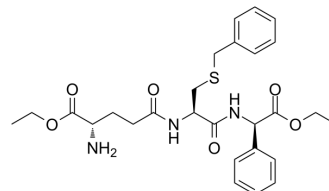


## Ezatiostat

<b>Cat. No.:</b>	HY-13634A
<b>CAS No.:</b>	168682-53-9
<b>Molecular Formula:</b>	C <sub>27</sub> H <sub>35</sub> N <sub>3</sub> O <sub>6</sub> S
<b>Molecular Weight:</b>	529.65
<b>Target:</b>	Gutathione S-transferase; Apoptosis
<b>Pathway:</b>	Metabolic Enzyme/Protease; Apoptosis
<b>Storage:</b>	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; Need ultrasonic)  
 Ethanol : ≥ 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

- 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
- 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
- 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<sup>[1][2]</sup>.

<b>IC<sub>50</sub> &amp; Target</b>	Glutathione S-transferase P1-1 (GSTP1) <sup>[1]</sup>
<b>In Vitro</b>	<p>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<sup>[1]</sup>.</p> <p>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 allows the cells to proliferate under stress conditions that induced high levels of apoptosis in the wild type cells<sup>[2]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
<b>In Vivo</b>	<p>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<sup>+/+</sup>) and not in GSTP1<sup>-/-</sup> animals<sup>[2]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

## 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.

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

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