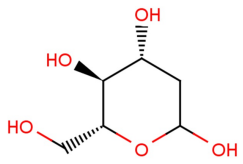
Data Sheet (Cat.No.T6742)

TargetMol
A DRUG SCREENING EXPERT

2-Deoxy-D-glucose

Chemical Properties



CAS No.:	154-17-6
Formula:	C6H12O5
Molecular Weight:	164.16
Appearance:	N/A
Storage:	0-4℃ for short term (days to weeks), or -20℃ for long term (months).

Biological Description

Description	2-Deoxy-D-glucose is an analog of glucose, which is a glycolytic inhibitor with antiviral activity.
Targets(IC_{50})	glycolysis: None
In vitro	2-Deoxy-D-glucose(2-DG) activates AKT function through phosphatidylinositol 3-kinase (PI3K) and is independent of glycolysis or mTOR inhibition. 2-DG treatments disrupts the binding between insulin-like growth factor 1 (IGF-1) and IGF-binding protein 3 (IGFBP3) so that the free form of IGF-1 could be released from the IGF-1·IGFBP3 complex to activate IGF-1 receptor (IGF1R) signaling. 2-DG-induced activation of many survival pathways can be jointly attenuated through IGF1R inhibition. 2-DG also induces time- and dose-dependent ERK phosphorylation[1]. 2-DG is readily transported into cells and is phosphorylated by hexokinase, but cannot be metabolized further and accumulates in the cell. This leads to ATP depletion and the induction of cell-death[2]. 2DG significantly suppresses proliferation, causes apoptosis and reduces migration of murine endothelial cells, inhibiting formation of lamellipodia and filopodia and causing disorganization of F-actin filaments in murine endothelial cell[5].
In vivo	Treatment of cancer patients with relatively high doses of 2-DG (greater than 200 mg/kg) was largely ineffective in managing tumor growth. Side effects of 2-DG included elevated blood glucose levels, progressive weight loss with lethargy, and behavioral symptoms of hypoglycemia[2]. 2-DG enhances isoflurane-induced loss of righting reflex in mice. By reducing metabolism, 2-DG treatment can decrease body temperature in rodent, enhancing sensitivity to anesthetics[3]. 2-DG diet significantly increased serum ketone body level and brain expression of enzymes required for ketone body metabolism. The 2-DG-induced maintenance of mitochondrial bioenergetics was paralleled by simultaneous reduction in oxidative stress. Further, 2-DG treated mice exhibited a significant reduction of both amyloid precursor protein (APP) and amyloid beta (Aβ) oligomers, which was paralleled by significantly increased α-secretase and decreased γ-secretase expression, indicating that 2-DG induced a shift towards a non-amyloidogenic pathway. 2-DG increased expression of genes involved in Aβ clearance pathways, degradation, sequestering, and transport. Concomitant with increased bioenergetic capacity and reduced β-amyloid burden, 2-DG significantly increased expression of neurotrophic growth factors, BDNF and NGF, thus reduces pathology in female mouse model of Alzheimer's disease[4].
Cell Research	2×103 H460 or H157 cells are seeded in 96-well cell culture plates. Cells are treated with 5 mM 2-DG only, 5 or 10 μM IGF1R inhibitor II only, or a combination of 2-DG and IGF1R inhibitor II. Cell growth inhibition is determined after 48 h by the CellTiter 96® AQueous nonradioactive cell proliferation assay. (Only for Reference) Cell lines: H460 or H157 cells
Animal Research	Animal Model: Adult C57BL/6J mice

Solubility Information

Solubility	DMSO: 16.4 mg/mL (100 mM) water: 16.4 mg/mL (100 mM) (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	6.092 mL	30.458 mL	60.916 mL
5 mM	1.218 mL	6.092 mL	12.183 mL
10 mM	0.609 mL	3.046 mL	6.092 mL
50 mM	0.122 mL	0.609 mL	1.218 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. The storage conditions and period of the stock solution: - 80 ℃ for 6 months; - 20 ℃ for 1 month. Please use it as soon as possible.

Reference

1. Zhong D, et al. J Biol Chem. 2009, 284(35):23225-33.
2. Marsh J, et al. Nutr Metab (Lond). 2008, 5:33.
3. Wang H, et al. Anesth Analg. 2015, 120(2):312-9.
4. Yao J, et al. PLoS One. 2011, 6(7):e21788.
5. Huang CC, et al. Dis Model Mech. 2015, 8(10):1247-54.

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