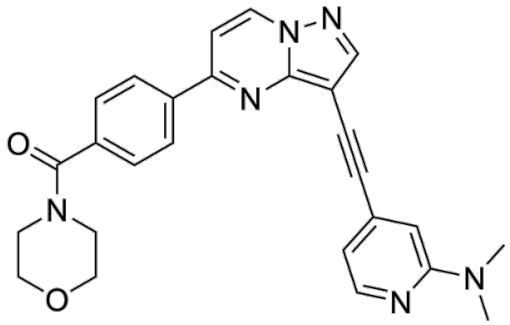
Product Data Sheet

Product Name: ETC-501
Cat. No.: GC79476

Chemical Properties

Cas. No.	3094990-88-9		
Formula	$C_{26}H_{24}N_6O_2$	M.Wt	452.51
Solubility	DMSO: 50 mg/mL (110.49 mM; Need ultrasonic)	Storage	Store at 4°C, away from moisture
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure			



Background

ETC-501 is a blood-brain barrier-permeable, orally active, and selective MNK1 / MNK2 inhibitor, with

an IC_{50} of 0.033 μM against MNK1 and 0.111 μM against MNK2. ETC-501 inhibits glioblastoma cell proliferation, impairs DNA damage repair function, delays cell cycle progression, and suppresses ribosome biogenesis. ETC-501 enhances Temozolomide -induced cellular senescence, attenuates the senescence-associated secretory phenotype, and increases cellular sensitivity to Navitoclax. ETC-501 is applicable to research related to glioblastoma [1].

In Vivo, ETC-501 (100 mg/kg; oral gavage; once daily; 21 days) enhances tumor cell senescence and attenuates the senescence-associated secretory phenotype when combined with TMZ in NSG mice bearing orthotopic or subcutaneous glioblastoma, and the additional combination with Navitoclax effectively eliminates senescent cells[1].

In Vitro, ETC-501 (0-20 μM; 24 h) dose-dependently inhibits eIF4E phosphorylation and reduces Cyclin D1 protein levels in LN-229 and T98G glioblastoma cells[1]. ETC-501 (0-10 μM) inhibits the viability of LN-229 and T98G glioblastoma cells with GI50 values of 4.035 μM and 6.122 μM, respectively, and reduces the tumor sphere-forming capacity and stem cell frequency of patient-derived GPCs[1]. ETC-501 (0-10 μM; 4 h) impairs DNA synthesis in LN-229, T98G and U-87MG glioblastoma cells in a dose-dependent manner[1]. ETC-501 (8 h) impairs G1-to-S phase progression in Palbociclib -synchronized LN-229 and U-87MG glioblastoma cells[1]. ETC-501 (5-10 μM; 14 days) induces cellular senescence in LN-229 and U-87MG glioblastoma cells[1]. ETC-501 (5-10 μM; 10 days) reduces the levels of key inflammatory cytokines and chemokines, including IL-6, IL-8 and CCL2[1]. ETC-501 (10 μM; 6 days) and TMZ drives LN-229 glioblastoma cells into a senescence priming state, and significantly enhances their sensitivity to Navitoclax -induced cell death and apoptosis[1].

References:
[1]. Nah GSS, et al. ETC-501 is a Brain Penetrant MNK Kinase Inhibitor that Potentiates TMZ-Induced Senescence and Sensitizes Glioblastoma Cells to Senolytic Therapy. Cancer Res. Published online January 22, 2026.