

Letter to the Editor

Impact of dietary carotenoid deprivation on macular pigment and serum concentrations of lutein and zeaxanthin

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Macular pigment (MP), composed of the dietary carotenoids, lutein (L), zeaxanthin (Z) and *meso*-Z (MZ), is known to possess antioxidant and photoprotective properties, and is believed to thereby confer protection against age-related macular degeneration (AMD). Accordingly, efforts to augment MP by means of dietary fortification and/or supplementation have been explored. Carotenoids cannot be synthesised by the body *de novo*, and are entirely of dietary origin. To date, the effect of dietary deprivation of the macular carotenoids has not been studied in human subjects. Here, we report findings from a unique experiment designed to investigate the effect of such dietary deprivation.

The experiment was approved by the Research Ethics Committee at the Dublin Institute of Technology. A single subject (J. L.) was deprived of dietary carotenoids by limiting food consumption to lean protein and eliminating all known sources of L and Z from the diet for a period of 42 d. Energy intake declined to approximately 6694 J/d (1600 cal/d). Heterochromatic flicker photometry was used to measure MP optical density (MPOD)^(1,2). BMI, MPOD at 0.25, 0.50, 1.0, 1.75 and 3.0° retinal eccentricity (using the Macular Metrics Densitometer™) and serum concentrations of L and Z were measured prior to commencement of carotenoid deprivation (days -14, -7 and 0), at regular intervals over the 42 d deprivation period (days 0 to 42) and then after a further 14 d repletion on a normal diet (day 56).

Central (0.25°) and average MPOD declined by 31 and 43%, respectively, during the period of dietary carotenoid deprivation (Fig. 1, Table 1), with trough levels being reached at day 21 (Table 1).

Serum concentrations of L and Z decreased by 71 and 62%, respectively, after 21 d of deprivation and stabilised thereafter (Table 1). There was also a 9% decrease in BMI (Table 1).

The observation of rapid depletion of serum L and Z concentrations over 21 d of dietary deprivation of the macular

carotenoids and recovery following resumption of a normal diet is consistent with rapid serum and plasma carotenoid depletion observed ($t_{1/2}$ 7 d) in larger studies in chicks⁽³⁾ and in human subjects⁽⁴⁾. The observed decline in MPOD, however, represents a novel finding, and although not directly comparable, is inconsistent with previous studies⁽⁵⁾. The trough plateau of MPOD and serum L and Z concentrations after 21 d of dietary deprivation of the macular carotenoids, observed here, may be explained by the release of adipose-stored L and Z as a consequence of weight loss (reflected in the falling BMI), offsetting continued decline of these parameters⁽⁶⁾.

The recovery of 40% of lost MPOD within 14 d of resumption of a normal diet, as observed here, is supported by recent evidence that supplementation with MP carotenoids, including MZ, augments MPOD across its spatial profile within 14 d⁽⁷⁾. Although an individual's MPOD is dependent on many factors, including dietary intake of the relevant carotenoids, gastrointestinal absorption, transport in serum and mechanisms influencing capture and stabilisation of these compounds in the central retina (including local tissue oxidative stresses and concentrations of co-antioxidants), our findings (albeit in a single subject) indicate that MP levels may be much more sensitive to dietary changes than hitherto believed.

Given the emerging evidence germane to the role of nutrition in AMD, and the important contribution of MP to visual performance and experience, researchers in this field should be aware that rapid fluctuations in MP, in response to a dramatic change in diet, could influence the interpretation of their findings. Furthermore, clinicians should give consideration to the possible adverse effects of nutritionally compromised diets on long-term ocular health and should reinforce, in patients, the importance of a stable and carotenoid-rich diet for optimisation of MP levels, visual health and risk of ocular disease such as AMD.

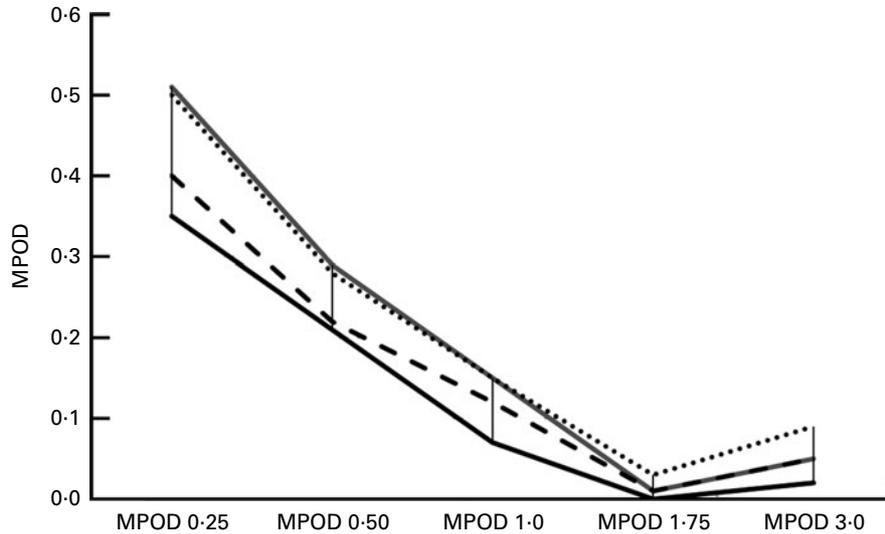


Fig. 1. Spatial profile of macular pigment (MP) during the first 21 d of carotenoid deprivation. MPOD, MP optical density. —, MPOD (day 0); ---, MPOD (day 7); ----, MPOD (day 14); —, MPOD (day 21).

Table 1. Macular pigment optical density (MPOD), serum lutein (L) and zeaxanthin (Z) concentrations and BMI during the experiment

Deprivation day	Central MPOD (0.25° retinal eccentricity)	Average MPOD	Serum L (μmol/l)	Serum Z (μmol/l)*	BMI (kg/m ²)
- 14	0.50	0.21			31.94
- 7	0.49	0.20			31.96
0	0.51	0.20	0.92	0.22	31.96
7	0.50	0.21	0.51	0.13	30.34
14	0.40	0.16	0.31	0.09	30.03
21	0.35	0.13	0.27	0.08	29.70
28	0.37	0.14	0.33	0.10	29.22
42	0.36	0.15	0.28	0.09	28.73
14 d repletion	0.42	0.17	0.63	0.17	28.09

*Total zeaxanthin, including Z and meso-Z.

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