Ezatiostat

Cat. No.: HY-13634A

CAS No.: 168682-53-9

Molecular Formula: $C_{27}H_{35}N_3O_6S$

Molecular Weight: 529.65

Target: Gutathione S-transferase; Apoptosis

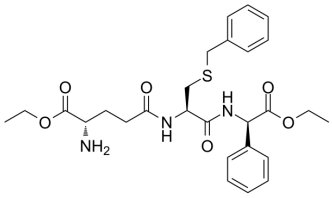
Pathway: Metabolic Enzyme/Protease; Apoptosis

Storage: Sealed storage, away from moisture

Powder -80°C 2 years

-20°C 1 year

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (188.80 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.8880 mL	9.4402 mL	18.8804 mL
5 mM	0.3776 mL	1.8880 mL	3.7761 mL
10 mM	0.1888 mL	0.9440 mL	1.8880 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: ≥ 2.75 mg/mL (5.19 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.75 mg/mL (5.19 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.75 mg/mL (5.19 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ezatiostat (TER199 free base; TLK199) is a tripeptide analog of glutathione and is a selective and orally active glutathione S-transferase P1-1 (GSTP1) inhibitor. Ezatiostat leads to JNK activation by inhibiting GSTP1. Ezatiostat stimulates both lymphocyte production and bone marrow progenitor proliferation. Ezatiostat has the potential for myelodysplastic syndrome (MDS) treatment[1][2].

IC_{50} & Target

Glutathione S-transferase P1-1 (GSTP1)[1]

In Vitro	Ezatiostat causes dissociation of the enzyme from the jun-N-terminal kinase/c-Jun (JNK/JUN) complex, leading to JNK activation by phosphorylation. The therapeutic action of ezatiostat appears to include both proliferation of normal myeloid progenitors as well as apoptosis of the malignant clone[1]. Selection of a resistant clone of an HL60 tumor cell line through chronic exposure to Ezatiostat (TLK199) results in cells with elevated activities of c-Jun NH2 terminal kinase (JNK1) and ERK1/ERK2, and allowes the cells to proliferate under stress conditions that induced high levels of apoptosis in the wild type cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Administration of Ezatiostat (TLK199), stimulates both lymphocyte production and bone marrow progenitor (colony-forming unit-granulocyte macrophage) proliferation, but only in glutathione S-transferase P1-1 ($GSTP1^{+/+}$) and not in $GSTP1^{-/-}$ animals[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Res. 2018 Dec;28(12):1171-1185.
- Adv Sci (Weinh). 2023 Jan 29;e2205262.
- Redox Biol. 2023 May.

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REFERENCES

[1]. Galili N, et al. Prediction of response to therapy with ezatiostat in lower risk myelodysplastic syndrome. J Hematol Oncol. 2012 May 6;5:20

[2]. Ruscoe JE, et al. Pharmacologic or genetic manipulation of glutathione S-transferase P1-1 (GSTpi) influences cell proliferation pathways. J Pharmacol Exp Ther. 2001 Jul;298(1):339-45.

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