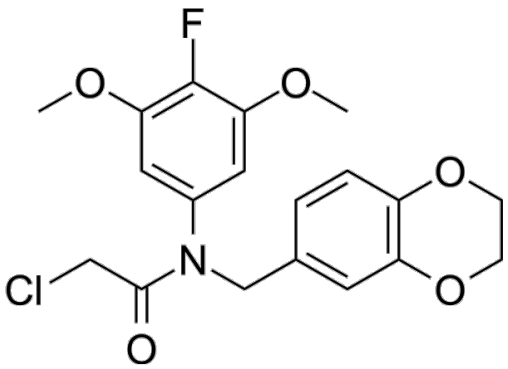
Product Data Sheet

Product Name: GPX4-IN-19
Cat. No.: GC81543

Chemical Properties

Cas. No.	3102894-57-2		
Formula	$C_{19}H_{19}ClFNO_5$	M.Wt	395.81
Solubility	DMSO: 50 mg/mL (126.32 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

GPX4-IN-19 is an effective GPX4 inhibitor (IC_{50} = 0.311 μM), covalently binds to the Sec 46 site of GPX4. GPX4-IN-19 shows strong anti-proliferative activity with high ferroptosis selectivity. GPX4-IN-

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19 causes intracellular Fe 2+ accumulation, leading to increased levels of lipid peroxides (LPOs) and reactive oxygen species (ROS), which induces ferroptosis and subsequently results in DNA damage. GPX4-IN-19 can be used for the study of Triple-Negative Breast Cancer (TNBC) [1].

In Vivo, GPX4-IN-19 (5 mg/kg, 10 mg/kg, i.p., every two days for 24 days) demonstrates a dose-dependent antitumor effect in the MDA-MB-231 tumor-bearing mouse model without causing significant weight loss, abnormal blood biochemical indicators, or organ pathological damage, showing good safety characteristics[1].

In Vitro, GPX4-IN-19 (Compound Y19) (72 h) exhibits effective anti-proliferative activity to six cancer cell lines (MCF-7, MDA-MB-231, PC-3, MDA-MB-468, PANC-1, and ASPC-1) (IC_{50}: 0.021-0.094 μM)[1]. GPX4-IN-19 (12.5-50 nM, 2 weeks) can effectively inhibit cell proliferation, and the addition of the inhibitor Ferrostatin-1 (Fer-1) reversed the inhibition of cell proliferation in MDA-MB-231 cells[1]. GPX4-IN-19 (50 nM, 8 h) can cause mitochondria in the cells to show typical morphological changes, such as rupture of the outer mitochondrial membrane, smaller mitochondrial volume, and reduction or absence of inner mitochondrial crista in MDA-MB-231 cells[1]. GPX4-IN-19 (12.5-50 nM, 8 h) induces dose-dependent increases in intracellular Fe2+ concentration, LPOs accumulation, MDA content, and ROS level in MDA-MB-231 cells[1]. GPX4-IN-19 (12.5-50 nM, 8 h) causes DNA damage in MDA-MB-231 cells by increasing the expression of γH2AX, and this DNA damage is significantly attenuated by the addition of inhibitors such as Fer-1[1]. GPX4-IN-19 (1 μM, 2 h) reduces the degradation temperature of GPX4 protein at the same temperature in MDA-MB-231 cells, indicating that GPX4-IN-19 reduced the thermal stability of GPX4 protein[1]. GPX4-IN-19 (12.5 nM, 25 nM, 50 nM, 8 h) exhibits significant resistance in the MDA-MB-231 cell lines that are resistant to GPX4 inhibitors RSL3 or ML162 (MDA-MB-231-RSL3 and MDA-MB-231-ML162)[1]. GPX4-IN-19 (12.5-50 nM, 4-12 h) reduces GPX4 levels in a concentration-dependent manner in MDA-MB-231 cells. However, time-course analysis reveals that GPX4 expression initially increases at 4 hours, with minimal change in xCT levels at this early time point, but subsequently decreases at 8 hours accompanied by a significant increase in xCT expression[1].

References:

[1]. Ning M, et al. Discovery of novel benzylaniline derivatives containing chloroacetamide as GPX4

inhibitors with potential efficacy in triple-negative breast cancer. Eur J Med Chem. 2025 Dec 15;300:118189.