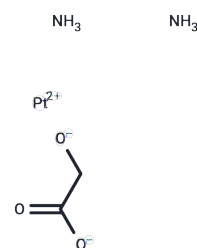


Nedaplatin

Chemical Properties

| | |
|-------------------|---|
| CAS No. : | 95734-82-0 |
| Formula: | C ₂ H ₈ N ₂ O ₃ Pt |
| Molecular Weight: | 303.17 |
| Storage: | Keep away from direct sunlight Powder: -20°C for 3 years <small>Actual storage temperature shall be subject to the COA.</small> |



Biological Description

| | |
|----------------------------|--|
| Description | Nedaplatin (NSC-375101D) is a derivative of cisplatin and DNA damage agent for tumor colony forming units (IC ₅₀ : 94 μM). Containing a novel ring structure in which glycolate is bound to the platinum by a bidentate ligand, nedaplatin forms reactive platinum complexes that bind to nucleophilic groups in DNA, resulting in intrastrand and interstrand DNA cross-links, apoptosis and cell death. |
| Targets(IC ₅₀) | DNA/RNA Synthesis |
| In vitro | Compared to the use of each of the three drugs individually, pre-treating with 5-FU continuously before Nedaplatin or Cisplatin (FN or FC treatment) demonstrates a synergistic enhancement in inhibiting tumor growth and extending survival time. |
| In vivo | Nedaplatin (Aqupla), a derivative of cisplatin, inhibits tumor clonogenic colony formation with an IC ₅₀ of 28.5 μg/mL. At concentrations ranging from 0.005 to 0.5 μg/mL, Nedaplatin suppresses the proliferation of SBC-3 cells by 2% to 98%, with an IC ₅₀ of 0.053 μg/mL. |
| Cell Research | The inhibition of cell (including human SCLC cell line SBC-3 and human NSCLC cell line PC-14) proliferation after drug treatments as the antitumor activity using a regrowth assay is measured. Briefly, cells are exposed to drugs alone or in combination for 6 days at 37°C in an atmosphere of 100% humidity with 5% CO ₂ ; the cells are then pipetted six to eight times until almost all cells appeared as single cells and counted with a counter. For each drug, concentration-effect curves are drawn as plots of the fraction of surviving cells (unaffected cell fraction, fu) versus drug concentration. The cell proliferation ratio of the treated:control cultures (T:C%) is calculated as follows: [(the number of treated cells on day 6)/(the number of treated cells on day 0)]/[(the number of control cells on day 6)/(the number of control cells on day 0)] × 100%. The IC ₅₀ is defined as the drug concentration required for a 50% reduction in the number of cells. Four or five independent experiments are carried out for each. To check the effect of the drug treatment schedule on the effect of the combination, the cells are treated either by simultaneous exposure to the two drugs or by sequential exposure to Nedaplatin followed by irinotecan (Nedaplatin→irinotecan) and vice versa (irinotecan→Nedaplatin) for 3 hours. For the sequential exposure treatment, cells are exposed to the first drug for 3 hours, washed in fresh medium once, and then immediately exposed to the second drug for 3 hours. The treated cells are cultured in drug-free medium until evaluation.(Only for Reference) |

Solubility Information

| | |
|------------|--|
| Solubility | DMSO: Insoluble,DMSO inactivates the activity of Nedaplatin. H2O: 9.21 mg/mL (30.38 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble) |
|------------|--|

Preparing Stock Solutions

| | 1mg | 5mg | 10mg |
|-------|-----------|------------|------------|
| 1 mM | 3.2985 mL | 16.4924 mL | 32.9848 mL |
| 5 mM | 0.6597 mL | 3.2985 mL | 6.597 mL |
| 10 mM | 0.3298 mL | 1.6492 mL | 3.2985 mL |
| 50 mM | 0.066 mL | 0.3298 mL | 0.6597 mL |

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

- Alberts DS, et al. Cancer Chemother Pharmacol, 1997, 39(6), 493-497.
 Wheate NJ et al. Dalton Trans, 2010, 39(35), 8113-8127.
 Kanzawa F, et al. Clin Cancer Res, 2001, 7(1), 202-209.
 Uchida N, et al. Eur J Cancer, 1998, 34(11), 1796-1801.
 Matsumoto M, et al. Jpn J Cancer Res, 2001, 92(1), 51-58.

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