Web: www.taiclone.com
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AT7867 (dihydrochloride)

Catalog No: tcsc1197

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
1431697-86-7

Formula:
$C_{20}H_{22}Cl_3N_3$

Pathway:
Stem Cell/Wnt;Protein Tyrosine Kinase/RTK;PI3K/Akt/mTOR;MAPK/ERK Pathway

Target:
PKA;PKA;Akt;Ribosomal S6 Kinase (RSK)

Purity / Grade:
>98%

Solubility:
10 mM in DMSO

Observed Molecular Weight:
410.77

Product Description

AT7867 dihydrochloride is a potent ATP-competitive inhibitor of **Akt1**/**Akt2**/**Akt3** and **p70S6K**/**PKA** with **IC_{50}**s of 32 nM/17 nM/47

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nM and 85 nM/20 nM, respectively.

IC50 & Target: IC50: 32 nM/17 nM/47 nM (Akt1/Akt2/Akt3), 85 nM/20 nM (p70S6K/PKA)[1]

In Vitro: The inhibition of AKT2 by AT7867 is shown to be ATP-competitive with a K_i of 18nM. AT7867 also displays potent activity against the structurally related AGC kinases p70S6K and PKA, but shows a clear window of selectivity against kinases from other kinase sub-families. In vitro growth inhibition studies show that AT7867 blocks proliferation in a number of human cancer cell lines. AT7867 appears to be most potent at inhibiting proliferation in MES-SA uterine, MDA-MB-468 and MCF-7 breast, and HCT116 and HT29 colon lines (IC_{50} values range from 0.9-3 μM), and least effective in the two prostate lines tested (IC_{50} values range from 10-12 μM) [1].

In Vivo: Following oral administration at 20 mg/kg, the elimination of AT7867 from plasma appears to be similar to that observed after i.v. administration. Plasma levels of AT7867 remain above 0.5 μM for at least 6 hours following an oral dose of 20 mg/kg. Assuming linear pharmacokinetics following i.v. administration, the bioavailability by the oral route is calculated to be 44%. In vivo pharmacodynamic (PD) biomarker studies are therefore performed with this model. Following pharmacokinetic and tolerability studies, doses of AT7867 (90 mg/kg p.o. or 20 mg/kg i.p.) are administered to athymic mice bearing MES-SA tumors and the phosphorylation status of GSK3β and S6RP in tumors is monitored over time. Clear inhibition of phosphorylation of the two markers of pathway activity is seen at 2 and 6 hours following treatment with AT7867. By 24 hours, total levels of both GSK3β and S6RP are greatly reduced[1].