

Product Data Sheet

Product Name: PJ34 hydrochloride

Cat. No.: GC10145

Chemical Properties

Cas. No. 344458-15-7

Chemical Name 2-(dimethylamino)-N-(6-oxo-5H-phenanthridin-2-yl)acetamide;hydrochloride

SMILES CN(C)CC(=O)NC1=CC2=C(C=C1)NC(=O)C3=CC=CC=C32.Cl

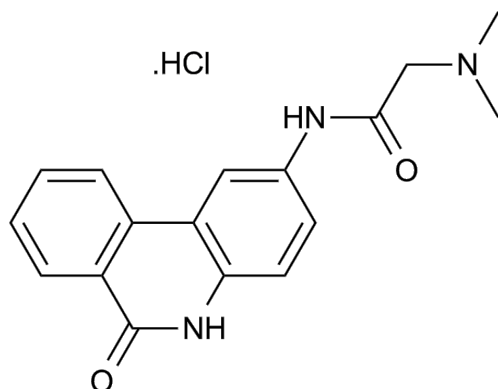
Formula $C_{17}H_{17}N_3O_2 \cdot HCl$ M.Wt 331.8

Solubility ≥ 16.6 mg/mL in DMSO; Water: 50 mg/mL (150.69 mM) Storage Store at $-20^{\circ}C$

General tips For obtaining a higher solubility, please warm the tube at $37^{\circ}C$ and shake it in the ultrasonic bath for a while. Stock solution can be stored below $-20^{\circ}C$ 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



Protocol

Cell experiment [1]:

Cell lines OVCAR-3 cells

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

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OVCAR-3 cells were grown in RPMI-1640 medium with 10% (v/v) fetal bovine serum (FBS), antibiotic/antimycotic solution at 37°C in 5% CO₂/atmosphere.

Preparation Method Cells were seeded in 96-well plates at a density of 5×10^3 cells and cultured overnight. After 48h of treatment with different concentrations of PJ34 hydrochloride (0, 5, 10, 25, 50, and 100μM), the cell viability was measured.

Reaction Conditions 0, 5, 10, 25, 50, and 100μM; 48h

Applications PJ34 hydrochloride treatment significantly inhibited the cell viability of OVCAR-3 cells in a dose-dependent manner.

Animal experiment [2]:

Animal models Male C57BL/6 mice

Male C57BL/6 mice (8 weeks old; 21-23g) were housed singly in a standard environment with food and water ad libitum. All mice were fed for 4 weeks with liquid diets. The pair-fed diet (PF) contained 18% protein, 35% fat, and 47% carbohydrate, whereas the alcohol-fed diet (AF) contained 18% protein, 35%

Preparation Method fat, 11% carbohydrate, and 36% alcohol. PJ34 hydrochloride (10mg/kg) was injected intraperitoneally three times a week for 4 weeks. The same volume of physiologic saline (0.1ml/20g per day; i.p.) was injected intraperitoneally three times a week as a vehicle treatment. At the end of the experiment, liver samples were harvested for analysis.

Dosage form 10mg/kg; three times a week for 4 weeks; i.p.

Applications PJ34 hydrochloride treatment significantly reduced the accumulation of TG in the liver of alcohol-fed mice.

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References:

- [1] Güler A E, Tuncer M C, Özdemir İ. Synergistic Antitumor Activity of Curcumin and the PARP1 Inhibitor PJ34 in Platinum-Sensitive and Resistant Ovarian Cancer Cells[J]. Cancers, 2026, 18(4): 620.
- [2] Huang S, Zhang B, Chen Y, et al. Poly (ADP-ribose) polymerase inhibitor PJ34 attenuated hepatic triglyceride accumulation in alcoholic fatty liver disease in mice[J]. The Journal of pharmacology and experimental therapeutics, 2018, 364(3): 452-461.

Background

PJ34 hydrochloride is an effective and specific PARP inhibitor with an IC₅₀ value of 110nM [1]. PJ34 hydrochloride promotes cell death by inhibiting PARP1-mediated DNA repair, and leads to a decrease in NF-κB expression and specifically blocks the mitosis of cancer cells[2]. PJ34 hydrochloride has been widely used to kill tumor cells and has been employed in the development of novel combined therapies to efficiently enhance the cytotoxicity of chemotherapy drugs[3].

In vitro, PJ34 hydrochloride treatment for 48 hours significantly inhibited the viability of OVCAR-3 cells and SKOV-3 cells, with the IC₅₀ values being 18.7μM and 28.5μM, respectively[4]. Treatment with 50μM PJ34 hydrochloride for 72 hours induced cell cycle arrest and apoptosis in HTLV-I-transformed MT-2 cells[5]. Treatment with 10μM PJ34 hydrochloride for 24 hours resulted in mitotic defects in M14 cells and promoted abnormal accumulation of actin in the nucleolus[6].

In vivo, PJ34 hydrochloride treatment via intraperitoneal injection at a dose of 10mg/kg three times weekly for 4 weeks alleviated the accumulation of triglycerides (TG) in the liver of alcohol-fed mice and reduced the hepatic gene expression of sterol regulatory element-binding protein 1c, DGAT1, and DGAT2[7]. Intraperitoneal injection of PJ34 hydrochloride (50μg) at 2 hours before and 6 hours after middle cerebral artery occlusion (MCAo) significantly reduced the cerebral infarct volume in SV/129 mice [8].

References:

- [1] Iwashita A, Tojo N, Matsuura S, et al. A novel and potent poly (ADP-ribose) polymerase-1 inhibitor, FR247304 (5-chloro-2-[3-(4-phenyl-3, 6-dihydro-1 (2 H)-pyridinyl) propyl]-4 (3 H)-

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- quinazolinone), attenuates neuronal damage in in vitro and in vivo models of cerebral ischemia[J]. The Journal of pharmacology and experimental therapeutics, 2004, 310(2): 425-436.
- [2] Cohen-Armon M. The modified phenanthridine PJ34 unveils an exclusive cell-death mechanism in human cancer cells[J]. Cancers, 2020, 12(6): 1628.
- [3] Mann M, Chauhan S S, Kumar S, et al. PJ34-mediated PARP-1 enzymatic activity inhibition in cervical cancer modulates the cellular response to cisplatin[J]. Cancer Research, 2016, 76(14_Supplement): 2152-2152.
- [4] Güler A E, Tuncer M C, Özdemir İ. Synergistic Antitumor Activity of Curcumin and the PARP1 Inhibitor PJ34 in Platinum-Sensitive and Resistant Ovarian Cancer Cells[J]. Cancers, 2026, 18(4): 620.
- [5] Bai X T, Moles R, Chaib-Mezrag H, et al. Small PARP inhibitor PJ-34 induces cell cycle arrest and apoptosis of adult T-cell leukemia cells[J]. Journal of hematology & oncology, 2015, 8(1): 117.
- [6] Chevanne M, Zampieri M, Caldini R, et al. Inhibition of PARP activity by PJ-34 leads to growth impairment and cell death associated with aberrant mitotic pattern and nucleolar actin accumulation in M14 melanoma cell line[J]. Journal of cellular physiology, 2010, 222(2): 401-410.
- [7] Huang S, Zhang B, Chen Y, et al. Poly (ADP-ribose) polymerase inhibitor PJ34 attenuated hepatic triglyceride accumulation in alcoholic fatty liver disease in mice[J]. The Journal of pharmacology and experimental therapeutics, 2018, 364(3): 452-461.
- [8] Abdelkarim G E, Gertz K, Harms C, et al. Protective effects of PJ34, a novel, potent inhibitor of poly (ADP-ribose) polymerase (PARP) in in vitro and in vivo models of stroke[J]. International journal of molecular medicine, 2001, 7(3): 255-260.

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