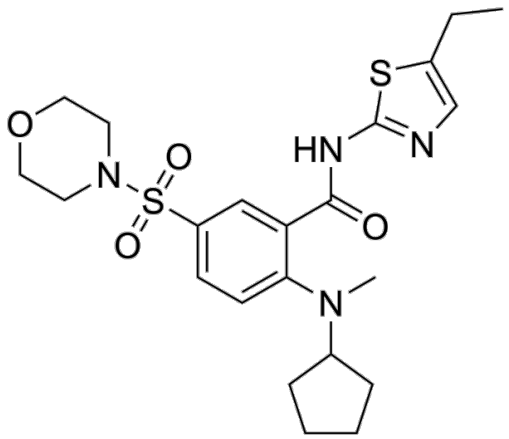
Product Data Sheet

Product Name: NGI-189
Cat. No.: GC79463

Chemical Properties

Cas. No.	2763063-26-7		
Formula	$C_{22}H_{30}N_4O_4S_2$	M.Wt	478.63
Solubility	DMSO: 100 mg/mL (208.93 mM; Need ultrasonic)	Storage	Store at -20°C
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

NGI-189 is a selective OST-A inhibitor. NGI-189 inhibits the STT3A catalytic subunit of the OST complex and reduces N-glycosylation of target glycoproteins. NGI-189 blocks oncogenic and bypass signaling, reduces phosphorylation of EGFR, AKT, p70S6K and S6RP, and induces cell cycle arrest and apoptosis. NGI-189 markedly suppresses tumor growth and induces tumor regression in

non-small cell lung cancer (NSCLC) xenograft models. NGI-189 can be used for the research of EGFR-mutant non-small cell lung cancer [1].

In Vivo, NGI-189 (10 mg/kg; i.p.; every other day; 8 total doses) elicits significant tumor growth delay in EGFR-mutant NSCLC PDX models as well as in gefitinib-resistant HCC827-GR NSCLC xenograft models[1]. NGI-189 (10 mg/kg; i.p.; every other day) induces significant tumor regression in osimertinib-resistant H1975-OR NSCLC xenografts[1]. NGI-189 (10 mg/kg; i.p.; every other day; 3 total doses) is well tolerated in mice, with no significant toxicity detected in blood work or organ histopathology[1].

In Vitro, NGI-189 (0.09 μM) potently inhibits N-glycosylation in ER-LucT reporter cells with an IC_{50} of 0.09 μM[1]. NGI-189 (5 μM; 5 days) inhibits proliferation of parental PC9 EGFR mutant NSCLC cells by ~70%, with significant rescue observed in PC9-CD8-EGFR-CL cells that express N-glycosylation-independent EGFR[1]. NGI-189 (0-25 μM; 24 h) preferentially inhibits the OST-A complex in HEK293 wild-type, STT3A KO, and STT3B KO cells, reducing N-glycosylation of EGFR and Halo3N in a dose-dependent manner, with complete inhibition of OST-A-dependent glycosylation achievable only in STT3B KO cells[1]. NGI-189 (10 μM; 24 h) reduces N-glycosylation of bypass PTK7, MET and inhibits downstream STAT3 signaling in Osimertinib -resistant H1975-OR and Gefitinib -resistant HCC827-GR NSCLC cells[1]. NGI-189 (5 μM) reduces clonogenic survival of parental PC9 EGFR-mutant NSCLC cells and shows significant rescue in PC9-CD8-EGFR-CL cells expressing N-glycosylation-independent EGFR; it also significantly decreases clonogenic survival of H3255, HCC-4006, and HCC-2935 EGFR-mutant NSCLC cells[1]. NGI-189 (5 μM; 24 h) inhibits EGFR and downstream AKT/p70S6K/S6RP signaling, and induces pro-apoptotic Bim protein expression in H3255, HCC-4006, and HCC-2935 EGFR mutant NSCLC cells[1]. NGI-189 (5 μM; 24 h) induces G1 phase cell cycle arrest in PC9 EGFR-mutant NSCLC cells, and triggers G1 arrest accompanied by marked cell death (sub-G1 population) in H3255 and HCC-2935 EGFR-mutant NSCLC cells[1].

References:
[1]. Baro M, et al. Redundancy of the OST catalytic subunit facilitates therapeutic targeting of N-glycosylation. Cell Chem Biol. 2025;32(6):839-853.e6.