For Research Use Only SKF-96365 hydrochloride



Catalog Number: CM03250

产品信息	Catalog Number: CM03250	分子量: 402.91	
	CAS号: 130495-35-1	溶解度: DMSO:1086mM H2O:1086mM	
	分子式: C ₂₂ H ₂₇ ClN ₂ O ₃	DM30.198.0 mm,H20.196.0 mm	С-снь
	主要靶点: Autophagy Potassium Channel Calcium Channel TRP/TRPV Channel Apoptosis		
	主要通路: 凋亡 代谢 离子通道 自噬		
体外活性	SKF-96365 exhibits protective activity again PC12 cells after MPP+ administration. SKF-9 cells. Because of its non-selective activity. S it not only blocks high-voltage-activated (H inhibits low-voltage -activated (LVA) T-type calcium homeostasis might dependent on co	nst MPP+ injury in PC 12 cells and significa 6365 does not exert effects on necrotic cel KF-96365 has been demonstrated to have VA) Ca2+ channels at typically utilized te: Ca2+ channels in HEK293 cells. The exact ell types and experimental models used[2	Intly inhibits apoptotic cell death in Il death induced by MPP+ insult in PC12 effects on multiple other Ca2+ channels: st concentrations, but also potently t effect of SKF-96365 on intracellular !].
体内活性	SKF-96365 treatment inhibited the calcium/ vitro and in vivo[4].	calmodulin-dependent protein kinase II γ	$^\prime$ (CaMKII γ)/AKT signaling cascade in
细胞实验	To investigate whether SKF-96365 cou PC12 cells are pretreated with SKF-96 µM) 30 min before MPP+ additio cell proliferation reagent WST-1. (Only	Ild protect PC12 cells from injury inc 365 in different concentrations (1 & on. The cells viability is measured 24 for Reference)	duced by MPP+ insult, cultured micro;M, 10 µM or 50 h after MPP+ insult by using the
描述	SKF-96365, a SOCE inhibitor, exhibits poten cells. SKF-96365 can induce cytoprotective a from the mitochondria into the cytoplasm. M dependent protein kinase II γ (CaMKII γ)/A abolished the effects of SKF-96365 on Y cell induced biological effects. SKF-96365 inhibi	t anti-neoplastic activity by inducing cell- utophagy to delay apoptosis by preventir lechanistically, SKF-96365 treatment inhil KT signaling cascade in vitro and in vivo. s, suggesting a critical role of the CaMKII γ ted hERG current in a concentration-deper	-cycle arrest and apoptosis in colorectal Y ng the release of cytochrome c (cyt c) bited the calcium/calmodulin- Overexpression of CaMKII γ or AKT / AKT signaling pathway in SFK-96365- ndent manner.
储存	Powder: -20°C for 3 years In solvent: -	80°C for 2 years	