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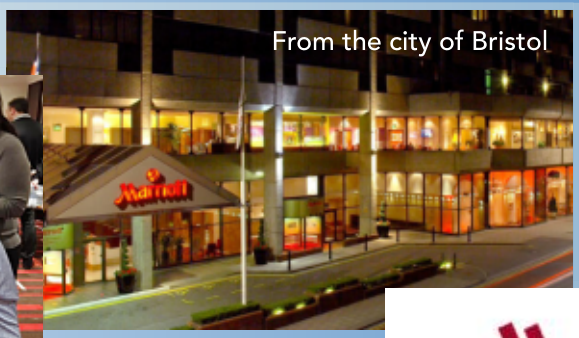

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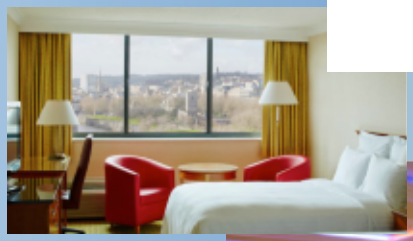

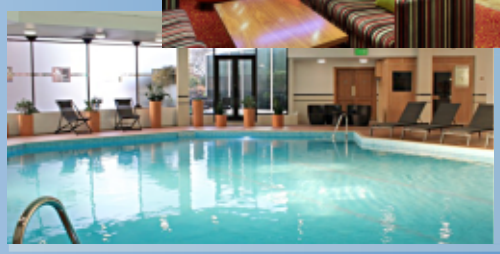

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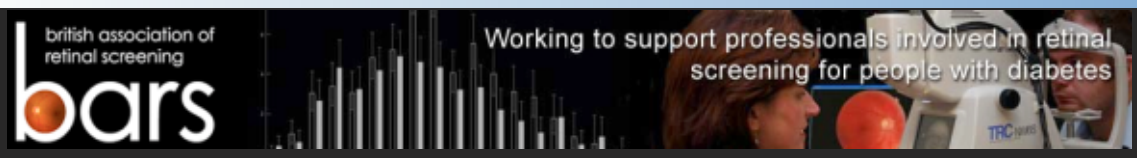

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DiabeticEyeJournal is published and produced by BARS with support of its founders from North Central London Diabetic Eye Screening Programme.

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• HAAG-STREIT UK, Training facility in Harlow, Essex
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• National DES Conference 2018
Friday 20 April 2018
Royal Society of Medicine, London W1G 0AE
www.rsm.ac.uk/events/events-listing.aspx

• South West Diabetes UK Professional Conference
Wednesday 9 May 2018
Taunton, Somerset, England
south.west@diabetes.org.uk

• Sharing and Caring - Multidisciplinary management
Thursday 10 May 2018
The Royal Society of Medicine, London W1G 0AE
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• RCOphth Congress
Monday 21 to Thursday 24 May 2018
Arena & Convention Centre (AAC), Liverpool
www.rcophth.ac.uk/events-and-courses/annual-congress-2018/

• Imaging in Endocrinology and Diabetes
Friday 13 July 2018
The Royal Society of Medicine, London W1G 0AE
www.rsm.ac.uk/events/events-listing.aspx

• EASDec 2018 Meeting
Thursday 24 to Saturday 26 May 2018
Belfast, Northern Ireland
www.easdec.org/pages/

• Skills in imaging, diagnosis and management of retinal diseases
Thursday 12 to 13 July 2018
Wellcome Trust, Euston Road, London NW1 2BE
www.rcophth.ac.uk/events-and-courses/

• BARS 18th Annual Conference
Thursday 27 to Friday 28 September 2018
Marriott Hotel City Centre, Bristol
www.eyescreening.org.uk

• EASD 2018 Meeting
Monday 1 to Friday 5 October 2018
Berlin, Germany
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DiabeticEyeJournal

From the Editor

Welcome to the 10th issue of DEJ! It is our anniversary edition, in our 5th year. We started from very humble beginnings in the North Central London DESP. There were three of us: Susanne Althauser, Jacqui Mansell and myself who brought it to life back in 2013. After a successful launch, Jacqui and I approached the British Association of Retinal Screening, who adopted the DEJ in its infancy and turned it into a national publication for Diabetic Eye Screening and Hospital Eye Service professionals. Five years on and it is still growing in its popularity and has a readership of more than a thousand members, plus every DESP and HES in the country who regularly receive it in their letter box.

We also have a wide on-line audience reaching as far as the clinicians of Moorfields Eye Hospital in United Arab Emirates. We hope that you will enjoy our March issue, which has a variety of articles including:

Musing on Microaneurysms, their importance and definition, by Stephen Aldington from Gloucesthershire Retinal Research and Education Group; Dry Eye Disease, the most common complaint of our patients, by Dr Penny Asbell Professor of Ophthalmlogy from Icahn School of Medicine at Mount Sinai in New York; and Uveitis, the condition that can irreversibly damage the retina, by Alice Thomas and Harry Petrushkin from Moorfields Eye Hospital NHS Foundation Trust in London. The changing role of graders, the distribution of workload within DES and how the workforce can be supported is discussed in an article by Phil Gardner, BARS chair. We also sum up the last 10 years of Diabetic Eye Screening in an article by our regular contributors at NDESP; and we look into the prognosis for the Future of Diabetes in an article by the editor of Update magazine for professionals, Susan Aldridge from Diabetes UK.

There is so much to explore! We hope you will enjoy DEJ and as always we are looking forward to your feedback!

Our aim is to make the next five years as productive as the last - so please raise your imaginary glass and Cheers!

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FRONT COVER IMAGE

Post-mortem microscopic image of microaneurysms from Hammersmith Hospital in London, pre-1980

COMMENTS and CONTRIBUTIONS

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ONLINE VERSION

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Contents

Musings on microaneurysms

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Introduction

The retinal microaneurysm (MA) is a frequently overlooked, apparently relatively insignificant feature of diabetic retinopathy (DR). Simply described it is a small red dot. It is usually the earliest clinically visible and probably most recognisable feature of DR. In the development of sight-threatening DR or need for laser, intravitreal or surgical intervention it seems to have no significant role. There is, however, much more to tell from the presence of the humble microaneurysm. Most importantly is that for the affected person it marks the point of transition from no diabetic eye disease to being in a changed status to "I have diabetic retinopathy". This is a highly significant psychological issue for many; the impact of which is commonly significantly underestimated by health professionals. It certainly is not all doom and gloom, but a threshold has been breached and for many this is a potentially worrying additional diagnosis.

Definition, description and development of microaneurysms

Some descriptions and definitions may help, as MAs are:

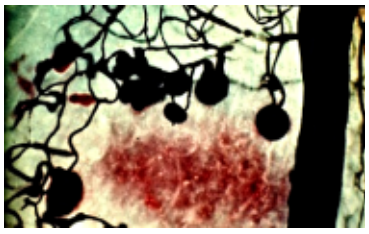
• A localised vascular out-pouching from a retinal capillary

• "A circular or near-circular red spot with sharp margins, with or without a central light reflex, less than 125 microns in largest dimension (the approximate width of a major vein crossing the optic disc margin)" (the ETDRS definition) [**ETDRS 1991**]

• Generally considered to be between 20 and 125 µm in diameter

• Much larger than the capillary from which it originates (**Figure 1**)

Figure 1.



Post-mortem microscopic image of microaneurysms with their capillary origins surrounding a cotton wool spot containing some acellular ghost capillary vessels. Indian ink, non-digest, magnification unknown. (source: Hammersmith Hospital London UK, pre-1980)

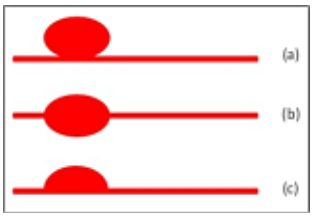
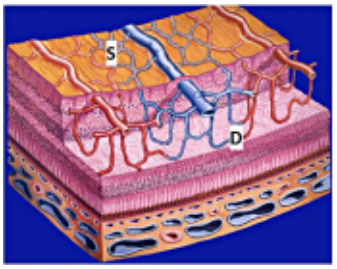


Figure 3. Types of microaneurysm (all assuming a straight capillary): (a) saccular, (b) fusiform or (c) focal bulge (redrawn from Moore, 1999)

Figure 2.

The inner retinal circulation showing arterial (red) to venous (blue) circulation through the superficial (S) and deep (D) capillary plexi. (source: Novartis)



Human retinal capillaries, from which all MAs develop, are located predominantly within the Ganglion Cell Layer (the superficial capillary plexus) or Inner Nuclear Layer (the deep capillary plexus). At 5 to 10 µm maximum diameter, retinal capillaries are far too narrow to be visible on colour images or clinical examination. The retinal vessels we see on examination or colour imaging are all larger than capillaries. Only when a defect or lesion of any appreciable size appears in the retinal layers can we see it without additional specialised imaging investigations. MAs however occur and are visible in both the superficial and deep capillary plexi (**Figure 2**) and their development and life-cycle may indeed differ between the two (or more) distinct populations. I briefly discuss this complex topic later. It is however not possible to differentiate the actual layer(s) in which the MA occur when using only 2-dimensional imaging techniques. Microaneurysms are categorised as being (a) 'saccular' if the dilatation (i.e. the MA) is asymmetric around the long axis of the associated vessel, (b) 'fusiform' if it is symmetric around the long axis of the associated vessel or (c) a 'focal bulge' if too small and irregularly shaped to be classified as (a) or (b) (**Figure 3**) [paraphrased and re-drawn from **Moore 1999**]. It is very common to find clusters of MAs lying close together, sometimes in different stages of individual development. These arise from a defective capillary, where the capillary vessel wall has been damaged sufficiently extensively for multiple MAs to develop along its length. (**Figure 1**)

To be visible on examination or standard colour retinal imaging, MA must still be connected to a patent capillary, or have been so recently connected as to have sufficient residual haemoglobin to remain 'red'. The MA is not actually red of course – it is the red blood cells containing haemoglobin (Hb) that are visible through the near-transparent wall or lumen of the MA or retinal vessel. Similarly, when observing fluorescein angiograms (FFA), for MA to appear as fluorescent 'dots' or 'spots', they absolutely have to be still connected to a patent capillary; otherwise they will not fill with plasma-bound sodium fluorescein and hence would not be visible against background hypofluorescence. Retinal haemorrhages however are not 'actively filled' with fluorescein and hence always appear hypofluorescent – an excellent way of distinguishing the dot haemorrhage from patent MAs on a fluorescein. Not that we have (or need) invasive FFA during routine diabetic retinal eye screening.

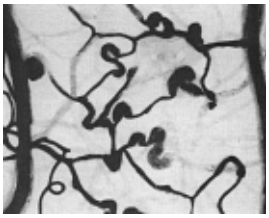
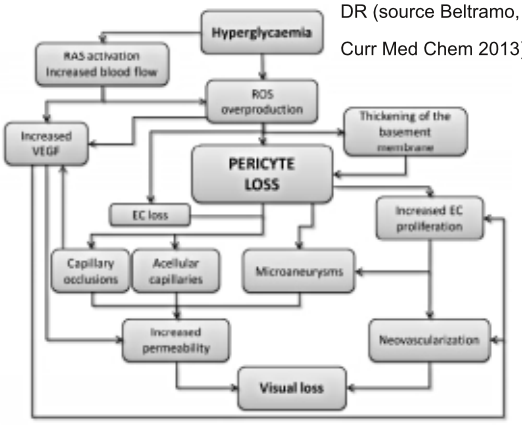


Figure 4.
MA predominantly originating from capillary loops (source Ashton, Br J Ophthalmol 1963)

Early work from Norman Ashton describing MAs in humans, proposed that they occurred only or predominantly from major deviations in the capillary – akin to a capillary loop – and it was on the outside 'apex' of such a loop that MAs were to be found [**Ashton 1950**]. However, Ashton's later work, using ink-injected and digested post-mortem retinae, showed that MAs occurred from such an abnormal capillary loop in only about 50-60% of cases, with the remaining proportion appearing on otherwise non-deviated capillaries [**Ashton 1963**]. Examples of both MA types are shown at **Figure 4**.

Figure 5. Mechanisms and consequences of pericyte loss in DR (source Beltramo, Curr Med Chem 2013)



There remains long-standing dispute as to whether MAs represent simply a structural herniation or 'diverticulation' of the retinal capillary or whether their genesis requires actual cell proliferation. This is too complex a topic for this short article but current thinking actually supports a middle ground, in that the development of a MA is far more complex and involved than can be described by one simple process. The retinal microaneurysm is however a visible structural manifestation indicating pre-existing retinal capillary cell damage and death. Capillary basement membrane thickening, endothelial cell death (and/or migration) and pericytes loss, individually or collectively but always invisibly, precede the formation of visible MAs [**Beltramo 2013**]. That publication gives a most useful illustration proposing a possible model of relationships in the loss of pericytes, development of MA and potential for more advanced DR (**Figure 5**). The major factor driving initial insult is of course hyperglycaemia. It seems clear there must be at least an element of cell proliferation in some MA, to explain the sheer size of the aberrant 'very large' MA when compared to the relatively narrow capillary from which it arose. There is just not enough potential give or stretch in a capillary wall to account for the larger MAs we sometimes see if it was all accounted for only by vessel wall herniation. However, this relationship has not yet been totally explored or explained and more needs to be done.

There is also still general dispute over the minimum and maximum size of a retinal feature sufficient to classify the 'red dot' as a microaneurysm rather than as a dot haemorrhage. Perhaps it does not really matter of course? However, with modern high-resolution retinal imaging, even without FFA, it is perfectly possible to identify retinal red dots in the region of 15-20 microns diameter on standard colour retinal images. These we would all call as MA. Smaller than that, if red dots are visible, then they are most likely to be capillary loops and not reliably classifiable as MA. In 1998 Moore and colleagues reported (from post-mortem eyes from four patients with established diabetes) that the mean diameter of MA as assessed using confocal scanning laser microscopy, ranged between 26 and 37 microns. The range of diameters found however was large: from 15 to 140 microns [**Moore 1999**]. Conversely, Wang and colleagues more recently published in vivo investigations from 50 eyes of 40 patients with diabetes and reported external MA diameters averaging 104μm and range of 43-266μm when using simultaneous FFA and SD-OCT [**Wang 2012**]. If we consider that a normal, non-diabetic retina has a mean total perifoveal thickness of 250-280μm (Zeiss Stratus, measuring ILM to top of BM) or 320-345μm (Heidelberg Spectralis, measuring ILM to bottom of BM), it is obvious that MA of any substantial size must therefore breach several retinal layers and cannot reside solely within the neat confines of just the Ganglion Cell or Inner Nuclear Layer [**Grover 2010, Spaide 2015, Campbell 2017**]. Evidence was added in the Wang 2012 publication, which reported that 157 of 173 (91%) MAs they detected (in patients with diabetes) involved more than one retinal layer with many MAs involving 3 or more adjacent layers. We therefore need to recognise that MAs are not tiny dots, located in discrete retinal layers, but commonly are features which affect several retinal layers. As an inherently abnormal section of retinal capillary, the MA is a potent and frequent source of intraretinal haemorrhage and of lipid-bearing plasma leakage into the (predominantly deeper) retinal layers: leakage manifest by retinal thickening and oedema and/or by the presence of intraretinal exudate.

Life-cycle of the microaneurysm

Unfortunately, the life-cycle of MAs is also somewhat opaque and complex. Some MAs appear and then disappear quickly – within months or even weeks. Many remain visible several years later. What is clear is that whilst the total count of the MAs number in an eye at any one time is a vitally important measure, it is the turnover rate (shown by new appearances and disappearances) which is the strongest indicator of the underlying damage being done to the eye in diabetes. Development of a MA indicates abnormality of the capillary from whence it arises. An affected capillary is prone to develop multiple MAs. There is clear evidence that the earliest clinically visible features of DR, particularly MAs, frequently occur temporal to the macular region at the 'lateral watershed' where superior and inferior temporal arcades meet.

Damaged capillaries are however prone to occlusion, causing the MA to apparently disappear. Hence appearance and disappearance of MAs are potentially equally sinister events. Essentially there are three main reasons for the 'disappearance' of an MA: internal thrombosis due to increasing stasis of the blood in the MA and hence deterioration of the (red) haemoglobin; separation from the host capillary (probably rare), where the haemoglobin quickly denatures so as to be not visibly red and (the most common reason) capillary occlusion i.e. non-perfusion of the capillary from which the MA arose. In truth, there is a possible fourth reason: recovery of the aberrant capillary vessel wall to the extent that the herniation or out-pouching relaxes sufficiently for the MA to disappear. This however only seems likely or possible in cases of relatively early-stage 'focal bulge' Mas, and presumes cessation or even reversal of any previous endothelial cell proliferation (if it had occurred at all).

However, Ashton postulated that many capillary occlusions are not end-stage infarctions from which there is no return but may be somewhat transient through being caused by increased pressure on the capillary bed by adjacent features (predominantly the cotton wool spot – but this would only be true for the superficial capillary plexus). Improvement in such a feature could theoretically cause re-canalisation of the aberrant occluded capillary or capillaries with the potential for re-appearance of MAs on that affected capillary once re-perfused [**Ashton 1963**]. This, in part, may go some way to explaining the differing rates of occurrence, disappearance and re-occurrence of some MA populations. Kohner and colleagues reported that "The disappearance rate was almost always higher in the early months of the study (11.6% and 9.3% per month) than later (0.8% and 1% per month) indicating more than one population of microaneurysms" [**Kohner 1970**]. Once again, this relationship has not yet been totally explored and further research needs to be done, particularly with the recent introduction of OCT angiography, allowing us to explore exact points of origin, involvement of adjacent layers and turnover of individual (still patent) features.

Microaneurysms as markers of future retinal damage

We must recognise that the first detection of even a single MA can be a traumatic event for a patient. Logically however, we have to ask how significant is such an event in the risk of development of sight-threatening DR or potential for future visual loss?

Many researchers, particularly the Wisconsin and Hammersmith groups, investigated the relationship between the presence, count or turnover of MA and future development of more severe DR or future visual loss [**Kohner 1970**, **Kohner 1986**, **Klein 1989**, **Klein 1995**, and **Kohner for UKPDS 1999**]. Each reported direct relationships between MA count and future risk of development of more severe DR. Klein's 1989 and 1995 papers reported significant relationships between baseline MA counts when patients with established type 2 diabetes were first studied and their retinopathy status 4- and 10-years later respectively. Kohner's 1999 publication reporting findings from the UKPDS group (of which I was a member), showed clear evidence of a near-linear relationship between MA count (even when only few MAs are present) in eyes of patients newly diagnosed with type 2 diabetes and recruited to the UK Prospective Diabetes Study and their retinopathy status 9 years later (**Figure 6**).

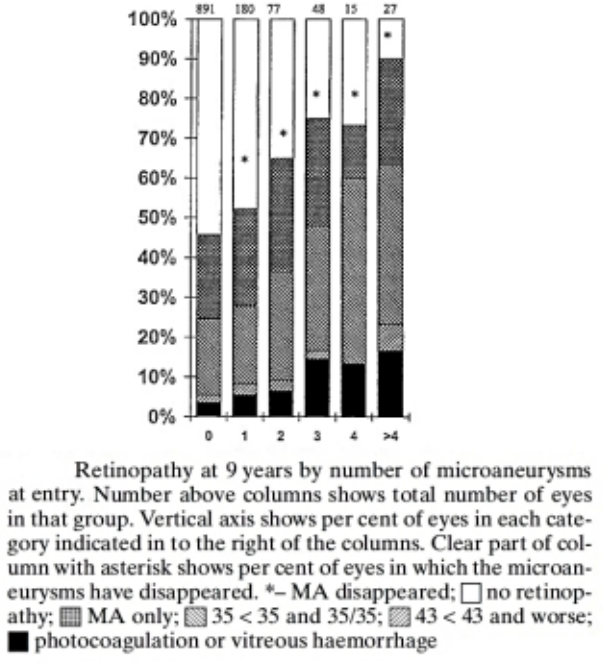


Figure 6. Retinopathy at 9 years in UKPDS by number of MA at baseline (source Kohner, Diabetologia 1999)

Importantly, we also showed that total subsequent 'regression' to zero MA in an eye becomes increasingly unlikely as one progresses from more than just a single MA at baseline. More recently, the DIRECT Study Group, of which I was also a member, reported similar relationships between baseline MA count in patients with minimal DR and future progression of ETDRS severity level in both type 1 and type 2 diabetes (**Figure 7**) [**Sjølie 2011**]. Once again, regression to fewer or zero MAs was inversely related to MA count at baseline, even when very few MAs were initially present.

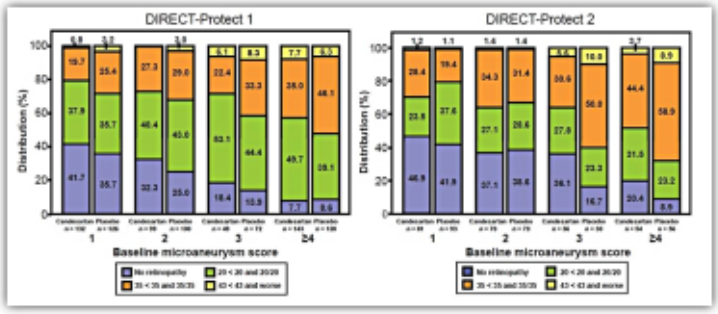


Figure 7. ETDRS retinopathy level at median 4.6 years in DIRECT by number of MA at baseline (source Sjølie, Diabet Med 2011)

All this evidence clearly supports the concept that MAs are not just a somewhat irrelevant finding but can be used in early retinopathy (in controlled conditions) as an effective marker for risk of future development of sight-threatening DR. Importantly, the DIRECT 2011 publication reported that baseline MA count was an independent predictor of future development of clinically significant macular oedema and/or proliferative retinopathy. The Hazard Ration (i.e. risk) was 1.12 (p=0.006) or 12% higher for patients with type 1 diabetes and 1.21 (p=0.003) or 21% higher for patients with type 2 diabetes for each MA score increase. Potential for clear markers for risk of development of such advanced directly sight-threatening features of DR are not simply academic statistical inferences but are highly relevant clinically issues to the patient and the healthcare system.

Our group recently reported findings from a project investigating the cost-effectiveness of various models of extending screening intervals [**Scanlon 2015**]. We found that annual screening of all patients (with any diabetes) for STDR was not cost-effective. Screening the entire cohort every 3 years was most likely to be cost-effective. We identified a low-risk group as those with no evidence of retinopathy in either eye on two consecutive screening occasions. When personalised intervals are applied, screening those in our low-risk groups every 5 years was found to be cost-effective. Screening high-risk groups (DR in both eyes twice) every 2 years further improved the cost-effectiveness of the programme. Extension of screening intervals to such a degree would be difficult to sell to persons with diabetes as anything other than more cost-cutting. As a result of this report however, the NHS Diabetic Eye Screening Programme in England (NDESP) and Public Health England are working to introduce extended, initially two-yearly, screening for those who can safely be identified as being at the lowest risk of development of STDR. This or similar approaches are also being applied in the other national screening programmes in the UK [**Four Nations DR Screening Intervals Group 2013**].

Equally importantly however, UKPDS [**Kohner 1999**] and DIRECT [**Sjølie 2011**] and many others have shown that good long-term control of diabetes and hypertension are essential tools in reducing the risk of progression of DR and of supporting the chance of some regression or certainly cessation of development of early DR. Retinal damage can at least be slowed if perhaps not reversed. It remains to be explored whether or not there is an actual threshold of retinal damage indicated by MA count or overall severity of DR, beyond which substantial regression is unlikely or impossible? What is clear however is that the sooner DR is detected, the more effective are interventions at preventing future visual impairment.

Computerised detection of microaneurysms and retinal lesions

Over 30 years ago, the advent of computer-assisted image processing systems gave us the first opportunity to chart turnover of individual microaneurysms with some degree of efficiency over manual plotting [**Baudoin 1984**, **Sleightholm 1984**]. At the time, image processing (certainly in ophthalmology) was in its relative infancy and so semi-automated systems were used to record the presence and location of retinal MAs. To ensure optimal image contrast, most systems used images from fluorescein angiograms rather than colour images. It was however still a very tedious and human-resource-intensive operation carried out only by trained observers. Many groups have since investigated automated detection of a MA with a view to monitoring turnover of individual features [**Goatman 2003**, **Bernardes 2009**, **Dupas 2010**, **Ribeiro 2013**, **Leicht 2014**, and **Solanki 2015**]. A veritable plethora of automated image analysis systems have being developed, each vying for a place in the market. This rapidly-advancing and exciting area of development is too massive to cover in detail here but some system of fully-automated detection of features of DR, including the microaneurysm, will be an inevitable inclusion in routine screening for DR in the near future. Where it fits in a screening pathway and how this will be judged by patients and professionals remains to be seen.

Microaneurysms in other ocular conditions

Microaneurysms are found not only in the retinae of patients with established diabetes but also in pre-diabetes and impaired glucose tolerance [**DPPRG 2007**]. They are also found in eyes of some patients with hypertension, vein occlusion, arteriosclerosis and some other systemic and ocular conditions [**Venkatramani 2004**, **Wong 2006**]. Indeed Ashton, in his seminal 1950 publication into early features of diabetic retinopathy, reported the presence of microaneurysms detected in 30 of 89 (34%) assessable post-mortem or post-enucleation eyes from patients without any diagnosed or confirmed diabetes [**Ashton 1950**]. Whilst Ashton freely admitted that the cases were heavily 'non-normal', in that the eyes and retinae were collected and examined for a variety of ocular or systemic conditions, they covered a wide range of non-diabetic retinal conditions: 10 cases of thrombotic glaucoma, 10 cases of chronic uveitis with secondary glaucoma and 10 other reasons including chronic glaucoma, one perforating ocular injury and even a melanoma. He did however point out that in nearly all cases the MAs were either extremely focal, "near an involving ocular feature" or were generally "few in number, small and found only in the retinal periphery". This is certainly not the pattern seen in DR, where MA and indeed many other features of early DR are found initially temporal to the macula, at the lateral vascular watershed (**Figure 8**), and are subsequently commonly concentrated in the posterior pole [**Aldington 2007**]. As DR progresses, their distribution progressively involves multiple retinal regions, as the capillaries throughout the eye become affected.

In reporting the Beaver Dam Study in Wisconsin USA, Klein found that 8.0% (336 of 4311) of people without known diabetes at baseline had evidence on 3-field 30° stereo colour images of features found in diabetic retinopathy [**Klein, 1992**]. Of these, 221 (66% of affected eyes) had MA only (notably, three-quarters of whom had just a single MA detected), 66 (20%) had a dot or blot haemorrhage only and 34 (10%) with one or more MA plus a haemorrhage (plus a few odd cases to make up the 100%). However (and this is particularly notable) when Klein and co-workers reported their 15-year follow-up on the Beaver Dam subjects who were not known to have diabetes or hypertension at baseline, they found that the presence of microaneurysms only, blot haemorrhages only, or 'any retinopathy' at baseline was associated with a significantly higher 15-year cumulative incidence of subsequent development of diabetes or hypertension [**Klein, 2006**]. The Odds Ratios (i.e. how strongly the presence or absence of property A is associated with the presence/absence of property B) for developing diabetes during the 15-year follow-up period were reported as 1.74 or 74% increased risk when 'any retinopathy' features were present at baseline compared to those without any retinopathy features. Very similar relationships were found for the development of hypertension over that 15-year period. There obviously was pre-existing and long-standing hyperglycaemia sufficient to cause some early retinal lesions, but not sufficiently overt to have been diagnosed with frank diabetes.

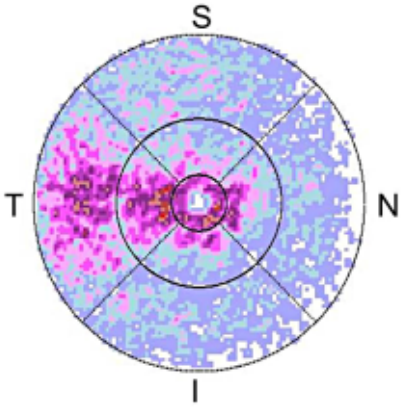


Figure 8. Perimacular microaneurysm 'heat map' from patients recruited to UK Prospective Diabetes Study (source Aldington, Diabetologia 2007)

Interestingly, Klein also reported that the cumulative incidence of new hypertension was very high over the following 15 years, as over 50% without any detected retinal lesions went on to subsequently develop hypertension. However, 66.3 to 84.7% of those with the various retinal lesions subsequently developed hypertension. It was clear that retinal features were detected in many people prior to diagnosis or reported symptoms of the underlying systemic condition be it diabetes, hypertension, or both. What we can never know is in how many patients apparently clear of retinal lesions, there were in fact such features lurking outside the fields that were imaged?

Summary and conclusions

I have shown that the retinal microaneurysm in diabetes is far from a benign and insignificant feature. Firstly, it marks the visibly detectable threshold between being free of diabetic retinopathy and having moved to a status of being a person with retinopathy. Breaching such a threshold is, however, not a disaster as much can be done to dramatically reduce the risk of development of more advanced and potentially sight-threatening DR (STDR). Secondly, presence of even a single MA indicates capillary damage and retinal cell death. The number and extent of MAs are directly related to future risk of developing more severe features of DR and indeed MAs are directly causative of some clinically important DR features such as retinal oedema and hard exudate. Early detection of DR, ideally before development of any STDR, through regular systematic screening is paramount and is hugely effective. Thirdly, development of advanced computing and technology now affords us the opportunity to radically improve methods of detection of the earliest features of DR in our ever-increasing population with diabetes.

This will also potentially reduce the tedium for staff when searching for the earliest visible features. Equally, development of personalised risk modelling including that through access to primary care risk data, will allow us to target screening at those most in need of it and at times when it can be most effectively applied. Finally, opportunistic detection of a MA in persons not previously known to have diabetes is a strong indicator of risk for future development of diabetes. Healthcare systems are however not funded to screen entire 'unaffected' populations for the presence of diabetes: it is not cost-effective and is probably not justifiable or wanted. We can however, increasingly identify persons who are at increased risk of development of diabetes or who have yet to develop any visual symptoms and apply our latest knowledge and technology to detect the earliest features of retinal damage, well before there is the risk of visual loss.

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Wales

The Welsh Background

Diabetic Eye Screening Wales (DESW) is designed to detect sight threatening retinopathy at an early stage, before visual loss occurs. The service was commissioned in July 2002 by the Welsh Government as part of the Welsh Eye Care Initiative risk reduction programme and is a vital element of the Diabetes National Service Framework. Every eligible registered person with diabetes in Wales is invited for retinal screening on an annual basis. DESW became operational in June 2003 and by December 2006 had achieved all Wales coverage; i.e. all known, eligible patients had been invited for screening at least once.

For the first 12 years, the organisation was known as the Diabetic Retinopathy Screening Service for Wales (DRSSW) and was hosted by Cardiff & Vale University Health Board. In April 2016 we transferred into Public Health Wales to join all the other national screening programmes under its charge. To coincide with that transfer, our name was changed to Sigrinio Llygaid Diabetic Cymru - Diabetic Eye Screening Wales.

The Welsh Context

When devolution was achieved in 1998, health and social care was one of the two major devolved responsibilities (along with education) that passed from Westminster to Cardiff. The Welsh Government has always treated health and social care together under a single ministerial portfolio and continued integration of the two has been a consistent strategy. There is no market economy in NHS Wales – it is almost entirely a publically led service.

The commissioning of a single national screening programme for diabetic retinopathy was one of the new, flagship services established by the fledgling administration. Since its inception DESW has developed into a stable, mature organisation which delivers a clinically excellent service to the diabetic population in Wales.

Another key strategy following devolution was the promotion and use of the Welsh language. Today all written correspondence with patients is bi-lingual and they also have the right to have their face to face services delivered in Welsh. Particularly in rural areas, many of our staff are first language Welsh speakers and routinely deliver their service this way.

The Welsh Model

From the start DESW was established as one national programme and the importance of this decision cannot be overstated. Having control over both strategy and operational resources is our cornerstone to delivering a high quality clinical service.

It allows us to maximise quality by minimising variation throughout the whole pathway. Wherever our service is encountered in Wales our users will be met by DESW employed staff, all trained and qualified to the same high standard. Their retinal images will be captured on the same model of camera, set up in the same way. All images are sent to one hugely experienced grading team in Cardiff, where they are graded against a single Welsh protocol, agreed with ophthalmologists' in Wales. Whenever referrals need to be made, all parties understand why and this has led to an efficient and effective pathway for our higher risk patients.

Some people have focussed on our single grading centre as being the key to our quality but it is the standardisation across the whole pathway that really delivers.

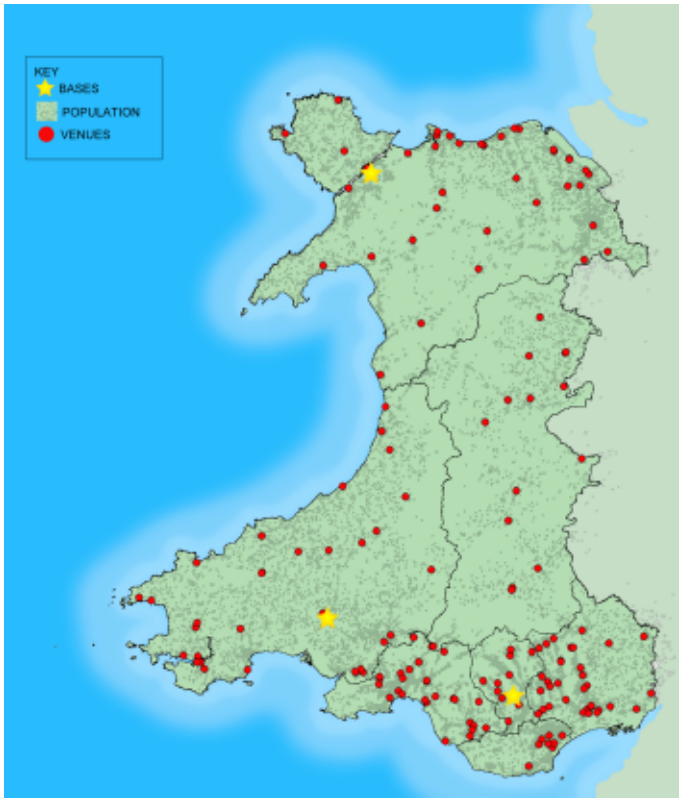
Because it is one national service, the programme is probably the largest in the UK. It is community based, delivering from clinical sites that allow all patients reasonable and equitable access. We operate a fleet of 34 small vans, with two mobile screening trucks. Through cooperation with local health boards we have access to approximately 120 clinic venues which generally gives us good national coverage. Each team consists of a Health Care Assistant, who collects the patient, performs a visual acuity test and administers Tropicamide, before they are seen by the Retinal Photographer for image capture, triage and uploading of images to the grading team. Two images (macular and nasal) are captured per eye.

One of our major challenges is delivering a recall service for a big and growing diabetic population over a very large (and often mountainous) surface area (20,735 km²).

Although originally established around three bases across Wales (Cardiff, Carmarthen and Bangor) we have been changing the model over the last few years, using locality sub bases and employing staff living locally. This dramatically cut time wasted in travel to and from clinics, which has been converted into more screening time, all using the same resource and with no compromise in quality. This is further enhanced by the increasing use of 'live' clinics with direct transfer of images, clinic lists, etc between the community venues and our administrative and grading centre in Cardiff. Regardless of connectivity, all of our clinics are now paperless. Deploying all of this, we have been able to deliver record numbers of invites and patients screened for each of the last four years.

DESW in Numbers (2017)

- Programme Size: 188,271
- New Registrations: 11,822
- Eligible, Active Population: 172,351
- Number of Clinics: 5,009
- Number of Invitations: 160,325
- Number Screened: 128,923
- Uptake: 80.4%
- Results Reported: 131,267
- Incidence of retinopathy: 28.7%
- Referral rate (DR): 2.6%
- Referral rate (non DR): 1.6%
- Number of staff: 100
- Vehicles: 34 Peugeot Expert panel vans, 2 articulated mobile screening units
- Fundus Cameras: 34 Canon CR2



External Stakeholders

Third Sector: We have carefully fostered close ties with a number of patient representative organisations, recognising the mutual value in working together for the benefit of our common constituency. For example, we've used our extensive mailing system to distribute Diabetes UK Cymru information leaflets, whilst they have been active advocates for our service – reminding people of the quality of the programme in Wales and how we can help protect their sight.

NHS Wales: We should never forget that we are not just in the business of risk reduction of sight loss. We actually refer quite small numbers into Opthalmology but every person who comes to our service is living with diabetes and we must link ourselves into that wider healthcare agenda. This means networking with other health professionals and looking for opportunities to make the lives of people living with diabetes that little bit better. As an example, we've recently joined the multidisciplinary team in a new diabetes centre in Llanelli – the first time ever in Wales.

Sgrinio Llygaid Diabetig Cymru
Diabetic Eye Screening Wales

Research: We work in collaboration with the team from the Diabetes Research Unit Cymru aimed at translational research that directly benefits patients. This is a very positive relationship which has produced a number of high value publications, including evidence for recall intervals based on clinical risk.

But perhaps the most significant is this recent publication:

Retrospective analysis of newly recorded certifications of visual impairment due to diabetic retinopathy in Wales during 2007–2015. BMJ Open 2017; Thomas RL, et al.

This showed that, since DESW was fully established, significant sight loss due to diabetes in Wales has halved. Quite an endorsement for the effectiveness of the whole pathway and one we are naturally very proud of in Wales.

Retrospective analysis of newly recorded certifications of visual impairment due to diabetic retinopathy in Wales during 2007–2015

Rebecca L Thomas, Stephen D Luzio, Rachel V North, Sanjiv Banerjee, Antra Zekite, Catey Bunce, David R Owens.

The aim of this study was to retrospectively analyse the changes in new certifications for both sight impairment (SI) and severe sight impairment (SSI, blindness) in Wales due to diabetic retinopathy/maculopathy between 2007 and 2015, derived from the national database at Moorfields Eye Hospital.

In 2014-15 there were 86 combined new certifications due to diabetic retinopathy compared with 108 in 2007-8. However, during this observation period there was a parallel increase of 52 229 (+39.8%) persons with diabetes in Wales. This equates to a reduction in new certifications over the observation period for SI and SSI combined from 82.4 to 46.9 per 100 000 (−43.1%). The fall in SI was -43.0% and -49.5% in SSI. During the same period the number of people referred by DESW to hospital eye services for sight threatening retinopathy fell from 3.4% in 2007 to 2.0% in 2015.

A key limitation of the study is the non compulsory and inconsistent process for certification of visual loss. The strength of the study relates to its nationwide coverage, a unified database and the period it covers is when a national screening programme (DESW) was introduced with the objective to reduce severe sight impairment (blindness) through early detection and treatment.

The study builds on a previous paper (Liew et al, BMJ Feb 2014) which showed that, for the first time in at least five decades, diabetic retinopathy/maculopathy is no longer the leading cause of certifiable blindness among working age adults in Wales and England. However this recent paper goes further by quantifying the actual reduction in the whole diabetic population in Wales for the very first time.

It is likely that the reduction is partly due to a combination of improvements in the management and treatment of diabetes and diabetic retinopathy but highlights the significant positive benefits of the introduction of a community based, national screening programme in Wales for the early detection of sight threatening diabetic retinopathy.

Coming Full Circle

In looking back, it would be remiss not to recognise the person who more than anyone else helped to make DESW what it is today. Professor David Owens was not just our first clinical director but the true architect of the service. It was his vision for a single national programme that prevailed from which, as described, the people of Wales have benefited. How fitting then that David was the principle investigator on the publication above that proved that this vision had become a reality.

Andrew Crowder

Sgrinio Llygaid Diabetig Cymru Diabetic Eye Screening Wales

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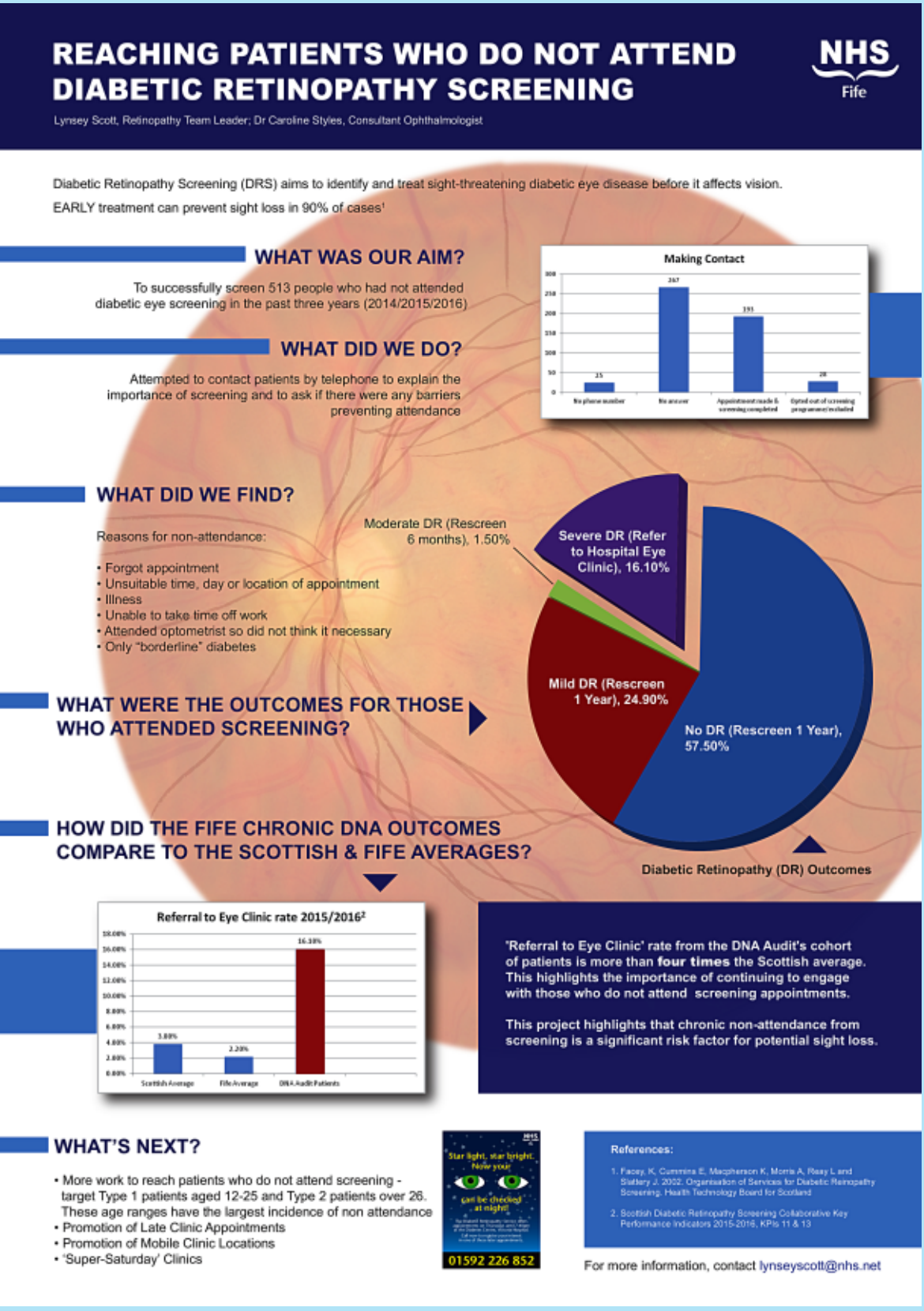
AT THE FOREFRONT OF OPHTHALMIC TECHNOLOGY

SUCCESS FOR FIFE DIABETIC RETINOPATHY SCREENING SERVICE



A project undertaken by NHS Fife's Diabetic Retinopathy Screening (DRS) Service received national recognition at the recent British Association of Retinal Screeners' (BARS) Annual Conference in Leeds.

First prize in the Poster Exhibition was awarded to Fife DRS for work done on looking at those patients who do not attend (DNA) their appointment for three consecutive years. The main purpose of the audit was to encourage these patients to attend, as their vision could be at risk, and to also identify any barriers to non-attendance. Examination of the outcomes for those who did attend for screening showed a four-fold increase in the number referred to the Hospital Eye Service due to referable retinopathy being present. This highlights the importance of everyone with diabetes attending annually to have their eyes screened.

DRS Team Leader, Lynsey Scott, was delighted to accept, on behalf of the team, the prize which includes the poster being submitted to the 2018 European Association for the Study of Diabetes Eye Complications (EASDec) Meeting and the abstract considered for publication in the European Journal of Ophthalmology. BARS also provide one free place at the 2018 EASDec Meeting in Belfast, including travel and accommodation.

Lynsey Scott
Team Leader / Advanced Diabetic Retinopsathy Specialist
Diabetes Centre, Victoria Hospital, Kirkcaldy, Scotland

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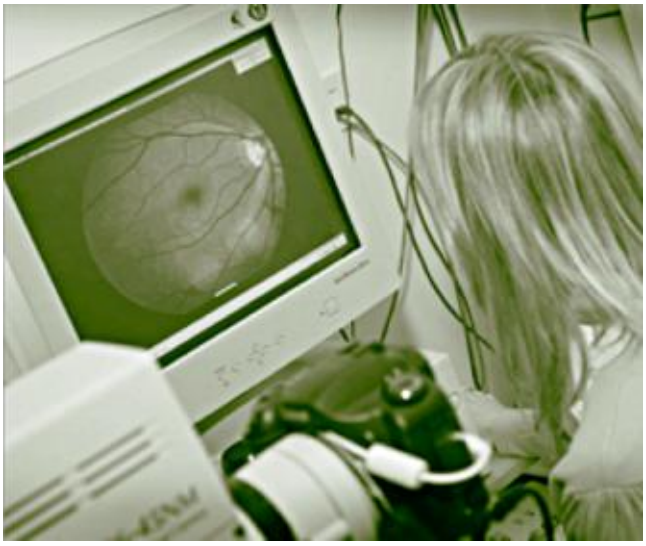
Diabetic Eye Screening - the last 10 years

The success of the NHS Diabetic Eye Screening Programme (NDESP) depends upon ensuring the early identification and appropriate treatment of patients with sight-threatening retinopathy.

The four UK nations were the first countries in the world to introduce systematic national screening programmes for diabetic retinopathy. The implementation of diabetic eye screening in England was announced in the 2003 Delivery Strategy for the National Service Framework for Diabetes and, by 2008, local retinal screening programmes covered the whole country.

External quality assurance (QA) was established in 2008, and when it started to look at programmes, it quickly became obvious there was a certain level of variation in almost every parameter.

It became apparent that the workforce delivering screening should be fully trained and accredited. A level 3 City & Guilds qualification was developed to meet the learning needs of anyone involved in the identification of sight-threatening diabetic retinopathy. The requirement to hold this certificate was introduced as a QA standard to ensure local programmes had trained and competent staff to carry out their screening roles in the most effective way.



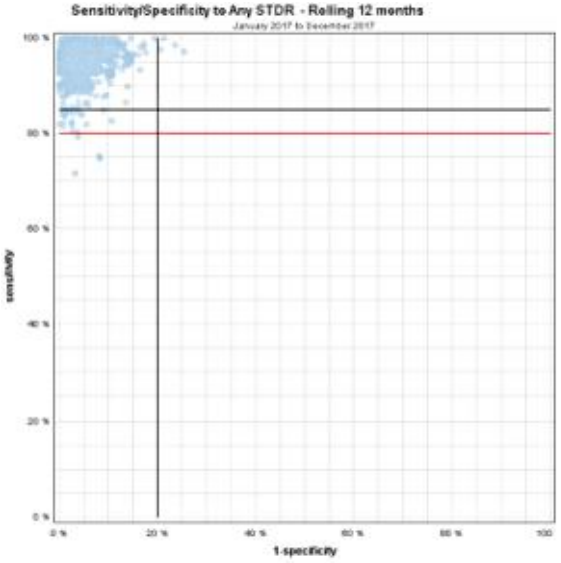
By 2012, annual screening for diabetic retinopathy was a well-established and essential component of effective healthcare for all people with diabetes aged 12 and across the UK. However, NDESP was aware of significant variation between local services in grading and referral processes. To address these issues, new grading criteria and a new common pathway for diabetic eye screening were implemented throughout England.

These developments introduced a more consistent approach to commissioning and screening delivery. It also meant more comparable data was collected so that NDESP could identify where quality needed improving.

The QA service developed and started to streamline its practices and QA visits, using evidence from newly introduced national standards. This ensured there was a mechanism to check local programmes were safe and effective.

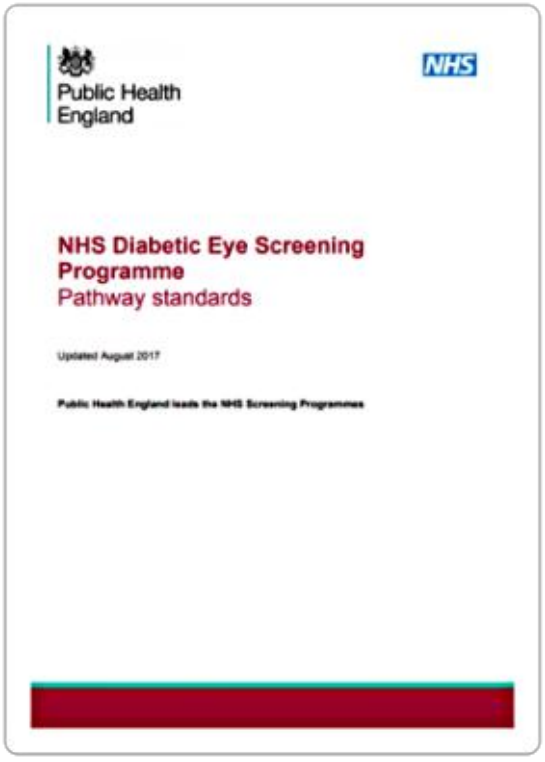
The process of cohort identification and transfer of patient information between GP practices and local screening programmes was another challenge. To address this, NDESP rolled out a major IT system called GP2DRS. This system streamlines the process of cohort identification so local programmes have easy access to the most up to date information for their diabetic patients. This is now available to all local programmes at no cost, and is the recommended tool for list validation and cohort identification.

In 2011, NDESP developed an online grading test and training (TAT) system. The soft launch of TAT introduced graders to the idea of testing their grading skills and knowledge against a consensus of grading results. The system was developed further and participation guidance was published. In 2015, local programmes were able to use this system as a performance monitoring tool for their graders. There are now 1,200 graders registered on the system and the test results show that English national graders have a high sensitivity and specificity to sight-threatening disease.



Each blue dot represents the sensitivity and specificity of an individual national grader participating in the TAT.

Those above and to the left of the black lines are all performing at the agreed national standard.



In August 2014, we updated the programme pathway standards to make sure they are fit for purpose and meet the requirements of changes in organisational structure across the NHS. The most recent versions of the pathway standards were introduced in April 2017.

When the NHS screening programmes moved into Public Health England in 2013, we reviewed the arrangements for training of screeners to make sure provision was equitable, accessible and sustainable. We developed a new national screener qualification to make sure there is a work-based training programme with a nationally recognised qualification and a pathway for screeners which supports career progression and opportunities in healthcare. After a lot of hard work and consultation, the level 3 Diploma for Health Screeners was launched in April 2016. Nearly 400 learners have registered for the new qualification to date.

In January 2016, following a review of the evidence against strict criteria, the UK National Screening Committee (UK NSC) recommended that the interval between screening tests should change from one year to two years for people with diabetes at low risk of sight loss. Screening this group less often will not only release capacity, but also lessen the inconvenience for this group in attending appointments every year.

This takes us to our current position in screening. NDESP is working closely with stakeholders to make sure that the introduction of extended screening intervals is safely implemented. This means having a robust IT system and proven consistent accurate grading in those local programmes moving over to the new pathway.

Between 1 April 2016 and 31 March 2017, we offered screening to nearly 3 million people with diabetes in England and nearly 2.5 million accepted the invitation.

Looking ahead

NDESP is looking at the role of optical coherence tomography (OCT) within the programme and whether we can develop best practice guidance to support local services that are looking at implementing an OCT service.

A health technology assessment has been commissioned to assess the role of scanning ophthalmoscopy in the imaging of the retina to detect diabetic retinopathy and whether we could use it within the screening programme at some point in the future.

Automation software to detect retinopathy is another future development we are keeping an eye on. We will look at the available evidence to determine what the potential may be for implementing automation in screening sometime in the future.

NDESP would like to take this opportunity to say a huge thank you to everyone working in the screening programme. Your hard work and dedication is having an enormous positive impact on the eye health of people with diabetes across the country. In fact, diabetic retinopathy is now no longer the leading cause of blindness in the working age population in England, due in part to the introduction of national screening.

Who knows what the else the next 10 years will bring?