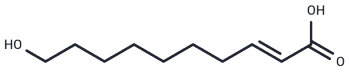
Data Sheet (Cat.No.T3250)

TargetMol

Royal Jelly acid

Chemical Properties



CAS No. : 14113-05-4

Formula: C10H18O3

Molecular Weight: 186.25

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Royal Jelly Acid (Queen Bee Acid) is a major fatty acid found in royal jelly that promotes neuronal growth and protection and reduces anxiety. Royal Jelly Acid induces reactive oxygen species (ROS)-mediated apoptosis in A549 cells, improves non-alcoholic fatty liver disease by activating the AMPK-α signaling pathway, and reduces bone loss by inhibiting the NF-κB signaling pathway downstream of FFAR4. Royal Jelly Acid also exhibits inhibitory effects against various bacteria and fungi, including Aspergillus niger, mold, and Staphylococcus aureus. By targeting aspartyl β-hydroxylase and inhibiting chondrocyte senescence, Royal Jelly Acid exerts a preventive and therapeutic effect against osteoarthritis.
Targets(IC50)	NF-κB,Endogenous Metabolite,NO Synthase,Parasite,IFNAR,VEGFR
In vitro	Methods: Animal models including mice and chickens were administered with 10-Hydroxy-2-decenoic acid via intra-articular injection, oral administration, subcutaneous injection and other routes, and indicators such as cartilage metabolism, tumor growth, liver protection, intestinal injury and bone metabolism were detected. Results: 1 10-Hydroxy-2-decenoic acid (10 mg/kg intra-articular injection, twice weekly for 6 weeks; 100 mg/kg oral administration, three times weekly for 7 weeks) improved cartilage anabolism, inhibited cartilage degeneration and alleviated surgery-induced knee pain in DMM mice [1]. 2 10-Hydroxy-2-decenoic acid (10 mg/kg intra-articular injection, twice weekly for 8 weeks; 100 mg/kg oral administration, three times weekly for 8 weeks) alleviated chondrocyte senescence and cartilage degeneration in naturally aged mice [1]. 3 10-Hydroxy-2-decenoic acid (0.5–2 mg/mL subcutaneous injection, single administration) completely inhibited tumor progression in AKR mice and ascites tumor-bearing mice [2]. 4 10-Hydroxy-2-decenoic acid (10–50 mg/kg oral administration, once daily for 4 weeks) exerted significant protective effects on MCD diet-induced NAFLD mice, alleviating lipid accumulation, liver injury and cell apoptosis [3]. 5 10-Hydroxy-2-decenoic acid (dietary supplementation at 1–5 g/kg, oral administration for 21 days) alleviated LPS-induced intestinal mucosal injury and growth performance decline in chickens through anti-inflammatory, antioxidant effects and regulation of intestinal flora [4]. 6 10-Hydroxy-2-decenoic acid (40 mg/kg oral administration, once daily for 4 weeks) inhibited osteoclast-mediated bone resorption without affecting bone formation in

In vitro	ovariectomized mice [10].
In vivo	Methods: Chondrocytes, tissue explants, various tumor cells, HUVECs, hepatocytes, Caenorhabditis elegans and other models were treated with different concentrations of 10-hydroxy-2-decenoic acid, and relevant indicators including cell proliferation, apoptosis, metabolism, senescence, signaling pathways, angiogenesis, antibacterial activity, antitumor activity and lifespan were detected. Results 1 10-Hydroxy-2-decenoic acid (0-40 nM, 0-6 days) promoted proliferation and anabolism, and inhibited apoptosis and catabolism in primary chondrocytes and human C28/I2 chondrocytes. 2 10-Hydroxy-2-decenoic acid (0-10 nM, 7 days) enhanced anabolism and inhibited catabolism in chondrocytes within human osteoarthritic cartilage explants. 3 10-Hydroxy-2-decenoic acid (0-200 μM, 1-48 h) bound to ASPH and regulated ASPH-dependent Asp-Arg-Hydrox domain-related cartilage metabolism in C28/I2 and HEK-293T cells. 4 10-Hydroxy-2-decenoic acid (0-10 nM, 48 h) reduced mRNA levels of SA-β-gal and the senescence marker genes p16 and p21 in C28/I2 cells. 5 10-Hydroxy-2-decenoic acid (0-10 nM, 48 h) inhibited chondrocyte senescence by regulating the ASPH/ERK/p53/p21 and ASPH/GSK3β/p16 pathways [1]. 6 10-Hydroxy-2-decenoic acid (2 mg/mL) exhibited antitumor activity against 6C3HED lymphoma cells under low pH conditions [2]. 7 10-Hydroxy-2-decenoic acid (50-150 μM) blocked FFA-induced lipid accumulation and apoptosis in AML12 cells by activating AMPK-α [3]. 8 10-Hydroxy-2-decenoic acid (0.07-1.13 mg/mL, 24 h) reduced biofilm mass and cell viability by disrupting exopolysaccharide structures, and inhibited hemolysin production in Staphylococcus aureus [5]. 9 10-Hydroxy-2-decenoic acid (20-500 μM, 11 days) inhibited VEGF-induced angiogenesis in HUVECs and fibroblasts. 10 10-Hydroxy-2-decenoic acid (20-500 μM, 3 days) inhibited VEGF-induced HUVEC proliferation at 500 μM, while no significant inhibitory effect was observed at 20-100 μM. 11 10-Hydroxy-2-decenoic acid (500 μM, 24 h) inhibited HUVEC migration [6]. 12 10-Hydroxy-2-decenoic acid (1-100 μM, 3-36 h) suppressed the growth of A549, NCI-H460 and NCI-H23 cells, with IC_{50} values of 22.68, 44.03 and 44.79 μM, respectively, and showed weak cytotoxicity against IMR90, L-02 and GES-1 cells. 13 10-Hydroxy-2-decenoic acid (30 μM, 3-24 h) elevated ROS levels and induced apoptosis in A549 cells by regulating the MAPK, NF-κB and STAT3 pathways. 14 10-Hydroxy-2-decenoic acid (30 μM, 3-24 h) induced cell cycle arrest in A549 cells by modulating the expression of cycle-related proteins. 15 10-Hydroxy-2-decenoic acid (30 μM, 3-24 h) inhibited A549 cell migration by regulating the TGF-β1 pathway [8]. 16 10-Hydroxy-2-decenoic acid (10-100 μM, 50 days) extended the mean and maximum lifespan of both daf-16 mutant and wild-type Caenorhabditis elegans via an IIS-DAF-16-independent mechanism [9]. 17 10-Hydroxy-2-decenoic acid (0.00004-20 mM) activated FFAR4 with an EC_{50} of 1.025 mM. 18 10-Hydroxy-2-decenoic acid (12.5-50 ng/mL, 3 days) inhibited osteoclastogenesis in

Solubility Information

Solubility	DMSO: 55 mg/mL (295.3 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween 80+45% Saline: 2 mg/mL (10.74 mM),Sonication is recommended. *Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.*

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	5.3691 mL	26.8456 mL	53.6913 mL
5 mM	1.0738 mL	5.3691 mL	10.7383 mL
10 mM	0.5369 mL	2.6846 mL	5.3691 mL
50 mM	0.1074 mL	0.5369 mL	1.0738 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

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