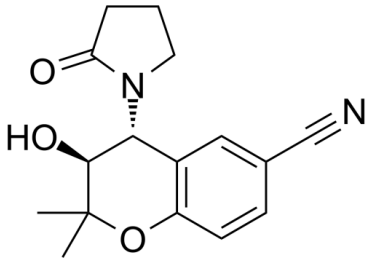
Data Sheet

Product Name:	Levcromakalim
Cat. No.:	CS-6950
CAS No.:	94535-50-9
Molecular Formula:	C16H18N2O3
Molecular Weight:	286.33
Target:	Potassium Channel
Pathway:	Membrane Transporter/Ion Channel
Solubility:	DMSO : ≥ 50 mg/mL (174.62 mM); H2O : < 0.1 mg/mL (insoluble)



BIOLOGICAL ACTIVITY:

Levcromakalim ((-)-Cromakalim) is an ATP-sensitive **K^+ channel** (K_{ATP}) activator. IC50 & Target: K^+ channel[1] **In Vitro:** Levcromakalim ((-)-Cromakalim) inhibits spontaneous contractions completely in a glibenclamide-sensitive manner. LevCromakalim (5 μM) inhibits spontaneous contractions, which are recovered by glibenclamide. Levcromakalim (1, 5 and 10 μM) inhibits phasic contractions to 34±21.1%, 20.1±20.0% and 0% of the control (n=5, respectively; P<0.05). Glibenclamide reverses the inhibition of spontaneous isometric contractions caused by LevCromakalim (5 μM) to 84±1.5% of the control (n=5; P<0.05). Levcromakalim (20 and 100 μM) also inhibits oxytocin (OXT) (10 nM)-induced phasic contractions to 34±21.4% and 14±12.6% of the control (n=6 and 4, respectively; P<0.05). Glibenclamide reverses the inhibition of spontaneous isometric contractions by LevCromakalim (100 μM) to 79±3.5% of the control (n=4; P<0.05). Tonic contraction by OXT is also suppressed by Cromakalim in a glibenclamide-sensitive manner[2].The function of the K_{ATP} channels is examined with the specific channel opener LevCromakalim (Cromakalim). LevCromakalim induces dose-dependent relaxation in both the young and old mesenteric artery (MAs); and there is no difference in relaxation with age. However, the relaxation is markedly reduced in response to the high-salt (HS) diet in the old MAs (P<0.05). Maximum dilations to Levcromakalim (10^{-4} M) are 97 ± 3% in the young MAs versus 98 ± 1% in the young salt arteries, while dilations are 99±0.7% in the old MAs when compared with 85 ± 5% in the old salt arteries (P<0.05)[3].

PROTOCOL (Extracted from published papers and Only for reference)

Kinase Assay: [3]Levcromakalim (Cromakalim) is dissolved in 10% DMSO and Krebs solution[3].

The endothelium-dependent relaxation is tested by performing concentration-response experiments with acetylcholine (ACh; 10 nM-10 μM). Typically, MAs are exposed to each dose of ACh for at least 6 minutes and maximal responses are determined. Function of the K_{ATP} channels are examined with 10 μM of glibenclamide (a selective K_{ATP} channel inhibitor) and Levcromakalim (Cromakalim) (10 nM to 100 μM), a K_{ATP} channel opener. The addition of glibenclamide to the arterial bath 10 minutes prior to ACh does not alter passive maximum internal diameters of any MAs in our groups.The vessel diameter changes are presented as percentages (%) of dilation of the preconstricted vessels, calculated[3].

References:

[1]. Matsumoto T, et al. Tunicamycin-Induced Alterations in the Vasorelaxant Response in Organ-Cultured Superior Mesenteric Arteries of Rats. Biol Pharm Bull. 2016;39(9):1475-81.

[2]. Hong SH, et al. Regulation of myometrial contraction by ATP-sensitive potassium (KATP) channel via activation of SUR2B and Kir 6.2 in mouse. J Vet Med Sci. 2016 Aug 1;78(7):1153-9.

[3]. Whidden MA, Altered potassium ATP channel signaling in mesenteric arteries of old high salt-fed rats. J Exerc Nutrition Biochem. 2016 Jun;20(2):58-64.

CAIndexNames:

2H-1-Benzopyran-6-carbonitrile, 3,4-dihydro-3-hydroxy-2,2-dimethyl-4-(2-oxo-1-pyrrolidinyl)-, (3S,4R)-

SMILES:

CC(O1)(C)[C@@H](O)[C@H](N2CCCC2=O)C3=C1C=CC(C#N)=C3

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