Taiclone®
Quality Guarantee

Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

WYE-132

Catalog No: tcsc0066

Available Sizes

Size: 2mg

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
1144068-46-1

Formula:
$C_{27}H_{33}N_7O_4$

Pathway:
PI3K/Akt/mTOR

Target:
mTOR

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 62 mg/mL (119.32 mM)

Alternative Names:
WYE-125132

Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Observed Molecular Weight:
519.6

Product Description

WYE-125132 (WYE-132) is a highly potent, ATP-competitive, and specific **mTOR** kinase inhibitor (**IC_{50}**: 0.19±0.07 nM; >5,000-fold selective versus PI3Ks). WYE-125132 inhibits **mTORC1** and **mTORC2**.

IC50 & Target: IC50: 0.19±0.07 nM (mTOR), 1179 nM (PI3Kα), 2380 nM (PI3Kδ), 1250 nM (hSMG1)[1]

In Vitro: WYE-125132 (WYE-132) potently inhibits recombinant mTOR via an ATP-competitive mechanism. WYE-125132 is a potent antiproliferative agent against a panel of cancer cell lines with IC_{50} values generally in the nanomolar range. In the typical 3-day dose-response studies, WYE-125132 exhibits a more profound antiproliferative activity than CCI-779 in MDA361 and other cells, as shown by the sharper inhibition at doses up to 10 μM. Fluorescence-activated cell sorting (FACS) analysis of inhibitor-treated (1 μM, 24 hours) MDA468, PC3MM2, U87MG, A549, and HCT116 cells indicates that WYE-125132 elicits a more profound increase in G_1-phase and a reduction in S-phase cells than CCI-779. The WYE-125132–induced cell death is evident at 10 and 30 nM (6.2% and 13%, respectively) and is dose dependent, reaching 47% at 1 μM and 59% at 3 μM[1].

In Vivo: A single i.v. administration of 50 mg/kg WYE-125132 (WYE-132) into tumor-bearing mice leads to suppression of P-S6K(T389) and P-AKT(S473) for at least 8 hours in PC3MM2, MDA361, HCT116, and HT29 tumors, whereas the steady-state level of P-AKT(T308) is not significantly reduced, indicating that the antitumor efficacy of WYE-125132 under such dosing regimens reflects the suppression of mTOR rather than PI3K. Oral administration of WYE-125132 causes dose-dependent tumor growth delay in the PI3K/mTOR- and HER2-hyperactive MDA361 tumors with significant antitumor activity at 5 mg/kg, which correlates with a suppression P-S6 and P-AKT(S473) but not P-AKT(T308). An optimal dose of 50 mg/kg WYE-125132 induces a substantial regression of large MDA361 tumors. WYE-125132 also causes a potent and substantial tumor growth delay in the PTEN-null U87MG glioma[1].