**Product Name**: Cisplatin

**Catalog Number**: T1564

**CAS Number**: 15663-27-1

**Molecular Formula**: H6Cl2N2Pt

**Molecular Weight**: 300.05

**Description**: Cisplatin, a DNA-crosslinking agent, is able to suppress DNA synthesis by conforming DNA adducts in cancer cells.

**Storage**: 2 years -80°C in solvent; 3 years -20°C powder;

**Solubility**

<table>
<thead>
<tr>
<th>Solvent</th>
<th>Solubility</th>
</tr>
</thead>
<tbody>
<tr>
<td>H2O</td>
<td>1 mg/mL (3.33 mM), Need ultrasonic and warming</td>
</tr>
<tr>
<td>DMF</td>
<td>14.17 mg/mL (47.23 mM), Need ultrasonic and warming</td>
</tr>
</tbody>
</table>

(< 1 mg/ml refers to the product slightly soluble or insoluble)

**Receptor (IC50)**

- Caspase-3
- Caspase-9
- DNA synthesis

**In vitro Activity**

Cisplatin (50 μM) produced time-dependent apoptosis in RPTCs, causing cell shrinkage, a 50-fold increase in caspase 3 activity, a 4-fold increase in phosphatidylserine externalization, and 5- and 15-fold increases in chromatin condensation and DNA hypoploidy, respectively. Mitochondrial membrane potential and ATP levels did not change at any time during cisplatin exposure [1]. High concentrations of cisplatin (800 μM) led to necrotic cell death over a few hours. Much lower concentrations of cisplatin (8 microM) led to apoptosis, which caused loss of the cell monolayer over several days. DNA electrophoresis of cells exposed to 800 μM cisplatin yielded a "smear" pattern, due to random DNA degradation [2].

**In vivo Activity**

In melanoma-bearing mice, cisplatin (4 mg/kg B.W.) reduced the size and weight of the solid tumors, and HemoHIM supplementation with cisplatin enhanced the decrease of both the tumor size and weight. In melanoma-bearing mice treated with cisplatin, HemoHIM administration also increased the activity of NK cells and Tc cells and the IL-2 and IFN-gamma secretion from splenocytes [3]. The maximum tolerated dose of cisplatin given weekly i.v. x 2 induced a tumor growth inhibition (GI) of 77.5% and 85.1% of the serous xenografts Ov.Ri(C) and OVCAR-3, respectively. The mucinous xenograft Ov.Pe was relatively resistant to cisplatin. The maximum tolerated dose of CTX, given i.p. x 2 with a 2-week interval, induced a GI between 52.9% and 59.7% for each of the 3 xenografts [4].

**Cell Assay**

Rabbit renal proximal tubules were isolated using the iron oxide perfusion method and grown in 35-mm tissue culture dishes under improved conditions as described previously. The cell culture medium was a 1:1 mixture of Dulbecco’s modified Eagle’s medium/Ham’s F-12 (without D-glucose, phenol red, or sodium pyruvate) supplemented with 15 mM HEPES buffer, 2.5 mM L-glutamine, 1 μM pyridoxine HCl, 15 mM sodium bicarbonate, and 6 mM lactate. Hydrocortisone (50 nM), selenium (5 ng/ml), human transferrin (5 μg/ml), bovine insulin (10 nM), and L-ascorbic acid-2-phosphate (50 μM) were added to fresh culture medium immediately before daily media change. In general, confluent RPTCs were treated with inhibitors or diluent control [typically DMSO at 0.1% (v/v)] for 30 min before treatment with cisplatin. Aliquots of RPTCs were used for various assays as detailed below [1].

Cell line: Leukemia L1210/0 cells

**Animal Experiment**

Animal Model: The human ovarian cancer xenografts OVCAR-3 in nude mice
Reference

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