

# PRX-08066

**Catalog No: tcsc0991**



## Available Sizes

**Size:** 5mg

**Size:** 10mg

**Size:** 25mg

**Size:** 50mg



## Specifications

**CAS No:**

866206-54-4

**Formula:**

$C_{19}H_{17}ClFN_5S$

**Pathway:**

Neuronal Signaling;GPCR/G Protein

**Target:**

5-HT Receptor;5-HT Receptor

**Purity / Grade:**

>98%

**Solubility:**

DMSO : 7 mg/mL (17.42 mM; Need ultrasonic)

**Observed Molecular Weight:**

401.89

## Product Description

PRX-08066 is a selective 5-hydroxytryptamine receptor 2B (5-HT<sub>2B</sub>R, IC<sub>50</sub>= 3.4 nM) antagonist that causes selective vasodilation of

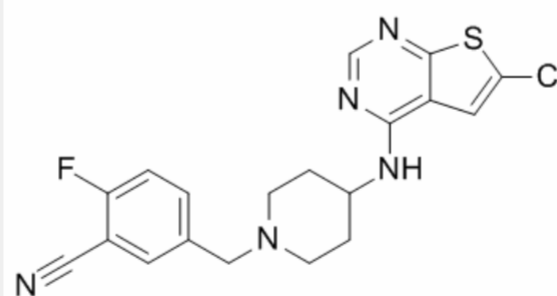
pulmonary arteries.

IC50 value: 3.4 nM [1]

Target: HT2B receptor

in vitro: PRX-08066 inhibits 5-HT-induced mitogen-activated protein kinase activation with IC50 of 12 nM and markedly reduces thymidine incorporation with IC50 of 3 nM in Chinese hamster ovary cells expressing the human 5-HT2BR, which suggests that PRX-08066 can potentially inhibit the pathologic 5-HT-induced vascular muscularization associated with PAH [1]. PRX-08066 inhibits cell proliferation with IC50 of 0.46 nM and with a maximum inhibition of 20% and 5-HT secretion with IC50 of 6.9 nM with a maximum inhibition of 30% in the 5-HT(2B) expressing SI-NET cell line, KRJ-I. PRX-08066 inhibits isoproterenol-stimulated 5-HT release with IC50 of 1.25 nM and a maximum inhibition of 60% in NCI-H720 cells. PRX-08066 (0.5 nM) significantly inhibits ERK phosphorylation in KRJ-I cells. PRX-08066 inhibits TGFβ1, CTGF and FGF2 transcription and secretion in KRJ-I cells. PRX-08066 decreases level of transcripts for Ki67 (84%) as well as Ki67 protein (36.8%) associated with an increase in caspase 3 transcript levels in KRJ-I cells. PRX-08066 decreases level of transcripts of TGFβ1, FGF2 and TPH1 in KRJ-I cells. PRX-08066 significantly increases the number of dead cells (34%) compared with untreated controls in KRJ-I cells. PRX-08066 causes a significant increase in dead/caspase 3 positive cells (76%) and caspase 3 activity (52%) in HEK293 cells [2].

in vivo: PRX-08066 (100 mg/kg) treated groups demonstrates less right ventricular hypertrophy and septal flattening than the monocrotaline control group in rats. PRX-08066 significantly reduces peak pulmonary artery pressure at 50 mg/kg and 100 mg/kg compared with monocrotaline control rats. PRX-08066 also significantly reduces right ventricle (RV)/body weight and RV/left ventricle + septum, compared with MCT-treated rats. PRX-08066 significantly attenuates the elevation in pulmonary artery pressure and RV hypertrophy and maintains cardiac function. PRX-08066 significantly reduces the hypoxia-dependent increase in right ventricular systolic pressure in both rats and mice without affecting the systemic mean arterial pressure in the animals [1]. PRX-08066 (100 mg/kg) significantly inhibits both right ventricular systolic pressure and right ventricular/left ventricular +septum weight elevations in rats. PRX-08066 (30 mg/kg) inhibits right ventricular systolic pressure and monocrotaline-induced ERK phosphorylation in whole lung homogenates in rats [3].



All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!