Chemical Properties

Product Name: Cycloheximide

Cas No.: 66-81-9
M.Wt: 281.4
Formula: C15H23NO4
Synonyms: Naramycin A; Actidione; 3-[2-(3,5-Dimethyl-2-oxocyclohexyl)-2-hydroxyethyl]glutarimide
Chemical Name: 4-[(2R)-2-[(1S,3S,5S)-3,5-dimethyl-2-oxocyclohexyl]-2-hydroxyethyl] piperidine-2,6-dione
Canonical SMILES: CC1CC(=O)C(C1)C(C2CC(=O)NC(=O)C2)O)C
Solubility: ≥14.07mg/mL in DMSO
Storage: Store at -20°C

General tips: For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shopping Condition: Evaluation sample solution: ship with blue ice
All other available size: ship with RT, or blue ice upon request

Biological Activity

Targets: Apoptosis
Pathways: Apoptosis Inducers

Description:
IC50: N/A
Cycloheximide is an inhibitor of protein biosynthesis in eukaryotic organisms widely used in biomedical research to inhibit protein synthesis in eukaryotic cells studied in vitro. Due to
significant toxic side effects, including teratogenesis, DNA damage, and other reproductive effects, cycloheximide is generally used only in in vitro research applications, but not suitable for human use as a therapeutic compound.

In vitro: Cycloheximide blocks the movement of peptidyl-tRNA from acceptor site to the donor site on reticulocyte ribosomes. This translocation reaction is dependent on the transfer enzyme, TF-II, and GTP hydrolysis. Cycloheximide has no effect on the ribosome dependent GTPase activity of TF-II or peptidyl transferase reaction by which peptides on tRNA in the donor ribosomal site are transferred to an amino acid on tRNA in the acceptor site [1].

In vivo: Cycloheximide treatment was effective in attenuating rat brain injury within a 6 hr therapeutic window after hypoxia-ischemia in a newborn rat pup model. These data support the possibility that protein synthesis inhibitors, as well as other anti-apoptotic strategies, may have therapeutic utility in hypoxic-ischemic (HI) events of the developing newborn brain even when treatment is delayed for up to 6 hr after the primary asphyxial insult [2].

Clinical trial: Up to now, cycloheximide is still in the preclinical development stage.

Reference:

Protocol

Cell experiment:

Cell lines SGBS preadipocytes

Preparation method The solubility of this compound in DMSO is &gt;10 mM. General tips for obtaining a higher concentration: Please warm the tube at 37 °C for 10 minutes and/or shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Reacting conditions 10 μg/ml, 9 hours

Applications Addition of CHX enhanced CD95-induced cleavage of caspase-8 into p43/p41 intermediate and p18 active fragments as well as proteolytic turnover of the proenzyme form of caspase-8 after 1, 3, 6, and 9 h. In addition, CHX increased cleavage of caspase-3 into the active p20/17 fragment at these time points. At later time points (24, 48, and 72 h), a decrease in the p55 pro-form of caspase-8 and the p35 pro-form of caspase-3 was observed. Interestingly, α-APO-1 alone induced caspase-8 and caspase-3 cleavage (3, 6, 9 h) although there is no induction of cell death after 24 h. Involvement of caspase-cleavage was confirmed by the use of the caspase inhibitor Z-VAD.fmk, which reduced CD95- and CHX-induced apoptosis. Apoptosis was rescued by ~50% pointing to a potential role of
caspase-independent cell death in SGBS preadipocytes.

**Animal experiment [3]:**

**Animal models**  
Sprague Dawley rat pups

**Dosage form**  
Intraperitoneal injection, 0.6 mg/kg, 0, 6, 12 or 24 hr

**Applications**  
The hypoxia-ischemia model was set up using the rat pups. The hypoxia-ischemia control group (HI) and hypoxia-ischemia were treated with cycloheximide treatment group at 0, 6, 12, 24 and 24 hr after HI (HI_0, 6, 12, 24), respectively. Infarct volume, as measured by morphometric analysis of infarct areas with TTC, was significantly reduced by 92% and 61% when cycloheximide was given 0 or 6 hr after HI respectively, but showed an insignificant trend in infarct reduction if cycloheximide was administered 12 hr after HI compared to the HI control group, and no protective effect was observed when administration was delayed until 24 hr after HI.

**Other notes**  
Please test the solubility of all compounds indoor, and the actual solubility may slightly differ with the theoretical value. This is caused by an experimental system error and it is normal.

**Reference:**


**Product Citations**


4. Cingöz O, Goff SP. "Cyclin-dependent kinase activity is required for type I interferon production."


Caution

FOR RESEARCH PURPOSES ONLY.

NOT FOR HUMAN, VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

Specific storage and handling information for each product is indicated on the product datasheet. Most ApexBio products are stable under the recommended conditions. Products are sometimes shipped at a temperature that differs from the recommended storage temperature. Short-term storage of many products are stable in the short-term at temperatures that differ from that required for long-term storage. We ensure that the product is shipped under conditions that will maintain the quality of the reagents. Upon receipt of the product, follow the storage recommendations on the product data sheet.

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