

Product Data Sheet

Product Name: Glimepiride

Cat. No.: GC10104

Chemical Properties

Cas. No. 93479-97-1

Chemical Name 4-ethyl-3-methyl-N-[2-[4-[(4-methylcyclohexyl)carbamoylsulfamoyl]phenyl]ethyl]-5-oxo-2H-pyrrole-1-carboxamide

SMILES CCC1=C(CN(C1=O)C(=O)NCCC2=CC=C(C=C2)S(=O)(=O)NC(=O)NC3CCC(CC3)C)C

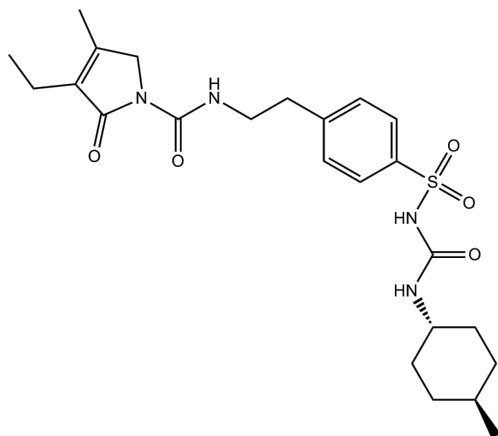
Formula $C_{24}H_{34}N_4O_3S$ M.Wt 490.62

Solubility ≥ 21.45 mg/mL in DMSO Storage Store at -20°C , dry, 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 HCAECs

Preparation Method HCAECs were incubated with 10 μ M Glimepiride, HGF (positive control) or vehicle in Krebs' buffer (118mM NaCl, 4.7mM KCl, 2.5mM CaCl₂, 1.2mM MgSO₄, 1.2mM KH₂PO₄, 11mM glucose, 25mM NaHCO₃, 20mM HEPES, pH 7.4) pre-warmed to 37°C; after 30min the supernatant was collected from the dishes and kept at 4°C. The released nitric oxide (NO) was quantified within 24h of each collection using a NOx-analyzing HPLC system.

Reaction
Conditions 10 μ M; 30min

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Applications Glimepiride induces NO production by HCAECs.

Animal experiment [2]:

Animal models 20 to 25g mice

Preparation Method During the experimental period, animals had ad libitum access to food and water; they were divided into four groups: Group 1 received saline as the normal control, Group 2 received intraperitoneal CdCl₂ (6.5mg/kg) for seven consecutive days as the CdCl₂ control, Group 3 received Glimepiride (4mg/kg; i.p.), and Group 4 was co-treated with CdCl₂ plus Glimepiride, with Glimepiride continued for 3 weeks. Twenty-four hours after the last dose, mice were anaesthetized with thiopental sodium (50mg/kg) and euthanized; blood collected via cardiac puncture into dry Eppendorf tubes was allowed to clot for 30min and then centrifuged at 2000g for 15min, and the serum was stored at -20°C for biochemical assays; both kidneys were excised—one fixed in 10% paraformaldehyde for histopathology and the other snap-frozen at -80°C for ELISA.

Dosage form 4mg/kg; 3 weeks; i.p.

Applications Glimepiride ameliorates renal toxicity induced by cadmium in mice.

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References:

- [1] Ueba H, Kuroki M, Hashimoto S, Umemoto T, Yasu T, Ishikawa SE, Saito M, Kawakami M. Glimepiride induces nitric oxide production in human coronary artery endothelial cells via a PI3-kinase-Akt dependent pathway. *Atherosclerosis*. 2005 Nov;183(1):35-9.
- [2] ElMahdy MK, Zaki MO, Al-Karmalawy AA, Abdo W, Alnasser SM, Antar SA. Glimepiride ameliorates renal toxicity induced by cadmium in mice: Modulation of Jun N terminal kinase (JNK)/nuclear factor kappa B (NF-κB) and phosphatidylinositol 3-kinases (PI3K)/protein kinase (AKT) pathways. *Life Sci*. 2022 Dec 15;311(Pt B):121184.

Background

Glimepiride is a second-generation sulfonylurea that stimulates pancreatic β cells to release insulin. Glimepiride is used for the treatment of type 2 diabetes mellitu as monotherapy as well as in combination with metformin or insulin^[1].

In vitro, Glimepiride (0, 0.3, 0.6, 1.2, 2.5 and 5 μ M; 1h) stimulated the release of CD14 from RAW 264 cells^[2]. Glimepiride (10 μ M; 1, 4 and 7 days) induced the proliferation and differentiation of rat osteoblasts in a high glucose microenvironment^[3]. Glimepiride (10 μ M; 30min) induces NO production by HCAECs^[4].

In vivo, Glimepiride (1, 2 or 4mg/kg; 4 days; p.o.) prevents 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine induced dopamine neurons degeneration through attenuation of glia activation and oxidative stress in mice^[5]. Glimepiride (4mg/kg; 3 weeks; i.p.) ameliorates renal toxicity induced by cadmium in mice^[6].

References:

- [1] Basit A, Riaz M, Fawwad A. Glimepiride: evidence-based facts, trends, and observations (GIFTS). [corrected]. *Vasc Health Risk Manag*. 2012;8:463-72.
- [2] Ingham V, Williams A, Bate C. Glimepiride reduces CD14 expression and cytokine secretion from macrophages. *J Neuroinflammation*. 2014 Jun 21;11:115.
- [3] Ma P, Gu B, Xiong W, Tan B, Geng W, Li J, Liu H. Glimepiride promotes osteogenic differentiation in rat osteoblasts via the PI3K/Akt/eNOS pathway in a high glucose microenvironment. *PLoS One*. 2014 Nov 12;9(11):e112243.

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- [4] Ueba H, Kuroki M, Hashimoto S, Umemoto T, Yasu T, Ishikawa SE, Saito M, Kawakami M. Glimepiride induces nitric oxide production in human coronary artery endothelial cells via a PI3-kinase-Akt dependent pathway. *Atherosclerosis*. 2005 Nov;183(1):35-9.
- [5] Oduola-Akande MD, Ishola IO, Olubodun-Obadun TG, Akande AJ, Adeyemi OO. Glimepiride Prevents 1-Methyl-4-Phenyl-1,2,3,6-Tetrahydropyridine Induced Dopamine Neurons Degeneration Through Attenuation of Glia Activation and Oxidative Stress in Mice. *Neurotox Res*. 2023 Jun;41(3):212-223.
- [6] ElMahdy MK, Zaki MO, Al-Karmalawy AA, Abdo W, Alnasser SM, Antar SA. Glimepiride ameliorates renal toxicity induced by cadmium in mice: Modulation of Jun N terminal kinase (JNK)/nuclear factor kappa B (NF- κ B) and phosphatidylinositol 3-kinases (PI3K)/protein kinase (AKT) pathways. *Life Sci*. 2022 Dec 15;311(Pt B):121184.

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