20-HETE

Cat. No.: HY-15598

CAS No.: 79551-86-3

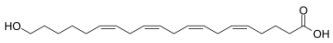
Molecular Formula: $C_{20}H_{32}O_3$

Molecular Weight: 320.47

Target: Potassium Channel; Endogenous Metabolite; Reactive Oxygen Species; NF-κB; NO Synthase; NADPH Oxidase

Pathway: Membrane Transporter/Ion Channel; Metabolic Enzyme/Protease; Immunology/Inflammation; NF-κB

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 110 mg/mL (343.25 mM; Need ultrasonic)
Ethanol : 6.67 mg/mL (20.81 mM; Need ultrasonic)

Preparing Stock Solutions

Mass / Solvent / Concentration	1 mg	5 mg	10 mg
1 mM	3.1204 mL	15.6021 mL	31.2042 mL
5 mM	0.6241 mL	3.1204 mL	6.2408 mL
10 mM	0.3120 mL	1.5602 mL	3.1204 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: 5.5 mg/mL (17.16 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 5.5 mg/mL (17.16 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.75 mg/mL (8.58 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

20-HETE (20-hydroxy Arachidonic Acid) is a CYP450 metabolite and a potent vasoconstrictor. 20-HETE is an endogenous inhibitor of the large-conductance Ca^{2+}-activated K^+ channel in renal arterioles. 20-HETE constricts smooth muscles, stimulates smooth muscle proliferation and migration. 20-HETE increases NADPH oxidase, ROS, and NF-κB activity. 20-HETE also inhibits endothelial NO synthase and inhibits apoptosis of pulmonary artery smooth muscle cells[1][2].

IC_{50} & Target

Human Endogenous Metabolite

In Vitro	Addition of 20-HETE to the bath (1-100 nM), reduces the frequency of opening of the large-conductance Ca2+-activated K+ channel recorded using cell-attached patches on vascular smooth muscle cells (VSM)[1]. 20-HETE (1-1000 nM) reduces the diameter of isolated perfused small renal arteries of the rat by approximately 15% tetraethylammonium (1 mM) blocked the vasoconstrictor response to 20-HETE (100 nM)[1]. 20-HETE induces the phosphorylation of ERK1/2, a MAPK that plays a pivotal role in the proliferation induced by the activation of receptor tyrosine kinases and G protein-coupled receptors. 20-HETE promotes platelet-derived growth factor-stimulated vascular smooth muscle cell migration via pathways that involve MEK and phosphatidylinositol 3-kinase activation[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	In Sprague-Dawley rats, administration of the 20-HETE inhibitor HET0016 or the 20-HETE antagonist 20-HEDE preventes DHT-induced increases in blood pressure as well as abrogates DHT-induced increases in the media-to-lumen ratio (M/L), media thickness, and collagen IV deposition in renal interlobar arteries. 20-HETE contributes to DHT-induced vascular remodeling independent of blood pressure elevation[2]. In Cyp4a14$^{-/-}$ mice, which display androgen-driven and 20-HETE-dependent hypertension, treatment with the 20-HETE antagonist abolishes remodeling of renal resistance arteries measured as media thickness and M/L. 20-HETE is a key regulator of microvascular remodeling in hypertension; its effect is independent of blood pressure elevation and androgen levels[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Zou AP, et al. 20-HETE is an endogenous inhibitor of the large-conductance Ca(2+)-activated K+ channel in renal arterioles. Am J Physiol. 1996 Jan;270(1 Pt 2):R228-37.

[2]. Ding Y, et al. 20-HETE induces remodeling of renal resistance arteries independent of blood pressure elevation in hypertension. Am J Physiol Renal Physiol. 2013 Sep 1;305(5):F753-63.

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

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