Nicorandil

Cat. No.: HY-B0341

CAS No.: 65141-46-0

Molecular Formula: $C_8H_9N_3O_4$

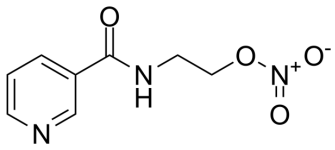
Molecular Weight: 211.17

Target: Potassium Channel

Pathway: Membrane Transporter/Ion Channel

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 33 mg/mL (156.27 mM)

H_2O : ≥ 20 mg/mL (94.71 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.7355 mL	23.6776 mL	47.3552 mL
5 mM	0.9471 mL	4.7355 mL	9.4710 mL
10 mM	0.4736 mL	2.3678 mL	4.7355 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (11.84 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (11.84 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Nicorandil (SG-75) is a potent potassium channel activator and targets vascular nucleoside diphosphate-dependent K^+ channels and cardiac ATP-sensitive K^+ channels (K_{ATP}). Nicorandil is a nicotinamide ester with vasodilatory and cardioprotective effects and has the potential for angina and forischemic heart diseases[1][2][3].

IC_{50} & Target

Potassium Channel[1].

In Vitro

Nicorandil (SG-75) activates ATP-sensitive K^+ channels composed of Kir6.2 and either sulfonylurea receptor (SUR) 2A or 2B[1].

Nicorandil is a vasodilatory drug used to treat angina. Nicorandil (SG-75) stimulates guanylate cyclase to increase formation of cyclic GMP (cGMP). cGMP activates protein kinase G (PKG) which phosphorylates and inhibits GTPase RhoA and decreases Rho-kinase activity. Reduced Rho-kinase activity permits an increase in myosin phosphatase activity, decreasing the calcium sensitivity of the smooth muscle. PKG also activates the sarcolemma calcium pump to remove activating calcium. PKG acts on K+ channels to promote K+ efflux and the ensuing hyperpolarization inhibits voltage-gated calcium channels. Overall, this leads to relaxation of the smooth muscle and coronary vasodilation[2][3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Nakae, I., et al., Effects of intravenous nicorandil on coronary circulation in humans: plasma concentration and action mechanism. J Cardiovasc Pharmacol, 2000. 35(6): p. 919-25.

[2]. Sauzeau, V., et al., Cyclic GMP-dependent protein kinase signaling pathway inhibits RhoA-induced Ca2+ sensitization of contraction in vascular smooth muscle. J Biol Chem, 2000. 275(28): p. 21722-9.

[3]. Mitsuhiko Yamada, et al. The nucleotide-binding domains of sulfonylurea receptor 2A and 2B play different functional roles in nicorandil-induced activation of ATP-sensitive K+ channels. Mol Pharmacol. 2004 May;65(5):1198-207.

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