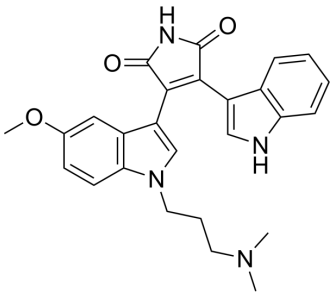
Go 6983

Cat. No.:	HY-13689
CAS No.:	133053-19-7
Molecular Formula:	$C_{26}H_{26}N_4O_3$
Molecular Weight:	442.51
Target:	PKC
Pathway:	Epigenetics; TGF-beta/Smad
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 34 mg/mL (76.83 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2598 mL	11.2992 mL	22.5984 mL
5 mM	0.4520 mL	2.2598 mL	4.5197 mL
10 mM	0.2260 mL	1.1299 mL	2.2598 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: ≥ 2.5 mg/mL (5.65 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (5.65 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.5 mg/mL (5.65 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Go 6983 is a pan-PKC inhibitor against for PKCα, PKCβ, PKCγ, PKCδ and PKCζ with IC_{50} of 7 nM, 7 nM, 6 nM, 10 nM and 60 nM, respectively.

IC_{50} & Target

PKCγ 6 nM (IC_{50})	PKCα 7 nM (IC_{50})	PKCβ 7 nM (IC_{50})	PKCδ 10 nM (IC_{50})
PKCζ	PKCμ		

	60 nM (IC_{50})	20000 nM (IC_{50})

In Vitro

Go 6983 inhibits PKCμ with IC_{50} of 20 μM, and the pther PKC isoenzymes can be suppressed by Go 6983 with IC_{50} values from 7 to 60 nM[1]. Go 6983 (100 nM) significantly reduces PMN adherence to the endothelium and infiltration into the myocardium compared with I/R + PMN hearts, and significantly inhibits superoxide release from PMNs by 90 +/- 2% in rat hearts[2]. Go 6983 (200 nM) has a reduced cardioprotective effect compared with the cardioprotective Go 6983 concentrations (50 and 100 nM) despite inhibiting PMN superoxide release by 99%[3].

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

PROTOCOL

Kinase Assay [1]

Phosphorylation reactions are carried out in a total volume of 100 μL, containing buffer C (50 mM Tris-HCl, pH 7.5, 10 mM β-mercaptoethanol), 4 mM $MgCl_2$, 10 μg PS, 100 nM TPA, 5 μL of a Sf158 cell extract as a source of recombinant PKCμ or of Sf9 cell extracts as a source of other recombinant PKC isoenzymes, 10 μg of syntide 2 as substrate, and 35 μM ATP containing 1 μ Ci [γ-^{32}P]ATP. In some experiments, PS and TPA are omitted or various inhibitors at concentrations indicated in the text are added. After incubation for 10 min at 30°C, the reaction is terminated by transferring 50 μL of the assay mixture onto a 20 mm square piece of phosphocellulose paper, which is washed 3 times in deionized water and twice in acetone. The radioactivity on each paper is determined by liquid scintillation counting.

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

CUSTOMER VALIDATION

- Nature. 2022 Jan;601(7894):600-605.
- Immunity. 2021 Sep 14;54(9):2042-2056.e8.
- Nat Commun. 2022 Nov 10;13(1):6796.
- Adv Sci (Weinh). 2023 Nov 22:e2304987.
- Nat Protoc. 2023 Feb 15.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Gschwendt M, et al. Inhibition of protein kinase C mu by various inhibitors. Differentiation from protein kinase c isoenzymes. FEBS Lett, 1996, 392(2), 77-80.

[2]. Peterman EE, et al. G0 6983 exerts cardioprotective effects in myocardial ischemia/reperfusion. J Cardiovasc Pharmacol, 2004, 43(5), 645-656.

[3]. Young LH, et al. G0 6983: a fast acting protein kinase C inhibitor that attenuates myocardial ischemia/reperfusion injury. Cardiovasc Drug Rev, 2005, 23(3), 255-272.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA