Profiles of Macular Pigment Optical Density and Their Changes Following Supplemental Lutein and Zeaxannthin

We congratulate Zeimer et al.[1] on their article, entitled "Profiles of macular pigment optical density and their changes following supplemental lutein and zeaxannthin," which, we believe, represents an important step toward a better understanding of the pathophysiology of macular pigment (MP).

In brief, Zeimer et al. have shown that supplemental lutein (L) and zeaxanthin (Z) fail to augment MP centrally in the presence of "ring-like structures," a finding that is consistent with our recent report.[2] Also, we concur that these "ring-like structures," identified on autofluorescence, are representative of the central "dips" identified on heterochromatic flicker photometry, where the spatial profile does not exhibit a central peak with its typical monotonic decline from the foveal center. It is unsurprising, therefore, that supplementation with all three macular carotenoids (L, Z, and meso-zeaxanthin [MZ]) does augment MP centrally, given that MZ is the dominant carotenoid at this location.[3] The importance of this finding rests on the fact that vision is sharpest centrally, where the dual optical benefits of MP (attenuation of chromatic aberration with consequential enhancement of contrast sensitivity, and attenuation of blue light scatter with enhancement of visual performance under conditions of glare) are of greatest importance.[4]

With respect to the putative protective role of MP for age-related macular degeneration (AMD),[5] however, we would urge a note of caution in Zeimer et al.'s interpretation of Dietzel et al.'s recent study.[6] If MP is protective against AMD, it exerts that protective effect through its passive (filtering) and active (free radical neutralizing, or antioxidant) properties,[7] thereby ameliorating the chronic and cumulative oxidative damage in the central retina, and inflammatory consequences of same (which will be dependent upon genetic background).[8,9] Therefore, MP (and MP profiles), some considerable time before the onset of AMD, should be the subject of interest, prompting our team to conduct a study demonstrating that a relative lack of MP,[10] and spatial profiles exhibiting a central "dip" (equivalent to "ring-like structures"),[11] are each and independently associated with established risk-factors for AMD before disease onset. Furthermore, and given that AMD (even early disease) is associated with loss of photoreceptors,[12] and given that L, Z, and MZ are intracellular compounds,[13] any atypical measurements of MP found in eyes afflicted with AMD are likely to be the consequence of these pathologic changes, and causality certainly cannot be inferred.

In summary, and once again, we congratulate Zeimer et al.[1] on their excellent contribution to this growing area of interest, but we believe the prevailing current body of evidence indicates that a lack of MP and the presence of "ring-like structures," before disease onset, are associated with risk for AMD.

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