ANTI-INFLAMMATORY EFFECTS OF LUTEIN IN RETINAL ISCHEMIC INJURY: IN VIVO AND IN VITRO STUDIES

S.-Y. Li¹, F.K. Fung¹, H.H. Chan², D. Wong¹,³, A.C. Lo¹,³

¹Eye Institute, The University of Hong Kong, ²School of Optometry, The Hong Kong Polytechnic University, ³Research Center of Heart, Brain, Hormone and Healthy Aging, The University of Hong Kong, Hong Kong, Hong Kong S.A.R.

Purpose: Lutein has been shown to protect retinal neurons from damage during retinal ischemia/reperfusion (I/R), possibly through both anti-oxidative and anti-apoptotic properties. As inflammation plays a critical role in I/R injury, the anti-inflammatory effect of lutein was investigated in the present study.

Methods: Unilateral retinal I/R was induced by the blockade of internal carotid artery using intraluminal method in C57Bl/6N mice. Ischemia was maintained for 2 hours followed by 22 hours of reperfusion during which either lutein or vehicle was administered. Electroretinography (ERG) and GFAP activation were examined. An in vitro model of induced hypoxia was also used to elucidate the effects of lutein on Muller cells. Western blotting of IL-1β, Cox-2, TNFα, and NFκB were performed.

Results: Lutein treatment minimized the deterioration in ERG response and activation of GFAP in the animal model of retinal I/R injury. Decreased levels of IL-1β and Cox-2, but not TNFα, were observed in the cell culture model of hypoxia. In addition, the level of nuclear fraction of NFκB was also decreased in the lutein treatment group.

Conclusions: Retinal function was preserved with lutein treatment. Reduced production of inflammatory factors from Muller cells was noted, suggesting an anti-inflammatory role of lutein. Together with our previous study, these results suggest that lutein protects the retina from ischemic/hypoxic damage by its anti-oxidative, anti-apoptotic and anti-inflammatory properties.

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