Rhosin hydrochloride

Cat. No.: HY-12646

CAS No.: 1281870-42-5

Molecular Formula: $C_{20}H_{19}ClN_6O$

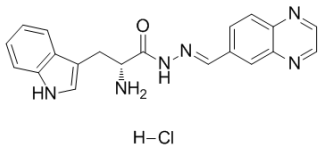
Molecular Weight: 394.86

Target: Ras; Apoptosis

Pathway: GPCR/G Protein; Apoptosis

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 : 100 mg/mL (253.25 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5325 mL	12.6627 mL	25.3254 mL
5 mM	0.5065 mL	2.5325 mL	5.0651 mL
10 mM	0.2533 mL	1.2663 mL	2.5325 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 (6.33 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.33 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.33 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Rhosin hydrochloride is a potent, specific RhoA subfamily Rho GTPases inhibitor. Rhosin hydrochloride specifically binds to RhoA to inhibit RhoA-GEF interaction with a K_d of ~ 0.4 uM, and does not interact with Cdc42 or Rac1, nor the GEF, LARG. Rhosin hydrochloride induces cell apoptosis[1][2]. Rhosin hydrochloride promotes stress resiliency through enhancing D1-MSN plasticity and reducing hyperexcitability[3].

IC_{50} & Target

Kd: 0.4 uM (RhoA)[1]

In Vitro	Rhosin hydrochloride dose-dependently reduces RhoA and p-MLC1 activities of MCF7 cell-derived mammospheres with an EC_{50} ~30-50 μM, and causes decreased size and reduced number of mammospheres in MCF7 cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Rhosin (40 mg/kg; i.p.) treatment prevents social avoidance caused by social defeat stress. Rhosin also blocks sucrose preference deficits induced by defeat in C57Bl6/J (Jackson) mice[3]. Rhosin (30 μM; bilateral, intra- Nucleus Accumbens (NAc) infusions) attenuates stress-induced social avoidance. Rhosin blocks stress-induced hyperexcitability in NAc dopamine 1 receptor medium spiny neurons (D1-MSNs). Rhosin prevents decreased excitatory transmission on NAc D1-MSNs. Rhosin enhances spine density in defeat mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	D1-GFP or D2-GFP hemizygote mice on a C57BL/6J background[3]
Dosage:	40 mg/kg
Administration:	I.p.
Result:	Rhosin was systemically administered 15 min prior to defeat to block RhoA activation. While defeat significantly reduced the time that experimental mice spent interacting with a novel mouse, Rhosin administration suppressed this effect without affecting locomotor behaviors.

CUSTOMER VALIDATION

- FASEB J. 2020 Jan;34(1):1481-1496.
- Cancer Biol Ther. 2018;19(12):1193-1203.
- Biochem Biophys Res Commun. 2019 Oct 29;519(1):134-140.
- J Breast Cancer. 2019 Apr 22;22(2):185-195.

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REFERENCES

[1]. Shang X, et al. Rational design of small molecule inhibitors targeting RhoA subfamily Rho GTPases. Chem Biol. 2012 Jun 22;19(6):699-710.

[2]. Shang X, et al. Small-molecule inhibitors targeting G-protein-coupled Rho guanine nucleotide exchange factors. Proc Natl Acad Sci U S A. 2013 Feb 19;110(8):3155-60.

[3]. Francis TC, et al. The Selective RhoA Inhibitor Rhosin Promotes Stress Resiliency Through Enhancing D1-Medium Spiny Neuron Plasticity and Reducing Hyperexcitability. Biol Psychiatry. 2019;85(12):1001-1010.

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