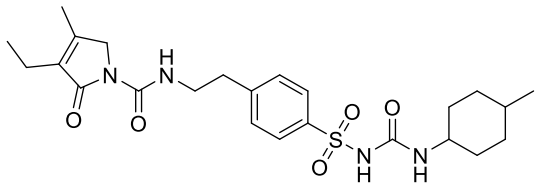
Product data sheet

MedKoo Cat#: 317971 Name: Glimepiride CAS#: 93479-97-1 Chemical Formula: $C_{24}H_{34}N_4O_5S$ Exact Mass: 490.22499 Molecular Weight: 490.62	
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:

Glimepiride is a third generation sulfonylurea compound, which increases the release of insulin from pancreatic beta cells. In addition, glimepiride increases the activity of intracellular insulin receptors. Glimepiride increases osteoblast proliferation and differentiation, which is thought to be related to its ability to activate the PI3K and Akt pathway. Furthermore, Glimepiride enhances intrinsic peroxisome proliferator-activated receptor γ activity. Glimepiride also increases protein expression of glucose transports 1 and 4, and is a potent KIR channel blocker.

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	11	22.42

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.04 mL	10.19 mL	20.38 mL
5 mM	0.41 mL	2.04 mL	4.08 mL
10 mM	0.20 mL	1.02 mL	2.04 mL
50 mM	0.04 mL	0.20 mL	0.41 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. Lawrence CL, Rainbow RD, Davies NW, Standen NB. Effect of metabolic inhibition on glimepiride block of native and cloned cardiac sarcolemmal K(ATP) channels. Br J Pharmacol. 2002 Jul;136(5):746-52. doi: 10.1038/sj.bjp.0704770. PMID: 12086984; PMCID: PMC1573398.

2. Liu F, Wang Y, Yan M, Zhang L, Pang T, Liao H. Glimepiride attenuates Aβ production via suppressing BACE1 activity in cortical neurons. Neurosci Lett. 2013 Dec 17;557 Pt B:90-4. doi: 10.1016/j.neulet.2013.10.052. Epub 2013 Oct 31. PMID: 24184877.

In vivo study

1. Niedowicz DM, Özcan S, Nelson PT. Glimepiride Administered in Chow Reversibly Impairs Glucose Tolerance in Mice. J Diabetes Res. 2018 Oct 29;2018:1251345. doi: 10.1155/2018/1251345. PMID: 30510962; PMCID: PMC6231393.

2. Ishola IO, Akataobi OE, Alade AA, Adeyemi OO. Glimepiride prevents paraquat-induced Parkinsonism in mice: involvement of oxidative stress and neuroinflammation. Fundam Clin Pharmacol. 2019 Jun;33(3):277-285. doi: 10.1111/fcp.12434. Epub 2018 Dec 7. PMID: 30451327.

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7. Bioactivity

Biological target:

Glimepiride (Glimperide) is a medium-to-long acting sulfonylurea anti-diabetic compound with an ED50 of 182 μg/kg.

In vitro activity

The effects of the sulphonylurea, glimepiride, currently used to treat type 2 diabetes, on ATP-sensitive K(+) (K(ATP)) currents of rat cardiac myocytes and on their cloned constituents Kir6.2 and SUR2A expressed in HEK 293 cells was investigated. Glimepiride blocked pinacidil-activated whole-cell K(ATP) currents of cardiac myocytes with an IC(50) of 6.8 nM, comparable to the potency of glibenclamide in these cells. Glimepiride blocked K(ATP) channels formed by co-expression of Kir6.2/SUR2A subunits in HEK 293 cells in outside-out excised patches with a similar IC(50) of 6.2 nM. Glimepiride was much less effective at blocking K(ATP) currents activated by either metabolic inhibition (MI) with CN(-) and iodoacetate or by the K(ATP) channel opener diazoxide in the presence of inhibitors of F(0)/F(1)-ATPase (oligomycin) and creatine kinase (DNFB). Thus 10 microM glimepiride blocked pinacidil-activated currents by >99%, MI-activated currents by 70% and diazoxide-activated currents by 82%. In inside-out patches from HEK 293 cells expressing the cloned K(ATP) channel subunits Kir6.2/SUR2A, increasing the concentration of ADP (1 - 100 microM), in the presence of 100 nM glimepiride, lead to significant increases in Kir6.2/SUR2A channel activity. However, over the range tested, ADP did not affect cloned K(ATP) channel activity in the presence of 100 nM glibenclamide. These results are consistent with the suggestion that ADP reduces glimepiride block of K(ATP) channels. These results show that glimepiride is a potent blocker of native cardiac K(ATP) channels activated by pinacidil and blocks cloned Kir6.2/SUR2A channels activated by ATP depletion with similar potency. However, glimepiride is much less effective when K(ATP) channels are activated by MI and this may reflect a reduction in glimepiride block by increased intracellular ADP.

Reference: Br J Pharmacol. 2002 Jul;136(5):746-52. https://www.ncbi.nlm.nih.gov/pmc/articles/pmid/12086984/

In vivo activity

The effect of glimepiride on fasting blood glucose, glucose tolerance, and insulin secretion was tested. It was also examined for the effect on glucagon, gluconeogenesis, and insulin sensitivity. Unexpectedly, glimepiride exposure in mice was associated with fasting hyperglycemia, glucose intolerance, and decreased insulin. There was no change in circulating glucagon levels or gluconeogenesis. The effect was dose-dependent, took effect by two weeks, and was reversed within three weeks after removal. Glimepiride elicited the same effects in all strains evaluated: four wild-type strains, as well as the transgenic Grn-/- and diabetic db/db mice. These findings suggest that the use of glimepiride as a hypoglycemic agent in mice should proceed with caution and may have broader implications about mouse models as a proxy to study the human pharmacopeia.

Reference: J Diabetes Res. 2018 Oct 29;2018:1251345. https://www.ncbi.nlm.nih.gov/pmc/articles/pmid/30510962/