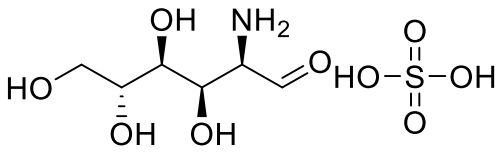
Product data sheet

MedKoo Cat#: 573274 Name: Glucosamine sulfate CAS: 29031-19-4 Chemical Formula: $C_6H_{15}NO_9S$ Molecular Weight: 277.244	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
Glucosamine sulfate is a natural sugar found in and around the fluid and cartilage that cushion joints.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	4.0	14.43
Water	90.0	324.62

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.61 mL	18.03 mL	36.07 mL
5 mM	0.72 mL	3.61 mL	7.21 mL
10 mM	0.36 mL	1.80 mL	3.61 mL
50 mM	0.07 mL	0.36 mL	0.72 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Lv C, Wang L, Zhu X, Lin W, Chen X, Huang Z, Huang L, Yang S. Glucosamine promotes osteoblast proliferation by modulating autophagy via the mammalian target of rapamycin pathway. Biomed Pharmacother. 2018 Mar;99:271-277. doi: 10.1016/j.biopha.2018.01.066. PMID: 29334671.
2. Lei X, Ma N, Liang Y, Liu J, Zhang P, Han Y, Chen W, Du L, Qu B. Glucosamine protects against radiation-induced lung injury via inhibition of epithelial-mesenchymal transition. J Cell Mol Med. 2020 Sep;24(18):11018-11023. doi: 10.1111/jcmm.15662. Epub 2020 Jul 22. PMID: 32700471; PMCID: PMC7521322.

In vivo study

1. Hwang JS, Kim KH, Park J, Kim SM, Cho H, Lee Y, Han IO. Glucosamine improves survival in a mouse model of sepsis and attenuates sepsis-induced lung injury and inflammation. J Biol Chem. 2019 Jan 11;294(2):608-622. doi: 10.1074/jbc.RA118.004638. Epub 2018 Nov 19. PMID: 30455348; PMCID: PMC6333887.
2. Lei X, Ma N, Liang Y, Liu J, Zhang P, Han Y, Chen W, Du L, Qu B. Glucosamine protects against radiation-induced lung injury via inhibition of epithelial-mesenchymal transition. J Cell Mol Med. 2020 Sep;24(18):11018-11023. doi: 10.1111/jcmm.15662. Epub 2020 Jul 22. PMID: 32700471; PMCID: PMC7521322.

7. Bioactivity
Biological target:

Product data sheet

Glucosamine sulfate (D-Glucosamine sulfate) is an amino sugar and a prominent precursor in the biochemical synthesis of glycosylated proteins and lipids, is used as a dietary supplement.

In vitro activity

The effects of glucosamine on osteoblasts was examined. The potential underlying mechanisms were explored. The results showed that glucosamine had a biphasic effect on the viability of hFOB1.19 osteoblasts. At low concentrations (<0.6 mM), glucosamine induced hFOB1.19 cell proliferation, whereas at high concentrations (>0.8 mM) it induced apoptosis. The autophagy inhibitor 3-methyladenine (3-MA) was used to verify that glucosamine modulated hFOB1.19 cell viability via autophagy. The induction of apoptosis by high concentrations of glucosamine was significantly exacerbated by 3-MA, whereas the promotion of cell proliferation by low concentrations of glucosamine was significantly suppressed by 3-MA. Autophagy was examined by western blot detection of autophagy-related proteins including LC3, Beclin-1, and SQSTM1/p62 and by immunofluorescence analysis of autophagosomes. Glucosamine activated autophagy in a time- and concentration-dependent manner. Investigation of the underlying mechanism showed that glucosamine inhibited the phosphorylation of m-TOR in a concentration-dependent manner within 48 h. These results demonstrated that glucosamine promoted hFOB1.19 cell proliferation and increased autophagy by inhibiting the m-TOR pathway, suggesting its potential as a therapeutic agent for osteoporosis.

Reference: Biomed Pharmacother. 2018 Mar;99:271-277. https://linkinghub.elsevier.com/retrieve/pii/S0753-3322(17)36142-5

In vivo activity

The aim of the current study was to investigate the effects of glucosamine (GlcN) on septic lethality and sepsis-induced inflammation using animal models of mice and zebrafish. GlcN pretreatment improved survival in the cecal ligation and puncture (CLP)-induced sepsis mouse model and attenuated lipopolysaccharide (LPS)-induced septic lung injury and systemic inflammation. GlcN suppressed LPS-induced M1-specific but not M2-specific gene expression. Furthermore, increased expressions of inflammatory genes in visceral tissue of LPS-injected zebrafish were suppressed by GlcN. GlcN suppressed LPS-induced activation of mitogen-activated protein kinase (MAPK) and NF-κB in lung tissue. LPS triggered a reduction in O-GlcNAc levels in nucleocytoplasmic proteins of lung, liver, and spleen after 1 day, which returned to normal levels at day 3. GlcN inhibited LPS-induced O-GlcNAc down-regulation in mouse lung and visceral tissue of zebrafish. Furthermore, the O-GlcNAcase (OGA) level was increased by LPS, which were suppressed by GlcN in mouse and zebrafish. OGA inhibitors suppressed LPS-induced expression of inflammatory genes in RAW264.7 cells and the visceral tissue of zebrafish. Stable knockdown of Oga via short hairpin RNA led to increased inducible nitric oxide synthase (iNOS) expression in response to LPS with or without GlcN in RAW264.7 cells. Overall, our results demonstrate a protective effect of GlcN on sepsis potentially through modulation of O-GlcNAcylation of nucleocytoplasmic proteins.

Reference: J Biol Chem. 2019 Jan 11;294(2):608-622. https://www.ncbi.nlm.nih.gov/pmc/articles/pmid/30455348/

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.