

Product Datasheet

Praliciguat

Catalog No. **BP-01911** · CAS **1628730-49-3** · Form **free base** · Physical state **solid** · Color **White to off-white** · Version **1.0** · Revision **2026-09-04**

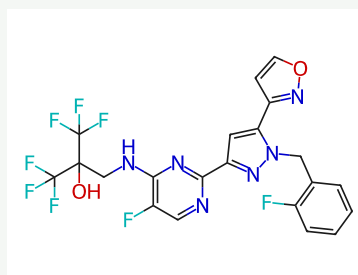
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Target Guanylate Cyclase; Apoptosis	Research area Cardiovascular Disease; Metabolic Disease
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Molecular formula **C₂₁H₁₄F₈N₆O₂**

Molecular weight **534.3621**

STRUCTURE



DESCRIPTION

Praliciguat (IW-1973) is a potent and orally active soluble guanylate cyclase stimulator, enhances NO signaling, acts as a vasodilator. Praliciguat (IW-1973) stimulates sGC in HEK-293 cells with an EC₅₀ of 197 nM.

BIOLOGICAL ACTIVITY

In Vitro

Praliciguat stimulates soluble guanylate cyclase in HEK-293 cells with an EC₅₀ of 197 nM.

Praliciguat demonstrates concentration-dependent relaxation of pre-contracted subcutaneous resistance arteries (EC₅₀ = 34.7 nM).

Praliciguat (10 μM, 48 h) inhibits the expression of proinflammatory cytokines and secretion of monocyte chemoattractant protein 1 in TNF-α-challenged proximal tubular epithelial cells (RPTC).

Praliciguat (1-10 μM, 48 h) inhibits TGF-β-mediated apoptosis in RPTC.

In Vivo

Praliciguat (1 mg/kg, p.o.) reduces arterial pressure (MAP) in normotensive and hypertensive rats.

Praliciguat (1-10 mg/kg, p.o.) attenuates proteinuria in obese ZSF1 rats model of diabetic nephropathy.

Praliciguat (1-10 mg/kg, p.o., daily for 2-7 weeks) attenuates inflammation, fibrosis, and end-organ damage in the Dahl rats model of cardiorenal failure.

SOLUBILITY

Soluble in DMSO 250 mg/mL

REFERENCES

[1]. Daniel P Zimmer, et al. Abstract 16938: IW-1973 is a Potent Soluble Guanylate Cyclase Stimulator in vitro and in vivo With Extensive Tissue Distribution.

[2]. Liu G, et al. Praliciguat inhibits progression of diabetic nephropathy in ZSF1 rats and suppresses inflammation and apoptosis in human renal proximal tubular cells. *Am J Physiol Renal Physiol.* 2020 Oct 1;319(4):F697-F711.

[3]. Shea CM, et al. Soluble guanylate cyclase stimulator praliciguat attenuates inflammation, fibrosis, and end-organ damage in the Dahl model of cardiorenal failure. *Am J Physiol Renal Physiol.* 2020 Jan 1;318(1):F148-F159.

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