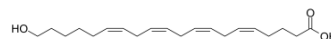


20-HETE

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
Pathway:	Membrane Transporter/Ion Channel; Metabolic Enzyme/Protease
Storage:	4°C, protect from light, stored under argon



SOLVENT & SOLUBILITY

In Vitro

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

DMSO : ≥ 3.2 mg/mL (9.99 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent		1 mg	5 mg	10 mg
	Concentration	Mass			
	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.

BIOLOGICAL ACTIVITY

Description

20-HETE(20-hydroxy Arachidonic Acid) is a potent vasoconstrictor produced in vascular smooth muscle (VSM) cells. It depolarizes VSM by blocking the open-state probability of Ca²⁺-activated K⁺-channels. IC₅₀ Value: Target: 20-Hydroxyeicosatetraenoic acid (20-HETE) is a cytochrome P450-derived arachidonic acid metabolite that has been shown to increase smooth muscle contractions and proliferation, stimulate endothelial dysfunction and activation and promote hypertension. *in vitro*: Addition of 20-HETE to the bath (1-100 nM), reduced the frequency of opening of the large-conductance Ca(2+)-activated K+ channel recorded using cell-attached patches on VSM [1]. In kidney, 20-HETE induces diuresis by inhibiting Na⁺-K⁺-ATPase in proximal tubules and Na⁺/K⁺/Cl⁻ cotransporter in the thick ascending limb of Henle's loop [2]. *in vivo*: In Cyp4a14(-/-) mice, which display androgen-driven and 20-HETE-dependent hypertension, treatment with 20-HETE antagonist abolished remodeling of renal resistance arteries measured as media thickness (24±1 vs. 15±1 μm) and M/L (0.29±0.03 vs. 0.17±0.01) [4]. The transgenic mice had overexpressed hepatic CYP4F2, high hepatic 20-HETE and fasting plasma glucose levels but normal insulin level. The GP activity was increased and the cAMP/PKA-PhK-GP pathway was activated in the transgenic mice compared with wild-type mice [5]. Clinical trial: Mechanisms of Response to Diesel Exhaust in Subjects With Asthma. Phase not specified

REFERENCES

- [1]. Zou AP, Fleming JT, Falck JR, 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]. Schwartzman M, Ferreri NR, Carroll MA, Renal cytochrome P450-related arachidonate metabolite inhibits (Na+ + K+)ATPase. *Nature*. 1985 Apr 18-24;314(6012):620-2.
- [3]. Ding Y, Wu CC, Garcia V, 20-HETE INDUCES REMODELING OF RENAL RESISTANCE ARTERIES INDEPENDENT OF BLOOD PRESSURE ELEVATION IN HYPERTENSION. *Am J Physiol Renal Physiol*. 2013 Jul 3. [Epub ahead of print]
- [4]. Lai G, Wu J, Liu X, 20-HETE induces hyperglycemia through the cAMP/PKA-PhK-GP pathway. *Mol Endocrinol*. 2012 Nov;26(11):1907-16.
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Caution: Product has not been fully validated for medical applications. For research use only.

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