elektron eye technology

Use of MPS II as part of a macular screening programme

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Overview

Age-related macular degeneration (AMD) is the most common cause of visual loss in the aged population globally[1]. The prevalence of AMD varies throughout the world. In the UK the disease accounts for 50% of blind and partial sight registrations. In Asian populations the figures are similar[2] whilst in African and Hispanic American demographics there appears to be a lower risk of AMD than that seen in Caucasians. Further studies are required to look at other populations and the effect of geographical location within the same country (rural to urban) and any changes in diet (traditional to western).

AMD is a complex disease, which is multifactorial and is usually sub-divided into non-exudative (dry) or exudative disease (wet). Dry AMD is the more common condition with an incidence of 80-90 per cent of all new cases[3]. The prevalence of both dry and wet AMD in the UK is 2.4 per cent at age 50 or over, increasing exponentially to 12.2 per cent at age 80 or over[4]. With the changing demographics of the population predicted due to increasing numbers of the elderly, these prevalence figures are expected to rise by a third between 2009 and 2020[2].

Recent data, however, suggests that AMD is affecting people at a far earlier age than previously thought. Examining statistics collated in Germany showed that within a patient group of 4,340 participants, 3.8% of patients aged 35-44 already show signs of AMD[27]. This contradicts previous wisdom on the matter and makes a compelling case with regards to screening examinations for the disease. Screening to identify those at risk of AMD should start at an earlier age, less than the traditional 40 years, if not for all persons over 16. At present there is no treatment for dry AMD and wet AMD cannot develop without the former[5]. If dry AMD is allowed to progress through its various stages, wet AMD will develop with an incidence of between 14-20 per cent over 5 years[6]. Although treatments have been developed for wet AMD (Visudyne®, Macugen® Lucentis®, Avastin® and Eylea®) these at best delay or stop the progression of further visual loss but do not restore sight to pre-AMD levels and are not without their own complications[7]. However the AREDS2 study has now shown that there is finally some good news for patients with dry AMD. By taking supplements to build up their macular pigment (MP), a statistically significant proportion of patients can halt progression of the disease. The details of these studies are outlined below.

Clinicians and community members greatly underestimate the impact of mild, moderate and severe AMD on a person's health-related quality of life[8]. Indeed many patients in this stage of the disease report that communication was poor and depression and anxiety has evolved[9].

It is thus important to (a) identify those in the population at risk; (b) employ a screening programme using evidence based methodology; and (c) manage the patient appropriately. This paper will discuss in detail these three aims and will present some case studies[10] that will reveal that prevention is much better than intervention.

Contents

Risk factors

There have been many risk factors associated with AMD. These can be categorised into low, medium and high risk groups based on the literature – as shown in the table below.

Some of these risk factors are modifiable and some are not. It is interesting that the "blue light hazard" has increased with the number of people now using technology, with emissions of these short wavelengths e.g., VDUs, mobile phones, and electronic tablets. In addition, many patients are being implanted with IOLs, which do not contain a yellow filter to absorb blue light as they are more expensive and can affect sleeping patterns in patients.[17] Combined with an older patient base and the advancement of surgical procedures to allow nearly all patients a choice of cataract surgery, this results in an significant increase of oxidative damage to the retina.

High risk	Medium risk	Low risk
Low macular pigment (MP)[11]	Blue light exposure[14]	Obesity BMI>25[28]
Smoking[12]	Caucasian ethnicity[15]	High cholesterol[29]
Age >50	Clear IOL[17]	Systemic hypertension[30]
Family history in first degree relatives		Excessive Sun exposure[16]
Pre-existing large soft confluent drusen in the same or fellow eye[13]		Vitamin D deficiency[31]

Signs and symptoms

Dry AMD		Wet AMD	
Signs	**Symptoms**	**Signs**	**Symptoms**
Hard drusen	Increased glare	Sub-retinal haemorrhages	Metamorphopsia
Soft drusen	Reduced contrast	Sub-retinal fluid	Scotoma(ta)
Retinal pigment epithelial (RPE) changes	Reduced vision[18]	Disciform scarring	Acquired colour vision defect[18]

Macular pigment

At the posterior of the eye, macular pigment (MP) is distributed centrally and decreases paracentrally, concentrated in the fibers of Henley and outer axon segments of the photoreceptors. It is able to absorb high-energy blue light (400-500nm), with characteristic peak absorption of 460nm and is a powerful antioxidant and free radical scavenger that also reduces chromatic aberration. In essence this is the body's natural blue light retinal filter.

AMD occurs through chronic oxidative damage to the photoreceptors at the retina, caused by long-term chronic and cumulative exposure to shortwave blue light[34,35.]

Using the analogy of glaucoma screening, where nerve fibre loss can occur before field loss, low MP can occur before (and after) drusen formation. It is therefore prudent to enhance MP using a management plan, e.g. diet, lifestyle and supplementation, in order to potentially reduce or even prevent drusen formation, reduce the cascade from hard to soft drusen, and reduce (or eliminate) the symptoms of glare, poor contrast and poor visual acuity. In practice, it is advisable to tell patients at risk that the goal is to achieve "stability of their AMD". In dry AMD the signs and symptoms are usually slow and insidious whereas in wet AMD the signs and symptoms can occur suddenly over a short period and referral to secondary eye-care is required for consideration of medical treatments.

Screening

The retina is particularly susceptible to oxidative stress due to the high levels of in-site reactive oxygen intermediates (ROI) production, which is due to a combination of high metabolic activity and light exposure[32]. Epidemiological studies validate the protective role of antioxidants[33.] Since early retinal signs of AMD are subtle or non-existent, with generally no symptoms, having a fast, reliable method for measuring and monitoring MP - one of the four main risk factors - is vital for early detection of those patients who are most at risk of developing the disease early. Using a screener such as the MPS II to detect, manage and monitor MP provides advanced eye-care for the practitioner.

Supplementation

It is now 13 years since the publication of AREDS[23], where it was shown that the use of general antioxidants vitamins C, E and beta-carotene, in combination with zinc, reduced the risk of AMD in patients with non-central geographic atrophy in one or both eyes by 19%, or advanced AMD or vision loss due to AMD in one eye by 25%. Long term use of the AREDS supplements appears both safe and protective and the effects have been shown to be long lasting.

Following on from AREDS, laboratories began to synthesize the retinal carotenoids lutein, zeaxanthin (L/Z) and meso-zeaxanthin (Z), which constitute the yellow MP. This enabled manufacturers to add the carotenoids to the AREDS formula and for suppliers to provide it to patients in the form of supplements.

MP is entirely of dietary origin with meso-zeaxanthin an isomer of zeaxanthin formed in the retinal tissue with lutein[24]. In 2013 came the publication of AREDS2[25]. This was a multi-centre, randomized, double-blind, placebo controlled trial containing 4302 patients at high risk of progression to advanced AMD. The trial was designed to assess the effects of oral supplementation of macular xanthophylls (lutein and zeaxanthin) and/or long-chain omega-3 fatty acids (docosahexaenoic acid) [DHA] and eicosapentaenoic acid [EPA]) on the progression to advanced AMD. An additional goal of the study was to assess whether forms of the AREDS nutritional supplement with reduced zinc and/or no beta-carotene work as well as the original supplement in reducing the risk of progression to advanced AMD.

There were eleven separate treatment groups contained in the study. Sub-group analysis of the L/Z patient group clearly reported the benefits of supplementation, with 26% reduction in the risk of progression to advanced AMD, beyond the effects of the AREDS supplement (25%), in persons with the lowest dietary intake of L/Z. Other notable conclusions were that (a) beta-carotene actively competes with lutein as an antioxidant and should be removed from formulations giving a risk reduction of 18%; (b) beta-carotene is also contraindicated for patients who smoke as this increased risk of lung cancer; (c) omega-3 in its present form was deemed not to increase or decrease the effectiveness of the formulation as an antioxidant; and (d) the amount of zinc oxide could be reduced from 80mg to 25mg and still produce the same effect.

When supplements are started they should be continued no matter how high the MP density score becomes to protect the retina against the blue light hazard.

For comparison, trying to obtain supplementation from green vegetables alone would require approx. 1kg of broccoli per day, or 100mg of kale or spinach digested per day. Given that this is not a feasible way to gain L/Z and increase MP, a far more balanced approach is to adopt a "macular controlled diet" of reduced omega-6 fatty acids, increased omega-3, leafy green vegetables, yellow/orange fruits, egg yolks and supplementation (if advised). When supplements are started they should be continued no matter how high the MP density score becomes to protect the retina against the blue light hazard. If the regime is discontinued MP levels will fall over time and make the retina vulnerable to blue light hazard once again. They are not contraindicated for any other medication, except advice is recommended from the patient's GP is they are taking vitamin E supplements. They are available over the counter as food supplements in many countries such as the UK and in Europe are available on prescription.

MPOD test and scores

Screening for MP can be performed on all patients over 16 or for a sub-group, e.g. over 40 as a standard protocol (or those younger with any identified risk factors). As with other diseases, the AMD risk factors discussed are cumulative with respect to risk. Put simply, the greater the number of risk factors, the greater the need for intervention through supplementation, diet and lifestyle. If only a single risk factor such as smoking exists, a smoking cessation therapy may be recommended, but when risk factors are coupled to low MP scores (<0.25), the practitioner may want to suggest a more direct approach, e.g. AREDS2 supplements and re-measure MP density after 6 months to determine the effect of the treatment.

The MPS II is an innovative device that uses a patented approach to measuring the density of the MP. The result is a macular pigment optical density (MPOD) score from zero to one*. The test is very fast, simple to perform and easy to interpret. There are many studies now published using this device.

A large multi-centre published study revealed that the average MP density using the original MPS II (called MPOD) was 0.3319. The average MPOD score of all our patients in the practice who undertook MP screening was 0.36 (sample size: 152). The former study was conducted in North America and the latter in Scotland.

$$MPOD = \log_{10}\left(B_{periph}/B_{centre}\right)$$

Where*

- B is luminance in cd/m2 measured using light of constant incident intensity with central wavelength 465nm and bandwidth 25nm.
- B_{periph} is the value measured in the periphery of the eye (where MP is absent), B_{centre} the value at the centre, where MP is at its peak. The MPS II measures MPOD in the range from zero (equal absorption at periphery and peak) to 1 (10x absorption); although MPOD values in excess of 1 are possible the instrument will not report these values since they represent extremely high MP levels whose exact measurement is not relevant to practitioners or patients

When an MPOD reading is taken using the MPS II, the eye-care professional needs to understand how best to leverage this reading to create an AMD prevention or management strategy that best fits the patient.

As discussed, AMD is a complex disease, with many risk factors influencing a particular patient's susceptibility to early onset of the disease. There is a consensus that the interaction between genetics/lifestyle and environment all predispose to AMD. In other words, an individual's risk for AMD is dependent on the interplay of variables seen in Table 1.

A supplement regime needs to take into account both the MPOD reading and the other cumulative risk factors. Consulting with other leading practitioners over the years has led to the formation of the Red/Amber/Green (RAG) guidelines seen in Figure 1. Adopting this approach - using the MPS II MPOD score for the poorer eye - gives the following broad guidance:

- MP optical density score is very low <0.25 - supplementation advised with or without any other risk factors present
- MP optical density score is quite low 0.25-0.5 - take supplements if one or more key risk factors are present
- MP optical density score is good 0.50-0.75 - take supplements if two or more risk factors are present
- MP optical density score is very good >0.75 - no need to take supplements.

The prevention toolkit for any person wishing to protect themselves against early onset AMD includes education, making proactive diet and lifestyle choices, supplementation (when appropriate) and blocking of blue light through appropriate choice of spectacle lenses.

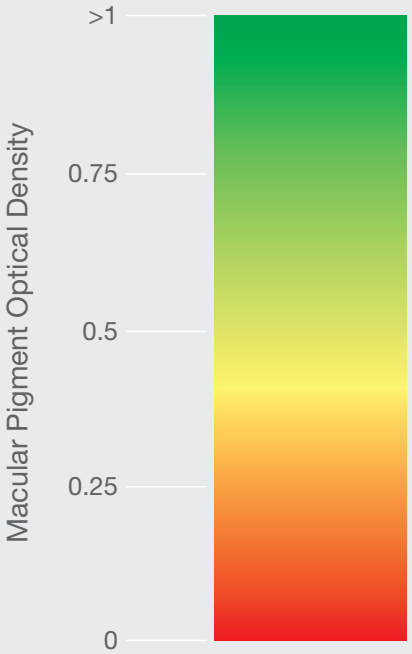
Whilst there can be discussion about the finer details, this pragmatic and intuitive assessment can be used to develop a coherent strategy around prevention and management of AMD. As more experience is gained in the field, this guide will continue to refine and develop.

The guide also serves to educate the patient on the number of modifiable risk factors that they may have, enabling them to make proactive diet and lifestyle choices early on. Building up MPOD over the decades before AMD is likely to develop (50-60's) will provide far greater protection against blue light, making the retina less susceptible to oxidative damage and ergo AMD. A patient can reduce the risk of early onset AMD by 25-50% simply by cessation of smoking cigarettes. The prevention toolkit for any person wishing to protect themselves against early onset AMD includes education, making proactive diet and lifestyle choices, supplementation (when appropriate) and blocking of blue light through appropriate choice of spectacle lenses.

Figure 1. After an MPOD reading is taken, the next step is to use the Red/Amber/Green (RAG) guidelines

Key Risk Factors:
Smoking, Family History, Soft Drusen, Age>50

Some Other Risk Factors:
Blue Light Exposure, BMI>25, Excessive Sun Exposure, High Cholesterol, Vit. D deficiency



MPOD is very good.
No need to take supplements.

MPOD is good.
Take supplements if *at least* 2 risk factors (1 key) are present.

MPOD is quite low.
Take supplements/wear lenses if 1 key risk factor is present.

MPOD is very low.
Take supplements and wear lenses to reduce blue light.

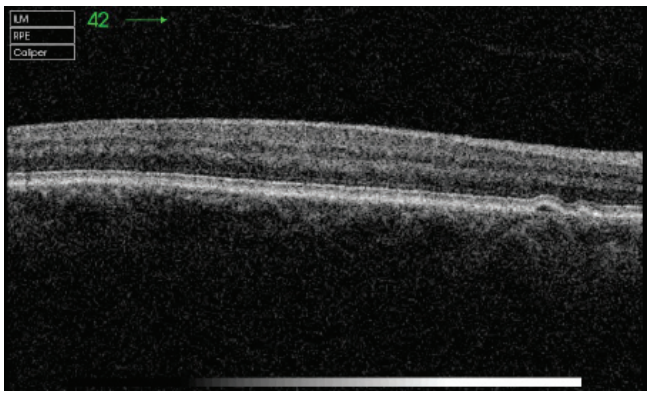
Case studies

Patient MM: Female, 76 years, positive FH, non-smoker; drusen seen peripherally on the fundus.

An OCT scan captured an elevation in the RPE within the central arcades, which increased suspicion of risk (Figure 2).

Figure 2

Patient MM: 76 years
OCT B-Scan



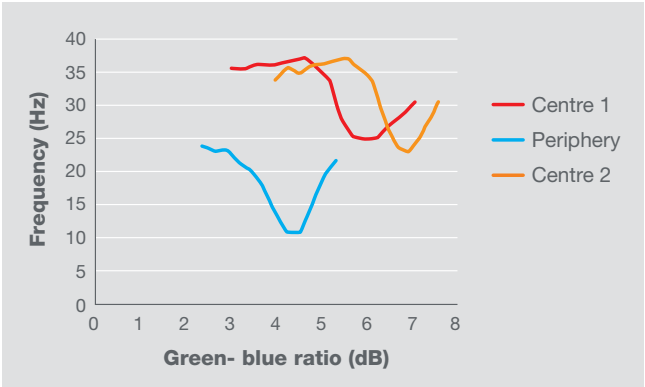
MP measurement using an MPOD (now the MPS II) revealed the patient's score was below the average being 0.30. After informed consent macular supplementation was commenced. Within six months L/Z supplementation had improved the MP score to 0.45) (Figure 3), visual acuity (VA) stable, fundus appearance same.

Drusen is not a dormant sign to be ignored. These are focal deposits of debris external to the RPE basal lamina and internal to the collagenous layer of Bruch's membrane. They contain acute phase reactants and are inflammatory markers as a result of oxidative injury and are the pathogenesis of AMD. They represent loss of photoreceptors, retinal pigment epithelium (RPE) cell dysfunction and reduced foveolar choroidal circulation. Again thinking about glaucoma, this is like measuring the first subtle signs, such as nerve fibre loss, before a visual field defect occurs.

This patient will be kept on supplements and reviewed every six months, which is the average time from nutrients ingested and within the blood stream to transfer their effects to the retinal appearance and function.

Figure 3

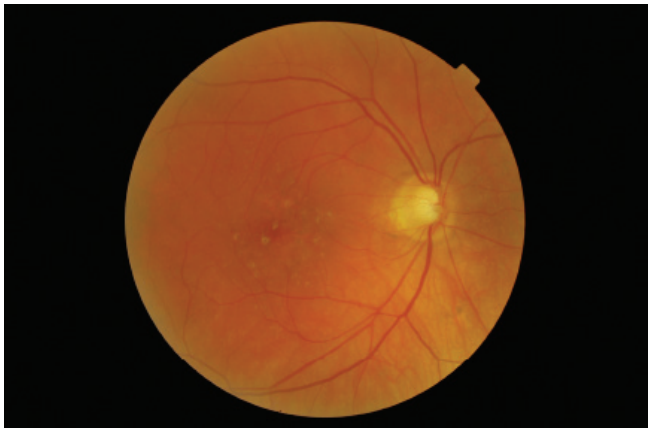
Patient MM: 76 years
Initial MPOD: 0.30 (Nov 2011)
Second MPOD: 0.45 (Apr 2012)



The second central measurement has shifted to the right over time, indicating an increase in MP. Measurement at the periphery – where there is no MP - remains consistent in both readings as this is dictated by the lens age, which has not changed to any significant extent in this short time frame.

Figure 4

Patient GW: 67 years
Initial MPOD: 0.46, VA 6/9 (Mar 2011)



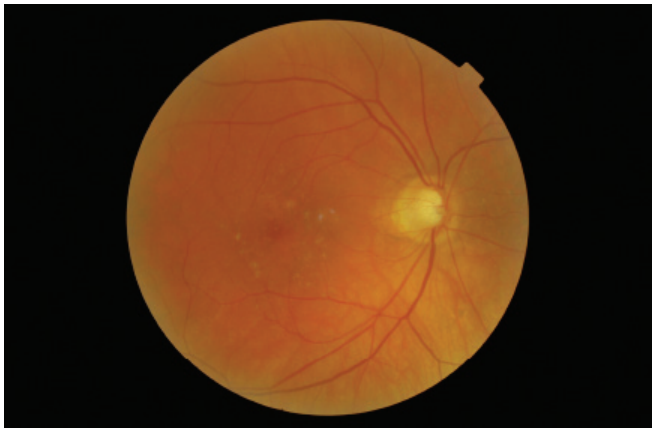
Patient GW was first seen for a routine eye examination. This patient had drusen in both eyes and fundus photographs were taken (Figure 4).

The patient only had central vision in one eye due to dense amblyopia since birth. There was no family history of AMD and the patient did not smoke. Symptoms included reduced VA to 6/9 and glare, especially driving at night, even whilst wearing varifocals with a reflection free coating. MPOD was measured at 0.46. The patient was started on supplements and given advice on diet and exercise.

In April 2012, on follow up eye examination, fundus photos (Figure 5) revealed less drusen, improved VA to 6/6 and the resolution of glare. MPOD had increased to 0.64, which as a log scale represent over 100% improvement in MP. NB: Drusen can appear to resolve spontaneously, however this can also be due to RPE atrophy[26]. In November 2011 the practice invested in an OCT and this revealed drusen on the RPE but no retinal thinning (atrophy) thus it represented true reduction in risk.

Figure 5

Patient GW: 68yrs
Second MPOD: 0.64, VA 6/6, less central drusen (Apr 2012)



In November 2012, as part of the beta-testing, the patient was measured on the MPS II at 0.72 after 2.5 years of supplementation and their VA remained at 6/6 with a similar drusen count and resolution of glare.

Note: There is not a magic tablet that will cure dry AMD. There are many supplements on the market which vary in efficacy, some of which have been formulated on scientific evidence based on ongoing research. The supplements given above included (Vitamin C 60mg; Vitamin E 10mg; Zinc 10mg; Cooper 500ug; Selenium 25ug essential eatty acids: EPAX quality fish oil 330mg containing omega-3: EPA (40%) and DHA (20%) Retinoids: FloraGlo Lutein 10mg and Zeaxanthin 2mg and Reservatrol 1mg). However, this is a multi-factorial disease and the addition of lifestyle changes, e.g. smoking cessation, better diet, more exercise, blue filter protection on spectacles/sunglasses etc., should also be discussed and proactively addressed as part of an ongoing management strategy.

Conclusion

AMD is affecting people at a far younger age than previously thought[27], which in turn means that it is vital to screen patients early to identify those most at risk of developing the condition. By measuring macular pigment (MP) and identifying all other risk factors that may be present, appropriate preventative management strategies can be implemented. From a socio-economic standpoint, if nothing else, it is no longer a viable option to wait for patients to develop wet AMD. Treatment is extremely costly, outcomes are varied and people's life quality is severely impaired at this late stage. Screening people early for low MP and increasing pigment over time can be seen to be an insurance policy against sight loss. Supplementation has also proved to be effective for those many millions of patients worldwide who have dry AMD[25] demonstrating that the combination of MP screening and supplementation is a viable (preventative) management strategy for AMD. Screening on the MPS II is a simple test that can be performed by support staff and does not require the patient to take eye drops to dilate the pupils. The test is therefore non-invasive, quick to perform and provides instant results that can be fed into a comprehensive management programme.

Please visit **www.elektron-eye-technology.com** for further information on AMD, MPOD and the MPS II device.

References

1 Nunes RP, Rosenfield PJK, de Amorium Garcia Filho CA, Yehoshua Z, Martidis A, Tennant MTS. Section 6. Macular Disorders, Chapter 6.28. Macular Degeneration. In: M Yanoff, JS Duker, editors. Ophthalmology. 4th Ed. Elsevier Saunders, 2013; p580-599.

2 AMD Age-Related Macular Degeneration. GER Group, 2010 ISBN 978-989-96792-0-7

3 Jonisch J, Shah G, Chapter 3. Diagnosis of Age-Related macular Degeneration. In: AC Ho, CD Regillo, editors. Age-related Macular Degeneration Diagnosis and Treatment, Springer, 2011;p23-39.

4 Mousavi M, Armstrong RA. Genetic risk factors and age related macular degeneration (AMD). Journal of Optometry, 2013; 6: 176-184.

5 Moshfeghi DM. The Pathophysiology of wet AMD (online) 2009. Available from www.lucentis.com/media/hcp/download/PathophysiologyWetAMD_brochure.pdf

6 Gurwood AS, Hutchinson JK, Myres MD. AMD: Counteracting Conversion. Optometric Management (online) 2011. Available from www.optometricmanagement.com/ articleviewer.aspx?articleid=105537.

7 Chakravarthy U, Harding SP, Rogers CA et al. Alternative treatments to inhibit VEGF in age-related choroidal neovascularization: 2-year findings of the IVAN random controlled trial. The Lancet, 2013:382(9900)1258-1267.

8 Stein JD, Brown MM, Brown GC et al. Quality of life with macular degeneration: perceptions of patients, clinicians and community members. Br J Ophthalmol 2003:87:8-12.

9 Rovner BW, Casten RJ. Activity loss and depression in age-related macular degeneration. American Journal Geriatic Psychiatry 2002; 10(3), 305-310.

10. Mackie S The Rise of supplements-seeing is believing Optician 11/05/12 p12-13.

11 Nolan JM, Stack J, et al. Risk factors for age-related maculopathy are associated with a relative lack of macular pigment. Exp Eye Res 2007; 84(1), 61-74.

12 Khan JC, Thurlby DA, et al. Smoking and age related macular degeneration:the number of packs of cigarette smoking is a major determinant of risk for both geographic atrophy and choroidal neovascularisation; Br J Ophthalmol, 2006; 90, 75-80.

13 Smith W, Mitchell P. Family history and age-related maculopathy:the blue mountains eye study. Clin and Experimental Ophthalmology 1998; 26(3), 203-206.

14 Lu L, Hackett SF, Mincey A, et al. Effect of different types of oxidative Stress in RPE Cells; J Cell Physiol, 2006; 206, 119-25.

15 Chen Y, Bedwell M, Zhang K. Age-related macular degeneration: Genetics and Environmental factors of the disease. Molecular Interventions, 2010(5).

16 Beatty SB, Murray IJ, Hanson DB et al. Macular pigment and Risk for Age-Related Macular Degeneration in subjects for a Northern European Population. Investigative Ophthalmology and Visual Science, 2001 (42)2439-446.

17 Mianster MA, Turner PL. Blue-blocking IOLs decrease photoreception without providing significant photo protection; Surv Ophthalmol, 2010; 55(3), 272-283.

18 Kanski. Clinical Ophthalmology. A Systematic Approach.

19 Van Der Veen RLP, Berendschot TTJM, et al. A new desktop instrument for measuring macular pigment optical density based on a novel technique for setting flicker thresholds; Ophthal.Physiol.Opt, 2009; 29, 127-137.

20 Murray I, Makridaki M and Carden D. Measuring MP in Practice Optician 01/04/11 p15-17

21 Howells O, Eperjesi F and Barlett H Improving the repeatability of heterochromatic flicker photometry for measurement of macular pigment optical density Graefes Arch Clin Ophthalmol 2012 DOI 10.1007/s00417-012-2127-0

22 Clarke JJJ A study of Troxler's Effect Optica Acta: International Journal of Optics 1960 Volume 7, Issue 3

23 AREDS Report No 8. A randomised, 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; Arch Ophthalmol, 2001; 119, 1417-1436.

24 Sommerberg O, Keunen JEE, et al. Fruits and vegetables that are sources of lutein and zeaxanthin:the macular pigment ion human eyes. Br J Ophthalmol, 1998; 82(8), 907-910.

25 AREDS2 Research Group. "Lutein/Zeaxanthin and Omega-3 Fatty Acids for Age-Related Macular Degeneration. The Age-Related Eye Disease Study 2 (AREDS2) Controlled Randomized Clinical Trial." JAMA, published online May 5, 2013.

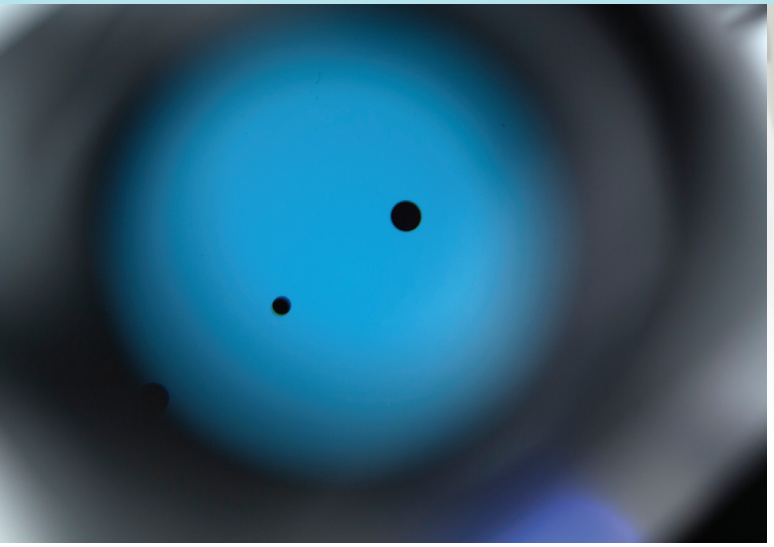
26 Bressler NM, Munoz B, et al. 5-year incidence and disappearance of drusen and retinal-pigment epithelial abnormalities. Arch Ophthalmol 1995; 113(3), 301-308.

27 C. A. Korb et al. (2014), Prevalence of age-related macular degeneration in a large European cohort: Results from the population-based Gutenberg Health Study, Graefe's Archive for Clinical and Experimental Ophthalmology,

28 Schaumberg D; Christen W, Hankinson S, Glynn R. Body mass index and the incidence of visually significant age-related maculopathy in men. Arch Ophthalmol 2001: 119:1259-1265

29 Van Leeuwen R, Klaver CC, Vingerling JR et al. Cholesterol and age-related macular degeneration: is there a link? Am J Ophthalmol 2004; 137:750-2

30 Sperduto R, Hiller R. Systemic hypertension and age-related maculopathy in the ramingham Study. Arch Ophthalmol 1986; 104:216-219



31 Parakh N, Chappell RJ, Millen AE et al. Association between vitamin D and age-related macular degeneration in the thirs national health and nutrition examination survey, 1988 through 1994. PMID 17502506. Arch Ophthalmol 2007, 125: 661-69

32 Beatty s, Koh H, Phil M et al. The role of oxidative stress in the pathogenesis of age-related macular degeneration. Surv Ophthalmol 2000; 45 :115-134

33. Chiu CJ, Taylor a. Nutritional antioxidants and age-related cataract and maculopathy. Exp Eye Res 2007: 84 (2): 229-245

34. Tomany SC, Cruickshanks KJ, Klein R, et al. Sunlight and the 10-year incidence of age-related maculopathy—The Beaver Dam eye study. Arch Ophthalmol 2004;122:750–7.

35. SanGiovanni JP, Chew EY, Clemons TE, et al. The relationship of dietary carotenoid and vitamin A, E, and C intake with age-related macular degeneration in a case control study: AREDS Report No. 22. Arch Ophthalmol 2007; 125:1225–32..

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Addendum

Tips and suggestions for data collection with MPS II

Using an analogy of investigations for glaucoma, it is noted that performing a visual field examination without any practitioner/patient interaction introduces a lot of noise in the data; this is the same for the MPS II. So I would suggest the following examination procedure:

a) Put a full aperture (or reduced aperture) trial lens in the front slot of the machine with the reading Rx. If there is no access to these, use the patient's spectacles (near SV, varifocals or bifocals) or contact lenses (distance Rx with over-readers, multifocals or monovision) but refrain from using transition lenses or lenses with tinting or blue-blocking filters. If the patient does not wear spectacles or contact lenses, no additional lens is required as the HFP procedure is relatively insensitive to blur. Occlude the other eye using an eye patch. Perform in mesopic or scotopic lighting. The newest version of the software can record many variables (e.g. medication, lifestyle etc.) however minimum requirement is to record the patient's name, age, gender and whether they have had any IOL. Take a central only measurement of the RE then LE using Standard test mode and save the results. Reports can be generated of real time or time progression of MPOD and the supported platforms are Windows 7, and above, which are available in several languages.

b) Inform the patient that this test requires concentration and a consistent approach, however, they should be encouraged to blink naturally. (Many will advise them to blink each time they press the button). When the patient is certain they detect flicker on the central spot they must press and release the button quickly. Repeating this at every presentation consistently – in other words ensuring that the button is pressed at the same time for each presentation – eliminates noise from the test and ensures it will be accepted first time. There will be a short familarisation test before the main test to check the speed of their response, this sets the initial blue/green ratio and is recorded as open squares on the screen of a laptop (or another computer). This takes approximately 30 seconds and only if the responses are so inconsistent will an error message appear stating range too high and start again. This only occurred rarely during beta analyses. When this is over the middle spot will go black temporarily. Keep watching (don't let the patient move their head), as the second phase of the test will take place immediately the screen lights up again. NB: The flickering central light may appear slightly bleached out which is a normal after-image.

c) Watch the screen and if the patient is seen to be pressing the button too quickly or indeed losing concentration between responses, the test should be paused and the patients reminded of the original instructions. Alternately, the test should be repeated later or on a different day. Constant communication cannot be over-emphasised in my opinion but views vary on this. Patients can be encouraged with phrases like "you're doing well", "look for the flicker" and "you're nearly finished" as silence may cause the patient to question if they are performing the test correctly. On average depending on MP this measurement takes approximately 60 seconds to complete. The repeatability of HFP measurements has previously been studied and amended scoring techniques have reduced the standard deviation (SD)[21]. However, with the new protocol of a central only measurement any "noise" in repeatability testing is minimised and thus measurements taken at different visits can be compared with confidence.

d) Only if co-existing pathology (e.g. diabetic maculopathy and AMD) is present will it be necessary to take both central and peripheral measurements (using Detailed test mode). The patient will complete a central measurement first and should be informed to stare at the top of the red target (i.e. left target for RE and vice-versa). During the peripheral part of the Detailed test the patient will use their side vision to view the peripheral blue flickering target. They will want to glance at this, but they should be informed to "resist the temptation", as if they stare at the central target directly again, it forfeits the results as a central measurement would be repeated. Half-way through the test, it is advisable to pause and encourage the patient to look at the bottom of the red spot from then on, but again only pressing the button when they see the peripheral blue flickering target. This prevents the blue target from disappearing due to the "Troxler Effect"[22]. If a patient has a cataract or a tinted IOL and the testing is being performed in order to ascertain the absolute MPOD then use the Detailed test mode. However, if the patient is being screened for an improvement in their MPOD through supplementation, then the Standard (central only) test mode is fit for this purpose as the central measurement it uses is relative to the last central measurement taken and thus the effect of lens yellowing is independent of the result.

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