

Product Data Sheet

Product Name: Uridine
Cat. No.: GC13742

Chemical Properties

Cas. No. 58-96-8

Chemical Name 1-[(2R,3R,4S,5R)-3,4-dihydroxy-5-(hydroxymethyl)oxolan-2-yl]pyrimidine-2,4-dione

SMILES C1=CN(C(=O)NC1=O)C2C(C(C(O2)CO)O)O

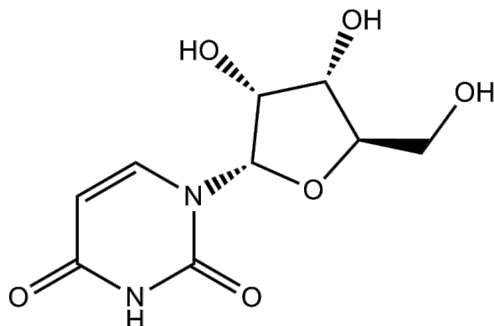
Formula $C_9H_{12}N_2O_6$ M.Wt 244.2

Solubility $\geq 71.4\text{mg/mL}$ in DMSO Storage Store at 4°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.

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

Structure



Protocol

Cell experiment [1]:

Cell lines AML12 cells

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

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To avoid the adverse effects of hepatocyte injury produced during perfusion, the mouse hepatocyte cell line (AML12, the mouse hepatocyte cell line) was also used to check the protective effects of Uridine on hepatocytes apoptosis caused by CCl₄. Before 4h prior to treatment with 10mmol/L CCl₄ for 10h, the hepatocytes were treated with Uridine (200µg/ml) or the vehicle (DMSO). Cell Preparation Method apoptosis rate was assessed using a PE Annexin V apoptosis detection kit according to the manufacturer's instructions. The cell samples were analysed with a FACSCalibur flow cytometer (Becton-Dickinson), and cells that are viable are Annexin V-PE and 7-AAD negative; cells that are in early apoptosis are Annexin V-PE positive and 7-AAD negative; and cells that are in late apoptosis or already dead are both Annexin V-PE and 7-AAD positive.

Reaction Conditions 200µg/ml; 10h

Applications Uridine reduced hepatocyte apoptosis induced by CCl₄, significantly decreased the expression of Caspase-3 and Bax, and upregulated the expression of Bcl-2.

Animal experiment [1]:

Animal models C57BL/6J mice

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Preparation Method	The establishment of the experimental mouse model was approved by the Animal Ethics Committee of the Intervention and Cell Therapy Center of Peking University Shenzhen Hospital. 30 healthy C57BL/6J mice were chosen (6–7 weeks old, weighing between 19 ± 2g) and randomly divided them into control group, model group and Uridine treatment group. Model group: CCl₄ was mixed with olive oil into a 10% oily solvent, the mice from the model group were injected intraperitoneally with the mixed solution at 0.6μl/g twice per week for 8 weeks; Control group: the C57BL/6J mice were injected intraperitoneally with the same volume of olive oil at the same time point (as negative control); Uridine treatment group, the mice were given Uridine by drinking water (10–20mg/kg) for 6 weeks. Twenty-four hours after the last injection, the eyeballs of experimental animals were removed and blood was collected. The mice were anaesthetized and perfused with saline. The liver tissues were collected for a series of biological analyses.
Dosage form	10-20mg/kg/day for 6 weeks; oral
Applications	Uridine alleviated liver fibrosis in mice with CCl₄-induced liver fibrosis model, and inhibited the expression of pro-inflammatory cytokines and the activation of the NF-κB signaling pathway.

References:

[1] Zheng W V, Li Y, Cheng X, et al. Uridine alleviates carbon tetrachloride-induced liver fibrosis by regulating the activity of liver-related cells[J]. Journal of Cellular and Molecular Medicine, 2022, 26(3): 840-854.

Background

Uridine is a nucleoside that contains uracil and is linked to the ribose ring through a β-N1-glycosidic bond [1]. Uridine regulates enzymes and intermediates in the Uridine metabolism (such as UTP, DHODH, and UPase) and is associated with glucose homeostasis, lipid metabolism, and amino acid metabolism [2-3].

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In vitro, Uridine (6-24mM; 7 days) dose-dependently inhibits the proliferation of leukemia cells HL-60 and induces HL-60 to differentiate into mature cells with monocyte and granulocyte characteristics [1]. Uridine (0.2-0.4mg/mL; 10h) reduces hepatocyte apoptosis caused by CCl₄, significantly reduces the expression of Caspase-3 and Bax, and upregulates Bcl-2 expression [4].

In vivo, Uridine (10-20mg/kg; oral; 6 weeks) alleviates liver fibrosis in mice with CCl₄-induced liver fibrosis model and inhibits the expression of pro-inflammatory cytokines and the activation of the NF- κ B signaling pathway [4]. Uridine (30mg/kg; iv; single dose) significantly increases the UDP and UTP contents in the myocardium of rats with acute myocardial ischemia (AMI) and ischemia/reperfusion injury (I/R), reduces CrP and ATP levels, and significantly reduces the size of the ischemic area in the myocardium [5].

References:

- [1] Sokoloski JA, et al., Effects of uridine on the growth and differentiation of HL-60 leukemia cells. Leuk Res. 1991;15(11):1051-8.
- [2] Zhang Y, Guo S, Xie C, et al. Uridine metabolism and its role in glucose, lipid, and amino acid homeostasis[J]. BioMed research international, 2020, 2020(1): 7091718.
- [3] Chenna Narendra S, Chalise J P, Magnusson M, et al. Local but not systemic administration of uridine prevents development of antigen-induced arthritis[J]. PloS one, 2015, 10(10): e0141863.
- [4] Zheng W V, Li Y, Cheng X, et al. Uridine alleviates carbon tetrachloride-induced liver fibrosis by regulating the activity of liver-related cells[J]. Journal of Cellular and Molecular Medicine, 2022, 26(3): 840-854.
- [5] Krylova IB, Selina EN, Bulion VV, et al. Uridine treatment prevents myocardial injury in rat models of acute ischemia and ischemia/reperfusion by activating the mitochondrial ATP-dependent potassium channel. Sci Rep. 2021;11(1):16999.

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