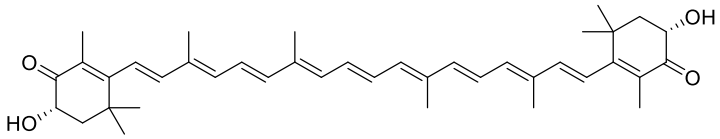
Product data sheet

MedKoo Cat#: 600113 Name: Astaxanthin CAS#: 472-61-7 Chemical Formula: $C_{40}H_{52}O_4$ Exact Mass: 596.38656 Molecular Weight: 596.84	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
Astaxanthin is a keto-carotenoid. Astaxanthin, unlike several carotenes and one other known carotenoid, is not converted to vitamin A (retinol) in the human body. Like other carotenoids, astaxanthin has self-limited absorption orally and such low toxicity by mouth that no toxic syndrome is known. It is an antioxidant with a slightly lower antioxidant activity in some model systems than other carotenoids. However, in living organisms the free-radical terminating effectiveness of each carotenoid is heavily modified by its lipid solubility, and thus varies with the type of system being protected. While astaxanthin is a natural dietary component, it can also be used as a food supplement. The supplement is intended for human, animal, and aquaculture consumption. The commercial production of astaxanthin comes from both natural and synthetic sources.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	51.0	85.45

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	1.68 mL	8.38 mL	16.75 mL
5 mM	0.34 mL	1.68 mL	3.35 mL
10 mM	0.17 mL	0.84 mL	1.68 mL
50 mM	0.03 mL	0.17 mL	0.34 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Jia BY, Xiang DC, Shao QY, Zhang B, Liu SN, Hong QH, Quan GB, Wu GQ. Inhibitory effects of astaxanthin on postovulatory porcine oocyte aging in vitro. Sci Rep. 2020 Nov 19;10(1):20217. doi: 10.1038/s41598-020-77359-6. PMID: 33214659; PMCID: PMC7677382.
2. Chao CT, Yeh HY, Tsai YT, Yuan TH, Liao MT, Huang JW, Chen HW. Astaxanthin Counteracts Vascular Calcification In Vitro Through an Early Up-Regulation of SOD2 Based on a Transcriptomic Approach. Int J Mol Sci. 2020 Nov 12;21(22):8530. doi: 10.3390/ijms21228530. PMID: 33198315; PMCID: PMC7698184.

In vivo study

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1. Wang W, Liu T, Liu Y, Yu L, Yan X, Weng W, Lu X, Zhang C. Astaxanthin attenuates alcoholic cardiomyopathy via inhibition of endoplasmic reticulum stress-mediated cardiac apoptosis. Toxicol Appl Pharmacol. 2021 Feb 1;412:115378. doi: 10.1016/j.taap.2020.115378. Epub 2021 Jan 2. PMID: 33352188.
2. Zhuge F, Ni Y, Wan C, Liu F, Fu Z. Anti-diabetic effects of astaxanthin on an STZ-induced diabetic model in rats. Endocr J. 2021 Apr 28;68(4):451-459. doi: 10.1507/endocrj.EJ20-0699. Epub 2020 Dec 2. PMID: 33268598.

7. Bioactivity

Biological target:

Astaxanthin is a modulator of PPARγ and a potent antioxidant with antiproliferative, neuroprotective and anti-inflammatory activity.

In vitro activity

In this study, the blastocyst yield, and diameter and total cell number per blastocyst were improved when the oocytes were treated with Ax (astaxanthin) during aging in vitro, suggesting that Ax could enhance the capacity of embryos to develop to the blastocyst stage and the embryo quality. The maternal gene in oocytes is required for preimplantation embryo development. Differences in maternal gene expression lead to the lower developmental potential of aged oocytes. For instance, ZAR1 plays essential role in transition from oocyte to embryo, and its expression in this study was found to be reduced in aged oocytes. Furthermore, Ax treatment was helpful to maintain the maternal gene expression for C-MOS, CCNB1, BMP15, CDX2 and POU5F1 in aged oocytes. The results also showed that the mRNA levels of CDX2 and POU5F1 genes were also increased in resultant blastocysts from Ax-treated aged oocytes. These two genes are required for differentiation of trophectoderm during early embryo development. Therefore, the above findings provided the most direct proof of the beneficial effects of Ax for aged oocytes.

Reference: Sci Rep. 2020; 10: 20217. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7677382/

In vivo activity

The severe ER stress observed in response to ethanol was notably inhibited in the presence of AST (astaxanthin), demonstrating the anti-ER stress function of AST. ER stress can be activated by three UPR pathways, PERK-eIF2α-ATF4, ATF6 and IRE1-XBP1, which were also examined in this study. Vardiac levels of p-PERK, p-eif2α, ATF4, ATF-6, p-IRE1, and XBP-1 were all significantly increased in ethanol-treated mice (Fig. 3E–H), indicating that the three UPR pathways were all involved in ethanol-induced cardiac ER stress. However, activation of the three UPR pathways induced by ethanol was significantly inhibited by AST treatment (Fig. 3E–H), suggesting that AST may affect the same upstream regulatory factors of the three pathways. Based on these results, expression of cardiac GRP78 was assessed. Results showed that ethanol induced, while AST reversed, cardiac GRP78 expression (Fig. 3E&I), indicating that cardiac GRP78 is the target by which AST inhibited all UPR pathways induced by ethanol.

Reference: Toxicol Appl Pharmacol. 2021 Feb 1; 412: 115378.
https://www.sciencedirect.com/science/article/pii/S0041008X20305007?via%3Dihub

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.