

Product Data Sheet

Product Name: Ginkgolide B

Cat. No.: GN10453

Chemical Properties

Cas. No. 15291-77-7

SMILES O=C1O[C@@]2([H])C([C@@]([C@]([C@@]3([H])OC4=O)([C@]4([H])C([H])([H])[H])O[H])1O[C@]5([H])OC6=O)(C3([H])O[H])C5([C@@]6([H])O[H])C(C(C([H])([H])[H])(C([H])([H])[H])C([H])([H])[H])([H])C2([H])[H]

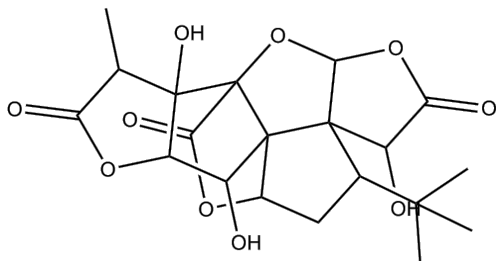
Formula $C_{20}H_{24}O_{10}$ M.Wt 424.4

Solubility DMF: 25 mg/ml, DMF:PBS(pH 7.2) (1:1): 0.5 mg/ml, DMSO: 14 mg/ml Storage Store at 2-8°C Dry, sealed and protect from light

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines Human umbilical vein endothelial cells

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Human umbilical vein endothelial cells (HUVECs) were cultured in RPMI-1640 supplemented with 10% (v/v) FBS and grown in a humidified incubator containing 5% CO₂ at 37°C. Ginkgolide B or RIF was dissolved in DMSO and diluted with RPMI-1640. The final concentration of DMSO never exceeded 1% of the total culture volume. The cells were seeded in 6-, 12-, or 24-well culture plates for each experiment and treated with various concentrations (30–300 μM) of ginkgolide B for 24h. Then cells were collected for further analyses.

Preparation Method
Reaction Conditions 30–300 μM; 24h

Applications Ginkgolide B dose-dependently increased PXR nuclear translocation, upregulated CYP3A4 and MDR1 expression, attenuated doxorubicin- or staurosporine-induced apoptosis, and suppressed TNF-α-induced THP-1 cell adhesion and expression of VCAM-1 and E-selectin.

Animal experiment [2]:

Animal models Sprague-Dawley rats

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Preparation Method	A total of 150 male clean healthy Sprague-Dawley rats, weighing 250–280g and aged 8 weeks were randomly assigned to three groups: sham, middle cerebral artery occlusion (MCAO) and MCAO + Ginkgolide B ($n = 50$ per group). A cerebral ischemia/reperfusion model was established. In brief, a 4-0 monofilament nylon thread was inserted into the right common carotid artery to obstruct the middle cerebral artery (MCA) for 90 minutes, and then the thread was withdrawn. In the sham group, the thread was inserted into the right common carotid artery without reaching the bifurcation between the MCA and anterior cerebral artery. Ginkgolide B at 20mg/kg was intraperitoneally administered immediately and 6 hours after ischemia, and thereafter once daily. In the sham and MCAO groups, an equal volume of normal saline (2mL) was intraperitoneally administered with the same schedule. In each group, rats received intraperitoneal injection of bromodeoxyuridine (BrdU) at 50mg/kg three times (once every 4 hours) within 12 hours. Then, 4 hours after the last injection of BrdU, the rats were sacrificed for assessing the proliferation of NSCs. At 3, 7 and 14 days after surgery, neurological function was evaluated ($n=5$ per time point for each of the three groups). These rats were then sacrificed for immunohistochemistry, real-time quantitative polymerase chain reaction (RT-PCR) and western blot assay ($n=5$ per group, for a total of 45 rats for each of the three assays).
Dosage form	20mg/kg/day for 14 days; i.p.
Applications	Ginkgolide B significantly reduce the volume of cerebral infarction and the neurological deficit score.

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References:

- [1] Zhou T, You WT, Ma ZC, et al. Ginkgolide B protects human umbilical vein endothelial cells against xenobiotic injuries via PXR activation. *Acta Pharmacol Sin.* 2016;37(2):177-186.
- [7] Zheng PD, Mungur R, Zhou HJ, Hassan M, Jiang SN, Zheng JS. 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;13(7):1204-1211.

Background

Ginkgolide B is a terpene lactone extracted from Ginkgo biloba leaves and is a specific antagonist of platelet activating factor (PAF), with an IC₅₀ of 3.6μM^[1]. Platelet activating factor (PAF) is a phospholipid inflammatory mediator that rapidly activates platelets, white blood cells and endothelial cells by binding to PAFR, amplifying inflammatory and thrombotic responses^[2]. Ginkgolide B has antioxidant, anti-inflammatory, anti-tumor, and anti-apoptotic activities and is commonly used for research on ischemia/reperfusion injury, neuroinflammation, and tumors^[3-5].

In vitro, treatment of Human umbilical vein endothelial cells (HUVECs) with Ginkgolide B (30–300μM; 24h) dose-dependently increased PXR nuclear translocation, upregulated CYP3A4 and MDR1 expression, attenuated doxorubicin- or staurosporine-induced apoptosis, and suppressed TNF-α-induced THP-1 cell adhesion and expression of VCAM-1 and E-selectin^[6].

In vivo, in rat model of cerebral ischemia/reperfusion injury, Ginkgolide B(20mg/kg; i.p.; 14days) significantly reduce the volume of cerebral infarction and the neurological deficit score, increased the proportion of nestin-, neuron-specific enolase- and glial fibrillary acid protein-positive cells, increased the mRNA expression of brain-derived neurotrophic factor(BDNF) and epidermal growth factor(EGF), and increased the expression levels of BDNF and suppressor of cytokine signaling 2(SOCS2) in the ischemic penumbra^[7]. In pentobarbitone or ethyl carbamate-anaesthetized guinea-pigs, Ginkgolide B (1mg/kg; i.v.; 1h) inhibited bronchoconstriction, the hematocrit increase and the accompanying thrombopenia and leukopenia induced by PAF-acether^[8].

References:

- [1] Lamant V, Mauco G, Braquet P, Chap H, Douste-Blazy L. Inhibition of the metabolism of platelet

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activating factor (PAF-acether) by three specific antagonists from Ginkgo biloba. *Biochem Pharmacol.* 1987;36(17):2749-2752.

[2] Venable ME, Zimmerman GA, McIntyre TM, Prescott SM. Platelet-activating factor: a phospholipid autacoid with diverse actions. *J Lipid Res.* 1993;34(5):691-702.

[3] Koltai M, Tosaki A, Hosford D, Braquet P. Ginkgolide B protects isolated hearts against arrhythmias induced by ischemia but not reperfusion. *Eur J Pharmacol.* 1989;164(2):293-302.

[4] Lee CW, Lin HC, Wang BY, et al. Ginkgolide B monotherapy reverses osteoporosis by regulating oxidative stress-mediated bone homeostasis. *Free Radic Biol Med.* 2021;168:234-246.

[5] Ahlemeyer B, Krieglstein J. Pharmacological studies supporting the therapeutic use of Ginkgo biloba extract for Alzheimer's disease. *Pharmacopsychiatry.* 2003;36 Suppl 1:S8-S14.

[6] Zhou T, You WT, Ma ZC, et al. Ginkgolide B protects human umbilical vein endothelial cells against xenobiotic injuries via PXR activation. *Acta Pharmacol Sin.* 2016;37(2):177-186.

[7] Zheng PD, Mungur R, Zhou HJ, Hassan M, Jiang SN, Zheng JS. 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;13(7):1204-1211.

[8] Desquand S, Touvy C, Randon J, et al. Interference of BN 52021 (ginkgolide B) with the bronchopulmonary effects of PAF-acether in the guinea-pig. *Eur J Pharmacol.* 1986;127(1-2):83-95.

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