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Astaxanthin Induces Cytotoxicity through Down-Regulating Rad51 Expression in Human Lung Cancer Cells

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Abstract : Astaxanthin has been demonstrated to exhibit a wide range of beneficial effects including anti-inflammatory and anti-cancer properties. However, the molecular mechanism of astaxanthin-induced cytotoxicity in non-small cell lung cancer (NSCLC) cells has not been identified. Rad51 plays a central role in homologous recombination and high levels of Rad51 expression are observed in chemo- or radioresistant carcinomas. In this study, astaxanthin treatment inhibited cell viability and proliferation of two NSCLC cells, A549 and H1703. Treatment with astaxanthin decreased Rad51 expression and phospho-AKT protein level in a time and dose-dependent manner. Furthermore, expression of constitutively active AKT (AKT-CA) vector significantly rescued the decreased Rad51 protein and mRNA levels in astaxanthin-treated NSCLC cells. Combined treatment with PI3K inhibitors (LY294002 or wortmannin) and astaxanthin further decreased the Rad51 expression in NSCLC cells. Knockdown of Rad51 enhanced astaxanthin-induced cytotoxicity and growth inhibition in NSCLC cells. These findings may have implications for the rational design of future drug regimens incorporating astaxanthin for the treatment of NSCLC.

Keywords : astaxanthin, cytotoxicity, AKT, non-small cell lung cancer, PI3K
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