DMOG

Cat. No.: HY-15893

CAS No.: 89464-63-1

Molecular Formula: $C_6H_9NO_5$

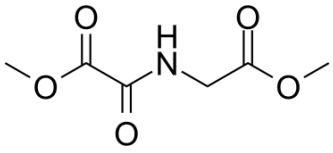
Molecular Weight: 175.14

Target: HIF/HIF Prolyl-Hydroxylase; Autophagy

Pathway: Metabolic Enzyme/Protease; Autophagy

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 : ≥ 50 mg/mL (285.49 mM)

H_2O : 50 mg/mL (285.49 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.7097 mL	28.5486 mL	57.0972 mL
5 mM	1.1419 mL	5.7097 mL	11.4194 mL
10 mM	0.5710 mL	2.8549 mL	5.7097 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 (14.27 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (14.27 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (14.27 mM); Clear solution
4. Add each solvent one by one: PBS
   Solubility: 150 mg/mL (856.46 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

DMOG (Dimethyloxallyl Glycine) is a cell permeable and competitive inhibitor of HIF-PH, which results in HIF-1α stabilisation and accmulation in vitro and in vivo[1]. DMOG is an α-ketoglutarate analogue and inhibits α-KG-dependent hydroxylases. DMOG acts as a pro-angiogenic agent and plays a protective role in experimental model of colitis and diarrhoea via HIF-1

	related signal[2][4]. DMOG induces cell autophagy[5].
IC_{50} & Target	HIF-1α prolyl hydroxylase[1]
In Vitro	DMOG efficiently suppresses hydroxyproline synthesis in intact cells, but shows only weakly active in the microsomal system [1]. DMOG reduces FGF-2-induced proliferation and cyclin A expression by inhibiting prolyl hydroxylase activity in HPASMC[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	DMOG inhibits endogenous HIF inactivation, and induces angiogenesis in ischaemic skeletal muscles of mice[2]. Up-regulation of hypoxia-inducible factor-1α by DMOG enhances the cardioprotective effects of ischemic postconditioning in hyperlipidemic rats[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]	To analyze DNA synthesis as an index of cellular proliferation, VSMC are plated in 48-well plates (5,000 per square centimeter) in growth medium, incubated overnight, and serum-deprived (1% FCS) for 24 h. Replicate wells are then stored at −70°C for baseline (day 0) cell counts, and fresh medium with or without growth factors is added to the remaining wells, which are incubated 72-96 h in 20 or 5% O_2. Days 0 and 3 or 4 cell counts are determined by lysing cells in a buffer containing a fluorescent dye, which has minimal fluorescence by itself but fluoresces when bound to DNA or RNA. Absolute cell numbers are calculated by comparing the fluorescence of specimens with that of a standard curve similarly prepared using a known number of cells. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Two groups of mice (C57Bl6) are used. One group (n=11) receives dimethyloxalylglycine (DMOG) i.p. (8 mg in 0.5 mL saline) on days 1, 3, 5, 7 and 9 while the animals in the other group are injected with sterile saline (0.5 mL) at the same intervals (n=6). A third group is treated with DMOG without ligation (n=4) and four unoperated mice serve as controls. After 11 days mice are terminally anaesthetized and the extensor digitorum longus (EDL) and tibialis anterior (TA) muscles excised. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Death Differ. 2020 Dec 7.
- Theranostics. 2020 Jun 12;10(16):7409-7421.
- Front Immunol. 2018 Jul 23;9:1667.
- J Biol Chem. 2015 Jan 16;290(3):1389-94.
- J Neurotrauma. 2019 Dec 15;36(24):3394-3409.

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REFERENCES

[1]. Baader E, et al. Inhibition of prolyl 4-hydroxylase by oxalyl amino acid derivatives in vitro, in isolated microsomes and in embryonic chicken tissues. Biochem J. 1994 Jun 1;300 (Pt 2):525-30.

[2]. Milkiewicz M, et al. Inhibition of endogenous HIF inactivation induces angiogenesis in ischaemic skeletal muscles of mice. J Physiol. 2004 Oct 1;560(Pt 1):21-6.

[3]. Schultz K, et al. Prolyl hydroxylase 2 deficiency limits proliferation of vascular smooth muscle cells by hypoxia-inducible factor-1{alpha}-dependent mechanisms. Am J

Physiol Lung Cell Mol Physiol. 2009 Jun;296(6):L921-7.

[4]. Li X, et al. Up-regulation of hypoxia-inducible factor-1α enhanced the cardioprotective effects of ischemic postconditioning in hyperlipidemic rats. Acta Biochim Biophys Sin (Shanghai). 2014 Feb;46(2):112-8.

[5]. Singh A, et al. Hypoxia-inducible factor (HIF) prolyl hydroxylase inhibitors induce autophagy and have a protective effect in an in-vitro ischaemia model.Sci Rep. 2020 Jan 31;10(1):1597.

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

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