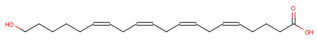
Data Sheet (Cat.No.T14021)

TargetMol
A DRUG SCREENING EXPERT

20-HETE

Chemical Properties

CAS No.:	79551-86-3
Formula:	C20H32O3
Molecular Weight:	320.47
Appearance:	N/A
Storage:	0-4℃ for short term (days to weeks), or -20℃ for long term (months).



Biological Description

Description	20-HETE (20-hydroxy Arachidonic Acid) is a CYP450 metabolite and a potent vasoconstrictor and it is an endogenous inhibitor of the large-conductance Ca2+-activated K+ channel in renal arterioles. 20-HETE increases NADPH oxidase, ROS, and NF-κB activity, and it also inhibits endothelial NO synthase and inhibits apoptosis of pulmonary artery smooth muscle cells[1][2]. 20-HETE constricts smooth muscles, stimulates smooth muscle proliferation and migration.
Targets(IC_{50})	Human Endogenous Metabolite: None
In vitro	20-HETE promotes platelet-derived growth factor-stimulated vascular smooth muscle cell migration via pathways that involve MEK and phosphatidylinositol 3-kinase activation[2]. 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 (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]. 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].
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 is a key regulator of microvascular remodeling in hypertension; its effect is independent of blood pressure elevation and androgen levels[2]. 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.

Solubility Information

Solubility	Ethanol: 6.67 mg/mL (20.81 mM) DMSO: 3.2 mg/mL (9.99 mM) (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	3.12 mL	15.602 mL	31.204 mL
5 mM	0.624 mL	3.12 mL	6.241 mL
10 mM	0.312 mL	1.56 mL	3.12 mL
50 mM	0.062 mL	0.312 mL	0.624 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. The storage conditions and period of the stock solution: - 80 ℃ for 6 months; - 20 ℃ for 1 month. Please use it as soon as possible.

Reference

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.

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