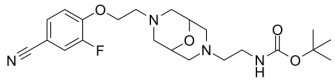
AZD-1305

Cat. No.:	HY-111245
CAS No.:	872045-91-5
Molecular Formula:	$C_{22}H_{31}FN_4O_4$
Molecular Weight:	434.5
Target:	Potassium Channel; Calcium Channel; Sodium Channel
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	AZD-1305 is an antiarrhythmic agent and atrial selective sodium channel/potassium channel blocker, which can significantly prolongs action potential duration and reduces excitability, cause atrial selective ERP prolongation and acute termination of atrial fibrillation. AZD1305 can be used for atrial fibrillation research[1][2].
In Vitro	AZD-1305 (1,3 μM) induces atrial selective PRR in isolated coronary-perfused preparations [1]. AZD-1305 (1,3 μM) produces a greater use-dependent reduction of maximum rate of rise of the action potential upstroke (V_{max}) in atrial versus ventricular preparations[1]. AZD-1305 (1,3 μM) causes a greater increase in sodium channel–mediated parameters conduction velocity and diastolic threshold of excitation in atrial versus ventricular coronaryperfused preparations[1]. AZD-1305 (5 μM) induces greater tonic and steadystate inhibitions of atrial sodium channels than ventricular sodium channels in isolated coronary-perfused preparations[1]. AZD-1305 (1-10 μM) causes a significant decrease in V_{max}, action potential amplitude, and takeoff potential in canine pulmonary vein sleeve preparations, and significantly increases action potential duration at 90% repolarization[2]. AZD-1305 (1-10 μM) significantly increases the basic cycle length at which 1:1 activation is maintained in canine pulmonary vein sleeve preparations, and pretreatment with amiodarone significantly potentiates the effect of AZD1305[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	AZD-1305 (steady state plasma concentrations of 1.2 and 4.5 μM, i.v.drip) produces a greater prolongation of repolarization and ERP in canine atrium of Beagle dogs than in ventricle[1]. AZD-1305 (steady state plasma concentrations of 1.2 and 4.5 μM, i.v.drip) increases conduction time and depress excitability in atria versus ventricles of Beagle dogs [1]. AZD-1305 (steady state plasma concentrations of 1-3 μM, i.v.drip) terminates persistent AF and prevents induction of AF in an ACh-mediated model of AF in Beagle dogs [1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Alexander Burashnikov, et al. AZD1305 exerts atrial predominant electrophysiological actions and is effective in suppressing atrial fibrillation and preventing its reinduction in the dog. J Cardiovasc Pharmacol. 2010 Jul;56(1):80-90.

[2]. Charles Antzelevitch, et al. AZD1305 Exerts Atrial Predominant Electrophysiological Actions and Is Effective in Suppressing Atrial Fibrillation and Preventing Its Reinduction in the Dog. [J] Cardiovasc Pharmacol. 2010, Volume 56□Number 1.

[3]. Serge Sicouri, et al. Electrophysiologic and antiarrhythmic effects of AZD1305 in canine pulmonary vein sleeves. THE JOURNAL OF PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS. 2010, Volume 334Number 1.

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