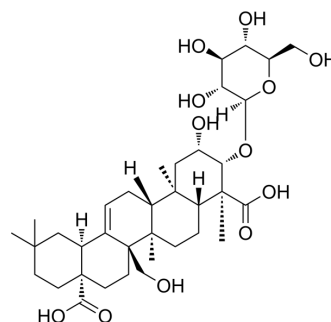


Tenuifolin

Cat. No.:	HY-N0702
CAS No.:	20183-47-5
Molecular Formula:	C ₃₆ H ₅₆ O ₁₂
Molecular Weight:	680.82
Target:	Beta-secretase; Cholinesterase (ChE)
Pathway:	Neuronal Signaling
Storage:	-20°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro	DMSO : 100 mg/mL (146.88 mM; Need ultrasonic)					
	Preparing Stock Solutions	Solvent Concentration	Mass	1 mg	5 mg	10 mg
		1 mM		1.4688 mL	7.3441 mL	14.6882 mL
		5 mM		0.2938 mL	1.4688 mL	2.9376 mL
		10 mM		0.1469 mL	0.7344 mL	1.4688 mL
Please refer to the solubility information to select the appropriate solvent.						
In Vivo	<ol style="list-style-type: none"> Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.5 mg/mL (3.67 mM); Clear solution Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (3.67 mM); Clear solution Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (3.67 mM); Clear solution 					

BIOLOGICAL ACTIVITY

Description	Tenuifolin is effective and has a protective action. Tenuifolin inhibits β-secretase decreases Aβ protein secretion, suppresses Aβ ₂₅₋₃₅ secretion, and subsequently caspase-3 and caspase-9 become active. Tenuifolin's ability to lower AChE activity, increase at the same time, increase the ability of the upper glands, and improve the ability to read and remember. Research on tenuifolin's potential for use in urinary disease (AD).
IC₅₀ & Target	AChE
In Vitro	Tenuifolin (50 μM; 24 h) protects SH-SY5Y cells against Aβ ₂₅₋₃₅ (20 μM)-induced apoptosis, and decreased the mRNA level of caspase-3 and -9 ^[2] .

Tenuifolin (50 μ M, 100 μ M; 24 h) inhibits A β ₂₅₋₃₅ (20 μ M)-induced the loss of mitochondria-membrane potential in SH-SY5Y cells^[2].

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

In Vivo

Tenuifolin (20-80 mg/kg; po; single dose) induces beneficial effects on learning and memory, prevents loss and apoptosis of neurons in APP/PS1 mice^[2].

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

REFERENCES

[1]. Ki-Yeol Yoo, et al. Terpenoids as Potential Anti-Alzheimer's Disease Therapeutics. *Molecules* 2012, 17(3), 3524-3538

Caution: Product has not been fully validated for medical applications. For research use only.

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