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Produktinformation

- Forschungsprodukte & Biochemikalien
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See the following pages for more information!

Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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Linolelaidic acid

Cat. No.: HY-W071746

CAS No.: 506-21-8

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

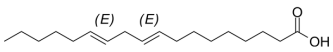
Molecular Weight: 280.45

Target: Endogenous Metabolite; Parasite; Apoptosis

Pathway: Metabolic Enzyme/Protease; Anti-infection; Apoptosis

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (445.71 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / 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.

BIOLOGICAL ACTIVITY

Description

Linolelaidic acid (Linoelaidic acid) is an omega-6 trans fatty acid (TFA) that is an essential nutrient with oral activity. Linolelaidic acid can be added to enteral nutrition (oral), parenteral nutrition (intravenous), and infant formula. Linolelaidic acid has anti-inflammatory and anti-parasitic ((Parasite)) activities, and can induce Apoptosis. Linolelaidic acid is useful for research in infections[1][2][3].

In Vitro

Linolenic Acid shows inhibitory activity against P. falciparum strains D10 (the chloroquine sensitive strain) and Dd2 (the chloroquine resistant strain), with IC_{50} values of 4.12 μg/mL and 5.04 μg/mL, respectively[2].

Linolelaidic acid (50 μM, 24 h) induces apoptosis, cell cycle arrest andinflammation in human umbilicalvein endothelial cells through lipid rafts[3].

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

Cell Viability Assay[3]

Cell Line:	Human umbilicalvein endothelial cells (HUVECs) (Methyl-β-cyclodextrin (HY-101461)-induced lipid raft disruption model)
Concentration:	50 μM

Incubation Time:	24 h
Result:	Resulted in a significant decrease in cell viability.

Apoptosis Analysis[3]

Cell Line:	Human umbilicalvein endothelial cells (HUVECs) (Methyl-β-cyclodextrin (HY-101461)-induced lipid raft disruption model)
Concentration:	50 μM
Incubation Time:	24 h
Result:	Resulted in a significant increase in the number of apoptotic cells and the cell population in the G1 phase.

Western Blot Analysis[3]

Cell Line:	Human umbilicalvein endothelial cells (HUVECs) (Methyl-β-cyclodextrin (HY-101461)-induced lipid raft disruption model)
Concentration:	50 μM
Incubation Time:	24 h
Result:	Led to a significant increase in the expression levels of pro-apoptotic proteins (caspase-3, -8, Bax, p53) and inflammatory factors (vascular cell adhesion molecule-1, intercellular adhesion molecule, E-selectin, and nitric oxide), while the expression level of the anti-apoptotic protein Bcl-2 was significantly decreased.

In Vivo

Linolenic Acid (10 mg/kg, p.o., once daily for 4 days) shows anti-parasitic activity in the P. berghei (ANKA) strain-induced malaria C57BL/6 mouse model[2].

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

Animal Model:	P. berghei (ANKA) strain-induced malaria C57BL/6 mice model (7-to-10-week old)[3]
Dosage:	10 mg/kg
Administration:	Oral gavage (p.o.), once daily for 4 days
Result:	Inhibited the growth of P. berghei by 70%.

Animal Model:	High-fat diet-fed streptozotocin (HFD-STZ) rat model[2]
Dosage:	500 μg/kg
Administration:	Oral gavage (p.o.), once daily for 4 weeks, 30 min myocardial ischemia followed by 4 or 6 h reperfusion after 4 weeks
Result:	Significantly improved the instantaneous first derivation of left ventricle pressure, reduced infarct size, plasma creatine kinase and lactate dehydrogenase activities, and apotosis at the end of reperfusion in HFD-STZ diabetic rats. Not only significantly reduced tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6) concentrations but reduced the increase in superoxide production and malonaldialdehyde formation and simultaneously enhanced the antioxidant capacity in the diabetic hearts.

	Increased myocardial PI3K expression and Akt phosphorylation in diabetic but not normal rats.

REFERENCES

[1]. Melariri P, et al. In vitro and in vivo antimalarial activity of linolenic and linoleic acids and their methyl esters[J]. Adv Stud Biol, 2012, 4(7): 333-49.

[2]. Li J, et al. Linolelaidic acid induces apoptosis, cell cycle arrest and inflammation stronger than elaidic acid in human umbilical vein endothelial cells through lipid rafts[J]. European Journal of Lipid Science and Technology, 2017, 119(7): 1600374.

[3]. Jay Whelan, et al. Linoleic acid. Adv Nutr. 2013 May 1;4(3):311-2.

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