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Phorbol 12-myristate 13-acetate

Cat. No.: HY-18739

CAS No.: 16561-29-8

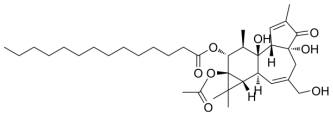
Molecular Formula: $C_{36}H_{56}O_8$

Molecular Weight: 616.83

Target: PKC; SphK; NF-κB

Pathway: Epigenetics; TGF-beta/Smad; Immunology/Inflammation; NF-κB

Storage: 4°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 (162.12 mM; Need ultrasonic)
Ethanol : 100 mg/mL (162.12 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.6212 mL	8.1060 mL	16.2119 mL
	5 mM	0.3242 mL	1.6212 mL	3.2424 mL
	10 mM	0.1621 mL	0.8106 mL	1.6212 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 (4.05 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (4.05 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.05 mM); Clear solution
4. Add each solvent one by one: 10% EtOH >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (4.05 mM); Clear solution
5. Add each solvent one by one: 10% EtOH >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (4.05 mM); Suspended solution; Need ultrasonic
6. Add each solvent one by one: 10% EtOH >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.05 mM); Clear solution

BIOLOGICAL ACTIVITY

Description Phorbol 12-myristate 13-acetate (PMA), a phorbol ester, is a dual SphK and protein kinase C (PKC) activator[1][2]. Phorbol 12-

	myristate 13-acetate is a NF-κB activator. Phorbol 12-myristate 13-acetate induces differentiation in THP-1 cells[3][7].
IC_{50} & Target	PKC 11.7 nM (EC50) NF-κB
In Vitro	In order to examine the role of PKC in p38MAPK phosphorylation, the cells are stimulated with the PKC activator, Phorbol 12-myristate 13-acetate (PMA) (100 nM), which mimics the binding of DAG, the natural activator of PKC, to the C1 region of the PKCs. p38MAPK phosphorylation by PMA is observed in the two cell types similar to that observed by GnRH in αT3-1 cells, that is, a slow sustained activation (3.2-fold and 3.6-fold, respectively at 30 min). The paradoxical findings that PKCs activated by GnRH and PMA play a differential role in p38MAPK phosphorylation may be explained by differential localization of the PKCs. Basal, GnRH- and PMA- stimulation of p38MAPK phosphorylation in αT3-1 cells is mediated by Ca^{2+} influx via voltage-gated Ca^{2+} channels and Ca^{2+} mobilization, while in the differentiated LβT2 gonadotrope cells it is mediated only by Ca^{2+} mobilization[2]. THP-1 cells are differentiated into macrophage-like cells (THP-1 macrophages) by incubation in the presence of PMA (200 ng/mL; 1-5 days), which leads to a macrophage-like phenotype characterized by changes in morphology and increased cell surface expression of CD11 and CD14[3]. In the monocytic cell line THP-1, PMA results in a more differentiated phenotype than VD3, according to adherence, loss of proliferation, phagocytosis of latex beads, and expression of CD11b and CD14[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Phorbol 12-myristate 13-acetate can be used in animal modeling to construct eczema-like models. Phorbol 12-myristate 13-acetate (PMA) is a PKC agonist, which reverses the damage induced by 5-hydroxydecanoic acid (5-HD). Thus, activation of the mitoKATP protected mitochondrial function in SOD and MDA via the PKC pathway[4]. 1. Induction of oedema at ear[8] Background PMA induces a pronounced inflammatory response mediated by protein kinase C (PKC), specifically activating PLA2 to trigger inflammation. Specific Mmodeling Methods Mice: Swiss mouse • Female • 25-30 g Administration: Topically applied in one ear • 100 μg/mL in 20 μL (2 μg/ear) vehicle • single dose Note Modeling Indicators Appearance monitoring: The thickness difference between the left and right ears increases significantly. Indicator changes: Increased vascular permeability. □□□□: □□□□:Hydroxyachillin; Indomethacin (HY-14397) 2. Induction of oedema at feet[9] Background PMA induces a pronounced inflammatory response mediated by protein kinase C (PKC), specifically activating PLA2 to trigger inflammation. Specific Mmodeling Methods Rats: Wistar • male • adult with weight of 200-220 g Mice: Swiss albino • male • 25-30 g Administration: Topically applied in one ear • 2.5 μg in 20 μL vehicle • single dose Note Administration should be conducted 4 h before mouse were killed. Modeling Indicators Appearance monitoring: The quality difference between the left and right ears increases significantly. Indicator changes: Stimulate macrophages to produce superoxide anions. □□□□: Carrageenan (HY-125474); Histamine (HY-B1204); Serotonin (HY-B1473A); Prostaglandin E2 (PGE2) (HY-101952) □□□□: MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]

αT3-1 and LβT-2 cells are grown in monolayer cultured in DMEM in humidified incubator 5% CO_2 at 37°C. Serum starvation is with 0.1% FCS in the same medium for 16 h. GnRH and PMA are then added for the length of time as indicated. In general, α T3-1 cells are transiently transfected by ExGen 500 or by jetPRIME, while LβT2 cells only by jetPRIME transfection reagent. For experiments with dominant-negative (DN) PKCs, αT3-1 cells (in 6 cm plates) are transfected with 1.5 μg of p38α-GFP with 3 μg of control vector, pCDNA3, or with 3 μg of the DN-PKCs constructs. For LβT2 cells, transfections are performed (in 10 cm plates) with 4 μg of p38α-GFP along with 9 μg of control vector, pCDNA3, or with 9 μg of the DN-PKCs constructs. Approximately 30 h after transfection, the cells are serum starved (0.1% FCS) for 16 h and later stimulated with GnRH or PMA, washed twice with ice-cold PBS, treated with the lysis buffer, followed by one freeze-thaw cycle. Cells are harvested; following centrifugation (15,000×g, 15 min, 4°C) supernatants are taken for immunoprecipitation experiments[2].

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

Animal Administration [3]

Rats[3]

All experiments qre performed with male Wistar rats (weighing 250-280 g). One hundred and thirty-five Wistar rats are randomly divided into seven groups. (1) Rats in the sham group (n=21) are given a lateral cerebral ventricle injection of 0.9% normal saline; (2) Rats in the IR group (n=21) are given a lateral cerebral ventricle injection of 0.9% normal saline 30 min before middle cerebral artery occlusion (MCAO); (3) Rats in the Carbenoxolone (CBX) group (n=21) are given a lateral cerebral ventricle injection of CBX (5 μg/mL×10 μL) 30 min before MCAO; (4) Rats in the Sch-6783 group (n=21) are given a lateral cerebral ventricle injection of DZX (2 mM×30 μL) 30 min prior to MCAO; (5) Rats in the 5-HD group (n=21) are given a lateral cerebral ventricle injection of 5-HD (100 mM×10 μL), and after 10 min, DZX is injected 15 min prior to MCAO; (6) The rats in the DZX + Ro group (n=15) are given a lateral cerebral ventricle injection of DZX, and after 10 min, Ro-31-8425 (400 μg/kg) is injected 15 min prior to MCAO; (7) The rats in the 5-HD+PMA group (n=15) are given an intraperitoneal injection of PMA (200 μg/kg) after the injection of 5-HD and DZX.

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

CUSTOMER VALIDATION

- Cell. 2023 Nov 9;186(23):5114-5134.e27.
- Cell Res. 2023 Jun 19.
- Signal Transduct Target Ther. 2023 Aug 9;8(1):290.
- Mil Med Res. 2022 Aug 23;9(1):46.
- Protein Cell. 2021 Oct 22;1-21.

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REFERENCES

[1]. Sergio E. Alvarez, et al. Autocrine and paracrine roles of sphingosine-1-phosphate. TRENDS in Endocrinology and Metabolism Vol.18 No.8

[2]. Mugami S, et al. Differential roles of PKC isoforms (PKCs) and Ca2+ in GnRH and phorbol 12-myristate 13-acetate (PMA) stimulation of p38MAPK phosphorylation in immortalized gonadotrope cells. Mol Cell Endocrinol. 2017 Jan 5;439:141-154.

[3]. Hou S, et al. Mechanism of Mitochondrial Connexin43's Protection of the Neurovascular Unit under Acute Cerebral Ischemia-Reperfusion Injury. Int J Mol Sci. 2016 May 5;17(5). pii: E679.

[4]. Zhang T, et al. MPTP-Induced Depletion in Basolateral Amygdala via Decrease of D2R Activation Suppresses GABAA Receptors Expression and LTD Induction Leading to Anxiety-Like Behaviors. Front Mol Neurosci. 2017 Aug 7;10:247.

[5]. Schwende H, et al. Differences in the state of differentiation of THP-1 cells induced by phorbol ester and 1,25-dihydroxyvitamin D3. J Leukoc Biol. 1996;59(4):555-561.

[6]. Starr T, et al. The phorbol 12-myristate-13-acetate differentiation protocol is critical to the interaction of THP-1 macrophages with Salmonella Typhimurium. PLoS One. 2018;13(3):e0193601. Published 2018 Mar 14.

[7]. Heng-Ching Wen, et al. PMA inhibits endothelial cell migration through activating the PKC-δ/Syk/NF-κB-mediated up-regulation of Thy-1. Sci Rep. 2018 Nov 2;8(1):16247.

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