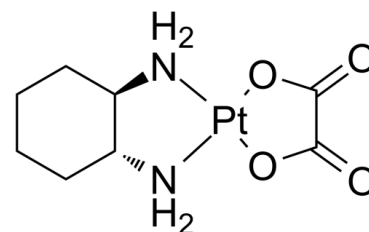


## Data Sheet

<b>Product Name:</b>	Oxaliplatin
<b>Cat. No.:</b>	CS-0992
<b>CAS No.:</b>	61825-94-3
<b>Molecular Formula:</b>	C <sub>8</sub> H <sub>14</sub> N <sub>2</sub> O <sub>4</sub> Pt
<b>Molecular Weight:</b>	397.29
<b>Target:</b>	Autophagy; DNA Alkylator/Crosslinker
<b>Pathway:</b>	Autophagy; Cell Cycle/DNA Damage
<b>Solubility:</b>	H <sub>2</sub> O : 1.7 mg/mL (4.28 mM; Need ultrasonic and warming); DMF : 2 mg/mL (5.03 mM; Need ultrasonic)



### BIOLOGICAL ACTIVITY:

Oxaliplatin is a **DNA synthesis** inhibitor. Oxaliplatin causes DNA crosslinking damage, prevents DNA replication and transcription and causes cell death. Oxaliplatin time-dependently inhibits human melanoma cell lines C32 and G361 with IC<sub>50</sub> values of 0.98 μM and 0.14 μM, respectively. Oxaliplatin induces cell **autophagy**<sup>[1][2]</sup>. IC<sub>50</sub> & Target: IC<sub>50</sub>: DNA synthesis<sup>[1]</sup> **In Vitro:** Oxaliplatin acts through the formation of DNA-adducts. Oxaliplatin induces primary and secondary DNA lesions leading to cell apoptosis<sup>[1]</sup>. Oxaliplatin inhibits human melanoma cell lines C32 and G361 with IC<sub>50</sub> values of 0.98 μM and 0.14 μM, respectively<sup>[2]</sup>. Oxaliplatin 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<sub>50</sub> 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<sup>[3]</sup>. **In Vivo:** Oxaliplatin (10 mg/kg, i.p.) significantly reduces tumor volume and apoptotic index in the nude mice bearing hepatocellular HCCLM3 tumors<sup>[4]</sup>. Oxaliplatin (5 mg/kg, i.v.) is effective on T-leukemia-lymphoma L40 AKR with T/C of 1.77. Oxaliplatin is efficient on intracerebrally grafted L1210 leukemia, MA 16-C xenografts, B16 melanoma xenografts, Lewis lung xenografts and C26 colon carcinoma xenografts<sup>[5]</sup>. Oxaliplatin induces impairment of retrograde neuronal transport in mice<sup>[6]</sup>.

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

**Cell Assay:** <sup>[3]</sup>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<sub>2</sub> 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<sup>3</sup> cells/50nL/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:** Oxaliplatin is dissolved in water.<sup>[4]</sup>HCC tumor models produced by HCCLM3 are established in nude mice by subcutaneous injection of 5×10<sup>5</sup> 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×b<sup>2</sup>/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 cisplatin and carboplatin. *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 and carboplatinum. *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.

## 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

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

Tel: 732-484-9848 Fax: 888-484-5008 E-mail: sales@ChemScene.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA