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REPLY

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REPLY

We thank Mathis et al for their comments on our publication[1] in which we demonstrated that 49% of persons ≥60 years of age with early age-related macular degeneration (AMD) had subretinal drusenoid deposits (SDD, also called reticular pseudodrusen) assessed with multimodal imaging including spectral-domain optical coherence tomography. We especially thank Mathis et al for bringing to our attention that their group had previously reported 71% prevalence of SDD in patients with early AMD in their retina clinic-based study,[2] and not 31%, as we incorrectly cited.

In eyes at different AMD stages and in eyes in good macular health,[1,3] SDD follow the topography of rod photoreceptors[4] and are frequently found along the superior vascular arcades and in the peripapillary area nasal to the optic nerve head. Owing to this topography, most SDD lies outside the Early Treatment Diabetic Retinopathy Study grid, and thus out of the range of commonly used diagnostic technologies and progression metrics. In our study we determined prevalence within an expanded ascertainment area, namely, spectral-domain optical coherence tomography volumes of both macula and optic nerve head.[1] Likewise, Mathis et al determined prevalence within a larger macular volume (30°×25°) than typically used in clinic-based studies.[2]

We agree wholeheartedly that the 49% to 71% prevalence of SDD in early AMD reported in these 2 publications underscores the urgency of understanding the role of these recently recognized lesions in the progression to advanced AMD. We further echo the importance of extramacular, periarcade spectral-domain optical coherence tomography scans in addressing these questions. Currently, physicians are unlikely to examine optical coherence tomography images outside the macula in the absence of pathology via en face imaging, which we showed is rare with sparse SDD lesions not forming a pattern.

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We appreciate the interest of Mathis et al in our hypothesis that the biogenesis of both SDD and drusen involves an outer retinal lipid-recycling program, with participation of the retinal pigment epithelium squarely in the middle. Fortunately, new data on the regulation of cholesterol homeostasis in outer retina are now emerging from laboratory studies.[5] Thus, our understanding of the biology of both SDD and drusen will be eventually clarified.

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