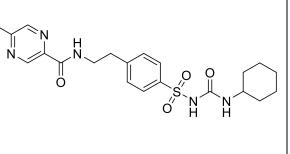
Product data sheet



MedKoo Cat#: 317972			
Name: Glipizide			
CAS#: 29094-61-9	\backslash		
Chemical Formula: C ₂₁ H ₂₇ N ₅ O ₄ S			
Exact Mass: 445.17838			
Molecular Weight: 445.54			
Product supplied as:	Powder		
Purity (by HPLC):	\geq 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:

Glipizide is a short-acting, second-generation, blood-glucose-lowering agent belonging to the sulfonylurea class that increases the release of endogenous insulin from β -cells by blocking potassium channels. The drug-formulated version of Glipizide, marketed as Glucotrol or under generic label, is most commonly prescribed to lower blood sugar levels in type 2 diabetes. Glipizide is rapidly absorbed, has a very quick onset of action and a short half-life. This agent is extensively metabolized in the liver and the metabolites as well as the unchanged form are excreted in the urine

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	53.0	118.96

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.24 mL	11.22 mL	22.44 mL
5 mM	0.45 mL	2.24 mL	4.49 mL
10 mM	0.22 mL	1.12 mL	2.24 mL
50 mM	0.04 mL	0.22 mL	0.45 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. Nazim UM, Moon JH, Lee YJ, Seol JW, Kim YJ, Park SY. Glipizide sensitizes lung cancer cells to TRAIL-induced apoptosis via Akt/mTOR/autophagy pathways. Oncotarget. 2017 Oct 9;8(59):100021-100033. doi: 10.18632/oncotarget.21754. PMID: 29245957; PMCID: PMC5724999.

2. Qi C, Bin Li, Yang Y, Yang Y, Li J, Zhou Q, Wen Y, Zeng C, Zheng L, Zhang Q, Li J, He X, Zhou J, Shao C, Wang L. Glipizide suppresses prostate cancer progression in the TRAMP model by inhibiting angiogenesis. Sci Rep. 2016 Jun 13;6:27819. doi: 10.1038/srep27819. PMID: 27292155; PMCID: PMC4904209.

In vivo study

1. Yang G, Zeng G, Wu JP, Jiang O, Zeng YB, Huang SJ, Huang JJ, Wu DQ. Glipizide blocks renal interstitial fibrosis by inhibiting AKT signaling pathway. Eur Rev Med Pharmacol Sci. 2017 Feb;21(4):867-872. PMID: 28272693.

2. Qi C, Zhou Q, Li B, Yang Y, Cao L, Ye Y, Li J, Ding Y, Wang H, Wang J, He X, Zhang Q, Lan T, Lee KK, Li W, Song X, Zhou J, Yang X, Wang L. Glipizide, an antidiabetic drug, suppresses tumor growth and metastasis by inhibiting angiogenesis. Oncotarget. 2014 Oct 30;5(20):9966-79. doi: 10.18632/oncotarget.2483. PMID: 25294818; PMCID: PMC4259451.

Product data sheet



7. Bioactivity

Biological target:

Glipizide (CP 2872; K 4024) is a sulfonylurea class anti-diabetic agent that acts by partially blocking ATP-sensitive potassium (KATP) channels among β cells (IC50 = 6.4 nM) of pancreatic islets of Langerhans.

In vitro activity

To investigate the effect of glipizide on autophagy flux, whole cell lysates were included to western blot analysis. As shown in Figure2A, the protein levels of DR4 and DR5 were unchanged by glipizide at varying concentrations. Nevertheless, LC3-II increased, and p62 expression decreased after glipizide treatment in a dose-dependent manner (Figure2B). Immunocytochemistry results also supported that various concentrations of glipizide decreased p62 protein levels (Figure2C). A TEM assay suggested that numerous autophagic vacuoles and empty vacuoles were appeared in the cells treated with glipizide (Figure2D). The combined treatment with glipizide and TRAIL (tumor necrosis factor–related apoptosis-inducing ligand) enhanced intracellular apoptosis indicators Ac-cas3 and Ac-cas8 expression levels compare with the single treatment with TRAIL or glipizide (Figure2E). These results reveal that glipizide can induce autophagy in A549 cells.

Reference: Oncotarget. 2017 Nov 21; 8(59): 100021-100033. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5724999/

In vivo activity

Given that glipizide is capable of inhibiting angiogenesis in the chick embryo, it was speculated that glipizide may also suppress tumor-induced angiogenesis, thereby mitigating tumor growth and metastasis. To this end, a mouse xenograft model with mouse breast cancer 4T1 cells was employed. Glipizide (5 mg/kg) was administered daily for 14 days after subcutaneous inoculation of 4T1 cells into mouse mammary fat pad. Glipizide was found to significantly inhibit tumor growth (Figure2A) and weight (Figure2B). Compared with the DMSO treatment, the postprandial blood glucose levels of the mice treated with the glipizide significantly decreased 30 min later and returned to normal 12 h later (Supplemental Table S2). Glipizide treatment also significantly reduced lung metastasis when 4T1 cells were intravenously administered as compared with the DMSO or glimepiride group (Figure2C). Furthermore, glipizide was also found capable of inhibiting B16F10 melanoma growth and metastasis (Supplemental Figure S1). In addition, glipizide abolishes the CD31 staining for MVD (Figure2D). These results collectively suggest that glipizide inhibits the tumor growth and metastasis of malignant melanoma and breast cancer by impeding tumor-induced angiogenesis.

Reference: Oncotarget. 2014 Oct; 5(20): 9966–9979. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4259451/

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.