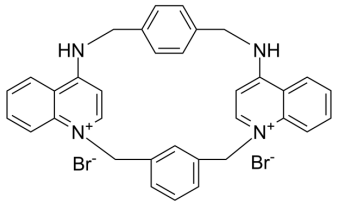
UCL 1684 dibromide

Cat. No.:	HY-108579
CAS No.:	199934-16-2
Molecular Formula:	$C_{34}H_{30}Br_2N_4$
Molecular Weight:	654.44
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)



BIOLOGICAL ACTIVITY

Description	UCL 1684 (dibromide) is a first nanomolar, non-peptidic small conductance calcium-activated potassium (SK) channel blocker. UCL 1684 (dibromide) is effective in preventing the development of atrial fibrillation due to potent atrial-selective inhibition of I_{Na}. UCL 1684 (dibromide) causes atrial-selective prolongation of ERP secondary to induction of postrepolarization refractoriness[1][2][3].
IC_{50} & Target	Potassium Channel[1]
In Vitro	UCL 1684 (dibromide) (0.5 μM; HEK cells) produces direct atrial-selective inhibition of sodium channel current (I_{Na}) and shifts SS inactivation of the cardiac sodium channels. UCL 1684 (dibromide) (0.5 μM) induces PRR, decreases V_{max}, increases DTE, and extends the shortest S1-S1 interval[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	UCL 1684 (dibromide) (3 mg/kg; i.v.) increases wenckebach cycle length to 115.0±5.1 % of baseline value[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Burashnikov A, et al. The Small Conductance Calcium-Activated Potassium Channel Inhibitors NS8593 and UCL1684 Prevent the Development of Atrial Fibrillation Through Atrial-Selective Inhibition of Sodium Channel Activity. J Cardiovasc Pharmacol. 2020;76(2)

[2]. Rosa JC, et al. Bis-quinolinium cyclophanes: 6,10-diaza-3(1,3),8(1,4)-dibenzena-1,5(1,4)- diquinolinacyclodecaphane (UCL 1684), the first nanomolar, non-peptidic blocker of the apamin-sensitive Ca(2+)-activated K+ channel. J Med Chem. 1998;41(1):2-5.

[3]. Diness JG, et al. Effects on atrial fibrillation in aged hypertensive rats by Ca(2+)-activated K(+) channel inhibition. Hypertension. 2011;57(6):1129-1135.

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