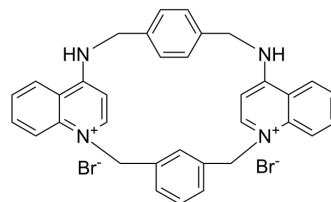


## UCL 1684 dibromide

<b>Cat. No.:</b>	HY-108579
<b>CAS No.:</b>	199934-16-2
<b>Molecular Formula:</b>	C <sub>34</sub> H <sub>30</sub> Br <sub>2</sub> N <sub>4</sub>
<b>Molecular Weight:</b>	654.44
<b>Target:</b>	Potassium Channel
<b>Pathway:</b>	Membrane Transporter/Ion Channel
<b>Storage:</b>	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	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 <sub>Na</sub> . UCL 1684 (dibromide) causes atrial-selective prolongation of ERP secondary to induction of postrepolarization refractoriness <sup>[1][2][3]</sup> .
<b>IC<sub>50</sub> &amp; Target</b>	Potassium Channel <sup>[1]</sup>
<b>In Vitro</b>	UCL 1684 (dibromide) (0.5 μM; HEK cells) produces direct atrial-selective inhibition of sodium channel current (I <sub>Na</sub> ) and shifts SS inactivation of the cardiac sodium channels. UCL 1684 (dibromide) (0.5 μM) induces PRR, decreases V <sub>max</sub> , increases DTE, and extends the shortest S1-S1 interval <sup>[1]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.
<b>In Vivo</b>	UCL 1684 (dibromide) (3 mg/kg; i.v.) increases wenckebach cycle length to 115.0±5.1 % of baseline value <sup>[3]</sup> . 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):164-172.
- [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<sup>+</sup> 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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**Caution: Product has not been fully validated for medical applications. For research use only.**

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