Editorial: Cellular debris—a key to the aging macula

The greatest socioeconomic problem confronting the Western World is the progressive aging of its population. This change presents special problems for the ophthalmologist, as all surveys show a dramatic increase of prevalence of blindness with increasing age. A review of the principal diagnoses given in relation to blind registration in people over 65 shows that almost 50% of blind registrations result from macular disease.

It is perhaps significant that over the last few years many research groups throughout the world have directed their efforts towards understanding the aging processes occurring in the outer retina. Many studies seem to indicate that with increasing age the constant renewal of the light sensitive membranes of photoreceptor cells may present problems for the retinal pigment epithelium and in turn Bruch's membrane.¹ The implications are that the constant phagocytosis of disc membranes and their subsequent degradation by lytic enzyme systems within the pigment epithelium eventually result in the progressive accumulation of debris within pigment epithelial cells.² By the fourth decade extracellular debris is also seen accumulating in the innermost layers of Bruch's membrane. Sarks³ has elegantly demonstrated in her clinicopathological studies that in many individuals the extracellular deposits, possibly genetically predetermined, play a significant part in the generation of age related macular disease. The spectrum of disturbances in the aging retina ranges from extensive subpigment epithelial deposits, through drusen, to pigment epithelial detachments and subretinal neovascular complexes. Extracellular debris may not only be a causal agent in loss of function in the aging macula, but may also account for some of the variations or patterns seen on clinical examination.

A recent paper⁴ has postulated as a causal agent the constant deposition of the waste products of phospholipid degradation by the pigment epithelium, eventually resulting in an aggregation of lipid within the innermost layers of Bruch's membrane, the presence of such a hydrophobic layer impeding the passage of water moving from the retina towards the choroid.⁵ In elderly people such a barrier may well be the causal agent of pigment epithelial detachments, in that as the pigment epithelium pumps water from the subretinal space towards the choroid it literally pumps itself off Bruch's membrane.

Clinically, Hoskin *et al*⁶ subdivided pigment epithelial detachments into those in which hyperfluorescence of the detached pigment epithelium was uneven and those in which it was even. This subdivision was considered an important indicator in prognosis, as even hyperfluorescence indicated a predisposition for the detached epithelium to rip. The authors postulated that the dichotomy in clinical appearance of these two types of detachments may arise through differences in cleavage planes as the cells leave Bruch's membrane. Detachments with uneven hyperfluorescence were postulated to occur as a result of the pigment epithelium becoming detached from Bruch's membrane and taking with it both its basement membrane and some of the underlying extracellular debris which generally lies between the basement membrane and the inner collagenous layer of Bruch's membrane. In contrast in those lesions with even hyperfluorescence the concept was that the pigment epithelium became detached, leaving its basement membrane behind.

The scanning electron microscope study presented in this issue by Yamamoto and Yamashita is of particular interest in relation to the possible locations of differential cleavage planes within Bruch's membrane. Although the cleavage planes illustrated result from deliberate histological techniques, they show clearly that the pigment epithelium can be displaced from its basement membrane and that the basement membrane itself may be detached from the underlying inner collagenous layer of Bruch's membrane, and, in its becoming so, debris lying between the basement membrane and the inner collagenous layer may either remain on the collagenous layer or become detached from it and adhere to the basement membrane of the pigment epithelium.

JOHN MARSHALL

References

- 1 Hogan MJ. Role of the retinal pigment epithelium in macular disease. *Ophthalmology* 1972; **76**: 64–80.
- 2 Feeney-Burns L, Hilderbrand ES, Eldridge S. Ageing human RPE: morphometric analysis of macular, equatorial and peripheral cells. *Invest Ophthalmol Vis Sci* 1984; **25**: 195–200.
- 3 Sarks SH. Aging and degeneration in the macular region: A clinicopathological study. *Br J Ophthalmol* 1976; **60**: 324–41.
- 4 Bird AC, Marshall J. Retinal pigment epithelial detachments in the elderly. *Trans Ophthalmol Soc UK* 1986; **105**: 674–82.
- 5 Fisher RF. The influence of age on some ocular basement membranes. *Eye* 1987; **1**: 184–9.
- 6 Hoskin A, Bird AC, Sehmi K. Tears of detached retinal pigment epithelium. *Br J Ophthalmol* 1981; **65**: 417–22.