17-ODYA

Cat. No.: HY-101016

CAS No.: 34450-18-5

Molecular Formula: $C_{18}H_{32}O_2$

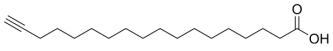
Molecular Weight: 280.45

Target: Cytochrome P450; Apoptosis

Pathway: Metabolic Enzyme/Protease; Apoptosis

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (89.14 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.5657 mL	17.8285 mL	35.6570 mL
5 mM	0.7131 mL	3.5657 mL	7.1314 mL
10 mM	0.3566 mL	1.7828 mL	3.5657 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: 2.5 mg/mL (8.91 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.91 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

17-ODYA is a CYP450 ω-hydroxylase inhibitor. 17-ODYA is also a potent inhibitor (IC_{50}<100 nM) of the formation of 20-hydroxyeicosatetraenoic acid (20-HETE), epoxyeicosatrienoic acids and dihydroxyeicosatrienoic acids by rat renal cortical microsomes incubated with arachidonic acid. 17-ODYA completely attenuates the isoproterenol (ISO)-induced apoptosis, and necrosis in cultured cardiomyocytes[1][2][3]. 17-ODYA is a click chemistry reagent, it contains an Alkyne group and can undergo copper-catalyzed azide-alkyne cycloaddition (CuAAc) with molecules containing Azide groups.

IC_{50} & Target

CYP450 ω-hydroxylase

In Vivo

Infusion of 17-ODYA (16.5 nmol/min) directly into the renal cortical interstitium of rats produced a diuresis and a natriuresis which were associated with an increase in renal papillary blood flow in the absence of changes in renal blood flow, cortical blood flow or glomerular filtration rate[2].

?17-ODYA (0.28 mg/kg; intracoronary; infusion for 2 to 3 minutes; dogs) markedly inhibits 20-HETE production during ischemia-reperfusion and produces a profound reduction in myocardial infarct size[3].

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

REFERENCES

[1]. Jiang S, et al. β-adrenergic Receptor-stimulated Cardiac Myocyte Apoptosis: Role of Cytochrome P450 ω-hydroxylase. J Cardiovasc Pharmacol. 2017;70(2):94-101.

[2]. Zou AP, et al. Effects of 17-octadecynoic acid, a suicide-substrate inhibitor of cytochrome P450 fatty acid omega-hydroxylase, on renal function in rats. J Pharmacol Exp Ther. 1994;268(1):474-481.

[3]. Nithipatikom K, et al. Inhibition of cytochrome P450omega-hydroxylase: a novel endogenous cardioprotective pathway. Circ Res. 2004;95(8):e65-e71.

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