Progression from early- to late- AMD and development of GA and/or neovascular AMD is a complex process. The underlying mechanisms that cause an eye to develop GA versus neovascular AMD are not fully understood. No reliable genetic or environmental risk factors have been identified to predict whether a patient will develop one form or the other.1 Both types can occur simultaneously in the same eye, or simultaneously in different eyes.2 It has in fact been suggested that GA and neovascular AMD are not mutually exclusive diseases, but that they lie on the same disease continuum. Eyes developing both types may in fact be at a more advanced stage than either GA or neovascular AMD alone.2

The rate of progression varies between patients; some people progress quickly to late-stage disease (either GA or neovascular AMD), whereas others may progress slowly over several years.3

Long-term epidemiological studies evaluating the natural history and progression of AMD, including the Blue Mountain Eye Study and Beaver Dam Eye study, have reported that the presence of large drusen and pigmentary abnormalities are associated with a greater long-term risk of developing late-stage AMD (either GA or neovascular AMD).4 Recent studies have also reported the presence of reticular pseudodrusen (RPD) as a strong predictor for progression to late-stage AMD, particularly GA.5,6 RPD are subretinal deposits that appear on colour images as yellowish interlacing networks in the fundus.7

An analysis of an Australian community-based cohort (the Melbourne Collaborative Cohort Study) reported a prevalence of RPD of 0.41% in a population aged 48–86 years (87 of

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Progres
tion to Late-AMD  
(GA & Neovascular AMD)

21,130 participants). The study found that the presence of RPD was linked with development of late-stage AMD, and this association was stronger with GA than with neovascular AMD.\(^6\) The precise role of RPD in the progression of AMD is still unclear. However, the strong correlation between the presence of RPD and GA, and the observation that GA lesions tend to develop in regions of the macula where RPD lesions exist, has led to the suggestion that RPD lesions are an early manifestation of the process leading to GA.\(^5\)

The proportion of people reported to develop both GA and neovascular AMD in the same eye varies widely between clinical studies. The Beaver Dam Eye Study reported that among eyes with GA at baseline, 11% progressed to neovascular AMD after 5 years; the development of neovascular AMD was more frequent if it was present in the fellow eye.\(^8\) Another study investigating the GA/neovascular AMD phenotype reported that GA usually occurs prior to the development of neovascular AMD.\(^1\)

Epidemiological studies conducted for the US and UK populations have shown that GA and neovascular AMD occur with approximately equal prevalence, among people with late-stage AMD.\(^9,10\)

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