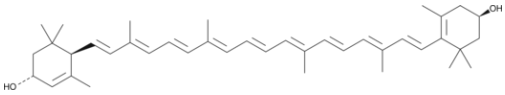
Product Data Sheet

Product Name: Lutein
Cat. No.: GC44093

Chemical Properties

Cas. No.	127-40-2		
SMILES	O[C@@H]1CC(C)(C)[C@@H](/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)C(C)=C1		
Formula	$C_{40}H_{56}O_2$	M.Wt	568.9
Solubility	Chloroform: 5 mg/ml,DMF: 10 mg/ml,DMF:PBS (pH 7.2) (1:1): 0.5 mg/ml,DMSO: 5 mg/ml	Storage	Store at -20°C
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	Primary mammary epithelial cells (PmECs)、MCF-7 cells、MDA-MB-468 cells
Preparation Method	Cells were grown in 96-well plates and treated with Lutein (0.5-2.0μM) or DMSO for 48h. Cell viability was measured by MTT assay.
Reaction Conditions	0.5-2.0μM; 48h

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Applications	Lutein treatment inhibits cell growth in human breast cancer cells but not in normal mammary epithelial cells.

Animal experiment [2]:

Animal models	C57BL/6N male mice
Preparation Method	Two-hour cerebral ischemia was induced by unilateral middle cerebral artery occlusion (MCAo) in mice. Either Lutein (0.2mg/kg) or vehicle was given to mice intraperitoneally 1 h after MCAo and 1 h after reperfusion. Neurological deficits were evaluated at 22h after reperfusion while survival rate was assessed daily until 7 days after reperfusion. Brains were cut into 2 mm-thick coronal slices and stained with 2% 2,3,5-triphenyltetrazolium chloride to determine the infarct size after MCAo. Paraffin-embedded brain sections were prepared for TUNEL assay and immunohistochemistry. Protein lysate was collected for Western blotting experiments.
Dosage form	0.2mg/kg; i.c.v.
Applications	Higher survival rate, better neurological scores, smaller infarct area and smaller infarct volume were noted in the Lutein-treated group. Immunohistochemistry data showed a decrease of immunoreactivity of nitrotyrosine, poly (ADP-ribose) and NFκB in the Lutein-treated brains. Western blotting data showed decreased levels of Cox-2, pERK, and pIκB, but increased levels of Bcl-2, heat shock protein 70 and pAkt in the Lutein-treated brains.

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References:
[1] Gong X, Smith J R, Swanson H M, et al. Carotenoid lutein selectively inhibits breast cancer cell growth and potentiates the effect of chemotherapeutic agents through ROS-mediated mechanisms[J]. Molecules, 2018, 23(4): 905.
[2]Li S Y, Yang D, Fu Z J, et al. Lutein enhances survival and reduces neuronal damage in a mouse model of ischemic stroke[J]. Neurobiology of disease, 2012, 45(1): 624-632.

Background

Lutein is a naturally occurring carotenoid with multiple biological activities and is widely found in green leafy vegetables, corn, and egg yolks[1]. Lutein is the main pigment component of the macula of the human retina and has strong antioxidant and blue light filtering properties[2]. As an important dietary nutrient, Lutein can play a positive role in visual health and neuroprotection by reducing oxidative stress and inflammatory responses[3].

In vitro, Lutein (0.5-2.0μM) treatment of human breast cancer cell lines (MCF-7 and MDA-MB-468 cells) for 48h inhibited the growth of breast cancer cells, but had no effect on normal breast epithelial cells, induced breast cancer cell cycle arrest, and significantly increased the level of ROS in breast cancer cells[4]. Lutein (1μM) pretreatment of ARPE-19 cells for 12h protected the cells from tert-butyl peroxide (tBHP)-induced oxidative stress and cell death[5]. Pretreatment of peripheral blood mononuclear cells (PBMCs) from patients with stable angina (SA) with Lutein (1, 5, 25μM) for 24h reduced LPS-induced secretion of IL-6, IL-1β, and TNF, and decreased the expression of IL-6, IL-1β, and TNF mRNA in a dose-dependent manner[6].

In vivo, Lutein (0.2mg/kg) was administered intraventricularly to treat ischemic stroke model mice, which improved the survival rate and neurological function score of the mice, reduced the infarct area and volume, and reduced the immunoreactivity of nitric oxide tyrosine, poly ADP ribose, and NFκB in brain tissue. It also reduced the levels of Cox-2, pERK, and pIκB in brain tissue, and increased the levels of Bcl-2, heat shock protein 70, and pAkt[7].

References:
[1] Fuad N I N, Sekar M, Gan S H, et al. Lutein: A comprehensive review on its chemical, biological activities and therapeutic potentials[J]. 2020.

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[2] Kijlstra A, Tian Y, Kelly E R, et al. Lutein: more than just a filter for blue light[J]. Progress in retinal and eye research, 2012, 31(4): 303-315.
[3] Mrowicka M, Mrowicki J, Kucharska E, et al. Lutein and zeaxanthin and their roles in age-related macular degeneration—neurodegenerative disease[J]. Nutrients, 2022, 14(4): 827.
[4] Gong X, Smith J R, Swanson H M, et al. Carotenoid lutein selectively inhibits breast cancer cell growth and potentiates the effect of chemotherapeutic agents through ROS-mediated mechanisms[J]. Molecules, 2018, 23(4): 905.
[5] Gong X, Draper C S, Allison G S, et al. Effects of the macular carotenoid lutein in human retinal pigment epithelial cells[J]. Antioxidants, 2017, 6(4): 100.
[6] Chung R W S, Leanderson P, Lundberg A K, et al. Lutein exerts anti-inflammatory effects in patients with coronary artery disease[J]. Atherosclerosis, 2017, 262: 87-93.
[7] Li S Y, Yang D, Fu Z J, et al. Lutein enhances survival and reduces neuronal damage in a mouse model of ischemic stroke[J]. Neurobiology of disease, 2012, 45(1): 624-632.

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