Azimilide dihydrochloride

Cat. No.: HY-18600A

CAS No.: 149888-94-8

Molecular Formula: $C_{23}H_{30}Cl_3N_5O_3$

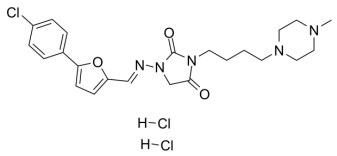
Molecular Weight: 530.88

Target: Potassium Channel

Pathway: Membrane Transporter/Ion Channel

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 50 mg/mL (94.18 mM; Need ultrasonic)

DMSO : 2 mg/mL (3.77 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.8837 mL	9.4183 mL	18.8366 mL
5 mM	0.3767 mL	1.8837 mL	3.7673 mL
10 mM	0.1884 mL	0.9418 mL	1.8837 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

Azimilide (NE-10064) dihydrochloride is a class III antiarrhythmic compound, inhibits I(Ks) and I(Kr) in guinea-pig cardiac myocytes and I(Ks) (minK) channels expressed in Xenopus oocytes.IC50 value:Target: in vitro: Azimilide dihydrochloride blocked HERG channels at 0.1 and 1 Hz with IC50s of 1.4 microM and 5.2 microM respectively. Azimilide dihydrochloride blockade of HERG channels expressed in Xenopus oocytes and I(Kr) in mouse AT-1 cells was decreased under conditions of high [K+]e, whereas block of slowly activating I(Ks) channels was not affected by changes in [K+]e [1]. Azimilide dihydrochloride suppressed the following currents (Kd in parenthesis): IKr (< 1 microM at -20 mV), IKs (1.8 microM at +30 mV), L-type Ca current (17.8 microM at +10 mV), and Na current (19 microM at -40 mV). Azimilide dihydrochloride was a weak blocker of the transient outward and inward rectifier currents (Kd > or = 50 microM at +50 and -140 mV, respectively). Azimilide dihydrochloride blocked IKr, IKs, and INa in a use-dependent manner. Furthermore, azimilide reduced a slowly inactivating component of Na current that might be important for maintaining the action potential plateau in canine ventricular myocytes [2]. In guinea pig ventricular myocytes, Azimilide (0.3-3 microM) dihydrochloride significantly prolonged action potential duration (APD) at 1 Hz. At 3 Hz, Azimilide (0.3-1 microM) dihydrochloride increased APD only slightly, and at 10 microM decreased APD and the plateau potential. Azimilide dihydrochloride potently blocked the rapidly activating component of the delayed rectifier, IKr (IC50 0.4 microM), and inhibited IKs (IC50 3 microM) with nearly 10-fold less potency [3].in vivo: Azimilide (10 mg/kg intravenously, i.v.) dihydrochloride reduced ($p < 0.05$) the incidence (8 of 12) of

PES-induced ventricular tachycardia (VT). The cycle length of induced VT was not prolonged by Azimilide (0.245 +/- 0.046 s predrug vs. 0.301 +/- 0.060 s postdrug) dihydrochloride. Azimilide dihydrochloride increased ventricular effective refractory period (VERP 166 +/- 5 ms predrug vs. 194 +/- 13 ms postdrug, p = 0.013), prolonged QTc interval (310 +/- 12 ms predrug vs. 350 +/- 16 ms postdrug, p = 0.004) and prolonged the effective refractory period (ERP) of noninfarcted myocardium (p = 0.045) [4].

CUSTOMER VALIDATION

- Biochem Biophys Res Commun. 2016 Apr 29;473(2):396-402.
- Exp Ther Med. 2023, 25(1): 1-14.

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REFERENCES

[1]. Busch AE, et al. Blockade of HERG channels by the class III antiarrhythmic azimilide: mode of action. Br J Pharmacol. 1998 Jan;123(1):23-30.

[2]. Yao JA, et al. Azimilide (NE-10064) can prolong or shorten the action potential duration in canine ventricular myocytes: dependence on blockade of K, Ca, and Na channels. J Cardiovasc Electrophysiol. 1997 Feb;8(2):184-98.

[3]. Fermini B, et al. Use-dependent effects of the class III antiarrhythmic agent NE-10064 (azimilide) on cardiac repolarization: block of delayed rectifier potassium and L-type calcium currents. J Cardiovasc Pharmacol. 1995 Aug;26(2):259-71.

[4]. Black SC, et al. Protection against programmed electrical stimulation-induced ventricular tachycardia and sudden cardiac death by NE-10064, a class III antiarrhythmic drug. J Cardiovasc Pharmacol. 1993 Dec;22(6):810-8.

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