# Data Sheet

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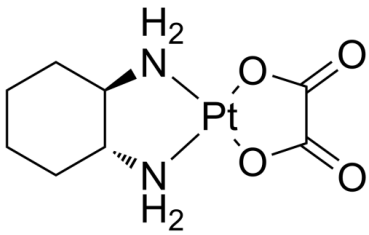
| **Product Name:** | Oxaliplatin |
| **Cat. No.:** | CS-0992 |
| **CAS No.:** | 61825-94-3 |
| **Molecular Formula:** | $C_8H_{14}N_2O_4Pt$ |
| **Molecular Weight:** | 397.29 |
| **Target:** | Apoptosis; DNA/RNA Synthesis |
| **Pathway:** | Apoptosis; Cell Cycle/DNA Damage |
| **Solubility:** | DMF : 1.67 mg/mL (ultrasonic);$H_2O$ : 2.17 mg/mL (ultrasonic;warming;heat to 60°C);DMSO : 20.83 mg/mL (ultrasonic;warming) |




## BIOLOGICAL ACTIVITY:

Oxaliplatin is a **DNA synthesis** inhibitor. Oxaliplatin causes DNA crosslinking damage, prevents DNA replication and transcription and induces apoptosis. Oxaliplatin can be used for cancer research[1][2][3]. IC50 & Target:IC50: DNA synthesis[1] ***In Vitro:*** Oxaliplatin (24-72 hours; 2-128 μM; HCC, HCCLM3 and Hep3B cells) inhibits cell growth and induces apoptosis[1].

Oxaliplatin (10 μM; 15-240 mins; CEM cells ) induces primary and secondary DNA lesions, including DNA cross-links (ISC) and DNA-protein cross-links (DPC)[2].

Oxaliplatin (0.01 to 100 μM; 24 hours) potently inhibits bladder carcinoma cell lines RT4 and TCCSUP, ovarian carcinoma cell line A2780, colon carcinoma cell line HT-29, glioblastoma cell lines U-373MG and U-87MG, and melanoma cell lines SK-MEL-2 and HT-144 with $IC_{50}$ of 11 μM, 15 μM, 0.17 μM, 0.97 μM, 2.95 μM, 17.6 μM, 30.9 μM and 7.85 μM, respectively[3]. ***In Vivo:*** Oxaliplatin (5-10 mg/kg; i.p.; for 32 days; nude mice) inhibits tumor growth[1].

## PROTOCOL (Extracted from published papers and Only for reference)

**Cell Assay:** [3]Typically, cells are plated into 96-well plates on day 0 and exposed to Oxaliplatin on day 1; the sulforhodamine-B assay is carried out 48 h after Oxaliplatin exposure. The plates are incubated at 37°C in 5% $CO_2$ and 100% relative humidity at all times except when adding Oxaliplatin and during the final assay period. The initial number of cells plated for the assay ranged from 2-20×$10^3$ cells/50/nL/well. The numbers of cells for plating and the drug exposure time are based on pilot studies using the criteria that (a) the cells in control wells are still in the log phase of growth on the day of the assay; (b) the maximum absorbance for the untreated controls on the day of the assay is in the range of 1.0 to 1.5; and (c) cells go through > 2 doublings during the drug exposure. Eight wells are used per concentration. The plates are read at 570 and/or 540 nm using a Biotek Instruments model EL309 microplate reader interfaced with an IBM PC-compatible computer. **Animal Administration:** [4]HCC tumor models produced by HCCLM3 are established in nude mice by subcutaneous injection of 5×$10^5$ HCCLM3 cells in 0.2 mL of serum-free culture medium into the left upper flank region. Three days later, the mice are randomLy assigned to receive one of the following three treatments: i) a weekly intraperitoneal (i.p.) injection of distilled water (control group, n=8); ii) a weekly i.p. injection of oxaliplatin at 5 mg/kg (low dose group, n=7); or iii) a weekly i.p. injection of oxaliplatin at 10 mg/kg (high dose group, n=7). Tumor growth is monitored by measuring two bisecting diameters of each tumor with a caliper every 5 days. The tumor volume is calculated using the formula ($V=a\times b^2/2$), with a as the larger diameter and b as the smaller diameter. Mice are euthanized by day 32 after oxaliplatin administration. Tumors of each group are completely removed, weighed, photographed, and fixed in 10% formalin/PBS or stored in liquid nitrogen for histological examination.

## References:


[1]. Raymond E, et al. Oxaliplatin: a review of preclinical and clinical studies. Ann Oncol. 1998 Oct;9(10):1053-71.

[2]. Mohammed MQ, et al. Oxaliplatin is active in vitro against human melanoma cell lines: comparison with NSC 119875 and NSC 241240. Anticancer Drugs. 2000 Nov;11(10):859-63.

[3]. Pendyala L, et al. In vitro cytotoxicity, protein binding, red blood cell partitioning, and biotransformation of oxaliplatin. Cancer Res. 1993 Dec 15;53(24):5970-6.

[4]. Wang Z, et al. Oxaliplatin induces apoptosis in hepatocellular carcinoma cells and inhibits tumor growth. Expert Opin Investig Drugs. 2009 Nov;18(11):1595-604

[5]. Mathé G, et al. Oxalato-platinum or 1-OHP, a third-generation platinum complex: an experimental and clinical appraisal and preliminary comparison with cis-platinum. Biomed Pharmacother. 1989;43(4):237-50.

[6]. Schellingerhout D, et al. Impairment of retrograde neuronal transport in oxaliplatin-induced neuropathy demonstrated by molecular imaging. PLoS One. 2012;7(9):e45776. doi: 10.1371/journal.pone.0045776. Epub 2012 Sep 20.

[7]. Park GY, et al. Phenanthriplatin, a monofunctional DNA-binding platinum anticancer drug candidate with unusual potency and cellular activity profile. Proc Natl Acad Sci U S A. 2012 Jul 24;109(30):11987-92.

[8]. Yi Yao, et al. Comparative proteomic analysis of colon cancer cells in response to oxaliplatin treatment. Biochim Biophys Acta. 2009 Oct;1794(10):1433-40.

[9]. Garrett MJ, et, al. Capecitabine, Oxaliplatin, and Bevacizumab (BCapOx) Regimen for Metastatic Colorectal Cancer. Hosp Pharm. 2017 May;52(5):341-347.


## CAIndexNames:

Platinum, [(1R,2R)-1,2-cyclohexanediamine-κN1,κN2][ethanedioato(2-)-κO1,κO2]-, (SP-4-2)-

## SMILES:

O=C(O1)C(O[Pt]21[NH2][C@@H]3CCCC[C@H]3[NH2]2)=O

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