

A review of the putative causal mechanisms associated with lower macular pigment in diabetes mellitus

Grainne Scanlon, James Loughman, Donal Farrell and Daniel McCartney

Technological University, Dublin

Purpose: Macular Pigment confers potent antioxidant and anti-inflammatory effects at the macula, and may therefore, protect retinal tissue from the oxidative stress and inflammation associated with ocular disease and aging. There is a body of evidence implicating oxidative damage and inflammation as underlying pathological processes in diabetic retinopathy, a major cause of vision impairment and blindness. Macular pigment has therefore become a focus of research in diabetes. This review explores the currently available evidence pertaining to MP levels in diabetes, and illuminates the potential metabolic perturbations implicated in MP depletion in diabetic eye disease.

Methods: The review was carried out in two stages. Firstly we identified all relevant published articles from human and animal studies which reported on the relationship between MP (lutein and/or zeaxanthin and/or meso-zeaxanthin) and diabetes (Type 1 & Type 2), up until the year 2019. The second part of the search involved identifying publications which investigated the relationship between the metabolic perturbations typically associated with diabetes, and Type 2 diabetes in particular (e.g. adiposity/dyslipidaemia) and MP. PubMed, Google Scholar, EMBASE, Mendeley, Medline Plus

and Scopus were used to search for literature of relevance to MP and diabetes.

Results: Metabolic co-morbidities commonly associated with Type 2 diabetes such as overweight/obesity, dyslipidaemia, hyperglycaemia and insulin resistance, may have added and independent relationships with MP. Increased adiposity and dyslipidaemia may adversely affect MP by compromising the availability, transport, and assimilation of these dietary carotenoids in the retina. Furthermore, carotenoid intake may be compromised by the dietary deficiencies characteristic of Type 2 diabetes, thereby further compromising redox homeostasis.

Conclusion: Candidate causal mechanisms to explain the lower MP levels reported in diabetes include increased oxidative stress, inflammation, hyperglycaemia, insulin resistance, overweight/obesity and dyslipidaemia; factors, which may negatively affect redox status, and the availability, transport and stabilisation of carotenoids in the retina. Further study in a diabetic population is warranted to fully elucidate these relationships.

e:grainne.scanlon@TUDublin.ie