Carboplatin

Cat. No.: HY-17393

CAS No.: 41575-94-4

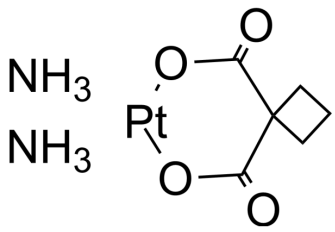
Molecular Formula: $C_6H_{12}N_2O_4Pt$

Molecular Weight: 371.25

Target: DNA Alkylator/Crosslinker; Autophagy; DNA/RNA Synthesis

Pathway: Cell Cycle/DNA Damage; Autophagy

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 8.33 mg/mL (22.44 mM; Need ultrasonic; DMSO can inactivate Carboplatin's activity)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6936 mL	13.4680 mL	26.9360 mL
	5 mM	0.5387 mL	2.6936 mL	5.3872 mL
	10 mM	0.2694 mL	1.3468 mL	2.6936 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
Solubility: 10 mg/mL (26.94 mM); Clear solution; Need ultrasonic and warming and heat to 60°C

BIOLOGICAL ACTIVITY

Description

Carboplatin (NSC 241240) is a DNA synthesis inhibitor which binds to DNA, inhibits replication and transcription and induces cell death. Carboplatin (NSC 241240) is a derivative of CDDP and a potent anti-cancer agent.

IC_{50} & Target

DNA Alkylator[1]

In Vitro

Carboplatin is an antitumor agent, with an increased DNA-binding activity in the presence of nucleophiles and human breast cancer MCF-7 cell cytoplasmic extracts[1].

Carboplatin is less cytotoxic to human ovarian cells such as A2780, SKOV3, IGROV1 and HX62 than 17-AAG, with IC_{50}s of 6.177, 12.442, 2.233 and 116.068 μM, respectively. Moreover, Carboplatin does not affect HSP90 or change the activity of 17-AAG to inhibit HSP90[2].

Carboplatin reduces the viability of Brca1 (IC_{50}, 3.4 μM) and Brca2 cells (IC_{50}, 1.9 μM). Carboplatin (25 μM) combined with ABT-888 also shows an apoptotic effect in BRCA1 cells[3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo	Carboplatin (60 mg/kg, i.p.) shows a modest effect on the tumor, but significantly inhibits tumor growth in combination with 17-AAG in mice bearing A2780 human ovarian cancer xenografts[2]. Carboplatin (25 mg/kg, p.o.) combined with ABT-888 delays tumor growth in Brca2 xenografts[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Exponentially growing A2780 cells are plated in 96 well microtitre plates. For experiments studying the effect of sequence of exposure to 17-AAG or Carboplatin, cells are exposed to increasing concentrations of 17-AAG or Carboplatin for 24 h. A period of 24-h exposure to the first agent is chosen so that the A2780 cells will be exposed to the first drug for at least one doubling time (18-24 h). The cells are then washed with sterile phosphate buffered saline and the medium is replenished. Following this, the second drug (to which the cells are not exposed to in the first 24 h) or medium is added for 96 h. SRB assays are carried out. All experiments are carried out in triplicate[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] The A2780 human ovarian cancer cell line is grown as a subcutaneous xenograft in female athymic NCr nude mice (nu/nu) by injecting 4×10^6 cells in each flank. Mice with established tumors corresponding to a mean volume of 0.69 mm^3 are randomized into groups (six animals each) for treatment with either control vehicle (43% ethanol, 33% polypropylene glycol and 24% cremaphor diluted 1:7 with sterile water) days 1-4, 17-AAG (80 mg/kg intraperitonially, days 1-4), Carboplatin (60 mg/kg IP day 0) or a combination of 17-AAG (80 mg/kg IP days 1-4) and Carboplatin (60 mg/kg IP day 0). Tumor growth is assessed three times weekly and tumor volumes are calculated according to a validated formula: volume = $4.19 \times (a/4 + b/4)^3$, where a is the longer and b the shorter diameter. Tumor volumes are then expressed as a proportion of the volume at the start of treatment (relative tumor volume)[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cancer Discov. 2017 Sep;7(9):984-998.
- J Clin Invest. 2024 Mar 7:e172716.
- Cell Rep Med. 2023 Aug 10;101151.
- Cell Rep Med. 2023 Jan 10;100911.
- Genome Med. 2016 Oct 31;8(1):116.

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REFERENCES

[1]. Natarajan G, et al. Increased DNA-binding activity of cis-1,1-cyclobutanedicarboxylatodiammineplatinum(II) (carboplatin) in the presence of nucleophiles and human breast cancer MCF-7 cell cytoplasmic extracts: activation theory revisited. Biochem Pharmacol. 1999 Nov 15;58(10):1625-9.

[2]. Banerji U, et al. An in vitro and in vivo study of the combination of the heat shock protein inhibitor 17-allylamino-17-demethoxygeldanamycin and carboplatin in human ovarian cancer models. Cancer Chemother Pharmacol. 2008 Oct;62(5):769-78.

[3]. Clark CC, et al. Enhancement of synthetic lethality via combinations of ABT-888, a PARP inhibitor, and carboplatin in vitro and in vivo using BRCA1 and BRCA2 isogenic models. Mol Cancer Ther. 2012 Sep;11(9):1948-58.

[4]. Dela Cruz FS, et al. A case study of an integrative genomic and experimental therapeutic approach for rare tumors: identification of vulnerabilities in a pediatric poorly differentiated carcinoma. Genome Med. 2016 Oct 31;8(1):116.

[5]. Hall MD, Telma KA, Chang KE, et al. Say no to DMSO: dimethylsulfoxide inactivates cisplatin, carboplatin, and other platinum complexes. Cancer Res. 2014;74(14):3913-3922.

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