

Product Data Sheet

Product Name: D-+-Galactosamine

Cat. No.: GC11746

Chemical Properties

Cas. No. 1772-03-8

Chemical Name (2R,3R,4R,5R)-2-amino-3,4,5,6-tetrahydroxyhexanal hydrochloride

SMILES N[C@@]([C@](O)([H])[C@](O)([H])[C@@](O)([H])CO)([H])C=O.Cl

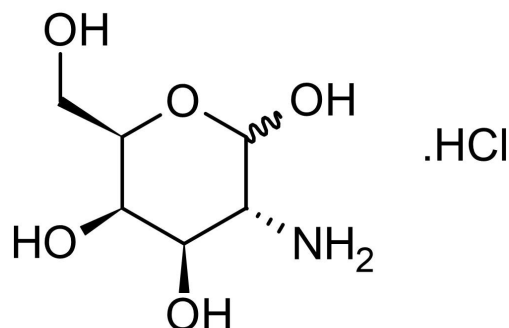
Formula $C_6H_{13}NO_5 \cdot HCl$ M.Wt 215.63

Solubility $\geq 21.6\text{mg/mL}$ in Water Storage Store at RT

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 Hepatocytes

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

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Preparation Method Cells were treated with PGE1 (1mM) 2 h before D-+-Galactosamine (5mM) and then cultured for 24 h. Apoptosis was determined by DNA fragmentation and caspase-3, -6, -8, and -9 activation in hepatocytes. Necrosis was determined by measuring lactate dehydrogenase (LDH) activity in the culture medium.

Reaction Conditions 5mM; 0-24h

Applications D-+-Galactosamine significantly induced cell apoptosis 2 hours after hepatotoxin administration and significantly increased cell necrosis 24 hours after administration.

Animal experiment [2]:

Animal models Sprague-Dawley rats

Preparation Method The rats were separated into four groups of 10 rats each: (1) control animals (0.9% NaCl i.p.); (2) control animals given antioxidants (100 mg/kg/day ascorbic acid, 100 mg/kg/day alpha tocopherol, 15 mg/kg/day beta carotene, and 0.2 mg/kg/day sodium selenate, p.o.); (3) animals given only a single dose of D-+-Galactosamine (500mg/kg, i.p.) (dissolved in 0.9% NaCl); (4) animals given D-+-Galactosamine and antioxidants. D-+-Galactosamine was administered on day 3, and rats were sacrificed 7 hours after the last antioxidant administration. Blood samples were collected and liver tissue samples were obtained for biochemical and histological analyses.

Dosage form 500mg/kg; i.p.

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Applications

D-+-Galactosamine administration caused significant damage to liver tissue. Serum aspartate and alanine aminotransferase, γ -glutamyl transpeptidase, alkaline phosphatase, lactate dehydrogenase, sialic acid, and uric acid levels were increased. Serum and liver glutathione levels were decreased, and serum superoxide dismutase, glutathione peroxidase, glutathione-S-transferase, catalase activities, and lipid peroxidation values were increased in the D-+-Galactosamine group.

References:

[1]Siendones E, JIMÉNEZ-GÓMEZ Y, Montero J L, et al. PGE1 abolishes the mitochondrial-independent cell death pathway induced by D-galactosamine in primary culture of rat hepatocytes[J]. Journal of gastroenterology and hepatology, 2005, 20(1): 108-116.

[2]Catal T, Tunali S, Bolkent S, et al. An antioxidant combination improves histopathological alterations and biochemical parameters in D-galactosamine-induced hepatotoxicity in rats[J]. European Journal of Biology, 2017, 76(1): 14-19.

Background

D-+-Galactosamine is a highly selective hepatotoxin that causes liver damage primarily through the production of free radicals and the consumption of uracil nucleotides^[1]. D-+-Galactosamine is a 6-carbon amino sugar derived from galactose that is used to induce hepatitis in the liver of rodents^[2]. D-+-Galactosamine poisoning can also lead to renal insufficiency, so renal failure is often associated with end-stage liver damage^[3].

In vitro, D-+-Galactosamine (5mM) treatment of mouse primary hepatocytes for 24h significantly induced apoptosis 2h after hepatotoxin administration, significantly increased cell necrosis 24h after administration, induced caspase-3 activation and DNA fragmentation in hepatocytes, but did not change the activity of caspase-6, -8, -9, SMases or cytochrome c release^[4]. Treatment of rat primary hepatocytes with D-+-Galactosamine (1-40 mM) for 24 h reduced mitochondrial dehydrogenase activity and glutathione content in a dose-dependent manner, and increased the

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concentration of lipid peroxide MDA^[5].

In vivo, a single intraperitoneal injection of D-+-Galactosamine (500 mg/kg) in Sprague-Dawley rats caused significant damage to liver tissue, increased serum aspartate and alanine aminotransferase, γ-glutamyl transpeptidase, alkaline phosphatase, lactate dehydrogenase, sialic acid and uric acid levels, increased serum superoxide dismutase, glutathione peroxidase, glutathione-S-transferase, catalase activities and lipid peroxidation values, and reduced serum and liver glutathione levels^[6]. A single intraperitoneal injection of D-+-Galactosamine (1400 mg/kg) in Wistar rats induced acute liver failure with severe parenchymal necrosis and severe lobular and periportal inflammatory infiltration in the liver^[7].

References:

- [1] Endo Y, Shibazaki M, Yamaguchi K, et al. Enhancement by galactosamine of lipopolysaccharide (LPS)-induced tumour necrosis factor production and lethality: its suppression by LPS pretreatment[J]. British journal of pharmacology, 1999, 128(1): 5-12.
- [2] Saracyn M, Zdanowski R, Brytan M, et al. D-Galactosamine intoxication in experimental animals: is it only an experimental model of acute liver failure?[J]. Medical Science Monitor: International Medical Journal of Experimental and Clinical Research, 2015, 21: 1469.
- [3] Sinha M, Manna P, Sil P C. Amelioration of galactosamine-induced nephrotoxicity by a protein isolated from the leaves of the herb, Cajanus indicus L[J]. BMC complementary and alternative medicine, 2007, 7: 1-18.
- [4] Siendones E, JIMÉNEZ-GÓMEZ Y, Montero J L, et al. PGE1 abolishes the mitochondrial-independent cell death pathway induced by D-galactosamine in primary culture of rat hepatocytes[J]. Journal of gastroenterology and hepatology, 2005, 20(1): 108-116.
- [5] Kučera O, Lotková H, Kand'ár R, et al. The model of D-galactosamine-induced injury of rat hepatocytes in primary culture[J]. Mesenchymal stem cells isolated from the human bone marrow: Cultivation, phenotypic and changes, 2006, 49(1): 59-65.
- [6] Catal T, Tunali S, Bolkent S, et al. An antioxidant combination improves histopathological alterations and biochemical parameters in D-galactosamine-induced hepatotoxicity in rats[J]. European Journal of Biology, 2017, 76(1): 14-19.
- [7] Éboli L P C B, Netto A A S, Azevedo R A, et al. Evaluating the best time to intervene acute liver failure in rat models induced by d-galactosamine[J]. Acta Cirurgica Brasileira, 2016, 31(12): 783-792.

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