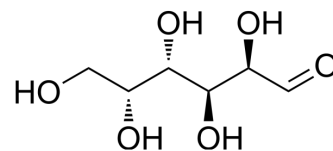


D-Galactose

Cat. No.:	HY-N0210		
CAS No.:	59-23-4		
Molecular Formula:	C ₆ H 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

H₂O : 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

- Add each solvent one by one: PBS
Solubility: 100 mg/mL (555.06 mM); Clear solution; Need ultrasonic
- 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
- 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
- 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.

IC₅₀ & 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 models to create subacute aging models in mice and cataract models in rats. Chronic D-galactose exposure induces neurodegeneration by enhancing caspase-mediated apoptosis and inhibiting neurogenesis and neuronal migration in mice, as well as increasing oxidative damage. Moreover, D-galactose-induced toxicity in mice serves as a useful model for studying the mechanisms of neurodegeneration and the evaluation of neuroprotective drugs and agents. Oral administration of D-galactose leads to cognitive impairment in rats, accompanied by oxidative damage. Cognitive impairments are observed in the open field test at the 4th and 6th weeks post-administration, and spatial memory deficits are noted in the radial arm maze test after the 6th week. D-Galactose is a classic agent for inducing subacute aging and cataract models. D-galactose alters protein and peptide structures by reacting with their free amino groups, leading to the accumulation of advanced glycation end-products (AGEs) during nonenzymatic glycation.

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 Modeling Methods

Mice: C57BL/6J • female • 8-week-old

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

- Modeling Indicators

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 in the second day.

- Correlated Product(s): /

- Opposite 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.
- EMBO J. 2024 Sep 11.
- PLoS Biol. 2018 Oct 18;16(10):e2006483.
- J Cachexia Sarcopenia Muscle. 2024 May 16.
- Antioxidants (Basel). 2024 Jan 31;13(2):183.

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

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