Azimilide Dihydrochloride

**Cat. No.:** HY-18600A  
**CAS No.:** 149888-94-8

**Molecular Formula:** C₂₃H₃₀Cl₃N₅O₃  
**Molecular Weight:** 530.88

**Target:** Potassium Channel  
**Pathway:** Membrane Transporter/Ion Channel

**Storage:**  
- **Powder**  
  - 20°C: 3 years  
  - 4°C: 2 years  
- **In solvent**  
  - -80°C: 6 months  
  - -20°C: 1 month

**SOLVENT & SOLUBILITY**

<table>
<thead>
<tr>
<th>In Vitro</th>
<th>DMSO: 8.46 mg/mL (15.94 mM; Need ultrasonic and warming)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preparing Stock Solutions</td>
<td>Solvent Mass</td>
</tr>
<tr>
<td></td>
<td>Concentration</td>
</tr>
<tr>
<td>1 mM</td>
<td>1.8837 mL</td>
</tr>
<tr>
<td>5 mM</td>
<td>0.3767 mL</td>
</tr>
<tr>
<td>10 mM</td>
<td>0.1884 mL</td>
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</table>

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

**BIOLOGICAL ACTIVITY**

**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 potentely 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].

REFERENCES


