

Product Data Sheet

Product Name: DDO3602

Cat. No.: GC81720

Chemical Properties

Cas. No.

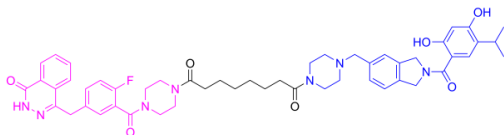
Formula $C_{51}H_{58}FN_7O_7$ M.Wt 900.05

Solubility 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

DDO3602 is a PARP1 HSPTAC (HSP90-mediated proteolysis-targeting chimera) degrader, with a DC_{50} of 490.3 nM against PARP1; its K_d values for PARP1 and HSP90 are 66.5 nM and 1.64 nM, respectively. DDO3602 induces the formation of an unnatural PARP1-HSP90 ternary complex, recruits E3 ubiquitin ligase, and promotes PARP1 degradation via the ubiquitin-proteasome pathway. DDO3602 induces G2/M cell cycle arrest, DNA damage, inhibits cell migration, and exhibits antiproliferative activity in breast cancer cells. DDO3602 can be used in breast cancer-related

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

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research^[1]. (Pink: PARP1 ligand; Blue: HSP90 ligand; Black: linker).

In Vivo, DDO3602 (10-20 mg/kg; i.p.; every other day; 21 days) potently inhibits MCF-7 xenograft tumor growth in immunocompromised mice^[1]. DDO3602 (20 mg/kg; i.v.; single dose) exhibits tumor-targeted pharmacokinetics in MCF-7 xenograft mice, with significantly higher accumulation in tumor tissue than normal tissues following a single 20 mg/kg intravenous dose^[1].

In Vitro, DDO3602 (0-3 μ M; 9 h) degrades PARP1 in human breast cancer MCF-7 cells in a dose-dependent manner, with a DC_{50} of 490.3 nM, and the degradation rate reaches 70.6% after incubation at 1 μ M for 9 h^[1]. DDO3602 (1 μ M; 0-24 h) induces significant degradation of PARP1 in human breast cancer MCF-7 cells within 3 h, with nearly complete depletion achieved by 9 h^[1]. DDO3602 (1 μ M; 0-24 h) degrades the pre-existing PARP1 protein in MCF-7 human breast cancer cells, rather than inhibiting its synthesis^[1]. DDO3602 (0-1 μ M) exerts a stronger inhibitory effect on colony formation in human breast cancer MCF-7 cells than in normal mammary epithelial MCF-10A cells^[1]. DDO3602 (0-10 μ M; 72 h) inhibits the viability of MCF-7 human breast cancer cells with an IC_{50} of 0.189 μ M; its potency is approximately 4-fold higher than that against MCF-10A normal mammary epithelial cells (IC_{50} = 0.744 μ M) after 72 h of treatment^[1]. DDO3602 (0.5-1 μ M; 24 h) induces dose-dependent G2/M cell cycle arrest in human breast cancer MCF-7 cells^[1]. DDO3602 (1 μ M; 24 h) induces significant DNA double-strand break formation in MCF-7 human breast cancer cells (detected by γ -H2A.X accumulation)^[1]. DDO3602 (0.1-1 μ M; 48 h) significantly inhibits the migration of MCF-7 cells^[1].

References:

[1]. Liu W, et al. HSP90 Mediates Targeted Degradation of Nonclient Protein PARP1 for Breast Cancer Treatment. Journal of medicinal chemistry. 2025 Oct 09;68(19):19933-19954.

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