Web: www.taiclone.com
Tel: +886-2-2735-9682
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Miransertib

Catalog No: tcsc5377

Available Sizes

Size: 1mg

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
1313881-70-7

Formula:
$C_{27}H_{24}N_6$

Pathway:
PI3K/Akt/mTOR

Target:
Akt

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 40.2 mg/mL (92.94 mM)

Alternative Names:
ARQ-092

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Observed Molecular Weight:
432.52

Product Description

Miransertib (ARQ-092) is an orally bioavailable, selective, and potent allosteric **Akt** inhibitor with **IC_{50}**s of 2.7 nM, 14 nM and 8.1 nM for **Akt1**, **Akt2**, **Akt3**, respectively.

IC50 & Target: IC50: 2.7 nM (Akt1), 14 nM (Akt2), 8.1 nM (Akt3)[1]

In Vitro: Miransertib (ARQ-092; Compound 21a) demonstrates high enzymatic potency against Akt1, Akt2 and Akt3, as well as potent cellular inhibition of Akt activation and the phosphorylation of the downstream target PRAS40. Miransertib shows strong affinity for un-phosphorylated fulllength Akt1 and potently inhibited the phosphorylated form of full-length Akt isoforms. In a large panel of cell lines derived from various tumor types, Miransertib shows potent anti-proliferative activity in cell lines containing PIK3CA/PIK3R1 mutations compared to those with wild-type (wt) PIK3CA/PIK3R1 or PTEN loss. Miransertib shows excellent inhibition of p-Akt (S473) and p-Akt (T308) in both AN3CA and A2780 cells. The inhibition of the downstream protein p-PRAS40 (T246) is observed with Miransertib (IC_{50}=0.31 μM)[1].

In Vivo: In a mouse pharmacokinetic study, (po at 100 mg/kg, iv at 5 mg/kg), Miransertib (ARQ-092; Compound 21a) shows an oral bioavailability of 23%. Miransertib results in 99%, 95% and 58% reductions in p-Akt (S473), p-Akt (T306) and p-PRAS40 (T246), respectively, after tumor-bearing mice are treated with 100 mg/kg po. The inhibition of phosphorylation is sustained at eight hours. The plasma concentration of Miransertib at one hour is 2.1 μM and decreased to 0.26 μM at 8 hours, while in the tumor, the concentration is 21.0 μM at one hour and 9.6 μM at 8 hours[1]. To determine the effects of Miransertib (ARQ-092) on cardiac function, echocardiographic analysis of $SHP2^{+/+}$ and $SHP2^{Y279C/+}$ littermates is conducted, either in the presence of orally administered vehicle or Miransertib (100 mg/kg/day), at 12, 14, and 16 weeks of age. By 12 weeks of age, $SHP2^{Y279C/+}$ mice show significant left ventricular hypertrophy, as indicated by decreased chamber dimension and increased posterior wall thickness compared with those of littermate controls; hypertrophy in these mice continued to progress over the 4 week time period. Treatment of the $SHP2^{Y279C/+}$ mice with Miransertib normalizes the hypertrophic cardiomyopathy (HCM) phenotype as early as 2 weeks following treatment, with levels comparable to those in $SHP2^{+/+}$ at this time point[2].