Data Sheet (Cat.No.T214433)

TargetMol

SU212

Chemical Properties

CAS No. :	1262219-89-5
Formula:	C25H27NO6
Molecular Weight:	437.49
Storage:	Powder: -20°C for 3 years | In solvent: -80°C for 1 year Actual storage temperature shall be subject to the COA.

Biological Description

Description	SU212 is a podophyllotoxin-derived ENO1 inhibitor and AMPK activator. It selectively induces oxidative phosphorylation in cells, reduces glycolytic activity and glucose uptake in tumor cells, and directly binds ENO1 without affecting pathways in normal cells. SU212 induces apoptosis and facilitates ENO1 degradation via proteasomal and autophagic pathways without inhibiting its catalytic activity. It activates AMPK independently of energy stress and without being affected by glucose or insulin levels, resulting in M phase arrest and apoptosis in triple-negative breast cancer (TNBC) cells, demonstrating potent in vitro antitumor activity. Additionally, SU212 inhibits tumor growth and metastasis in syngeneic, xenograft, and diabetic mouse models, showing excellent safety profiles. This compound can be utilized in research on TNBC, diabetes, and fatty liver disease.
Targets(IC50)	Apoptosis,Caspase,AMPK,Autophagy,mTOR
In vitro	SU212 exhibits lower toxicity and greater efficacy against triple-negative breast cancer (TNBC) cells (MDA-MB-231) compared to Etoposide, with an IC 50 of 0.26 μM. It inhibits 50% cell viability in human TNBC cells with IC 50 values: MDA-MB-468 at 0.1 μM, SUM159 at 0.24 μM, and BT549 at 0.037 μM; and in mouse TNBC cell lines: 4T1 at 0.85 μM, EMT6 at 0.18 μM, E0771 at 0.039 μM, and PY8119 at 0.31 μM. SU212 targets different pathways in MDA-MB-231 cells compared to Etoposide, promoting ENO1 degradation via proteasome and autophagy pathways, which can be partially blocked by MG132 or 3 MA. SU212 enhances the thermal stability of ENO1 and ENO3, demonstrating stronger interaction with ENO1 and dose-dependent effects in TNBC cell lines (MDA-MB-231, MDA-MB-468, and EMT6). SU212 also inhibits the overall oxygen consumption rate, extracellular acidification rate, and glycolysis rate in MDA-MB-231, MCF12A, and HEK293 cells without affecting normal cell glycolysis and viability. It suppresses tumor regeneration and relapse in TNBC cells and induces G2/M phase arrest in MDA-MB-468 and MDA-MB-231 cells. Treatment with SU212 decreases the abundance of microtubulin forms in both normal breast cells (MCF10A, MCF12A) and TNBC cells (MDA-MB-231 and MDA-MB-468). It induces 12-60% apoptotic cell death in MDA-MB-468 and MDA-MB-231 cells but not autophagic cell death. SU212 activates AMPK by phosphorylating the Thr172 site on AMPKα in MDA-MB-231 cells and significantly induces AMPKα activation in MDA-MB-468 and MDA-MB-231 cells. The compound inhibits lactate production in MDA-MB-468 and MDA-MB-231 cells, without affecting D-glucose, glucose-6-phosphate/fructose-6-phosphate, ATP, citrate, oxygen consumption rate (OCR), extracellular acidification rate (ECAR), or α-ketoglutarate levels within MDA-MB-231

In vitro	cells. It reduces cellular lipid content by 24-70% in MDA-MB-468 and MDA-MB-231 cells. Furthermore, SU212 significantly increases proteins related to oxidative phosphorylation while decreasing those associated with glycolysis and the pentose phosphate pathway in TNBC cell lines, with no such effect in normal breast cell lines. Finally, it shows AMPK activation-dependent cytotoxicity in MDA-MB-468 and MDA-MB-231 cells and activates AMPK independently of energy stress in TNBC cell lines.
In vivo	SU212 has been shown to reduce the glycolytic rate and overall glucose demand in a female NSG mouse model induced by MDA-MB-231 cells when administered intraperitoneally at 30 mg/kg once daily for 3 days. At doses ranging from 100-400 mg/kg given once, it is well tolerated in female C57BL/6 mice and SD rats. The compound, at 30 mg/kg administered intraperitoneally five times a week for either 21 or 24 days, does not induce liver or kidney toxicity in a syngeneic orthotopic TNBC model. In a heterozygous MMTV-PyMT transgenic female mouse model, a dose of 20 mg/kg given five times a week has a positive effect on tumor initiation and progression. Furthermore, in an orthotopic EMT6 mouse model of TNBC, 30 mg/kg of SU212 administered five times a week for 21 days induces ENO1 degradation, alters its subcellular location, and diminishes its function. In Lepr db (Db/Db) mouse models, SU212 at 10 mg/kg administered under hyperglycemic and hyperinsulinemic conditions for 32 days inhibits tumor growth and may aid in improving diabetes and fatty liver conditions. The dosage of 15 or 30 mg/kg over 21 days suppresses tumor progression in a luciferase-marked MDA-MB-231 xenograft mouse model. Moreover, 30 mg/kg for 30 days inhibits lung metastasis in a tail vein lung metastasis mouse model, while the same dose for 21 days in a 4T1 syngeneic mouse model shows strong anti-tumor growth and anti-metastatic activity by activating the AMPK pathway, with no significant weight loss or liver and kidney toxicity, while improving lipid metabolism.

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.2858 mL	11.4288 mL	22.8577 mL
5 mM	0.4572 mL	2.2858 mL	4.5715 mL
10 mM	0.2286 mL	1.1429 mL	2.2858 mL
50 mM	0.0457 mL	0.2286 mL	0.4572 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.