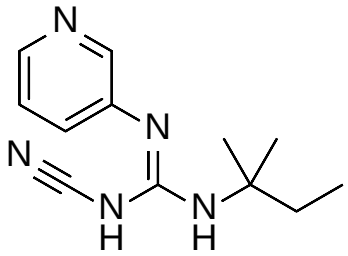
Product data sheet

MedKoo Cat#: 529713 Name: P-1075 CAS: 60559-98-0 Chemical Formula: $C_{12}H_{17}N_5$ Exact Mass: 231.1484 Molecular Weight: 231.303	
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:
P-1075, also known as PNU-83757 and U-83757, is a K-ATP channel agonist potentially for the treatment of erectile dysfunction, alopecia and arrhythmia.

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	250.0	1080.83

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	4.32 mL	21.62 mL	43.23 mL
5 mM	0.86 mL	4.32 mL	8.65 mL
10 mM	0.43 mL	2.16 mL	4.32 mL
50 mM	0.09 mL	0.43 mL	0.86 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. Oldenburg O, Yang XM, Krieg T, Garlid KD, Cohen MV, Grover GJ, Downey JM. P1075 opens mitochondrial K(ATP) channels and generates reactive oxygen species resulting in cardioprotection of rabbit hearts. J Mol Cell Cardiol. 2003 Sep;35(9):1035-42. doi: 10.1016/s0022-2828(03)00151-2. PMID: 12967626.

2. Löffler-Walz C, Hambrock A, Quast U. Interaction of K(ATP) channel modulators with sulfonylurea receptor SUR2B: implication for tetramer formation and allosteric coupling of subunits. Mol Pharmacol. 2002 Feb;61(2):407-14. doi: 10.1124/mol.61.2.407. PMID: 11809866.

In vivo study

1. Novakovic A, Pavlovic M, Milojevic P, Stojanovic I, Nenezic D, Jovic M, Ugresic N, Kanjuh V, Yang Q, He GW. Different potassium channels are involved in relaxation of rat renal artery induced by P1075. Basic Clin Pharmacol Toxicol. 2012 Jul;111(1):24-30. doi: 10.1111/j.1742-7843.2011.00855.x. Epub 2012 Jan 20. PMID: 22225832.

2. Gojkovic-Bukarica LC, Beleslin-Cokic BB, Novakovic AN, Peric MS, Markovic-Lipkovski JZ, Cirovic SZ, Nezic DG, Lesic AR, Kanjuh VI, Heinle H. The effects of potassium channel opener P1075 on the human saphenous vein and human internal mammary artery. J Cardiovasc Pharmacol. 2011 Jun;57(6):648-55. doi: 10.1097/FJC.0b013e3182145850. PMID: 21346595.

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7. Bioactivity

Biological target:

P-1075 is a potent activator of sulfonylurea receptor 2-associated ATP-sensitive potassium channels (SUR2-K_{IR}6).

In vitro activity

This study next tested whether P1075 causes rabbit cardiomyocytes to produce ROS in a K(ATP)-dependent fashion. Mitochondrial ROS production was monitored by the appearance of fluorescence as reduced MitoTracker Red was oxidized. P1075 (100 microM) led to a 44 +/- 9% increase in ROS generation ($P < 0.001$ vs. untreated cells), which was similar to the increase seen with 50 microM diazoxide, a selective mitoK(ATP) channel opener (49 +/- 9%, $P < 0.001$ vs. untreated cells).

Reference: J Mol Cell Cardiol. 2003 Sep;35(9):1035-42. https://pubmed.ncbi.nlm.nih.gov/12967626/

In vivo activity

P1075 induced a concentration-dependent relaxation of rat renal artery rings pre-contracted by phenylephrine. Glibenclamide, a selective K(ATP) channels inhibitor, partly antagonized the relaxation of rat renal artery induced by P1075. Tetraethylammonium (TEA), a non-selective inhibitor of Ca(2+)-activated K(+) channels, as well as iberiotoxin, a most selective blocker of large-conductance Ca(2+) -activated K(+) (BK(Ca)) channels, did not abolish the effect of P1075 on rat renal artery.

Reference: Basic Clin Pharmacol Toxicol. 2012 Jul;111(1):24-30. https://pubmed.ncbi.nlm.nih.gov/22225832/