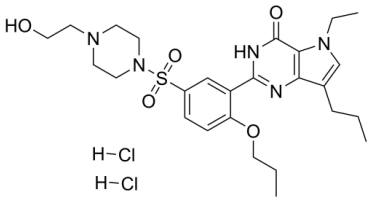
Data Sheet

Product Name:	Mirodenafil (dihydrochloride)
Cat. No.:	CS-3125
CAS No.:	862189-96-6
Molecular Formula:	$C_{26}H_{39}Cl_2N_5O_5S$
Molecular Weight:	604.59
Target:	Apoptosis; Glucocorticoid Receptor; Phosphodiesterase (PDE); Wnt; β-catenin
Pathway:	Apoptosis; Immunology/Inflammation; Metabolic Enzyme/Protease; Stem Cell/Wnt; Vitamin D Related/Nuclear Receptor
Solubility:	DMSO : ≥ 100 mg/mL (165.40 mM); H2O : 5 mg/mL (8.27 mM; ultrasonic and warming and heat to 60°C)



BIOLOGICAL ACTIVITY:

Mirodenafil (SK3530) dihydrochloride is an orally active, potent, reversible, and selective **phosphodiesterase 5 (PDE5)** inhibitor. Mirodenafil dihydrochloride is a **glucocorticoid receptor (GR)** modulator Mirodenafil dihydrochloride activates the **Wnt/β-catenin** signaling pathway by downregulating Dkk1 expression. Mirodenafil dihydrochloride can be used for the research of erectile dysfunction (ED), Alzheimer's disease (AD) and systemic sclerosis (SSc)[1][2][3]. ***In Vitro:*** Mirodenafil dihydrochloride (0-40 μM, 24 h) exerts neuroprotective functions via activating the cGMP/PKG/CREB signaling pathway[2].

Mirodenafil dihydrochloride (0-40 μM, 24 h) enhances neuronal survival by protecting the mitochondrial membrane potential and inhibiting apoptosis[2].

Mirodenafil dihydrochloride (0-40 μM) inhibits GSK-3β signaling, resulting in reduced tau phosphorylation, decreased Aβ production by inhibiting amyloidogenesis and activating the autophagosomal pathway[2].

Mirodenafil dihydrochloride inhibits the transcriptional activity of the glucocorticoid receptor (GR), and inhibits homodimerization of GR in HT-22 cells[2].

Mirodenafil dihydrochloride (0-100 μM, 24 h) inhibits TGF-β-induced phosphorylation of Smad2/3 and mRNA expression of the fibrosis marker in fibroblasts[3]. ***In Vivo:*** Mirodenafil dihydrochloride (4 mg/kg, IP, daily for 4 weeks) enhances the cognitive-behavioral performance in transgenic AD mice[2].

Mirodenafil dihydrochloride (0-10 mg/kg, Orally, daily for 3 weeks) ameliorates dermal fibrosis in a BLM-induced SSc mouse model by inhibiting the TGF-β signaling pathway, thereby suppressing the expression of collagen and profibrotic genes[3].

References:

[1]. Park HJ, et al. Mirodenafil for the treatment of erectile dysfunction: a systematic review of the literature. World J Mens Health. 2014 Apr;32(1):18-27.

[2]. Kang BW, et al. Phosphodiesterase 5 inhibitor mirodenafil ameliorates Alzheimer-like pathology and symptoms by multimodal actions. Alzheimers Res Ther. 2022 Jul 8;14(1):92.

[3]. Roh JS, et al. Mirodenafil ameliorates skin fibrosis in bleomycin-induced mouse model of systemic sclerosis. Anim Cells Syst (Seoul). 2021 Nov 3;25(6):387-395.

CAIndexNames:

4H-Pyrrolo[3,2-d]pyrimidin-4-one, 5-ethyl-3,5-dihydro-2-[5-[[4-(2-hydroxyethyl)-1-piperazinyl]sulfonyl]-2-propoxyphenyl]-7-propyl-, hydrochloride (1:2)

SMILES:

[H]Cl.[H]Cl.O=C1C(N(CC)C=C2CCC)=C2N=C(C3=CC(S(=O)(N4CCN(CCO)CC4)=O)=CC=C3OCCC)N1

Caution: Product has not been fully validated for medical applications. For research use only.

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