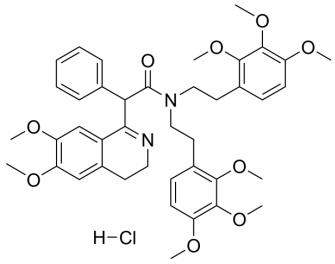
LOE 908 hydrochloride

Cat. No.:	HY-107756
CAS No.:	143482-60-4
Molecular Formula:	$C_{41}H_{49}ClN_2O_9$
Molecular Weight:	749.29
Target:	Calcium 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	LOE 908 hydrochloride is a non-selective cation channel (NSCC) inhibitor[1].
In Vitro	LOE 908 hydrochloride blocks cation conductance in a concentration-dependent manner with an IC_{50} of 560 nM, and blocks dihydropyridine-sensitive Ba^{2+} current through voltage-dependent Ca^{2+} channels with an IC_{50} of 28 μM in voltage-clamped A7r5 cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	LOE 908 (4 or 2 mg/kg followed by 160 or 80 mg/kg; i.v.) hydrochloride attenuates acute neuromotor dysfunction in rats[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Adult male Sprague-Dawley rats, lateral fluid percussion brain injury model[2]
Dosage:	4 mg/kg bolus followed by 160 mg/kg over 24 h or 2 mg/kg bolus followed by 80 mg/kg over 24 h
Administration:	Intravenous administration
Result:	Significantly improved neuromotor function at 48 h postinjury when compared to vehicle treatment.

REFERENCES

[1]. Krautwurst D, et al. The isoquinoline derivative LOE 908 selectively blocks vasopressin-activated nonselective cation currents in A7r5 aortic smooth muscle cells. Naunyn Schmiedebergs Arch Pharmacol. 1994 Mar;349(3):301-7.

[2]. Cheney JA, et al. The novel compound LOE 908 attenuates acute neuromotor dysfunction but not cognitive impairment or cortical tissue loss following traumatic brain injury in rats. J Neurotrauma. 2000 Jan;17(1):83-91.

Caution: Product has not been fully validated for medical applications. For research use only.

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