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View Abstract

CONTROL ID: 4051342**SUBMISSION ROLE:** Abstract Submission**AUTHORS****AUTHORS (LAST NAME, FIRST NAME):** Paraoan, Luminita I.¹; Suwanmanee, Gunticha^{1, 2}; Grimes, Daniel¹; Matei, Ioan V.¹; Manochantr, Sirikul^{2, 3}**INSTITUTIONS (ALL):** 1. Ocular Molecular Biology and Mechanisms of Disease, Edge Hill University, Liverpool, United Kingdom.
2. Thammasat University Faculty of Medicine, Khlong Nueng, Pathum Thani, Thailand.
3. Center of Excellence in Stem Research and Innovation, Thammasat University Faculty of Medicine, Khlong Nueng, Pathum Thani, Thailand.**Commercial Relationships Disclosure:** Luminita Paraoan: Commercial Relationship: Code N (No Commercial Relationship) | Gunticha Suwanmanee: Commercial Relationship: Code N (No Commercial Relationship) | Daniel Grimes: Commercial Relationship: Code N (No Commercial Relationship) | Ioan Matei: Commercial Relationship: Code N (No Commercial Relationship) | Sirikul Manochantr: Commercial Relationship: Code N (No Commercial Relationship)**Study Group:** (none)**ABSTRACT****TITLE:** The effects of fucoxanthin on ultraviolet A (UVA)-induced oxidative stress in human retinal epithelial cells**ABSTRACT BODY:****Purpose:** UVA irradiation causes the production of reactive oxygen species (ROS), mitochondrial dysfunction and DNA damage in RPE cells. Fucoxanthin, a marine carotenoid extracted from brown seaweed, is a bioactive compound with anti-inflammatory and antioxidant properties. Fucoxanthin was previously shown to significantly inhibit ROS generation and protect RPE cells from H₂O₂-induced oxidative stress cell damage. This study aims to investigate the protective effects of fucoxanthin against UVA-irradiated differentiated ARPE-19 cells.**Methods:** ARPE-19 cells were differentiated to a physiologically relevant RPE-like phenotype using α -MEM low serum-containing medium, supplemented with 1% N1, 0.25 mg/ml taurine, 20 ng/ml hydrocortisone, 0.013 ng/ml triiodo-thyronine and 10 nM nicotinamide for 4 weeks. Gene expression of RPE markers *RPE65*, *RLBP1*, and *RDH5* was investigated using real-time RT-PCR. To assess the impact of fucoxanthin-induced mitigation of UV cytotoxicity, the cells were pretreated with 1.25 μ M, 2.5 μ M and 5 μ M fucoxanthin for 3 days prior to UV treatment. Subsequently, fucoxanthin-treated cells were exposed to UVA at 40 J/cm² for 40 min, followed by the MTT assay. Intracellular ROS production and SOD activity were assessed by measuring the oxidative conversion of DCFH-DA to fluorescent DCF using a fluorospectrophotometer and colorimetric SOD activity assay, respectively.**Results:** Differentiated ARPE-19 exhibited cobblestone morphology and heavy pigmentation, compared with the fibroblast-like morphology and lack of pigmentation in the undifferentiated ARPE-19. RPE-specific markers *RPE65*, *RLBP1*, and *RDH5* in differentiated ARPE-19 were upregulated compared to undifferentiated ARPE-19. After UVA exposure, the cell metabolic activity of ARPE-19 cells was decreased to 35%, while cells pretreated with fucoxanthin at concentrations of 1.25 μ M, 2.5 μ M and 5 μ M for 72h showed significantly increased metabolic activity to 50.7%, 58.7% and 81.1% ($p < 0.05$) respectively, compared with the untreated group. Pretreatment with fucoxanthin similarly decreased intracellular ROS production compared with the untreated group.**Conclusions:** The results demonstrate that fucoxanthin has a cytoprotective effect and mitigates UVA-induced damage in differentiated ARPE-19 cells. The findings may lead to an additional strategy for AMD prevention and development of new therapeutic agents.

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