

Product Data Sheet

Product Name: AM 92016 hydrochloride

Cat. No.: GC10943

Chemical Properties

Cas. No. 133229-11-5

Chemical Name N-(4-(3-((3,4-dichlorophenethyl)(methyl)amino)-2-hydroxypropoxy)phenyl)methanesulfonamide hydrochloride

SMILES CN(CC(O)COC1=CC=C(NS(C)(=O)=O)C=C1)CCC2=CC(Cl)=C(Cl)C=C2.Cl

Formula $C_{19}H_{24}Cl_2N_2O_4S.HCl$ M.Wt 483.84

Solubility <48.38mg/ml in DMSO; <24.19mg/ml in Water Storage 4°C, 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.

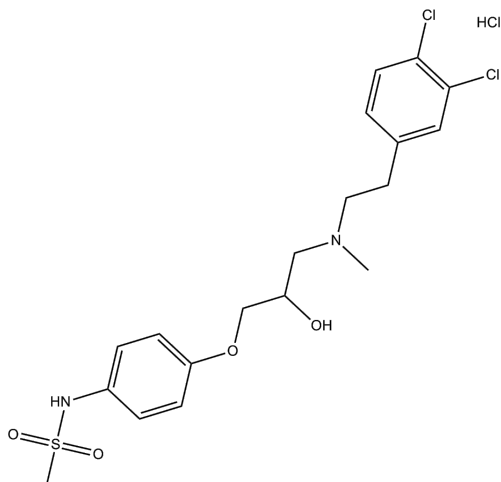
Caution: Product has not been fully validated for medical applications. For research use only.

Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

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Structure



Background

AM 92016 hydrochloride is a specific inhibitor of delayed rectifier potassium current [1].

Potassium channel is an ion channel and acts to reset the resting potential and shapes the action potential. Delayed rectifier potassium channel (IK) is activated by the influx of Na⁺ and discharges K⁺, which repolarizes the membrane. IK restricts the duration of the nerve impulse.

AM 92016 hydrochloride is a specific iK inhibitor. In guinea-pig and rabbit ventricular cells, AM 92016 significantly increased action potential duration with 20% and 90% repolarization levels, respectively. Also, AM 92016 (1 μM) inhibited IK activated by step depolarizations in a time-dependent way [1]. In rabbit sino-atrial node cells, AM 92016 (50 nM) significantly inhibited IK with IC₅₀ value of 40 nM in a concentration-dependent way [2]. In vascular smooth muscle cells (VSMC), AM92016 hydrochloride inhibited NO-induced ERK1/2 dephosphorylation [3].

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In guinea-pigs, AM 92016 (1-5 mg/kg) significantly increased heart rate, left ventricular systolic pressure, systolic arterial blood pressure and the contractile index dp/dt_{max} . AM 92016 exhibited proarrhythmic activity [4].

References:

- [1]. Connors SP, Gill EW, Terrar DA. Actions and mechanisms of action of novel analogues of sotalol on guinea-pig and rabbit ventricular cells. *Br J Pharmacol*, 1992, 106(4): 958-965.
- [2]. Lei M, Brown HF. Inhibition by Compound II, a sotalol analogue, of delayed rectifier current (i_K) in rabbit isolated sino-atrial node cells. *Naunyn Schmiedeberg's Arch Pharmacol*, 1998, 357(3): 260-267.
- [3]. Palen DI, Belmadani S, Lucchesi PA, et al. Role of SHP-1, Kv.1.2, and cGMP in nitric oxide-induced ERK1/2 MAP kinase dephosphorylation in rat vascular smooth muscle cells. *Cardiovasc Res*, 2005, 68(2): 268-277.
- [4]. Hagerty MJ, Wainwright CL, Kane KA. The in-vivo cardiovascular effects of a putative class III anti-arrhythmic drug, AM 92016. *J Pharm Pharmacol*, 1996, 48(4): 417-421.

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