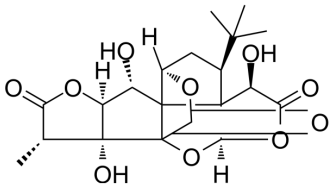
Ginkgolide B

Cat. No.:	HY-N0784
CAS No.:	15291-77-7
Molecular Formula:	$C_{20}H_{24}O_{10}$
Molecular Weight:	424.4
Target:	Apoptosis; Platelet-activating Factor Receptor (PAFR)
Pathway:	Apoptosis; GPCR/G Protein
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (235.63 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Mass / Solvent / Concentration	1 mg	5 mg	10 mg
1 mM	2.3563 mL	11.7813 mL	23.5627 mL
5 mM	0.4713 mL	2.3563 mL	4.7125 mL
10 mM	0.2356 mL	1.1781 mL	2.3563 mL

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

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 5 mg/mL (11.78 mM); Suspended solution; Need ultrasonic and warming and heat to 44°C
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.89 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (4.90 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.90 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ginkgolide B (BN-52021), a terpene lactone, is a potent platelet activating factor antagonist. Ginkgolide B protects endothelial cells via the activation of PXR from injuries induced by xeno- and endobiotics. Ginkgolide B can pass through the brain-blood barrier. Ginkgolide B has anti-oxidant, anti-inflammatory, anti-tumor, and anti-apoptotic activity. Ginkgolide B has marked neuroprotective effects against ischemia-induced impairments[1][2].

In Vitro

Ginkgolide B (BN-52021; 30-150 μM; 24 h) inhibits the up-regulation of VCAM-1 and E-selectin protein expression induced by TNF-α (10 ng/mL)[1].
Ginkgolide B (30-150 μM; 24, 48 h) increases CYP3A4 mRNA and MDR1 expression[1].
Ginkgolide B (30-150 μM; pretreated for 24 h) reduces Staurosporine-induced (500 nM; 6 h) apoptosis compared with the positive controls, which exhibited marked apoptosis compared with negative controls. Ginkgolide B does not affect Doxorubicin (5 μM; 24 h) induced apoptosis[1].
Ginkgolide B regulates PXR activity and does not affect PXR expression[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	HUVECs
Concentration:	30, 60, 150 μM
Incubation Time:	24 h
Result:	Inhibited the up-regulation of VCAM-1 and E-selectin protein expression induced by TNF-α.

RT-PCR[1]

Cell Line:	HUVECs
Concentration:	30, 60, 150 μM
Incubation Time:	24, 48 h
Result:	Increased CYP3A4 mRNA and MDR1 expression after 24 h, and this effect was dose-dependent after 48 h.

In Vivo

Ginkgolide B (BN-52021; 20 mg/kg; Intraperitoneally; immediately and 6 hours after ischemia, and thereafter once daily; 14 days) decreases the neurological deficit score[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male clean healthy Sprague-Dawley rats, weighing 250-280 g and aged 8 weeks[2]
Dosage:	20 mg/kg
Administration:	Intraperitoneally; immediately and 6 hours after ischemia, and thereafter once daily; 14 days
Result:	Decreased the neurological deficit score, increased the proportion of nestin-, neuron-specific enolase- and glial fibrillary acid protein-positive cells. Caused down-regulation of NSE protein at day 14.

CUSTOMER VALIDATION

- Biofactors. 2019 Nov;45(6):950-958.
- Neuropharmacology. 2024 Mar 14:250:109907.
- Research Square Print. 2022 Jun.

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REFERENCES

[1]. Tao Zhou, et al. Ginkgolide B protects human umbilical vein endothelial cells against xenobiotic injuries via PXR activation. Acta Pharmacol Sin. 2016 Feb;37(2):177-86.

[2]. Pei-Dong Zheng, et al. Ginkgolide B promotes the proliferation and differentiation of neural stem cells following cerebral ischemia/reperfusion injury, both in vivo and in vitro. Neural Regen Res. 2018 Jul;13(7):1204-1211.

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

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