

Product Data Sheet

Product Name: Avenanthramide C

Cat. No.: GC82316

Chemical Properties

Cas. No. 116764-15-9

Formula $C_{16}H_{13}NO_6$ M.Wt 315.28

Solubility DMSO: 50 mg/mL (158.59 mM; Need ultrasonic) 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.

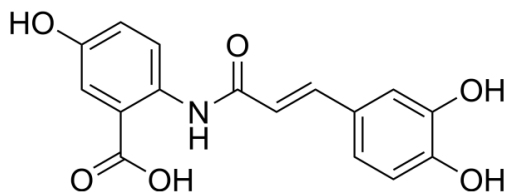
Caution: Product has not been fully validated for medical applications. For research use only.

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Structure



Background

Avenanthramide C is an orally active polyphenolic compound that penetrates the blood-brain barrier. Avenanthramide C is derived from oats. Avenanthramide C inhibits NF-κB nuclear translocation and inhibits the phosphorylation of PI3K, PLCγ1, Lyn, Syk, and Akt. Avenanthramide C inhibits the expression and activity of MMP-9 and inhibits MAPK phosphorylation. Avenanthramide C scavenges DPPH radicals, reduces intracellular ROS, and inhibits pro-inflammatory cytokine release and adhesion molecule expression. Avenanthramide C inhibits cell degranulation. Avenanthramide C attenuates active systemic anaphylaxis and inhibits passive cutaneous anaphylaxis. Avenanthramide C ameliorates PHZ-induced blood stasis. Avenanthramide C inhibits neuroinflammation, restores long-term potentiation, and improves recognition and spatial memory in Alzheimer's disease models. Avenanthramide C can be used for research on allergic inflammation, thrombosis, atherosclerosis, and Alzheimer's disease [1] [2] [3] [4] [5].

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In Vivo, Avenanthramide C (0.1-10 mg/kg; p.o.; administered on days 9, 11, and 13) dose-dependently attenuates ovalbumin-induced active systemic anaphylaxis by suppressing the decrease in body temperature and reducing elevated serum histamine, IgE, and IL-4 levels^[1]. Avenanthramide C (0.1-10 mg/kg; p.o.; single dose; 1 h before DNP-HSA challenge) dose-dependently inhibits IgE-mediated passive cutaneous anaphylaxis^[1]. Avenanthramide C (6 mg/kg; p.o.; daily; 2 weeks) rescues impaired long-term potentiation in 5XFAD Alzheimer's disease model mice^[5]. Avenanthramide C (6 mg/kg; p.o.; daily; 2 weeks) rescues impaired long-term potentiation in Tg2576 Alzheimer's disease model mice^[5]. Avenanthramide C (6 mg/kg; p.o.; once daily; 2 weeks) inhibits neuroinflammation in the hippocampus of 5XFAD and Tg2576 Alzheimer's disease model mice^[5].

In Vitro, Avenanthramide C (0.01-100 μ M; 12 h) is not cytotoxic up to 100 μ M in RBL-2H3 cells^[1]. Avenanthramide C (1-100 nM; 1 h) concentration-dependently inhibits β -hexosaminidase release in DNP-HSA-stimulated RBL-2H3 cells^[1]. Avenanthramide C (1-100 nM; 1 h) concentration-dependently inhibits histamine release in DNP-HSA-stimulated RBL-2H3 cells^[1]. Avenanthramide C (1-100 nM) suppresses DNP-HSA-stimulated intracellular calcium elevation in a concentration-dependent manner in RBL-2H3 cells^[1]. Avenanthramide C (1-100 nM; 1 h) inhibits DNP-HSA-stimulated phosphorylation of PI3K and PLC γ 1 in RBL-2H3 cells^[1]. Avenanthramide C (1-100 nM; 1 h) concentration-dependently inhibits the gene expression and release of IL-4, IL-6, and TNF- α in DNP-HSA-stimulated RBL-2H3 cells^[1]. Avenanthramide C (1-100 nM; 1 h) inhibits the phosphorylation of Lyn, Syk, and Akt and suppresses I κ B α degradation and NF- κ B nuclear translocation in DNP-HSA-stimulated RBL-2H3 cells^[1]. Avenanthramide C (0-100 μ M; 24 h) dose-dependently suppresses the TNF- α -induced increase in MMP-9 mRNA levels in HASMC cells^[3]. Avenanthramide C (100 μ M) inhibits the MAPK signaling pathway by decreasing phosphorylation of ERK, JNK, and p38, and reduces I κ B phosphorylation in TNF- α -stimulated HASMC cells^[3]. Avenanthramide C (50 μ M; 2 h) restores impaired LTP in ex vivo hippocampal slices from 5XFAD mice^[5]. Avenanthramide C (50 μ M; 2 h) restores impaired LTP in ex vivo hippocampal slices from Tg2576 mice^[5]. Avenanthramide C (1.0625-100 μ g mL⁻¹; 30 min) acts as a potent free radical scavenger in the cell-free DPPH assay with an IC₅₀ of 7.38 μ g mL⁻¹^[2]. Avenanthramide C (6.25-100 μ g mL⁻¹; 24 h) is non-toxic to HUVECs at concentrations up to 25 μ g mL⁻¹ but exhibits cytotoxic effects at 50 and 100 μ g mL⁻¹^[2]. Avenanthramide C (6.25-25 μ g mL⁻¹; 24 h) dose-dependently

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attenuates intracellular ROS accumulation induced by t-BOOH in HUVECs^[2].

References:

- [1]. Dhakal H, et al. Avenanthramide C from germinated oats exhibits anti-allergic inflammatory effects in mast cells. Scientific reports. 2019 May 03;9(1):6884.
- [2]. Wang LW, et al. Avenanthramide C from underutilized oat glumes exhibits dual anti-oxidant and anti-thrombotic effects in oxidative damage and thrombosis models. Food & function. 2025 Jul 01;16(13):5380-5392.
- [3]. Park J, et al. Avenanthramide C Suppresses Matrix Metalloproteinase-9 Expression and Migration Through the MAPK/NF- κ B Signaling Pathway in TNF- α -Activated HASMC Cells. Frontiers in pharmacology. 2021;12:621854.
- [4]. Matsukawa T, et al. Occurrence of avenanthramides and hydroxycinnamoyl-CoA:hydroxyanthranilate N-hydroxycinnamoyltransferase activity in oat seeds. Zeitschrift fur Naturforschung. C, Journal of biosciences. 2000;55(1-2):30-6.
- [5]. Ramasamy VS, et al. Avenanthramide-C Restores Impaired Plasticity and Cognition in Alzheimer's Disease Model Mice. Molecular neurobiology. 2020 Jan;57(1):315-330.

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