

H-89 (dihydrochloride)

Catalog No: tcsc3387



Available Sizes

Size: 10mg

Size: 50mg

Size: 100mg

Size: 200mg

Size: 500mg



Specifications

CAS No:

130964-39-5

Formula:

$C_{20}H_{22}BrCl_2N_3O_2S$

Pathway:

Stem Cell/Wnt;Protein Tyrosine Kinase/RTK;Autophagy

Target:

PKA;PKA;Autophagy

Purity / Grade:

>98%

Solubility:

DMSO : ≥ 50 mg/mL (96.29 mM)

Alternative Names:

Protein kinase inhibitor H-89 dihydrochloride

Observed Molecular Weight:

519.28

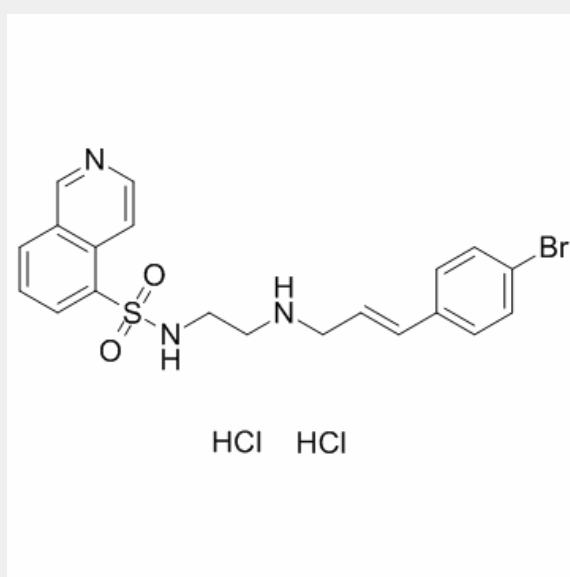
Product Description

H-89 (dihydrochloride) is a potent inhibitor of cyclic AMP-dependent protein kinase (**protein kinase A**) with **IC₅₀** of 48 nM and has weak inhibition on PKG, PKC, Casein Kinase, and others kinases.

IC50 & Target: IC50: 48 nM (protein kinase A)

In Vitro: H-89 inhibits protein kinase A, in competitive fashion against ATP. H-89 causes a dose-dependent inhibition of the forskolin-induced protein phosphorylation, with no decrease in intracellular cyclic AMP levels in PC12D cells. H-89 significantly inhibits the forskolin-induced neurite outgrowth from PC12D cells. H-89 (30 μM) inhibits significantly cAMP-dependent histone IIb phosphorylation activity in PC12D cell lysates^[1]. H-89 (1-2 μM) significantly slows the repriming rate in rat skinned fibres, most likely due to it deleteriously affecting the T-system potential. H-89 (10-100 μM) inhibits net Ca²⁺ uptake by the SR and affects the Ca²⁺-sensitivity of the contractile apparatus in rat skinned fibres^[2].

In Vivo: H-89 (0.2 mg/100g, i.p.) significantly increases seizure latency and threshold in PTZ-treated animals. H-89 (0.05, 0.2 mg/100 g, i.p.) prevents the epileptogenic activity of bucladesine (300 nM) with significant increase of seizure latency and seizure threshold^[3].



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