

Product Data Sheet

Product Name: (±)13-HODE

Cat. No.: GC19461

Chemical Properties

Cas. No. 18104-45-5

Chemical Name (±)-13-hydroxy-9Z,11E-octadecadienoic acid

SMILES CCCCC(O)\C=C\C=C\CCCCCCCC(=O)O

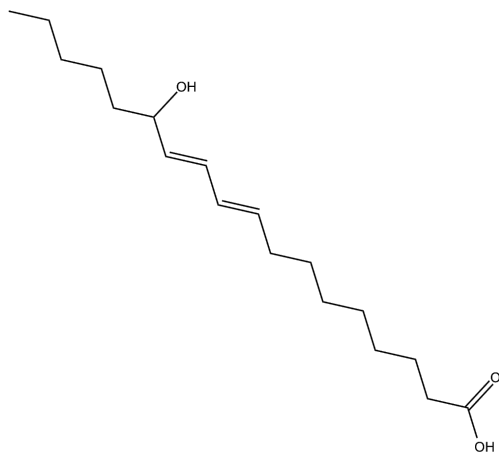
Formula $C_{18}H_{32}O_3$ M.Wt 296.4

Solubility 50mg/mL in DMSO, 50mg/mL in DMF, 50mg/mL in Ethanol Storage Store at -20°C, protect from light

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 MC38 cells and HCA-7 cells

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

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Preparation Method MC38 and HCA-7 cells were seeded into 96-well plates at a density of 1×10^4 cells per well in 200 μ L medium and cultured for 16 hours. The cells were then starved for 24 hours in medium containing 0.1% fetal bovine serum. Subsequently, various concentrations (0, 0.1, 1, 10 μ M) of ($\pm$)13-HODE were added to the wells for an additional 4 hours. BrdU solution was added to each well for 4 hours to measure cell proliferation.

Reaction Conditions 0, 0.1, 1, 10 μ M; 4h

Applications ($\pm$)13-HODE significantly inhibited the proliferation of both MC38 and HCA-7 colon cancer cells.

Animal experiment [2]:

Animal models LDL receptor knockout mice

Preparation Method Mice were fed diets containing varying levels of fat and cholesterol, mice were fed a high-fat diet supplemented with 13-hydroxylinoleic acid (13-HODE) at a dose of 8mg for 13 weeks.

Dosage form 8mg of ($\pm$)13-HODE per diet for 13 weeks

Applications ($\pm$)13-HODE led to an increase of over 100% in the aortic lesion area of the mice, ($\pm$)13-HODE also increased plasma total cholesterol and low-density lipoprotein cholesterol in the mice and enhanced oxidative stress

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References:

- [1] Kwong SC, Abd Jamil AH, Rhodes A, et al. Fatty acid binding protein 7 mediates linoleic acid-induced cell death in triple negative breast cancer cells by modulating 13-HODE. *Biochimie*. 2020 Dec;179:23-31.
- [2] Khan-Merchant N, Penumetcha M, Meilhac O, et al. Oxidized fatty acids promote atherosclerosis only in the presence of dietary cholesterol in low-density lipoprotein receptor knockout mice. *J Nutr*. 2002 Nov;132(11):3256-62.

Background

(±)13-HODE (13-hydroxyoctadecadienoic acid) is a racemic mixture of 13(S)-HODE and 13(R)-HODE, which is formed through the oxidation of linoleic acid by lipoxygenases or cyclooxygenases^[1]. (±)13-HODE plays diverse roles in biological systems. In inflammation, (±)13-HODE can modulate the production of pro-and anti-inflammatory cytokines. (±)13-HODE can act on immune cells such as macrophages and lymphocytes, influencing their activation and function^[2]. (±)13-HODE can either contribute to or alleviate oxidative damage depending on the cellular context. (±)13-HODE may induce the generation of reactive oxygen species (ROS), (±)13-HODE can upregulate antioxidant defense mechanisms^[3]. (±)13-HODE can interact with specific receptors or intracellular signaling pathways. (±)13-HODE may activate protein kinase C (PKC) pathways, affecting cell proliferation, differentiation, and apoptosis^[4].

In vitro, treatment of human umbilical vein endothelial cells (HUVEC) with (±)13-HODE (20-50µM) for 24 hours increased the expression of intercellular adhesion molecule-1 (ICAM-1), but had no significant effect on the expression of vascular cell adhesion molecule-1 (VCAM-1) and E-selectin^[5]. In vitro, MDA-MB-231 cells were divided into two groups: FABP7-overexpressing cells (231-FABP7) and control cells (231-RFP). Both groups were treated with 12.5mM linoleic acid or vehicle for 72 hours. When 25nM (±)13-HODE was added to the linoleic acid-treated 231-FABP7 cells, the cell death was significantly alleviated^[6].

In vivo, (±)13-HODE was administered at a dose of 8mg to low-density lipoprotein receptor knockout (LDL receptor knockout) mice for 13 weeks. (±)13-HODE led to an increase of over 100% in the aortic lesion area of the mice. (±)13-HODE also increased plasma total cholesterol and low-density

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lipoprotein cholesterol in the mice and enhanced oxidative stress^[7].

References:

- [1] Brash AR. Lipoxygenases: occurrence, functions, catalysis, and acquisition of substrate. J Biol Chem. 1999 Aug 20;274(34):23679-82.
- [2] Serhan CN, Petasis NA. Resolvins and protectins in inflammation resolution. Chem Rev. 2011 Oct 12;111(10):5922-43.
- [3] Buchanan MR, Horsewood P, Brister SJ. Regulation of endothelial cell and platelet receptor-ligand binding by the 12- and 15-lipoxygenase monohydroxides, 12-, 15-HETE and 13-HODE. Prostaglandins Leukot Essent Fatty Acids. 1998 May;58(5):339-46.
- [4] Nishizuka Y. Intracellular signaling by hydrolysis of phospholipids and activation of protein kinase C. Science. 1992 Oct 23;258(5082):607-14.
- [5] Friedrichs B, Toborek M, Hennig B, et al. 13-HPODE and 13-HODE modulate cytokine-induced expression of endothelial cell adhesion molecules differently. Biofactors. 1999;9(1):61-72.
- [6] Kwong SC, Abd Jamil AH, Rhodes A, et al. Fatty acid binding protein 7 mediates linoleic acid-induced cell death in triple negative breast cancer cells by modulating 13-HODE. Biochimie. 2020 Dec;179:23-31.
- [7] Khan-Merchant N, Penumetcha M, Meilhac O, et al. Oxidized fatty acids promote atherosclerosis only in the presence of dietary cholesterol in low-density lipoprotein receptor knockout mice. J Nutr. 2002 Nov;132(11):3256-62.

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