

Product Data Sheet

Product Name: D-Allose

Cat. No.: GC62921

Chemical Properties

Cas. No. 2595-97-3

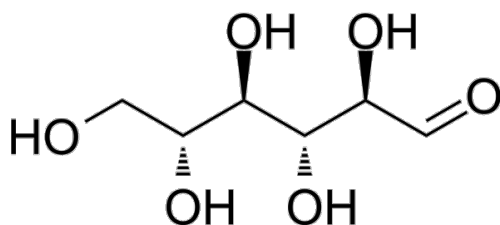
Formula $C_6H_{12}O_6$ M.Wt 180.16

Solubility DMSO : 100 mg/mL (555.06 mM; Need ultrasonic); H_2O : 100 mg/mL (555.06 mM; Need ultrasonic) Storage Store at 2-8°C, protect from light

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



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

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Protocol

Cell experiment [1]:

Cell lines MIA PaCa-2 cells

Preparation Method MIA PaCa-2 cells were cultured in Dulbecco's Modified Eagle's medium (DMEM) supplemented with 10% fetal bovine serum and 4.5g/l glucose. Cells were seeded in 96-well plates at a density of 3000 cells/well, with the lateral wells left empty for water addition. Cells were treated with different concentrations of D-Allose (0.01, 0.1, 1, 10, and 100mM) for 48 hours, and cell proliferation was analyzed.

Reaction Conditions 0.01, 0.1, 1, 10, and 100mM; 48h

Applications D-Allose treatment decreased the cell viability of MIA PaCa-2 cells in a dose-dependent manner.

Animal experiment [2]:

Animal models Male BALB/c (nu/nu) nude mice

Preparation Method Male BALB/c (nu/nu) nude mice, 5 weeks old and weighing 20g, were housed in a specific pathogen clearance grade (SPF) animal house at 24±2°C temperature and 55±5% humidity and maintained on a 12h light / 12h dark circadian rhythm. Mice received a standard diet and water AD libitum. U87MG cells (5×10^6 cells/100µl) were injected subcutaneously into the right abdomen of mice. When the tumor volume reached about 150mm³, the mice were randomly divided into two groups: the experimental group received D-Allose (100mg/kg; i.p.) once a day for 30 consecutive days; mice in the control group were intraperitoneally injected with normal saline. Tumor volume and body weight of mice were analyzed.

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Dosage form 100mg/kg/day for 30 days; i.p.

Applications D-Allose treatment suppressed tumor growth in the U87MG cell xenograft mouse models without affecting body weight.

References:

[1] Malm S W, Hanke N T, Gill A, et al. The anti-tumor efficacy of 2-deoxyglucose and D-allose are enhanced with p38 inhibition in pancreatic and ovarian cell lines[J]. Journal of Experimental & Clinical Cancer Research, 2015, 34(1): 31.

[2] Suzuki K, Ogawa D, Kanda T, et al. Antiproliferative effects of D-allose associated with reduced cell division frequency in glioblastoma[J]. Scientific Reports, 2023, 13(1): 19515.

Background

D-Allose is an ultra-low-calorie aldohexose that exhibits cryoprotective function on cell survival during freezing [1]. D-Allose protects mouse hippocampal HT-22 cells from oxygen-glucose deprivation and reperfusion (OGD/R) -induced injury by inhibiting the Gal-3/TLR4 signalling pathway [2]. D-Allose has been widely used to inhibit the proliferation of a variety of cancer cells and prevent cancer cells from taking up glucose[3].

In vitro, D-Allose treatment for 48 hours significantly inhibited the viability of MIA PaCa-2 cells with an IC₅₀ value of 53.25mM[4]. Treatment of cells with 40mM D-Allose for 48 hours inhibited the growth of PC-3 cells, induced apoptosis, and caused cell cycle arrest[5]. Treatment of RT112 cells with 50mM D-Allose for 24h inhibited cell viability, induced intracellular reactive oxygen species (ROS) production, and stimulated TXNIP expression[6].

In vivo, D-Allose treatment via intraperitoneal injection at a dose of 100mg/kg/day for 28 days significantly reduced tumor volume in a subcutaneous U87MG cell xenograft mouse model [7]. Intravenous administration of 0.4mg/g D-Allose 1 hour before ischemia significantly inhibited apoptosis, reduced blood brain barrier (BBB) permeability, and decreased inflammatory cytokine levels in the mice model of middle cerebral artery occlusion and reperfusion (MCAO/Rep) [8].

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References:

- [1] Chen Z, Chen J, Zhang W, et al. Recent research on the physiological functions, applications, and biotechnological production of D-allose[J]. Applied microbiology and biotechnology, 2018, 102(10): 4269-4278.
- [2] Huang Y, Thonusin C, Tokuda M, et al. The beneficial effects of D-allose and D-allulose on the brain under ischemic stroke and obese-insulin resistant conditions: evidence from in vitro to clinical studies[J]. Metabolic Brain Disease, 2025, 40(4): 1-15.
- [3] Noguchi C, Kamitori K, Hossain A, et al. D-allose inhibits cancer cell growth by reducing GLUT1 expression[J]. The Tohoku journal of experimental medicine, 2016, 238(2): 131-141.
- [4] Malm S W, Hanke N T, Gill A, et al. The anti-tumor efficacy of 2-deoxyglucose and D-allose are enhanced with p38 inhibition in pancreatic and ovarian cell lines[J]. Journal of Experimental & Clinical Cancer Research, 2015, 34(1): 31.
- [5] Naha N, Lee H Y, Jo M J, et al. Rare sugar D-allose induces programmed cell death in hormone refractory prostate cancer cells[J]. Apoptosis, 2008, 13(9): 1121-1134.
- [6] Tohi Y, Taoka R, Zhang X, et al. Antitumor effects of orally administered rare sugar D-allose in bladder cancer[J]. International Journal of Molecular Sciences, 2022, 23(12): 6771.
- [7] Suzuki K, Ogawa D, Kanda T, et al. Antiproliferative effects of D-allose associated with reduced cell division frequency in glioblastoma[J]. Scientific Reports, 2023, 13(1): 19515.
- [8] Huang T, Gao D, Hei Y, et al. D-allose protects the blood brain barrier through PPAR γ -mediated anti-inflammatory pathway in the mice model of ischemia reperfusion injury[J]. Brain research, 2016, 1642: 478-486.

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