EHNA hydrochloride

Cat. No.: HY-103160A

CAS No.: 58337-38-5

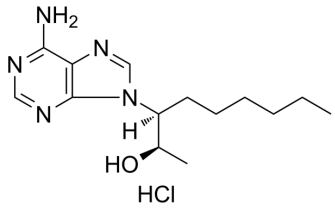
Molecular Formula: $C_{14}H_{24}ClN_5O$

Molecular Weight: 313.83

Target: Adenosine Deaminase; Phosphodiesterase (PDE); Influenza Virus

Pathway: Metabolic Enzyme/Protease; Anti-infection

Storage: -20°C, sealed storage, away from moisture
* In solvent : -80°C, 2 years; -20°C, 1 year (sealed storage, away from moisture)



Relative stereochemistry

SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (318.64 mM; Need ultrasonic)
DMSO : 83.33 mg/mL (265.53 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.1864 mL	15.9322 mL	31.8644 mL
5 mM	0.6373 mL	3.1864 mL	6.3729 mL
10 mM	0.3186 mL	1.5932 mL	3.1864 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 (6.63 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (6.63 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (6.63 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

EHNA hydrochloride is a potent and selective dual inhibitor of cyclic nucleotide phosphodiesterase 2 (PDE2)(IC_{50}=4 μM) and adenosine deaminase (ADA). EHNA hydrochloride exerts a concentration inhibition of the cGMP-stimulated PDE II (cGs-PDE)(IC_{50}:0.8 μM (human), 2 μM (porcine myocardium)), but has smaller inhibitory effect on the unstimulated PDE2 activity. EHNA hydrochloride play roles in mediating diverse pharmacological responses, including antiviral, antitumour and antiarrhythmic effects[1][2].

IC_{50} & Target

hPDE2A
0.8 μM (IC_{50})

In Vitro	EHNA completely ablates the ability of cyclic GMP to activate PDE2 activity, whilst having a much smaller inhibitory effect on the unstimulated PDE2 activity[2]. EHNA exhibits normal Michaelian kinetics of inhibition for the cyclic GMP-stimulated PDE2 activity with Hill plots near unity [2]. EHNA prevents dAdo degradation and increases mitochondrial dATP levels in fibroblasts[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Front Immunol. 04 October 2022.
- OncoImmunology. 2023, 12(1): 2152635.
- Eur J Pharmacol. 2021, 174077.

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REFERENCES

[1]. Podzuweit T, et al. Isozyme selective inhibition of cGMP-stimulated cyclic nucleotide phosphodiesterases by erythro-9-(2-hydroxy-3-nonyl) adenine.Cell Signal. 1995 Sep;7(7):733-8.

[2]. Michie AM, et al. Rapid regulation of PDE-2 and PDE-4 cyclic AMP phosphodiesterase activity following ligation of the T cell antigen receptor on thymocytes: analysis using the selective inhibitors erythro-9-(2-hydroxy-3-nonyl)-adenine (EHNA) and rolipram.

[3]. Blázquez-Bermejo C, et al. Increased dNTP pools rescue mtDNA depletion in human POLG-deficient fibroblasts. FASEB J. 2019 Jun;33(6):7168-7179.

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