

Product Data Sheet

Product Name: Opitor-0
Cat. No.: GC79122

Chemical Properties

Cas. No.

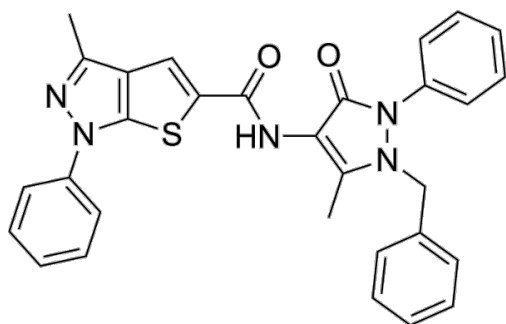
Formula $C_{30}H_{25}N_5O_2S$ M.Wt 519.62

Solubility DMSO: 35 mg/mL (67.36 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

Opitor-0 is a OPA1 GTPase inhibitor with an IC_{50} of 3.0 μ M. Opitor-0 disrupts mitochondrial cristae structure and respiratory efficiency, induces mitochondrial fragmentation, enhances cBID-mediated cytochrome c release, and restores the sensitivity of triple-negative breast cancer (TNBC) cells to ABT-737. Opitor-0 selectively reduces the migration and proliferation capacities of metastatic breast cancer cells, and induces cell death and apoptosis. Opitor-0 can be used in studies related to metastatic breast cancer and triple-negative breast cancer [1] [2].

Caution: Product has not been fully validated for medical applications. For research use only.

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In Vitro, Opitor-0 (50 μ M; 6-24 h) induces mitochondrial fragmentation in MCF10A, MCF10AT1 and MCF10CA1h breast cells; it selectively reduces mitochondrial respiratory efficiency and ATP-coupled respiration in metastatic MCF10CA1h and MCF10CA1a breast cancer cells^[1]. Opitor-0 (1-50 μ M; 5 h) induces concentration-dependent mitochondrial fragmentation in mtYFP-expressing MEF cells and HeLa cells^[2]. Opitor-0 (50 μ M; 20 h) selectively impairs the migratory capacity of metastatic MCF10CA1h and MCF10CA1a breast cancer cells, without affecting non-metastatic MCF10A and MCF10AT1 breast cancer cells^[1]. Opitor-0 (50 μ M; 24 h) selectively reduces the proliferative capacity of highly metastatic MCF10CA1a breast cancer cells, without affecting non-metastatic MCF10A, MCF10AT1 or moderately metastatic MCF10CA1h breast cancer cells^[1]. Opitor-0 (50 μ M) does not induce cell death in any tested breast cancer cell lines after 24 h of treatment, but causes a small yet significant increase in the level of cell death in metastatic MCF10CA1h and MCF10CA1a breast cancer cells after 72 h of treatment, without affecting non-metastatic MCF10A and MCF10AT1 breast cancer cells^[1]. Opitor-0 potently inhibits the GTPase activity of wild-type mini-rOpa1, with an IC₅₀ of 3.0 μ M^[2]. Opitor-0 (25-125 μ M; 30 min) potently enhances cBID-mediated cytochrome c release from purified mouse liver mitochondria^[2]. Opitor-0 (5-100 μ M; administered for 5 h after 12 h of preincubation with 10-20 μ M ABT-737) exerts a strong synergistic effect with the Bcl-2 inhibitor ABT-737 to induce caspase-dependent apoptosis in MDA-MB-231 triple-negative breast cancer cells^[2].

References:

- [1]. Diokmetzidou A, et al. Metastatic breast cancer cells are selectively dependent on the mitochondrial cristae-shaping protein OPA1. Cell death & disease. 2025 Jul 21;16(1):539.
- [2]. Pellattiero A, et al. Small molecule OPA1 inhibitors amplify cytochrome c release and reverse cancer cells resistance to Bcl-2 inhibitors. Sci Adv. 2025 Jul 4;11(27):eadx4562.

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