

عنوان مقاله:

The Investigation of the Induction of Diketocarotenoids Senescence in SHSY-&Y Cells

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خلاصه مقاله:

Introduction: Recently, studies of diketocarotenoids such as astaxanthin (Ax) and canthaxanthin (Cx) with powerful antioxidant have focused on numerous biological mechanisms such as singlet oxygen quenching, radical scavenging, anti-diabetic, anti-carcinogenesis, anti-inflammatory, anti-obesity and anti-melanogenesis activities. There is evidence demonstrating that diketocarotenoid confers neuroprotective effects in experimental models of chronic neurodegenerative disorders and neurological diseases. This study used Ax and Cx to detect its role on senescence of SHSY-&Y Cells. Methods: In this study, the sample included the cell control group (SH-SY&Y cell line) that did not receive Ax and Cx, , and the experimental group that received Ax and Cx (Yo mM). Ax and Cx were treated with SH-SYAY cell line at FA hours. To measure the expression of BAX, Bcl-Y and PPARy different groups were compared by real-time PCR analysis. The cell senescence effects of Ax and Cx, a β-galactosidase (SA-β-gal) senescence assay was evaluated. The results were analyzed by the one-way analysis of variance (ANOVA) using Prism version 5.0 software. Results: The results showed that treatment with Ax and Cx (Yo mM) for FAh induced apoptosis and senescence. The BAX and Bcl-Y gene expression analysis revealed a significant impact of Ax and Cx in apoptosis induction (P<...Δ). The measuring of cell senescence also indicated that Ax and Cx exhibited a senescence inductive activity as determined by an increase in β-galactosidase activity and PPARγ gene expression (P<o.o\alpha). Conclusion: It appears that Ax and Cx have therapeutic properties in SH-SYaY cells and can cause the proliferation of these cells to cease. The results suggest that Ax and Cx treatment may be beneficial for therapy of neuroblastoma and .neurodegenerative disorders

كلمات كليدى:

Astaxanthin, Canthaxanthin, Apoptosis, Senescence, Neuroblastoma, SHSY-\(\Delta\)Y Cells

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