Product Datasheet

GNE-140 racemate

Catalog No. **BP-02523** · CAS **1802977-61-2** · Form **free base** · Physical state **other** · Color **White to off-white** · Version **1.0** · Revision **2026-09-10**

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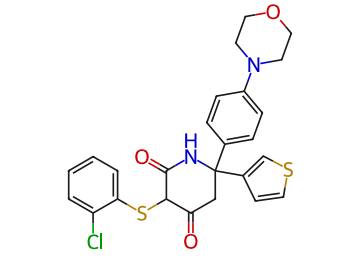
Target	Research area
Lactate Dehydrogenase; p38 MAPK; EGFR; Akt; Caspase; Reactive Oxygen Species (ROS)	**Inflammation/Immunology; Cancer**

Molecular formula	$C_{25}H_{23}ClN_2O_3S_2$
Molecular weight	**499.0450**

STRUCTURE



DESCRIPTION

GNE-140 racemate is a racemic mixture of (R)-GNE-140 and (S)-GNE-140 , and also a tautomer of GNE-140 . GNE-140 racemate is an orally active lactate dehydrogenase inhibitor, with an IC50 of 3 nM against human LDHA and an IC50 of 5 nM against LDHB. GNE-140 racemate reduces the phosphorylation level and total protein expression of p38 MAPK, and inhibits EGF-induced AKT phosphorylation. GNE-140 racemate decreases lactate production, regulates glycolysis, the pentose phosphate pathway and nucleotide biosynthesis, increases extracellular pH, inhibits cell proliferation, migration and invasion, and induces caspase-3 activation and ROS accumulation. GNE-140 racemate can be used in research related to breast cancer, cystic fibrosis and pancreatic cancer .

BIOLOGICAL ACTIVITY

GNE-140 racemate is a racemic mixture of (R)-GNE-140 and (S)-GNE-140. GNE-140 racemate is also a potent lactate dehydrogenase A (LDHA) inhibitor.

SOLUBILITY

Soluble in DMSO

In vitro	DMSO : 20 mg/mL

REFERENCES

[1].Khajah MA, et al. The effect of lactate dehydrogenase inhibitors on proliferation, motility and invasion of breast cancer cells highlights a new role for lactate. Molecular medicine reports. 2024 Jan;29(1):12.

[2].Valdivieso ÁG, et al. Impairment of CFTR activity in cultured epithelial cells upregulates the expression and activity of LDH resulting in lactic acid hypersecretion. Cellular and molecular life sciences : CMLS. 2019 Apr;76(8):1579-1593.

[3].Boudreau A, et al. Metabolic plasticity underpins innate and acquired resistance to LDHA inhibition. Nature chemical biology. 2016 Oct;12(10):779-86.

[4].Ji JJ, et al. Kallistatin/Serpina3c inhibits cardiac fibrosis after myocardial infarction by regulating glycolysis via Nr4a1 activation. Biochimica et biophysica acta. Molecular basis of disease. 2022 Sep 01;1868(9):166441.
