

Sapanisertib

Catalog No: tcsc0557

Available Sizes

Size: 5mg

Size: 10mg

Size: 100mg

Size: 200mg

Size: 500mg

Size: 500mg

Size: 2g

Size: 2g

Specifications

1224844-38-5

Formula:

 $C_{15}H_{15}N_7O$

Pathway:

PI3K/Akt/mTOR;Autophagy

Target:

mTOR;Autophagy

Purity / Grade:

>98%

Solubility:

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DMSO : ≥ 83.3 mg/mL (269.29 mM)

Alternative Names:

INK-128;MLN0128

Observed Molecular Weight:

309.33

Product Description

Sapanisertib (INK-128) is a ATP-dependent **mTOR1/2** inhibitor with an **IC**₅₀ of 1 nM for mTOR kinase.

IC50 & Target: IC50: 1 nM (mTOR), 219 nM (PI3Kα), 5293 nM (PI3Kβ), 230 nM (PI3Kδ), 221 nM (PI3Kγ)^[2]

Ki: 1.4 nM (mTOR), 152 nM (PI3Kα), 4700 nM (PI3Kβ), 165 nM (PI3Kγ)^[2]

In Vitro: Sapanisertib (INK-128) exhibits an enzymatic inhibition activity against mTOR and more than 100-fold selectivity to PI3K kinases^[1]. Sapanisertib (INK-128) selectively decreases the expression of YB1, MTA1, vimentin and CD44 at the protein but not transcript level in PC3 cells. Sapanisertib (INK-128) decreases the invasive potential of PC3 prostate cancer cells. Furthermore, Sapanisertib (INK-128) inhibits cancer cell migration starting at 6 h of treatment, precisely correlating with when decreases in the expression of pro-invasion genes are evident, but preceding any changes in the cell cycle or overall global protein synthesis^[2].

In Vivo: In a ZR-75-1 breast cancer xenograft model, Sapanisertib (INK-128) shows tumor growth inhibition efficacy at a dose of 0.3 mg/kg/day^[1]. 4EBP1 and p70S6K1/2 phosphorylation is completely restored to wild-type levels after treatment with INK128 in PtenL/L mice. Sapanisertib (INK-128) treatment results in a 50% decrease in prostatic intraepithelial neoplasia (PIN) lesions in PtenL/L mice and induces programmed cell death in multiple cancer cell lines in mice^[2].



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