PE17) Lutein Acts Via Antioxidant Pathway in the Oxidative –stressed Cellular Senescence

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We observed the induction of HO-1, NQO1, and the activation of Nrf2 on Lutein treatment. As is known, important roles of HO-1 and NQO1 are to protect cells from various stress. These results indicated that HO-1 induction via Lutein activated cellular protective or/and anti-oxidative effects. We confirmed the SIRT1, SIRT3 expression regulation of Lutein in H_2O_2 treated ARPE-19 cells. SIRT3 was increased by H_2O_2 and Lutein treatment, this data that Lutein is associated with inhibition of senescence. Also, the phosphorylation of SIRT1 was increased after treatment with H_2O_2 and Lutein in a dose-dependent manner. Therefore, our data indicated that Lutein effectively protected against H_2O_2-induced senescence via up-regulation of SIRT1, SIRT3.

In conclusion, the results suggested that Lutein, prevents H_2O_2-induced cellular senescence by oxidative stress by scavenge cellular ROS and decreased SA-b-gal positive cells. Furthermore, Lutein up-regulated the HO-1 and NQO1 and protected ARPE-19 cells. Also Lutein restored cell cycles though p53-p21 pathway regulation, Lutein up-regulated the SIRT1 and SIRT3, it protected ARPE-19 cells against cellular senescence by oxidative stress. Therefore, Oxidative stress induced cellular senescence is one of the important factors in the pathogenesis of Age-related Macular Degeneration (AMD). However, we confirmed that Lutein involvement about senescence regulation and protective of ARPE-19 cells. Therefore, it may be a potential therapeutic strategy for treatment of retinal-based disease.