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产品名称：盐酸阿齐利特
产品别名：Azimilide Dihydrochloride；阿齐利特二盐酸盐；NE-10064 Dihydrochloride

生物活性：

Description

Azimilide Dihydrochloride (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 blocked HERG channels at 0.1 and 1 Hz with IC50s of 1.4 microM and 5.2 microM respectively. Azimilide 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 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 was a weak blocker of the transient outward and inward rectifier currents (Kd > or = 50 microM at +50 and -140 mV, respectively). Azimilide 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) significantly prolonged action potential duration (APD) at 1 Hz. At 3 Hz, Azimilide (0.3-1 microM) increased APD only slightly, and at 10 microM decreased APD and the plateau potential. Azimilide 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.) 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). Azimilide 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].

Solvent&Solubility

In Vitro:

DMSO : 8.46 mg/mL (15.94 mM; Need ultrasonic and warming)

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

*请根据产品在不同溶剂中的溶解度选择合适的溶剂配制储备液；一旦配成溶液，请分装保存，避免反复冻融造成的产品失效。

储备液的保存方式和期限：-80°C, 6 months; -20°C, 1 month。-80°C 储存时，请在 6 个月内使用，-20°C 储存时，请在 1 个月内使用。

[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

References	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.