Biacetyl monoxime

Cat. No.: HY-Y0413

CAS No.: 57-71-6

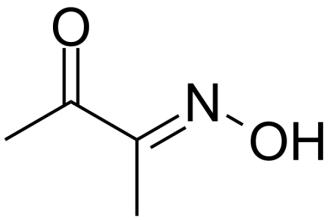
Molecular Formula: $C_4H_7NO_2$

Molecular Weight: 101.1

Target: Na+/K+ ATPase; Myosin

Pathway: Membrane Transporter/Ion Channel; Cytoskeleton

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (989.12 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	9.8912 mL	49.4560 mL	98.9120 mL
	5 mM	1.9782 mL	9.8912 mL	19.7824 mL
	10 mM	0.9891 mL	4.9456 mL	9.8912 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: ≥ 2.08 mg/mL (20.57 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (20.57 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (20.57 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Biacetyl monoxime (Diacetyl monoxime), a myosin ATPase inhibitor, is a skeletal and cardiac muscle contraction inhibitor. Biacetyl monoxime is also a well-characterized non-competitive inhibitor of chemical and motile activity of skeletal muscle myosin-II. Biacetyl monoxime induces sarcoplasmic reticulum Ca^{2+} release[1][2][3].

In Vitro

Biacetyl monoxime (Diacetyl monoxime) (50 mM, 6 and 48 h) decreases cellulase secretion in C. cinerea[1].
Biacetyl monoxime (50 mM, 2 and 4 h) disrupts the localization of the Golgi apparatus, but not that of the endoplasmic reticulum[1].
Biacetyl monoxime (0-30 mM) induces SR Ca^{2+} release (no efflux inhibitors) in a concentration-dependent manner, with a maximal reduction of 72% of SR Ca^{2+} at pCa 6.0[2].

Biacetyl monoxime acts as a chemical phosphatase, which has led to speculation that dephosphorylation of key Ca^{2+} channel proteins may be involved in its inhibition of contraction[2].
Biacetyl monoxime does not inhibit the ATPase activity of two different myosin-I isoforms, myosin-V, or myosin-VI[3].
Biacetyl monoxime (0-50 mM) suppresses L-type Ca^{2+} current of single cardiac myocytes isolated from SHR and WKY rats[4].
Biacetyl monoxime significantly reduces the duration of both spontaneous and electrically stimulated action potentials of cultured neonatal rat cardiomyocytes[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Biacetyl monoxime (0-200 mg/kg; i.v.; once) shows hypotensive effect[4].
Biacetyl monoxime (0-205 mg/kg; i.p.; once) shows anticonvulsant effect against Picrotoxin (HY-101391)-induced convulsions[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male SHR and age-matched WKY rat[4]
Dosage:	5, 30, I00 and 200 mg/kg
Administration:	Intravenous administration, 1 mL/kg, once
Result:	Decreased arterial blood pressure for both strains, the SHR was significantly more responsive.

Animal Model:	Male mice (20 to 25 g)[5]
Dosage:	51, 103 and 205 mg/kg in combination with intraperitoneal injection of 3.0 mg/kg Picrotoxin (HY-101391)
Administration:	Intraperitoneal injection, once
Result:	Showed dose-dependent anticonvulsant effect against Picrotoxin-induced convulsions.

REFERENCES

[1]. Ostap EM. 2,3-Butanedione monoxime (BDM) as a myosin inhibitor. J Muscle Res Cell Motil. 2002;23(4):305-8.

[2]. Xiao YF, et al. Effects of 2,3-butanedione monoxime on blood pressure, myocardial Ca2+ currents, and action potentials of rats. Am J Hypertens. 1995 Dec;8(12 Pt 1):1232-40.

[3]. Brightman T, et al. 2,3-Butanedione monoxime protects mice against the convulsant effect of picrotoxin by facilitating GABA-activated currents. Brain Res. 1995 Apr 24;678(1-2):110-6.

[4]. Kohsuke Hashimoto, et al. The myosin ATPase inhibitor, 2,3-butanedione 2-monoxime, prevents protein secretion by the basidiomycete Coprinopsis cinerea. Biotechnol Lett. 2011 Apr;33(4):769-75.

[5]. R M Phillips, et al. 2,3-Butanedione 2-monoxime (BDM) induces calcium release from canine cardiac sarcoplasmic reticulum. Biochem Biophys Res Commun. 1996 Dec 4;229(1):154-7.

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