16-HETE

Cat. No.: HY-116444A

CAS No.: 128914-46-5

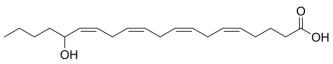
Molecular Formula: $C_{20}H_{32}O_3$

Molecular Weight: 320.47

Target: Na+/K+ ATPase

Pathway: Membrane Transporter/Ion Channel

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



BIOLOGICAL ACTIVITY

Description

16-HETE is arachidonic acid metabolite through subterminal hydroxylation by cytochrome P-450. 16-HETE exhibits vasodilatory and PMN inhibitory effects and serves as biomarker for early stages of non-alcoholic fatty liver disease[1][2][3].

In Vitro

16-HETE (0.01-1 μM) specificially suppresses PMN aggregation and adhesion, with no significant effects on platelets function and blood pressure[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

16-HETE (1-20 μg, i.a.) is stereospecificially involved in vasodilation, regulation of renal perfusion and in mechanisms of tubular transport with S- enantiomer in New Zealand white rabbit[2].

16-HETE (1 μg/kg/min) suppresses the increase of intracranial pressure (ICP) in a rabbit model of thromboembolic stroke[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	New Zealand White rabbit[2]
Dosage:	1-20 μg
Administration:	injection into artery
Result:	16S inhibited 60% ATPase activity at the concentration of 2 μM, while 16R enantiomer remained inactive.

Animal Model:	New Zealand White rabbit[1]
Dosage:	1 μg/kg/min
Administration:	6 hours constant infusion from Hours 1 to 2 after autologous clot embolization
Result:	Reduced infarction area and less increased ICP.

REFERENCES

[1]. Bednar MM, et al., 16(R)-hydroxyeicosatetraenoic acid, a novel cytochrome P450 product of arachidonic acid, suppresses activation of human polymorphonuclear

leukocyte and reduces intracranial pressure in a rabbit model of thromboembolic stroke. Neurosurgery. 2000 Dec;47(6):1410-8; discussion 1418-9.

[2]. Carroll MA, e al., Cytochrome P-450-dependent HETEs: profile of biological activity and stimulation by vasoactive peptides. Am J Physiol. 1996 Oct;271(4 Pt 2):R863-9.

[3]. Maciejewska D, et al., Metabolites of arachidonic acid and linoleic acid in early stages of non-alcoholic fatty liver disease--A pilot study. Prostaglandins Other Lipid Mediat. 2015 Sep;121(Pt B):184-9.

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