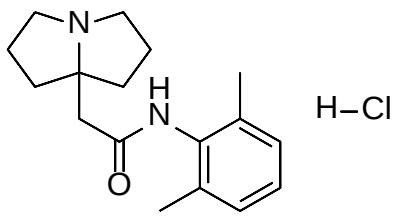
Product data sheet

MedKoo Cat#: 329454 Name: Pilsicainide HCl CAS: 88069-49-2 (HCl) Chemical Formula: $C_{17}H_{25}ClN_2O$ Molecular Weight: 308.85	
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:

Pilsicainide, also known as SUN 1165, is a drug used clinically in Japan to treat cardiac arrhythmias. It functions by blocking the fast inward movement of sodium ions through the Nav1.5 sodium channel that contributes to the rapid depolarization characteristic of phase 0 in the cardiac action potential. Pilsicainide is a pure sodium channel blocker, meaning it does not significantly affect any other cardiac channels including potassium and calcium channels.

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
DMF	20.0	64.76
DMSO	25.0	80.95
Ethanol	25.0	80.95
PBS (pH 7.2)	10.0	32.38

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.24 mL	16.19 mL	32.38
5 mM	0.65 mL	3.24 mL	6.48 mL
10 mM	0.32 mL	1.62 mL	3.24 mL
50 mM	0.07 mL	0.32 mL	0.65 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. Desaphy JF, Dipalma A, Costanza T, Bruno C, Lentini G, Franchini C, George A, Conte Camerino D. Molecular determinants of state-dependent block of voltage-gated sodium channels by pilsicainide. Br J Pharmacol. 2010 Jul;160(6):1521-33. doi: 10.1111/j.1476-5381.2010.00816.x. PMID: 20590641; PMCID: PMC2938822.
2. Wu LM, Orikabe M, Hirano Y, Kawano S, Hiraoka M. Effects of Na+ channel blocker, pilsicainide, on HERG current expressed in HEK-293 cells. J Cardiovasc Pharmacol. 2003 Sep;42(3):410-8. doi: 10.1097/00005344-200309000-00013. PMID: 12960687.

In vivo study

1. Fukuda K, Watanabe J, Yagi T, Wakayama Y, Nakano M, Kondo M, Kumagai K, Miura M, Shirato K, Shimokawa H. A sodium channel blocker, pilsicainide, produces atrial post-repolarization refractoriness through the reduction of sodium channel availability. Tohoku J Exp Med. 2011 Sep;225(1):35-42. doi: 10.1620/tjem.225.35. PMID: 21869589.
2. Yamakawa M, Sunagawa M, Shimabukuro M, Higa N, Takasu N, Kosugi T. Effect of sodium channel blocker, pilsicainide hydrochloride, on net inward current of atrial myocytes in thyroid hormone toxicosis rats. Thyroid. 2005 Jul;15(7):653-9. doi: 10.1089/thy.2005.15.653. PMID: 16053380.

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7. Bioactivity

Biological target:

Pilsicainide hydrochloride (SUN-1165) is an orally active sodium channel blocker and potent class Ic antiarrhythmic agent.

In vitro activity

Pilsicainide exhibited tonic and use-dependent effects comparable to those of mexiletine and flecainide on hNav1.4 channels. These use-dependent effects were abolished in the mutations F1586C and Y1593C within segment 6 of domain IV, suggesting that the interaction of pilsicainide with these residues is critical for its local anaesthetic action. Effects of pilsicainide were similar on skeletal muscle hNav1.4, brain hNav1.1 and heart hNav1.5 channels. The myotonic R1448C and G1306E hNav1.4 mutants were more and less sensitive to pilsicainide, respectively, due to mutation-induced gating modifications.

Reference: Br J Pharmacol. 2010 Jul;160(6):1521-33. https://pubmed.ncbi.nlm.nih.gov/20590641/

In vivo activity

This study explored the relationship between the reduction of sodium channel availability with a pure SCB (pilsicainide or tetrodotoxin) and atrial PRR using the left atrial appendage from male guinea pigs (each group, n = 3~10). Employing a standard microelectrode technique, this study evaluated APD measured at 90% repolarization (APD(90)) and the sodium channel availability, judged from the maximal rate of rise of action potential (Vmax). At a 500-msec basic cycle length (BCL), pilsicainide prolonged atrial ERP assessed by a single extra-stimulus in response to the decrement of the Vmax in a dose-dependent manner without affecting APD(90). Furthermore, pilsicainide increased the ERP and decreased the Vmax in a rate-dependent manner without APD(90) prolongation at a shorter BCL (200 msec).

Reference: Tohoku J Exp Med. 2011 Sep;225(1):35-42. https://pubmed.ncbi.nlm.nih.gov/21869589/