D-Mannitol

Cat. No.: HY-N0378

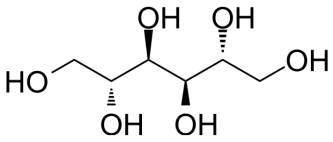
CAS No.: 69-65-8

Molecular Formula: $C_6H_{14}O_6$

Molecular Weight: 182.17

Target: Endogenous Metabolite; Apoptosis; Adrenergic Receptor; PGC-1α; PKA

Pathway: Metabolic Enzyme/Protease; Apoptosis; GPCR/G Protein; Neuronal Signaling; Stem Cell/Wnt; TGF-beta/Smad



Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year

SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 36 mg/mL (197.62 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.4894 mL	27.4469 mL	54.8938 mL
5 mM	1.0979 mL	5.4894 mL	10.9788 mL
10 mM	0.5489 mL	2.7447 mL	5.4894 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 (548.94 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

D-Mannitol (Mannitol) is an oral, resistant sugar widely used in the food and pharmaceutical industries to promote the absorption and retention of calcium and magnesium through cecal fermentation, while acting as a osmotic diuretic to reduce tissue edema. D-Mannitol can enhance brown fat formation, improve insulin effect, reduce blood sugar levels, And through the start the β_3-adrenergic receptor (β_3-AR), PGC1α and PKA induced by means of white fat cells into brown fat cells [1][2][3][4][5][6][7].

IC_{50} & Target			
	Microbial Metabolite	Human Endogenous Metabolite	Beta-3 adrenergic receptor

In Vitro

D-Mannitol (10 μM) can increase the number of mitochondria and promote the expression of brown fat related genes in

differentiated 3T3-L1 cell models[7].
D-Mannitol (10 μM; 7 days) in 3T3-L1 adipocyte cells has the effect of stimulating brown fat formation, enhancing insulin effect, reducing blood sugar levels, and promoting fat oxidation and the conversion of white fat to beige fat[7].
D-Mannitol (10 μM; 7 days) induce Browning of white adipocytes by activating β_3-adrenergic receptors (β_3-AR), PGC1α and PKA[7].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[7]

Cell Line:	3T3-L1 Adipocytes
Concentration:	10 μM
Incubation Time:	7 days
Result:	Increased expression of PGC1α, a key transcriptional coactivator of UCP1, also increased expression of UCP1 and PPARγ proteins, which helped to enhance the effects of insulin and lower blood sugar levels. Triggered the expression of the FGF21 gene, which is associated with thermogenic programming. Activation of AMPK and phosphorylation of ACC and HMGCR promoted β-oxidation, and eliminated UCP1 and ACC expression when AMPK was blocked.

RT-PCR[7]

Cell Line:	3T3-L1 Adipocytes
Concentration:	10 μM
Incubation Time:	
Result:	Increased the expression of brown fat associated genes such as PPARγ coactivator 1α (PGC1α), PR domain 16 (PRDM16), and UCP1. Increased the expression of genes related to energy expenditure and heat production.

In Vivo

D-Mannitol (orally, 7 or 28 days) promotes calcium and magnesium absorption and retention by fermentation in the cecum in male Wistar rat models[4].
D-Mannitol (0.5, 2.5, 1.5 g/kg; Intravenous injection (i.v.), 4-hour intervals×24h) in the rat model of ischemic cortex infarction repeatedly infusion, can lead to infarction area and the percentage of H_2O ipsilateral hemisphere reduce and decreased tissue pressure[6].
D-Mannitol (250, 500 mg/kg; Oral gavage (p.o.); 3 weeks) increases energy expenditure through lipid beta-oxidation in a mouse model, thereby contributing to the Browning of iWAT in vivo[7].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	4-wk-old growing male Wistar rats[4]
Dosage:	20-80 g/kg
Administration:	Oral gavage (p.o.), 28 days
Result:	Improved absorption and retention of calcium (Ca) and magnesium (Mg).

Animal Model:	9-wk-old growing male Wistar rats[4]
Dosage:	40 g/kg, 80 g/kg

Administration:	Oral gavage (p.o.), 7 days
Result:	Increased cecal weight and tissue weight, and reduced pH.

Animal Model:	Adult cats[5]
Dosage:	0.33 gm/kg
Administration:	Intraperitoneal injection (i.p.), 4-hour intervals, single dose or five doses
Result:	After multiple injections, the osmotic concentration gradient between vasogenic cerebral edema and plasma was reversed, which was associated with the worsening of vasogenic cerebral edema.

Animal Model:	Ischemic cortex infarction model in rats[6]
Dosage:	0.5, 2.5, 1.5 g/kg
Administration:	Intravenous injection (i.v.), 4-hour intervals, 24h
Result:	Repeated infusion results in a dose-dependent increase in plasma osmotic pressure and a dose-dependent decrease in the percentage of water in the ischemic cerebral tissue of the arterial cortex and ipsilateral hemisphere. Significantly reduced organizational stress.

Animal Model:	Mouse Model[7]
Dosage:	250 mg/kg, 500 mg/kg
Administration:	Oral gavage (p.o.), 3 weeks
Result:	Caused a significant decrease in the average body weight of the mice. Increased the conversion of white adipose tissue to brown tissue. Led to a significant increase in the expression of genes regulating mitochondrial heat production and lipid oxidation, such as Cidea, CPT1, UCP1, and PGC1α.

CUSTOMER VALIDATION

- Exp Biol Med. 2019 Oct;244(14):1193-1201.
- Neural Plast. 2015;2015:197392.
- Nephrology. 2023 Nov 27.
- Research Square Print. November 29th, 2022.

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[4]. Jeon HJ, et al. D-Mannitol Induces a Brown Fat-like Phenotype via a β3-Adrenergic Receptor-Dependent Mechanism. Cells. 2021;10(4):768. Published 2021 Mar 31.

[5]. Tan, K., et al., The mannitol operon repressor MtlR belongs to a new class of transcription regulators in bacteria. J Biol Chem, 2009. 284(52): p. 36670-9.

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[7]. Hanieh, H. and E. Sakaguchi, Effect of D-mannitol on feed digestion and cecotrophic system in rabbits. Anim Sci J, 2009. 80(2): p. 157-62.

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

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