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Produktinformation

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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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PRODUCT INFORMATION

Glimepiride-d_4

Item No. 27846

Formal Name: 3-ethyl-4-methyl-N-(2-(4-(N-((4-methylcyclohexyl)carbamoyl)sulfamoyl)phenyl)ethyl-1,1,2,2-d_4)-2-oxo-2,5-dihydro-1H-pyrrole-1-carboxamide

MF: $C_{24}H_{30}D_4N_4O_5S$

FW: 494.6

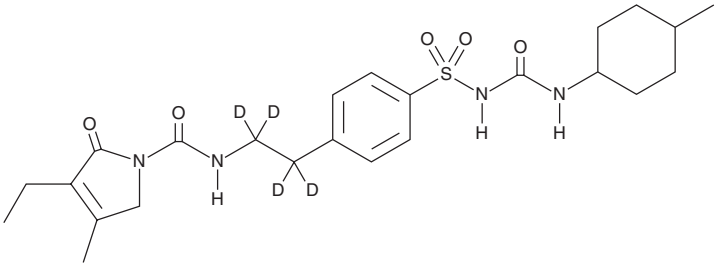
Chemical Purity: ≥98% (Glimepiride)

Deuterium Incorporation: ≥99% deuterated forms (d_1-d_4); ≤1% d_0

Supplied as: A solid

Storage: -20°C

Stability: ≥2 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Glimepiride-d_4 is intended for use as an internal standard for the quantification of glimepiride (Item No. 12090) by GC- or LC-MS. The accuracy of the sample weight in this vial is between 5% over and 2% under the amount shown on the vial. If better precision is required, the deuterated standard should be quantitated against a more precisely weighed unlabeled standard by constructing a standard curve of peak intensity ratios (deuterated *versus* unlabeled).

Glimepiride-d_4 is supplied as a solid. A stock solution may be made by dissolving the glimepiride-d_4 in the solvent of choice. Glimepiride-d_4 is soluble in organic solvents such as DMSO and dimethyl formamide, which should be purged with an inert gas. The solubility of glimepiride-d_4 in these solvents is approximately 3 and 10 mg/ml, respectively.

Description

Glimepiride is a long-acting sulfonylurea that inhibits ATP-sensitive potassium (K_{ATP}) channels in pancreatic β-cells (IC_{50} = 3 nM), which leads to the release of insulin.[1] At 20 µM, glimepiride has been shown to increase the activity of intracellular insulin receptors and to prevent insulin receptor downregulation during chronic insulin stimulation through a mechanism involving protein kinase C activation.[2] It has been reported to be less effective at inhibiting nonpancreatic K_{ATP} channels, and therefore contributes fewer cardiac actions, compared to earlier generations of sulfonylurea anti-type 2 diabetes treatments.[3]

References

1. Song, D.K. and Ashcroft, F.M. Glimepiride block of cloned β-cell, cardiac and smooth muscle K_{ATP} channels. *Br. J. Pharmacol.* **133(1)**, 193-199 (2001).
2. Hribal, M.L., D'Alfonso, R., Giovannone, B., *et al.* The sulfonylurea glimepiride regulates intracellular routing of the insulin-receptor complexes through their interaction with specific protein kinase C isoforms. *Mol. Pharmacol.* **59(2)**, 322-330 (2001).
3. Mocanu, M.M., Maddock, H.L., Baxter, G.F., *et al.* Glimepiride, a novel sulfonylurea, does not abolish myocardial protection afforded by either ischemic preconditioning or diazoxide. *Circulation* **103(25)**, 3111-3116 (2001).

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