γ-Linolenic Acid methyl ester

Cat. No.: HY-W009276

CAS No.: 16326-32-2

Molecular Formula: $C_{19}H_{32}O_2$

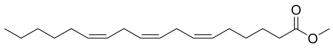
Molecular Weight: 292.46

Target: Apoptosis

Pathway: Apoptosis

Storage:

Pure form	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (341.93 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.4193 mL	17.0964 mL	34.1927 mL
	5 mM	0.6839 mL	3.4193 mL	6.8385 mL
	10 mM	0.3419 mL	1.7096 mL	3.4193 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.55 mM); Suspended solution

BIOLOGICAL ACTIVITY

Description

γ-Linolenic Acid methyl ester (Methyl GLA) is an esterified version of γ-Linolenic Acid (GLA), which is an ω-6 fatty acid, serves as melanoma cell proliferation inhibitors. γ-Linolenic Acid methyl ester inhibits ADP-induced blood platelet aggregation and induces apoptosis[1][2][3][4][5].

In Vitro

γ-Linolenic Acid methyl ester (1-4 μg/mL; 72 h) induces apoptosis in vitro A-549 lung cancer cell lines using SRB assay. However GLA is due to its action at the gene/oncogene level and by altering BCl-2 expression[1].

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

Apoptosis Analysis[1]

Cell Line:	A-549 lung cancer cell line
Concentration:	1, 2, 3, and 4 μg/mL

Incubation Time:	72 hours; observed at 24 h, 48 h, and 72 h
Result:	Showed cytotoxicity potentially due to the induction of apoptosis of tumor cells by augmenting free radical generation only in the tumor cells but not normal cells.

In Vivo

γ-Linolenic Acid methyl ester decreases the hepatic triglycerides and histological evidence of fatty liver induced by EtOH[2]. γ-Linolenic Acid methyl ester could significantly decrease levels of plasma total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-C), MDA, atherosclerosis index (AI) and liver TC, MDA, and increase levels of high density lipoprotein cholesterol (HDL-C) in both normal and hyperlipidemic rats[3].

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Animal Model:	Hepatic pathol rats model induced by EtOH (male Sprague-Dawley rats, 250-300 g)[2]
Dosage:	90 mL, 50-60 mL
Administration:	Intraperitoneal injection; once daily; administrated 90 mL during day 1-5 and day 9, 50-60 mL during day 6-8
Result:	Decreased the hepatic triglycerides of 25.6 mg/g compared with saline (40.2 mg/g) or olive oil (42.8 mg/g) treatment. Decreased liver index (the ratio of liver weight and body wight) in hyperlipidemic rats, but had no significant effect in normal rats.

REFERENCES

[1]. Jubie S, et al. Isolation of methyl gamma linolenate from Spirulina platensis using flash chromatography and its apoptosis inducing effect. BMC Complement Altern Med. 2015 Aug 4;15:263.

[2]. Segarnick DJ, et al. Gamma-linolenic acid inhibits the development of the ethanol-induced fatty liver. Prostaglandins Leukot Med. 1985 Mar;17(3):277-82.

[3]. Xiuqin K, et al. Studies on the hypolipidemic effects of gamma-linolenic acid methyl ester derived from Spirulina maxima[J]. Zhongguo hai Yang yao wu= Chinese Journal of Marine Drugs, 2003, 22(6): 30-34.

[4]. Williams, et al. Antithrombosis agent containing γ-linolenic acid or a functional derivative of it: Federal Republic of Germany, DE2749492[P]. 1978-05-11.

[5]. Hiyamuta, et al. Melanoma cell proliferation inhibitors containing γ-linolenic acid or its derivatives: Japan, JP2014141427[P]. 2014-08-07.

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