

Product Data Sheet

Product Name: Zeaxanthin

Cat. No.: GC45179

Chemical Properties

Cas. No. 144-68-3

SMILES O[C@H]1CC(C)(C)C(/C=C/C(C)=C/C=C/C(C)=C/C=C/C(C)=C(C)/C=C/C=C(C)/C=C/C2=C(C)C[C@@H](O)CC2(C)C)=C(C)C1

Formula C₄₀H₅₆O₂

M.Wt 568.9

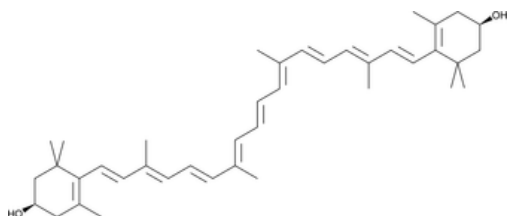
Solubility THF : ≥ 10 mg/mL (17.58 mM);
DMSO : < 1 mg/mL (insoluble or slightly soluble)

Storage -20°C, protect from light, unstable in solution, ready to use.

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines ARPE-19 cells

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Confluent cultured ARPE-19 cells were loaded with A2E alone or loaded with A2E (a lipofuscin fluorophore and photosensitizer) and then supplemented with 10 μ M lutein or Zeaxanthin for 72h. The cells were then exposed to blue light for 10min and harvested. The chymotrypsin-like activity of the proteasome in the cells was determined using a fluorogenic peptide as a substrate and levels of ubiquitin conjugates were determined by Western blotting analysis using levels of β -actin as a loading control.

Reaction
Conditions 10 μ M; 72h

Applications Incubation of A2E-containing retinal pigment epithelial cells (RPE) with lutein or Zeaxanthin before blue light irradiation significantly attenuated photooxidation-induced proteasome inactivation and also reduced the accumulation of intracellular ubiquitin conjugates.

Animal experiment [2]:

Animal models SD rats

Preparation Method Four-week-old male SD rats were randomly divided into three groups (n=8 in each group): rats fed a normal diet (control); rats fed a normal diet with daily oral gavage of a low dose of Zeaxanthin (8mg/kg/day); and rats fed a normal diet with daily oral gavage of a high dose of Zeaxanthin (24mg/kg/day). After 4 weeks of feeding, the rats were killed, and their serum and tissues including liver and heart were isolated for analysis.

Dosage form 8、24mg/kg/day for 4 weeks; p.o.

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Applications After supplementation with Zeaxanthin, the levels of GSH in the rat liver, heart, and serum increased, and the levels of 4-HNE decreased. The levels of carbonyl proteins in the serum also decreased.

References:

- [1]Bian Q, Gao S, Zhou J, et al. Lutein and zeaxanthin supplementation reduces photooxidative damage and modulates the expression of inflammation-related genes in retinal pigment epithelial cells[J]. Free Radical Biology and Medicine, 2012, 53(6): 1298-1307.
- [2]Zou X, Gao J, Zheng Y, et al. Zeaxanthin induces Nrf2-mediated phase II enzymes in protection of cell death[J]. Cell death & disease, 2014, 5(5): e1218-e1218.

Background

Zeaxanthin is a carotenoid pigment with multiple biological activities found in a variety of fruits and vegetables that can help treat or prevent age-related macular degeneration, glaucoma, and cataracts^[1]. Zeaxanthin is found in the macula of the eye, which mainly contains (3R,3'R)-Zeaxanthin and meso-Zeaxanthin forms^[2]. Zeaxanthin also has antioxidant, antiparasitic, anthelmintic and anti-osteoporotic effects^[3].

In vitro, treatment of ARPE-19 cells accumulating A2E (a lipofuscin fluorophore and photosensitizer) with Zeaxanthin (10 μ M) for 72h significantly attenuated photooxidation-induced proteasome inactivation, reduced the accumulation of intracellular ubiquitin conjugates, and reversed the changes in inflammatory-related gene expression caused by photooxidation^[4]. Pretreatment of retinal ganglion cells (RGC-5) with Zeaxanthin (1 μ M) for 1h significantly inhibited H₂O₂-induced cell viability decrease and nuclear condensation, and reduced ROS generation caused by serum deprivation^[5]. Zeaxanthin (5 μ g/mL) treatment of human corneal limbal and conjunctival epithelial cells protected the cells from UVB-induced cell death and reduced the secretion of proinflammatory cytokines IL-6, MCP-1, and IL-8^[6].

In vivo, Zeaxanthin (8, 24mg/kg) was administered to SD rats by gavage for 4 weeks, which increased the levels of glutathione (GSH) in the liver, heart, and serum of rats, reduced the levels of

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lipid and protein peroxidation marker 4-HNE, and reduced the levels of carbonyl proteins in serum^[7]. Zeaxanthin (25mg/kg) fed to non-alcoholic steatohepatitis (NASH) model mice for 6 weeks significantly reduced oxidative stress in the liver and prevented NASH-related inflammation and fibrosis^[8].

References:

- [1] Johra F T, Bepari A K, Bristy A T, et al. A mechanistic review of β -carotene, lutein, and zeaxanthin in eye health and disease[J]. *Antioxidants*, 2020, 9(11): 1046.
- [2] Sajilata M G, Singhal R S, Kamat M Y. The carotenoid pigment zeaxanthin—a review[J]. *Comprehensive reviews in food science and food safety*, 2008, 7(1): 29-49.
- [3] Bouyahya A, El Omari N, Hakkur M, et al. Sources, health benefits, and biological properties of zeaxanthin[J]. *Trends in Food Science & Technology*, 2021, 118: 519-538.
- [4] Bian Q, Gao S, Zhou J, et al. Lutein and zeaxanthin supplementation reduces photooxidative damage and modulates the expression of inflammation-related genes in retinal pigment epithelial cells[J]. *Free Radical Biology and Medicine*, 2012, 53(6): 1298-1307.
- [5] Nakajima Y, Shimazawa M, Otsubo K, et al. Zeaxanthin, a retinal carotenoid, protects retinal cells against oxidative stress[J]. *Current Eye Research*, 2009, 34(4): 311-318.
- [6] Huang Y, Shi C, Li J. The protective effect of zeaxanthin on human limbal and conjunctival epithelial cells against UV-induced cell death and oxidative stress[J]. *International Journal of Ophthalmology*, 2019, 12(3): 369.
- [7] Zou X, Gao J, Zheng Y, et al. Zeaxanthin induces Nrf2-mediated phase II enzymes in protection of cell death[J]. *Cell death & disease*, 2014, 5(5): e1218-e1218.
- [8] Chamberlain S M, Hall J D, Patel J, et al. Protective effects of the carotenoid zeaxanthin in experimental nonalcoholic steatohepatitis[J]. *Digestive diseases and sciences*, 2009, 54: 1460-1464.

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