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

S.-Y. Li[1], F.K. Fung[1], H.H. Chan[2], D. Wong[1,3], A.C. Lo[1,3]

[1]Eye Institute, The University of Hong Kong, [2]School of Optometry, The Hong Kong Polytechnic University, [3]Research 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.

COI/Financial disclosure: This research was supported by the grants from the Hong Kong Research Grants Council (GRF #HKU773210M) and the University Development Fund from The University of Hong Kong.