myopia Progression after Cessation of Atropine. *Ophthalmol* 2009; 116: 572-579.

12. Chia A, Lu QS, Tan D. (ATOM-2) Five-Year Clinical Trial on Atropine for the Treatment of Myopia 2: Myopia Control with Atropine 0.01% Eyedrops. *Ophthalmol* 2016; 123: 391-399.

13. WHO. The Impact of Myopia and High Myopia. Sydney, Australia: World Health Organization with the University of New South Wales; 2015.

14. Yam JC, Jiang Y, Tang SM et al. (LAMP) Low-Concentration Atropine for Myopia Progression Study: A Randomized, Double-Blinded, Placebo-Controlled Trial of 0.05%, 0.025%, and 0.01% Atropine Eye Drops in Myopia Control. *Ophthalmol* 2019; 126: 113-124.

15. Kollbaum PS, Jansen ME, Tan J et al. Vision performance with a contact lens designed to slow myopia progression. *Optom Vis Sci* 2013; 90: 205-214.

16. Sha J, Tilia D, Diec J et al. Visual performance of myopia control soft contact lenses in non-presbyopic myopes. *Clin Optom (Auckl)* 2018; 10: 75-86.

17. Bullimore MA. The Safety of Soft Contact Lenses in Children. *Optom Vis Sci* 2017; 94: 638-646.

18. McCrann S, Loughman J, Butler JS et al. Smartphone use as a possible risk factor for myopia. *Clin Exp Optom* 2021; 104: 35-41

19. Scheiman M, Zhang Q, Gwiazda J, Hyman L, Harb E, Weissberg E, et al. Visual activity and its association with myopia stabilisation. *Ophthalmic Physiol Opt* 2014; 34: 353-361.

Dr Sarah White
PhD
Director of Quit, Cancer Council Victoria
Smoking cessation advisor, Cancer Council Australia

When smoke gets in the eyes

Smoking cessation and optometrists

Cancers, respiratory and cardiovascular diseases are the key contributors to the staggering burden of preventable death and disease caused by tobacco use in Australia. However, highlighting health impacts that can greatly diminish quality of life – like loss of vision – can be a powerful tool to help people to stop smoking.

Increasing motivation for a quit attempt

Most people who smoke want to quit, and this includes people from populations with high smoking rates, such as people with socio-economic disadvantage, Aboriginal and Torres Strait Islander people,[1] people living with mental illness[2] and people who use alcohol and other drugs.[3] However, people who smoke often lack the motivation to 'get on with' making a quit attempt. We all know that change is hard; combine 'change' with an addiction, and it is very easy to understand why quit attempts often get put off to 'one day.'

New knowledge about the health impacts of smoking is enough to motivate some people to prioritise quitting, particularly if they have or know someone who has had a lived experience with a particular health issue.

The proportion of people aware that smoking increases the risk of poor eye health is relatively low, despite a specific graphic health warning on Australian cigarette packs that 'Smoking causes blindness.' A representative survey of smokers in Victoria, conducted in 2018 by the Cancer Council, found that 37.5 per cent of people who smoke had unprompted recall of smoking causing emphysema. However, only 5.2 per cent of people who smoke had unprompted recall of smoking causing 'eye problems' and 1.1 per cent had unprompted recall of smoking causing 'blindness.' Information on personal risk and how smoking causes 'eye problems' (obstruction and failure of blood supply to the macula caused by nicotine,[10] and damage caused by chemicals in the smoke) is almost certain to be new knowledge to the patient.

Every clinical interaction focused on eye health, though, is a golden opportunity for a very specific teachable moment that could motivate someone to quit. The saliency of being advised about one's personal risk of vision loss due to smoking – by an eye health professional when sitting in an optometry chair – is likely to be high. Critically, specific personalised information is more likely to be acted upon when it is delivered – not by a generic television advertisement or brochure laden with stock photographs – but by a trusted health professional who can also offer advice on how to quit.

Smoking and poor eye health

It is well established that smoking increases the risk of ocular disease and poor eye health. People who smoke cigarettes (and, it increasingly appears, use e-cigarettes) are more likely to develop age-related macular degeneration, cataracts, glaucoma, Graves' ophthalmopathy, dry eye disease and contact lens issues (infection and inflammation) compared to people who don't smoke.[4] Smoking is linked to increased risk of retinal blood clots[5] and uveitis, which tends to be more severe and recur more quickly in people who smoke.[6] People with diabetes who smoke are more likely to develop diabetic retinopathy[7-8] and women who smoke during pregnancy are more likely to have children with strabismus and hyperopia.[9] People exposed to second-hand smoke can experience symptoms including stinging and watery eyes and eye redness.

While smoking causes gradual and permanent damage, at least some of the risks associated with smoking can be reduced. The earlier people quit, the better for their eyes and overall health and wellbeing. It is never 'too late' to quit.

Advice for smoking cessation

Tobacco dependence is a clinical issue and, like any other chronic disease or drug dependency, it must be considered a vital component of holistic clinical care. People who smoke expect clinicians to address their smoking, and studies have shown that supportive, non-judgemental advice can, in the general practice setting at least, enhance the perceived quality of the care received. Critically, clinician advice with an offer of support increases the likelihood that a person who smokes will successfully quit.[11]

The 'Ask, Advise, Help' (AAH) brief advice model (Figure 1) is a streamlined way to structure a conversation about smoking and connect people who smoke to best-practice tobacco dependence treatment (TDT). Best-practice TDT is a combination of multi-session behavioural intervention (provided by Quitline in Australia) and pharmacotherapy (nicotine replacement therapy or stop smoking medications).[12]

In simple terms, pharmacotherapy helps alleviate some of the nicotine withdrawal symptoms (irritability, headaches, coughs, fatigue and constipation are common) while the behavioural intervention helps the client identify triggers and habits for smoking, develop practical strategies to manage triggers, habits and cravings, and shift the client's self-identity from smoker to non-smoker.

It's important to note that evidence-based behavioural intervention for smoking cessation involves a minimum of three sessions that employ tailored psycho-education, motivational interviewing and a range of counselling approaches, such as cognitive behavioural therapy and acceptance, and commitment therapy. Eye-health professionals are encouraged to refer their clients to the Quitline for behavioural intervention (and information on using pharmacotherapies), not to attempt to provide behavioural interventions themselves.

Figure 1.
The 'Ask, Advise, Help' (AAH) brief advice model

What does 'Ask, Advise, Help' for the optometrist look like in practice? It can be as simple as this:

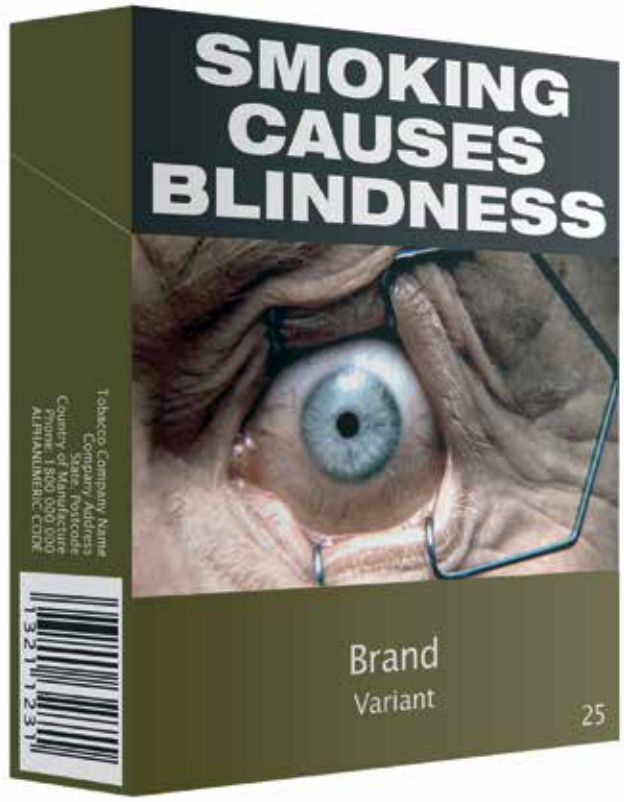
- **ASK:** 'Can I ask if you smoke?' ('Yes')
- **ADVISE:** Both salient risk and how to quit: 'OK, so I need to let you know that smoking increases your risk of some serious eye diseases (could be worsening your dry eyes/will make your cataracts grow faster/is a particular risk factor given you have diabetes etc.) and I really recommend you have a go at quitting. Combining stop smoking medications from your GP with coaching from the Quitline really increases your chances of quitting successfully.'
- **HELP:** 'Would you like me to get Quitline to give you a call to chat about this?'

In this conversation, 'Advise' and 'Help' are combined into one single 'speech' that takes just a couple of minutes to deliver. The key point, again, is to provide advice and encouragement: a) to quit because of a specific clinical risk (to prompt a quit attempt) and b) the recommended approach to quitting (to the use of evidence-based TDT and thus success).

Basing the advice on a specific clinical risk and offering some help to start the quitting journey ensures the client won't see the advice as being critical of their 'lifestyle choice' (an oxymoron when one is discussing an addiction!) to smoke.

Optometrists should advise their patients to visit their GP for appropriate pharmacotherapy (particularly as nicotine could play a role in the onset and/or progression of eye disease).[20] However, we strongly recommend optometrists actively refer clients to Quitline. Referrals to Quitline can be made via fax or by online form in most states (see www.quit.org.au/articles/about-quitline-13-7848/ for a list of state and territory resource pages). Research has shown that an active referral, rather than a recommendation to call, makes it more likely the patient will go on to both use the service and to make a quit attempt.

As trusted health professionals, optometrists can play a powerful role in both increasing awareness about the risks of poor eye health associated with smoking and helping their clients quit.



Incorporating fast, simple and effective brief advice into routine optometry practice could help thousands of Australians improve their eye health and also avoid the significant risks of early death and disease caused by smoking.

Optometry Australia's Institute of Excellence is hosting a CPD Module devised by Quit Victoria entitled 'Smoking cessation: brief advice essentials training for eye health professionals.' Log on today to find out more about how to provide fast, simple and effective brief advice to a smoker in a supportive manner. **https://lms.optometry.org.au/course/view.php?id=223**

1. Nicholson AK, Borland R, Davey ME et al. Predictors of wanting to quit in a national sample of Aboriginal and Torres Strait Islander smokers. *Med J Aust* 2015; 202: S26-S32.

2. Stockings E, Bowman J, McElwaine K et al. Readiness to quit smoking and quit attempts among Australian mental health inpatients. *Nicotine Tob Res* 2013; 15: 942-949.

3. Thurgood SL, McNeill A, Clark-Carter D et al. A Systematic Review of Smoking Cessation Interventions for Adults in Substance Abuse Treatment or Recovery. *Nicotine Tob Res* 2016: 18: 993-1001.

4. Makrynioti D, Zagoriti Z, Koutsojannis C et al. Ocular conditions and dry eye due to traditional and new forms of smoking: A review. *Cont Lens Anterior Eye* 2020; 43: 277-284.

5. Hayreh SS, Podhajsky PA, Zimmerman MB. Retinal artery occlusion: associated systemic and ophthalmic abnormalities. *Ophthalmology* 2009; 116 :1928-1936.

6. Yuen BG, Tham VM, Browne EN et al. Association between Smoking and Uveitis: Results from the Pacific Ocular Inflammation Study. *Ophthalmology* 2015; 122: 1257-1261.

7. Hammes HP, Kerner W, Hofer S et al. Diabetic retinopathy in type 1 diabetes-a contemporary analysis of 8,784 patients. *Diabetologia* 2011; 54: 1977-1984.

8. Groeneveld Y, Tavenier D, Blom JW et al. Incidence of sight-threatening diabetic retinopathy in people with Type 2 diabetes mellitus and numbers needed to screen: a systematic review. *Diabet Med* 2019; 36: 1199-1208.

9. Fernandes M, Yang X, Li JY et al. Smoking during pregnancy and vision difficulties in children: a systematic review. *Acta Ophthalmologica* 2015; 93: 213-223

10. Cınar E, Yuce B, Zengin MO et al. The Effect of Nicotine on Macular Microcirculation in Healthy Subjects. *Ophthalmic Surg Lasers Imaging Retina* 2019; 50: 691-700.

11. Stead LF, Buitrago D, Preciado N et al. Physician advice for smoking cessation. *Cochrane Database Syst Rev* 2013; 2013: Cd000165.

11. Kotz D, Brown J, West R. Prospective cohort study of the effectiveness of smoking cessation treatments used in the "real world". *Mayo Clin Proc* 2014; 89: 1360-1367.

Optometry AUSTRALIA

Institute of Excellence Quality Assured

Just like your best friend, we're here to support you

With the Optometry Board of Australia's new CPD system now in place, there is a lot you have to do to keep up with these latest requirements.

There are learning plans to complete. Hours to be recorded. Reflections to be written. For a busy optometrist with a full appointment schedule, keeping up with, and keeping track of your CPD obligations can be daunting.

Like any good friend, we are here to support you.

As an Optometry Australia member, you have access to a range of tools and systems that we have built specifically to remove this stress.

This includes access to an online learning plan so that you can nominate those courses you wish to undertake and then, once completed, create your reflection. And you can adapt, update and print your learning plan at any time throughout the year.

As a member, we will automatically upload hours of any CPD course that you complete, and that has been quality assured by Optometry Australia's Institute of Excellence. This means you don't have to track them or record if they are clinical, therapeutic, interactive or non-clinical CPD hours.

You can also participate in a growing range of quality and complimentary CPD programs developed and delivered by Optometry Australia including webcasts, a national virtual conference and CPD publications.

And to ensure that you have access to only the best CPD available, only quality assured programs that have met our exacting standards, can now be promoted via our Institute of Excellence and found in our searchable CPD and events listings.

We can do all this because we represent the largest community of optometrists in Australia and membership of our organisation means you are never alone.

Join our community today. Go to optometry.org.au for more information.

Systane®

DON'T LET DRY EYES HOLD YOU BACK

STAY FOCUSED with the Systane® family of products

2 DROPS, 1 FOCUSED YOU

ALWAYS READ THE LABEL. FOLLOW THE DIRECTIONS FOR USE. IF SYMPTOMS PERSIST, TALK TO YOUR HEALTH PROFESSIONAL.

Alcon