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Fundus Autofluorescence: Should I Be Using it in My Practice?

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Fundus autofluorescence (FAF) is an emerging technology with several research and clinical applications. FAF imaging has demonstrated utility in diagnosing macular and retinal disease, identifying disease progression, and monitoring response to therapy.[1-4] It has the potential to help elucidate pathological mechanisms, understand the natural history of progressive diseases, stratify risk factors for progression, and improve the quality of therapeutic trials.[1,2,5] This issue of *Ophthalmology Rounds* describes the strengths and limitations of FAF, and outlines the ocular disorders for which it is best suited.

Pathophysiology

The retinal pigment epithelium (RPE) is a polygonal monolayer situated between the neurosensory retina and the vascular choroid that plays a vital role in vision, and its dysfunction has been linked to blinding retinal diseases.[1,2] Accumulation of lipofuscin granules in the cytoplasm of post-mitotic RPE cells is seen with oxidative damage and aging,[2,6] but may also represent a common downstream pathological mechanism involved in age-related macular degeneration (AMD), and inherited retinal dystrophies.[1,2] Lipofuscin is an oxidative end product of the continuous RPE-mediated phagocytosis of the outer segments of photoreceptors.[1,7] Once formed, the RPE is unable to degrade or transport the lipofuscin material into the extracellular space and these granules thus accumulate in the cytoplasmic space; various toxic compounds within the lipofuscin granules may interfere with normal cellular functioning.[1] While the pathophysiology is not yet completely understood, fundus autofluorescence (FAF) imaging permits morphological imaging of the common downstream pathway.[5]

Autofluorescence is the emission of fluorescent light from a structure when it is illuminated by an exogenous source.[7,8] Lipofuscin is a composite of >10 fluorophores, each with discrete emission spectra;[6] this variability results in a broad range of LF excitation (300–600 nm) with a similarly broad emission spectrum (480–800 nm with a peak in the 600–640 nm region).[2] The fluorophores are notable for an extended system of conjugated double bonds that permit absorption of light and resultant fluorescent emission.[2]

FAF Image Acquisition

FAF images can be obtained with relative ease and speed. Primary methods of FAF imaging include confocal scanning laser ophthalmoscopy (cSLO) and modified fundus camera (mFC) photography, which have differing benefits and limitations.

cSLO

The cSLO obtains images in a series of scan lines using a low-power laser beam following a raster pattern. This technique ensures that the reflectance and autofluorescence are derived from the same optical plane, which allows it to also minimize autofluorescence from anterior structures.[1,2] The cSLO is able to obtain high-quality images of a large retinal area, up to a 55° field with a single image. To address the limitation of low intensity, multiple images are recorded and aligned using a software algorithm; this permits better visualization of the fundus and the production of a mean image. However, this limits the

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clinician to only evaluating localized topographical differences within one image; mean images cannot be used for quantification or absolute comparison between different images.[1,2]

The Heidelberg Retina Angiograph (HRA)-based systems are the only commercial cSLO units currently available for FAF imaging. These units utilize an excitation wavelength of 488 nm using an argon or solid-state laser. In order to block reflected light while permitting autofluorescent light to pass, a 500-mm barrier filter is inserted in front of the detector.[2]

mFC

The mFC images the entire retina with a single flash; however, it lacks the ability to differentiate the source of autofluorescence.[6] As such, fluorescence is detected from the RPE layer, as well as anterior and posterior structures. Using light in the blue range (488 nm), this limitation is particularly significant in patients with yellowing of the lens and nuclear opacities. Spaide modified the FC to use longer excitation wavelengths (580 nm, bandwidth 510–670 nm) and longer barrier filters (695 nm, bandwidth 675–715 nm), and was able to reduce the autofluorescent contribution from the lens allowing for improved image quality.[1,2] However, these modifications also alter the FAF images; for example, the normally dark optic nerve head appears brighter than the surrounding retina with the mFC, and further research is required to standardize this approach.[1]

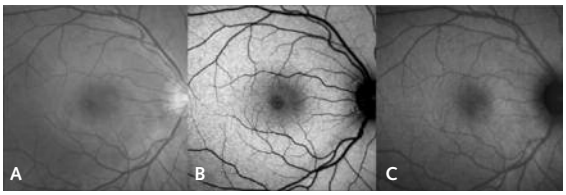
Limitations of FAF imaging

The intensity of FAF is about 2 orders of magnitude lower than that of intravenous fluorescein angiography (IVFA). Signal detection is also made difficult because of the autofluorescent properties of anterior structures in the eye, particularly the lens. These limitations present challenges when recording FAF, and are addressed uniquely by the different systems.[2]

Lipofuscin is composed of at least 10 different fluorophores, and the dominant fluorophore varies with the excitation wavelength utilized. As a result, each system may be recording intensity from different fluorophore sources. The early research has demonstrated similarities between the systems, although differences do exist; in particular, the cSLO demonstrates a darker signal over the retinal vessels and optic nerve.[2] These differences are evident in Figure 1, which shows a fundus photograph of a normal eye and normal FAF images obtained via cSLO and the mFC, respectively. Unfortunately, no currently available systematic comparison details the similarities and differences between the cSLO and mFC protocols in different pathological states.[1]

There is currently a lack of standardization between protocols with respect to laser powers, detector sensitivity, barrier filters, and technical factors such as eye movements, positioning of the chin rest, the camera-cornea distance, and adjusting for anterior structures.[1]

Figure 1: Photographs of a healthy eye. Note the different distribution of intensity in the fovea between confocal scanning laser ophthalmoscopy (cSLO) fundus autofluorescence (FAF) imaging (B) and fundus camera FAF imaging (C). This is due to different degrees of absorption of the excitation light by macular pigment.



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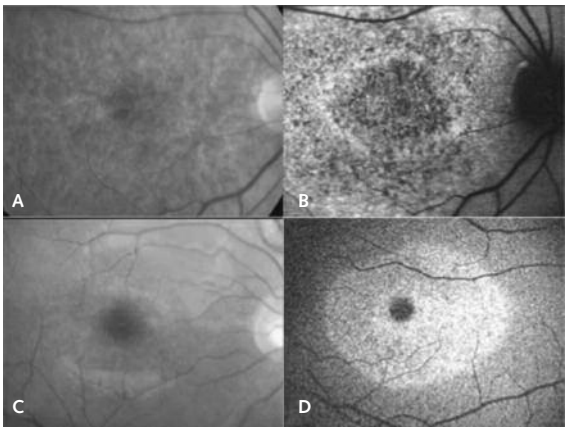
Interpretation of FAF Images

FAF images demonstrate the intensity of the signal on a grey scale; low pixel values appear dark and represent low intensity signals while high pixel values are bright and represent high intensity signals.[2] Deviation from normal is due to either a change in the quantity or quality of lipofuscin in the RPE or due to absorption or autofluorescence of anterior structures.[2]

Normal FAF

The FAF appearance of a normal eye has been consistently demonstrated. The optic nerve head appears dark because it lacks RPE, and thus lipofuscin. Due to absorption of light by blood, the retinal vessels also appear dark. In the macular area, there is absorption by luteal pigments with a corresponding decrease in FAF signal; the parafoveal area has a relatively higher

Figure 2: Colour fundus photography (CFP; A) and FAF (B) images demonstrate extensive disease of the macula in a patient with macular dystrophy and diminished vision with a known mutation of the retinal degeneration slow gene. Her 17-year-old son had no visual complaints and a normal-appearing fundus (C), but his FAF image (D) demonstrated a well-demarcated oval-shaped area of increased intensity in the central macular area, suggesting an abnormal phenotype.



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signal than the macula but is still relatively darker than the peripheral retina.[2,6] Utilizing pixel grey values, an FAF signal can be classified as decreased, normal, or increased intensity by qualitatively comparing it with the background signal in the same image.[1]

Increased and decreased FAF

The presence of lipofuscin within RPE results in hyperautofluorescence and indicates RPE pathology.[1,6,7] Increased FAF is also a marker of hereditary retinal disease and often precedes clinical disease (Figure 2).[8] Conversely, loss of autofluorescence signifies a decrease or absence of RPE and a diminished FAF signal[1,6,7] in keeping with RPE cell death or atrophy.[1,6-8]

Clinical Examples

AMD

AMD is a multifactorial disease that manifests with focal hypopigmentation and hyperpigmentation at the RPE level and drusen accumulation in the inner aspect of the Bruch membrane.[1,2,6,8] Colour fundus photography is a reproducible method of documenting and quantifying AMD; however, it is limited in distinguishing dead or nonfunctioning RPE from living depigmented RPE, and yellowish discolouration secondary to large drusen. Further, the predictive value of fundus photographs is poor; they are able to identify only 5%–7% of eyes that progress to late AMD within 5 years.[9]

Early and intermediate AMD

In early AMD, irregular pigmentation or drusen visible on fundus photography do not always directly correlate with FAF changes, and vice versa. FAF intensity within the vicinity of drusen may be increased, decreased, or normal.[1,2,6] FAF aberrations remote from fundoscopically evident alterations in early AMD may be suggestive of more widespread diseased areas and allow for earlier detection.[1] An 8-pattern classification system of FAF changes in early AMD has been developed: normal, minimal change, focal increased, patchy, linear, lacelike, reticular, and speckled.[2] Preliminary evidence suggests that patchy FAF may be more likely to progress to neovascular AMD. Focal changes are at higher risk for progression to geographic atrophy.[9]

A cause-and-effect relationship between lipofuscin accumulation and pathological progression of AMD has not been established.[8] There is some evidence to suggest that lipofuscin is toxic to RPE cells and increased lipofuscin is predictive of future cell death, while complete loss of autofluorescence signifies that cell death has already occurred.[7] Various lipofuscin compounds including A2-E (N-retinylidene-N-retinylethanolamine) may interfere with molecular mechanisms.[1,2]

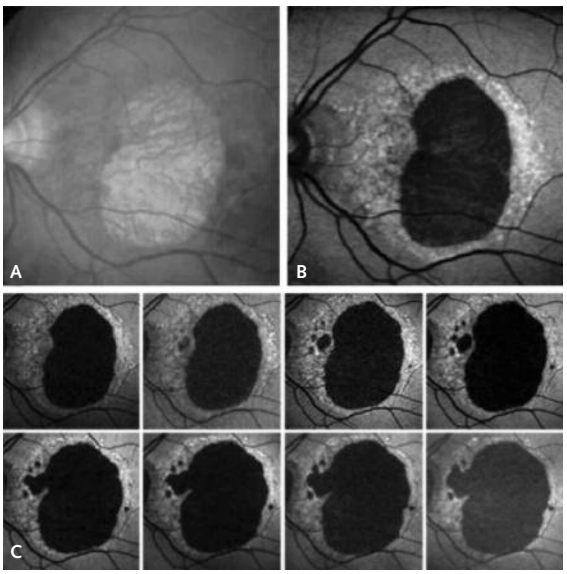
With respect to tracking disease progression, large drusen correspond well on fundus photographs and FAF images. However, change in drusen appearance on fundus photography may be better correlated with changes in FAF intensity on mFC-based than cSLO-based imaging. If using the latter, drusen on fundoscopic examination or photographs may need to be considered independently from FAF changes when monitoring disease progression.[1,2,9]

Geographic atrophy (GA)

In GA, the atrophic areas tend to enlarge over time and correspond to absolute scotomas and loss of retinal sensitivity.[5,6] It is hypothesized that excess lipofuscin in the RPE may be instrumental in the progression of GA; thus, increased FAF may precede the enlargement of preexisting atrophy or signal the development of new atrophic areas (Figure 3).[9]

FAF patterns have been identified at the junctional borders dividing normal retina from atrophic areas in AMD. These images have been classified into 4 primary phenotypes: focal, diffuse, banded, and patchy. The diffuse phenotype is further subclassified into reticular, branching, fine granular, trickling, and fine granular with peripheral punctate spots.[6,9] Atrophic progression was lowest in eyes without abnormal FAF (0.38 mm^2/year) and those with focal FAF abnormalities (0.81 mm^2/year), whereas eyes with diffuse FAF changes and banded FAF had greater progression (1.77 mm^2/year and 1.81 mm^2/year, respectively).[5,9]

Figure 3: Advanced age-related macular degeneration (AMD) with central geographic atrophy (GA) in the left eye on CFP (A) that corresponds to a similar kidney-shaped lesion of decreased intensity on FAF. The surrounding retina appears normal on the CFP, but there is increased signal around the atrophic regions on FAF (B) that may reflect retinal pigment epithelium (RPE) pathology. A series of FAF images over 6 years (C) demonstrate progression of atrophy in areas that were previously hyperautofluorescent.



Neovascular AMD

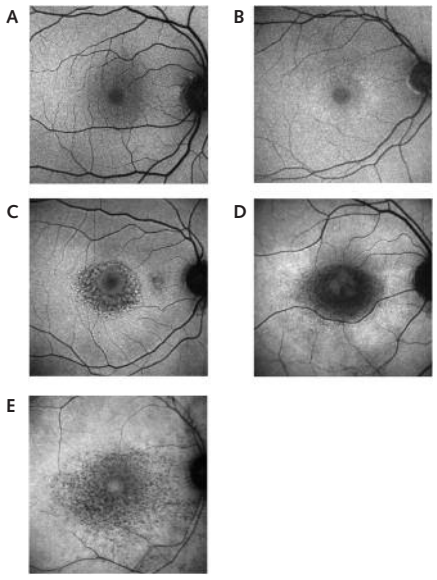
Hyperautofluorescence on FAF prior to treatment is associated with poor visual acuity before treatment, and is a negative prognostic factor for visual recovery following anti-VEGF treatment; in particular, increased intensity within 500 µm of the fovea leads to an unfavourable prognosis for visual recovery.[7]

Hydroxychloroquine toxicity

The retinopathy of hydroxychloroquine toxicity appears as a characteristic bilateral bull's-eye maculopathy – a ring of depigmented RPE with foveal sparing.[10] While the pathophysiology is not well understood, it is thought that the delayed progression is either due to decompensation of photoreceptor and RPE cells that were initially injured beyond repair or a continued reservoir of drug in the body due to drug clearance over a period of months.[11] FAF may show subtle RPE defects with reduced intensity or areas of early photoreceptor damage with increased autofluorescence that precede visual field loss (Figure 4).[10]

Recent American Academy of Ophthalmology guidelines recommend one of multifocal electroretinogram (mfERG), spectral domain optical coherence tomography (SD-OCT), or FAF as screening tools in managing patients on hydroxychloroquine.[10]

Figure 4: Variation of FAF findings in patients using hydroxychloroquine. (A) Normal FAF. (B) Pericentral ring of increased FAF. (C) Pericentral mottled loss of FAF with increased FAF in the adjacent peripheral area. (D) Pericentral ring with total loss of FAF with increased FAF in the adjacent peripheral area. (E) Mottled loss of FAF at the posterior pole and increased FAF in the adjacent peripheral retina.



Central serous chorioretinopathy (CSC)

FAF imaging findings in CSC vary with disease stage, and the extent of RPE involvement. Acute leaks will demonstrate minimal changes with some evidence of hyperautofluorescence locally of the serous detachment. The FAF findings become increasingly irregular with disease progression. Subretinal precipitates may appear as discrete areas of increased autofluorescence.[12] In eyes with chronic atrophic changes, FAF will demonstrate irregular hypoautofluorescence in these areas that can be subclassified as confluent or granular.[12,13] Confluent hypoautofluorescence appears as round or oval area of decreased FAF signal that is larger than one-quarter disc diameter. Granular hypoautofluorescence appears as a grainy or coarse area of decreased FAF signal that is greater than one-quarter disc diameter. A descending tract, or gutter, of decreased FAF intensity may be seen originating at the posterior pole and extending to below the level of the inferior arcade. These 3 findings and increased age are all independent predictors of decreased visual acuity in patients with CSC.

Retinal Dystrophies

Stargardt disease

In Stargardt disease, abnormal increased FAF intensity may be seen prior to detection of flecks or atrophy on clinical examination or fundus photography.[14] Uniform black central regions on FAF are associated with dense scotomas while patchy grey-black lesions on FAF represent areas of both scotomas and functioning retina.[15] Serial FAF imaging can be used to monitor disease progression. However, there is wide variation in pattern and degree between patients.[14,16]

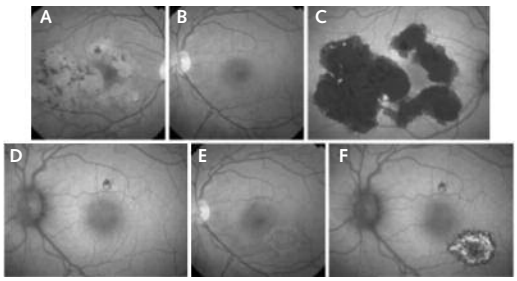
Retinitis pigmentosa (RP)

RP is a slowly progressive inherited disease that reduces visual function due to degeneration of photoreceptors and the RPE. More than half of RP patients have an abnormal high intensity parafoveal ring apparent on FAF. One-quarter of patients will demonstrate an abnormal central FAF with a high-density disciform lesion that spreads centrifugally from the fovea.[17] These ring structures demonstrate progressive constriction,[2] which is correlated with a loss of retinal sensitivity, visual acuity, and the inner segment/outer segment line on OCT.[17]

Best vitelliform macular dystrophy

Best disease presents with a characteristic finding of a circumscribed, yellow, vitelliform lesion.[18,19] Subretinal fluid may settle in the lesion, creating an appearance of a pseudohypopyon.[18]

Figure 5: CFP views of the right eye (A) of a patient with serpiginous choroidopathy with perifoveal geographic areas of chorioretinal atrophy with pigment mottling, and the left eye (B) with a small superior macular area of abnormal pigmentation at presentation. FAF images (C,D) at time of presentation demonstrate hypoautofluorescence corresponding to the areas of atrophic RPE. Two months later, there is evidence of a new large lesion with atrophic borders on both CFP (E) and FAF (F); note the mottled borders of the FAF image.



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The pseudohypopyon appears hyperautofluorescent on FAF imaging, suggesting excess accumulation of fluorophores.[2,19]

Posterior Uveitis

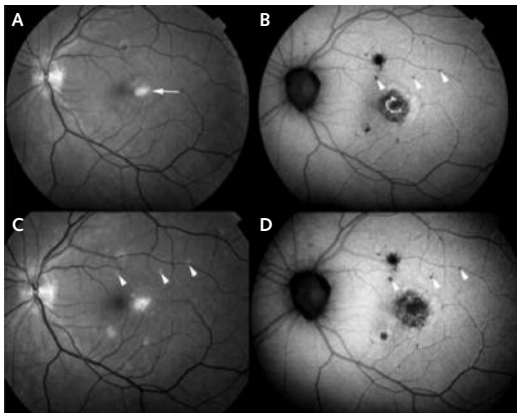
Birdshot chorioretinopathy

Birdshot chorioretinopathy is a rare posterior uveitis associated with human leukocyte antigen A29 and is characterized by hypopigmented choroidal lesions throughout the posterior pole.[20-22] On FAF imaging, birdshot lesions on fundus photography may appear hypoautofluorescent; however, birdshot fundus lesions do not always result in corresponding FAF changes.[20] Areas of hypoautofluorescence may be larger and more diffuse on FAF imaging in birdshot chorioretinopathy when compared to fundoscopic examination or fundus photography. This may reflect more widespread pathology than is visible and may be able to explain the vision and field loss in these eyes.[23] The lesions are more apparent on FAF, particularly in eyes with light fundus pigmentation that makes identification of birdshot lesions difficult.[20,21]

Serpiginous choroidopathy

Serpiginous choroidopathy is a rare bilateral chronic and recurring inflammatory disease of the inner choroid and RPE. Previously active areas with chorioretinal atrophy are hypoautofluorescent on FAF while areas of active choroiditis have been shown to be hyperautofluorescent.[23] The areas of hyperautofluorescence correlate well with the eventual areas of decreased signal, which suggests that FAF imaging is able to identify the involved RPE areas (Figure 5). Further, the clearly defined borders of the lesion permit evaluation of macular involvement.[24]

Figure 6: Fundus photographs and FAF images of the right eye of a 36-year-old woman with multifocal choroiditis and panuveitis with choroidal neovascularization that was treated with photodynamic therapy and corticosteroids (A,B). The baseline FAF image (B) reveals multiple hypoautofluorescent spots that do not correspond to any abnormalities on fundus photographs. Three years later, the patient developed sudden photopsias, and there are new fundus spots (C) that correspond to the previously identified FAF lesions. The repeat FAF (D) shows enlargement of the same lesions but no new spots in this eye.



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Serpiginous-like choroiditis

Serpiginous-like choroiditis presents with multifocal choroidal lesions in younger patients in tuberculosis-endemic areas. Fundus appearance is similar to that of serpiginous choroiditis. The acute stage FAF shows a poorly defined diffuse halo of increased intensity. Over 2-4 weeks, the halo fades as the lesion heals, and a thin well-defined rim of decreased intensity with central hyperautofluorescence appears. Months later, once the choroiditis lesions have healed, the lesion appears uniformly dark. This gradual sequence from bright to dark lesions reflects degeneration and atrophy of RPE cells during the healing phase. During the later stages of healing, fundus photographs may suggest inactive disease but a stippled pattern of grey-black mixed autofluorescence indicates ongoing metabolic activity.[25]

Multifocal choroiditis and panuveitis

Multifocal choroiditis and panuveitis presents with small round chorioretinal scars, in either a scattered or linear pattern, peripapillary scarring, possible choroidal neovascularization, vitreous cells, and chorioretinal infiltrates.[26] FAF imaging demonstrates hypoautofluorescence in areas with chori-

oretinal atrophy while acute exacerbations are marked by a hyperautofluorescent signal that improves with immunosuppressive therapy. Further, punctate hypoautofluorescence may be seen in areas without associated findings on fundus photographs and may reveal subclinical findings (Figure 6).[23] Typically, FAF lesions >125 µm are visible on colour fundus photography while smaller lesions are not. These smaller lesions are most often in the peripapillary and macular regions, and often become evident on follow-up colour fundus photographs. Lesions may be seen more clearly on FAF images than colour fundus photography and IVFA.[26]

Cytomegalovirus (CMV) retinitis

Recurrence of CMV retinitis can be difficult to detect in subtle or atypical cases, especially in the granular form, and FAF imaging may play a role in diagnosis. An increased FAF signal can be observed along the advancing edges of the lesion, and can be useful for detecting recurrence.[23,27] Increased autofluorescence is also reported in syphilitic chorioretinitis.[23]

Summary

FAF is a novel and emerging technique that permits rapid and noninvasive imaging of retinal diseases. While the exact pathophysiology of RPE dysfunction and the role of accumulated lipofuscin are not yet completely understood, FAF permits imaging of the common downstream pathway. It allows for topographic imaging of lipofuscin distribution and provides information not available through other techniques such as fundus photography, IVFA, or OCT.[12]

FAF imaging has a clear role in helping elucidate the pathophysiology of macular and retinal disease. Further research is required to standardize image acquisition protocols for both mFC and cSLO-based systems, and to determine the utility of FAF imaging in monitoring response to therapy, and identifying disease progression. Classification systems for prognostication are already being developed and will continue to be refined.

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