D-Galactose

Cat. No.: HY-N0210

CAS No.: 59-23-4

Molecular Formula: $C_6H_{12}O_6$

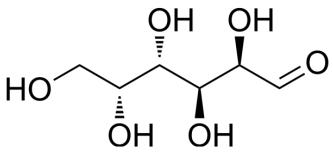
Molecular Weight: 180.16

Target: Endogenous Metabolite

Pathway: Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

H_2O : 62.5 mg/mL (346.91 mM; ultrasonic and warming and heat to 60°C)
DMSO : 50 mg/mL (277.53 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.5506 mL	27.7531 mL	55.5062 mL
5 mM	1.1101 mL	5.5506 mL	11.1012 mL
10 mM	0.5551 mL	2.7753 mL	5.5506 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (555.06 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (13.88 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (13.88 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (13.88 mM); Clear solution

BIOLOGICAL ACTIVITY

Description D-Galactose is a natural aldohexose and C-4 epimer of glucose.

IC_{50} & Target Human Endogenous Metabolite

In Vitro

Galactose is important for the survival and virulence of bacteria. In Escherichia coli galactose is utilized by the Leloir pathway. Two anomers of d-galactose are used for different purposes, α-d-galactose as a carbon source and β-d-galactose for induction of UDP-galactose synthesis for biosynthetic glycosylation[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

D-Galactose can be used in animal modeling to construct mouse subacute aging model and rat cataract model. Chronic D-galactose exposure induces neurodegeneration by enhancing caspase-mediated apoptosis and inhibiting neurogenesis and neuron migration in mice, as well as increasing oxidative damage. In addition, D-galactose-induced toxicity in mice is a useful model for studying the mechanisms of neurodegeneration and neuroprotective drugs and agents[2]. D-galactose given by oral route causes cognitive impairments in rats which are accompanied by oxidative damage. Cognitive impairments is observed in the open-field test in the 4th and 6th weeks after d-gal administration, as well as an impairment in spatial memory in the radial maze test after the 6th week of d-gal administration[3]. D-Galactose is a classic subacute aging and cataracts modeling agent. D-galactose changes the structure of protein and peptide by reacting with their free amine groups resulting in the accumulation of advanced glycation end (AGE) products through non-enzymatic glycation. AGEs give rise to age-related disorders. Rats and mice are generally used as animal models[4][5][6][7]. Dose reference for D-Galactose induction[4][5][6][7]:

(1) Model animal: female/male C57BL/6J mice

Subacute Aging Model: 50, 100 and 200 mg/kg/day, subcutaneous injection (s.c.), 8 weeks

Cataract Model: 200 mg/kg/day, subcutaneous injection (s.c.), 8 weeks

(2) Model animals: Albino-Wistar rats (120-130 g)

Subacute Aging Model: 300 mg/ml/kg, injected intraperitoneally (i.p.), 7 day

Cataract Model: 15 g/kg, injected intraperitoneally (i.p.), twice daily, 30 day

Induction of subacute aging model[5]

Background

D-galactose changes the structure of protein and peptide by reacting with their free amine groups resulting in the accumulation of advanced glycation end (AGE) products through non-enzymatic glycation. AGEs give rise to age-related disorders. Rats and mice are generally used as animal models.

Specific Mmodeling Methods

Mice: C57BL/6J • female • 8-week-old

Administration: 5, 100, 200 mg/day •s.c. • 8 weeks

Note

Modeling Record

Body quality changes: Impaired spatial learning and memory of mice at a dose of 100 mg/kg.

Induced locomotor impairment in LAT in a dose-dependent manner especially inthe second day.

Correlated Product(s):

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	female C57BL/6J mice[5]

Dosage:	50, 100 and 200 mg/kg/day, 8 weeks
Administration:	subcutaneous injection (s.c.)
Result:	Impaired spatial learning and memory of mice at a dose of 100 mg/kg. Induced locomotor impairment in LAT in a dose-dependent manner especially in the second day.

CUSTOMER VALIDATION

- Signal Transduct Target Ther. 2022 Sep 1;7(1):303.
- PLoS Biol. 2018 Oct 18;16(10):e2006483.
- J Cachexia Sarcopenia Muscle. 2024 May 16.
- Antioxidants (Basel). 2024 Jan 31;13(2):183.
- Cells. 2022, 11(20), 3270.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Haifeng Wei, et al. Behavioural study of the Haifeng Wei, et al. Behavioural study of the D-galactose induced aging model in C57BL/6J mice. Behav Brain Res. 2005, 157, 22.

[2]. Saida Haider, et al. A high dose of short term exogenous D-galactose administration in young male rats produces symptoms simulating the natural aging process. Life Sci. 2015.

[3]. Wenjing Feng, et al. Alginate Oligosaccharide Prevents against D-galactose-mediated Cataract in C57BL/6J Mice via Regulating Oxidative Stress and Antioxidant System. Curr Eye Res. 2021, 46, 6.

[4]. Lei Zhong, et al. Characterization of an i.p. D-galactose-induced cataract model in rats. J Pharmacol Toxicol Methods. 2021.

[5]. Csiszovszki Z, et al. Structure and function of the D-galactose network in enterobacteria. MBio. 2011 Jun 28;2(4):e00053-11.

[6]. Cui X, et al. Chronic systemic D-galactose exposure induces memory loss, neurodegeneration, and oxidativedamage in mice: protective effects of R-alpha-lipoic acid. J Neurosci Res. 2006 Aug 15;84(3):647-54.

[7]. Budni J, et al. Oral administration of d-galactose induces cognitive impairments and oxidative damage in rats. Behav Brain Res. 2016 Apr 1;302:35-43.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA