



Azimilide

Catalog No: tcsc3489

| Available Sizes |
|---|
| Size: 5mg |
| Size: 10mg |
| Size: 50mg |
| Size: 100mg |
| Specifications |
| CAS No: 149908-53-2 |
| Formula: C ₂₃ H ₂₈ CIN ₅ O ₃ |
| Pathway: Membrane Transporter/Ion Channel |
| Target: Potassium Channel |
| Purity / Grade: >98% |
| Solubility: 10 mM in DMSO |
| Alternative Names: NE-10064 |
| Observed Molecular Weight: 457.95 |





Product Description

Azimilide(NE-10064) 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 (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, NE-10064 (0.3-3 microM) significantly prolonged action potential duration (APD) at 1 Hz. At 3 Hz, NE-10064 (0.3-1 microM) increased APD only slightly, and at 10 microM decreased APD and the plateau potential. NE-10064 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: NE-10064 (10 mg/kg intravenously, i.v.) reduced (p

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