

Product Data Sheet

Product Name: NP1192
Cat. No.: GC80928

Chemical Properties

Cas. No. 2966791-41-1

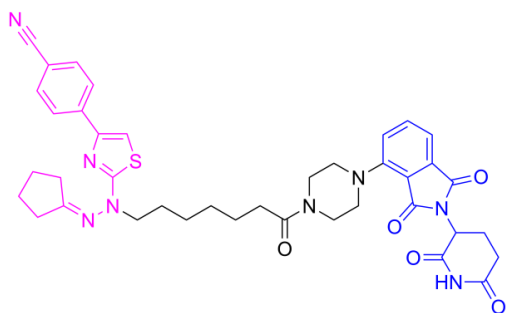
Formula $C_{39}H_{42}N_8O_5S$ M.Wt 734.87

Solubility DMSO: 100 mg/mL (136.08 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

NP1192 is a potent, selective PROTAC NAT10 degrader. NP1192 promotes NAT10 ubiquitination and degradation. NP1192 inhibits ac4C modification. NP1192 demonstrates dual inhibition of hypoxia-driven glycolysis and immunosuppression via NAT10/ HIF-1 α /PD-L1 axis disruption,

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

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achieving superior antitumor efficacy and synergizing with anti-PD-L1 both in vitro and in vivo.

NP1192 can be used for ovarian, cervical, and glioblastoma cancer research [1] [2] [3]. (Pink: NAT10 ligand; Blue: Cereblon ligand; Black: linker).

In Vivo, NP1192 (25 mg/kg, i.p., every 2 days for a week) synergizes with anti-PD-L1 to inhibit tumor growth, suppress glycolysis, and enhance CD8⁺ Teff cell immunity in U14-luc xenograft mouse model^[1].

In Vitro, NP1192 (0-20 μ M, 24-48 h) induces NAT10 degradation in SiHa (human) and U14 (murine) CCa cell lines in a dose-and time-dependent manner, through the ubiquitin-proteasome system (UPS) rather than via lysosomal pathways^[1]. NP1192 (36 h) inhibits cervical cancer (SiHa) cell growth with an IC₅₀ of 7.814 μ M (76.2 μ M in resistant cells) and additionally shows dose-dependent efficacy in organoids, with IC₅₀ values of 9.947, 3.048, and 13.76 μ M for ovarian (OV), cervical (CESC), and glioblastoma (GBM) models, respectively^[1]. NP1192 (20 μ M, 0-72 h and 2 weeks) significantly reduces cell viability, induces cell cycle arrest, and suppresses invasion and clonogenic growth in CCa cells^[1]. NP1192 (0.195-50 μ M, 1-8 days) induces a pronounced reduction in organoid size and number across tumor models (ovarian, cervical, and glioblastoma), while the corresponding normal organoids remained unaltered, indicating minimal cytotoxicity to nonmalignant tissues^[1]. NP1192 (20 μ M, 36 h) reprograms the hypoxic tumor microenvironment (TME) and metabolism in CCa cells by degrading NAT10 and thereby impairing HIF-1 α expression, which disrupts glycolysis as evidenced by reduced glucose uptake and lactate production alongside elevated ATP levels^[1].

References:

- [1]. Ao, K., et al. Targeted NAT10 Degradation by PROTAC NP1192 Suppresses Hypoxia-Adaptive Glycolysis and Reinvigorates CD8⁺ Effector T-Cell Function for Synergistic Cancer Immunotherapy. ACS Central Science. 2025.
- [2]. Zhou Q, et al. PROTAC technology for sensitizing melanoma to immune checkpoint therapy[J]. Journal of Experimental & Clinical Cancer Research, 2026.
- [3]. Xiong J, et al. Functional roles and mechanisms of NAT10-mediated RNA ac4C modification in normal development and cancer progression. Front Cell Dev Biol. 2026 Mar 6;14:1795359.

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