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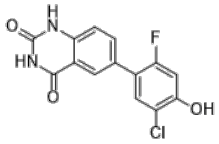
Data Sheet

Global Supplier of Chemical Probes, Inhibitors & Agonists.

Product Name	:	iMQT_020
Cat. No.	:	PC-25646
CAS No.	:	2463893-46-9
Molecular Formula	:	$C_{14}H_8ClFN_2O_3$
Molecular Weight	:	306.68
Target	:	Glutamate Transporter
Solubility	:	10 mM in DMSO



CAS: 2463893-46-9

Biological Activity

iMQT_020 (iMQT-020) is a first-in-class, selective, noncompetitive allosteric inhibitor of mitochondrial glutamine transporter **SLC1A5_var** with IC50 of 6.156 uM for mitochondrial glutamine transport, reprograms cancer cell metabolism, impairing both glycolysis and oxidative phosphorylation.
iMQT_020 not affect the level of glutamate transport into mitochondria, also does not inhibit the uptake of leucine, proline, and glutamate, which are not recognized substrates of SLC1A5_var.
iMQT_020 shows no activity against a panel of transporters and channels, including SLC1A5, SLC38A1, SLC38A2, SLC6A14, SLC6A19, SLC26A3, SLC26A4, SLC26A6, SLC26A7, SLC26A9, and ANO1.
iMQT_020 impairs glutamine anaplerosis and redox balance in pancreatic cancer cells, hinders mitochondrial metabolism, increasing mitochondrial ROS (MitoROS) levels, iMQT_020 also suppressesATP production, which is restored by DM-αKG supplementation.
iMQT_020 (10 uM) significantly inhibits glycolysis in MIA PaCa-2 cells.
iMQT_020 rewires metabolic reprogramming in pancreatic cancer cells, reduces oxygen consumption rate (OCR) in SLC1A5_var WT-overexpressing cells.
induces pancreatic cancer cell death in vitro (MIA PaCa-2, IC50=13.15 uM) lung cancer cell line NCI-H460 (IC50=7.612 uM), while sparing normal cells.
iMQT_020 significantly inhibited organoid growth, and DM-αKG supplementation rescued viability.
iMQT_020 exerts its cytotoxic effects through coordinated induction of apoptosis and ferroptosis, accompanied by cell cycle arrest.
iMQT_020 (75 mg/kg) inhibits cancer growth in mice bearing MIA PaCa-2 subcutaneous xenografts.
iMQT_020 modulates glutamine-dependent epigenetic modifications in cancer cells, leads to increased PD-L1 expression and suppressed MYC expression, increases the efficacy of immunotherapy by altering the tumor immune and metabolic landscape.

References

Sung Y, et al. ***Nat Commun.*** 2025 Nov 3;16(1):9690.