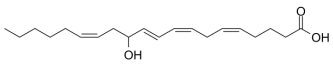
12-HETE

Cat. No.:	HY-113439
CAS No.:	71030-37-0
Molecular Formula:	$C_{20}H_{32}O_3$
Molecular Weight:	320.47
Target:	Apoptosis
Pathway:	Apoptosis
Storage:	Solution, -20°C, 2 years



BIOLOGICAL ACTIVITY

Description

12-HETE, a major metabolic product of arachidonic acid using 12-LOX catalysis, inhibits cell apoptosis in a dose-dependent manner. 12-HETE promotes the activation and nuclear translocation of NF-κB through the integrin-linked kinase (ILK) pathway[1].12-HETE has both anti-thrombotic and pro-thrombotic effects[2]. 12-HETE is a neuromodulator[3].

In Vitro

12-HETE participates in the inhibition of cell apoptosis by activating the ILK/NF-κB pathway, implying an important underlying mechanism that promotes the survival of ovarian cancer cells. 12-HETE facilitates cell survival by activating the integrin-linked kinase/NF-κB pathway in ovarian cancer. 12-HETE protects against cell apoptosis in ovarian cancer cells in a concentration-dependent manner. 12-HETE (1 μM) significantly decreases the activation of caspase-3 induced by serum deprivation (SD).12-HETE represses the increased activity of caspase-3 induced by SD in a concentration-dependent manner, with an IC_{50} value of 1.13 μM[1].

12-HETE (1 μM) facilitates the activation and nuclear translocation of NF-κB via ILK in ovarian cancer cells[1].

12-HETE inhibits insulin secretion, reduces metabolic activity and induces cell death in human islets. 12-HETE increases bovine platelet aggregation induced by thrombin and inhibits prostaglandin E1-induced elevation of intracellular cAMP levels. 12-HETE inhibits washed platelet (WP) aggregation[2].

The neuronal effects of 12-HETE include attenuation of calcium influx and glutamate release as well as inhibition of AMPA receptor (AMPA-R) activation[3].

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

Cell Viability Assay[1]

Cell Line:	Ovarian cancer OVCAR-3 and SKOV3 cells
Concentration:	0, 0.2, 0.5, and 1 μM
Incubation Time:	0, 24, 48, 72, and 96 hours
Result:	Inhibited the decrease in cell viability induced by SD in a dose-dependent manner. 1 μM 12-HETE treatment significantly mitigated the decrease in cell viability under conditions of SD.

Western Blot Analysis[1]

Cell Line:	Ovarian cancer OVCAR-3 and SKOV3 cells
Concentration:	1 μM

Incubation Time:	
Result:	Led to increased levels of NF-κB p65 phosphorylation. Caused a significant increase in the protein levels of nuclear NF-κB p65, which was accompanied by decreased levels of NF-κB p65 in the cytoplasm.

REFERENCES

[1]. Qian Liu, et al. 12-HETE facilitates cell survival by activating the integrin-linked kinase/NF-κB pathway in ovarian cancer. Cancer Manag Res. 2018 Nov 16;10:5825-5838.

[2]. Benedetta Porro, et al. Analysis, physiological and clinical significance of 12-HETE: a neglected platelet-derived 12-lipoxygenase product. J Chromatogr B Analyt Technol Biomed Life Sci. 2014 Aug 1;964:26-40.

[3]. Aidan J Hampson, et al. 12-hydroxyeicosatetrenoate (12-HETE) attenuates AMPA receptor-mediated neurotoxicity: evidence for a G-protein-coupled HETE receptor. J Neurosci. 2002 Jan 1;22(1):257-64.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA