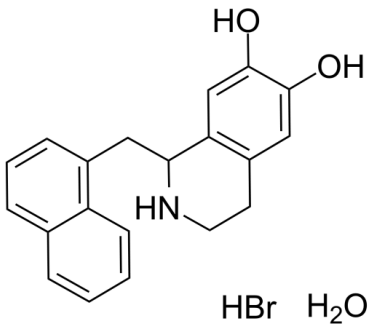
Data Sheet

Product Name:	YS-49 (monohydrate)
Cat. No.:	CS-2288
Molecular Formula:	$C_{20}H_{22}BrNO_3$
Molecular Weight:	404.30
Target:	Adrenergic Receptor; Akt; Angiotensin Receptor; PI3K
Pathway:	GPCR/G Protein; Neuronal Signaling; PI3K/Akt/mTOR
Solubility:	10 mM in DMSO



BIOLOGICAL ACTIVITY:

YS-49 (monohydrate) is a **PI3K/Akt** (a downstream target of RhoA) activator, to reduce RhoA/PTEN activation in the 3-methylcholanthrene-treated cells. YS-49 inhibits **angiotensin II (Ang II)**-stimulated proliferation of VSMCs via induction of heme oxygenase (HO)-1. YS-49 is also an isoquinoline compound alkaloid, has a strong positive inotropic action through activation of cardiac **β-adrenoceptors**[1][2][3]. IC50 & Target: PI3K/Akt[3] **In Vitro:** YS-49 (1-100 μM; 18 hours; RAVSMC and RAW 264.7 cells) concentration-dependently inhibits the accumulation of nitrite in both RAVSMC and RAW 264.7 exposed to lipopolysaccharide (LPS) plus INF-γ, with **IC_{50}** values of 22 μM and 30 μM, respectively[2].
YS-49 (10-100 μM; 18 hours; RAVSMC and RAW 264.7 cells) suppresses iNOS gene expression induced by LPS and/or cytokines in RAVSMC and RAW 264.7 cells at the transcriptional level[2]. **In Vivo:** YS-49 (5 mg/kg; intraperitoneal injection; 8 hours; male Sprague Dawley rats) treatment significantly reduces serum NOx levels in LPS-treated rats, the NOx levels reduce from 86 μM to 34 μM[2].

PROTOCOL (Extracted from published papers and Only for reference)

Cell assay [1] Confluent RAVSMC were cultured in serum-free DMEM for 24 h, washed with serum-free DMEM, and exposed for 18 h to LPS (300 ng ml-1) plus IFN-γ (10 u ml-1) in the presence or absence of YS 49 (10, 30 and 100 μm). Similarly, RAW 264.7 cells were also incubated with LPS (10 ng ml-1) plus IFN-γ (10 u ml-1) for 18?h in the presence or YS 49 (1, 10 and 100 μm). YS 49 was dissolved in sterile distilled water, filtered through a 0.2 mm filter, and administered 1 h prior to, or simultaneously, or 2, 4, 8 or 16 h after LPS plus IFN-γ.

References:

[1]. Sun JJ, et al. YS 49, 1-(alpha-naphtylmethyl)-6,7-dihydroxy-1,2,3,4-tetrahydroisoquinoline, regulates angiotensin II-stimulated ROS production, JNK phosphorylation and vascular smooth muscle cell proliferation via the induction of heme oxygenase-1. Life Sci. 2008 Mar 12;82(11-12):600-7.

[2]. Kang YJ, et al. Prevention of the expression of inducible nitric oxide synthase by a novel positive inotropic agent, YS 49, in rat vascular smooth muscle and RAW 264.7 macrophages. Br J Pharmacol. 1999 Sep;128(2):357-64.

[3]. Hsu YH, et al. RhoA-mediated inhibition of vascular endothelial cell mobility: positive feedback through reduced cytosolic p21 and p27. J Cell Physiol. 2014 Oct;229(10):1455-65.

CAIndexNames:

1-(naphthalen-1-ylmethyl)-1,2,3,4-tetrahydroisoquinoline-6,7-diol hydrobromide hydrate

SMILES:

OC1=CC2=C(C(CC3=C4C=CC=CC4=CC=C3)NCC2)C=C1O.O.Br

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