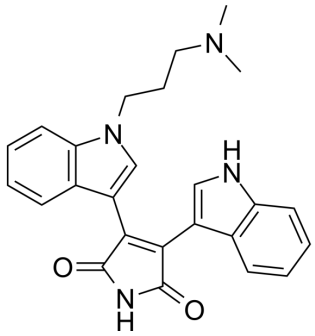
Bisindolylmaleimide I

Cat. No.:	HY-13867
CAS No.:	133052-90-1
Molecular Formula:	$C_{25}H_{24}N_4O_2$
Molecular Weight:	412
Target:	PKC; GSK-3
Pathway:	Epigenetics; TGF-beta/Smad; PI3K/Akt/mTOR; Stem Cell/Wnt
Storage:	4°C, protect from light * In solvent : -80°C, 1 years; -20°C, 6 months (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 32 mg/mL (77.67 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4272 mL	12.1359 mL	24.2718 mL
5 mM	0.4854 mL	2.4272 mL	4.8544 mL
10 mM	0.2427 mL	1.2136 mL	2.4272 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1 mg/mL (2.43 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Bisindolylmaleimide I (GF109203X) is a cell-permeable and reversible PKC inhibitor (IC_{50} of 20 nM, 17 nM, 16 nM, and 20 nM for PKCα, PKCβI, PKCβII, and PKCγ. Bisindolylmaleimide I is also a GSK-3 inhibitor[1][2][3].

IC_{50} & Target

Bovine brain PKC 10 nM (IC_{50})	PKCβII 16 nM (IC_{50})	PKCβI 17 nM (IC_{50})	PKCα 20 nM (IC_{50})
PKCγ 20 nM (IC_{50})	FDGFR 65 μM (IC_{50})		

In Vitro

Bisindolylmaleimide I (5 μM) inhibits α-thrombin-induced P47 phosphorylation[1].
Bisindolylmaleimide I (0-1 μM) inhibits DNA synthesis in quiescent swiss 3T3 cells[1].
Bisindolylmaleimide I (5 μM) reduces GSK-3 activity to 25.1±4.3% in adipocytes lysates[3].
Bisindolylmaleimide I (10 μM, 24 h) inhibits exosome and microvesicle (EMV) release from PC3 cells[4].

	Bisindolylmaleimide I (10 μM, 24 h) enhances cytotoxicity of 5-fluorouracil (HY-90006)[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Bisindolylmaleimide I (0.02 mg/kg, i.p.) reduced the inceased NLRP3, P-PKCα, and PKCα levels in mechanical ventilation (MV) group in mice[5]. Bisindolylmaleimide I (0-20 mg/kg, i.p.) reduces the mean frequency of Quinpirole-induced vomiting in shrews[6]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Quinpirole-treated shrews[2]
Dosage:	0-20 mg/kg
Administration:	i.p.
Result:	Reduced the mean frequency of Quinpirole-induced vomiting. Blocked Quinpirole-mediated ERK1/2 phosphorylation in shrew brainstems.

CUSTOMER VALIDATION

- Theranostics. 2021 Mar 11;11(11):5279-5295.
- Redox Biol. 2023 Apr 20, 102702.
- Redox Biol. 2021 Oct;46:102098.
- J Adv Res. 2022 Jul 13;S2090-1232(22)00156-4.
- Mucosal Immunol. 2021 Oct 22.

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REFERENCES

[1]. Hers I, et al. The protein kinase C inhibitors bisindolylmaleimide I (GF 109203x) and IX (Ro 31-8220) are potent inhibitors of glycogen synthase kinase-3 activity. FEBS Lett. 1999 Nov 5;460(3):433-6.

[2]. Kosgodage US, et al. Chloramidine/Bisindolylmaleimide-I-Mediated Inhibition of Exosome and Microvesicle Release and Enhanced Efficacy of Cancer Chemotherapy. Int J Mol Sci. 2017 May 9;18(5):1007.

[3]. Liu M, et al. Aerobic exercise alleviates ventilator-induced lung injury by inhibiting NLRP3 inflammasome activation. BMC Anesthesiol. 2022 Dec 1;22(1):369.

[4]. Belkacemi L, et al. Signal transduction pathways involved in dopamine D2 receptor-evoked emesis in the least shrew (Cryptotis parva). Auton Neurosci. 2021 Jul;233:102807.

[5]. Toullec D, et al. The bisindolylmaleimide GF 109203X is a potent and selective inhibitor of protein kinase C. J Biol Chem. 1991 Aug 25;266(24):15771-81.

[6]. Vetri F, et al. Impairment of neurovascular coupling in Type 1 Diabetes Mellitus in rats is prevented by pancreatic islet transplantation and reversed by a semi-selective PKC inhibitor. Brain Res. 2017 Jan 15;1655:48-54.

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