# Sapanisertib

**Cat. No.:** HY-13328

**CAS No.:** 1224844-38-5

**Molecular Formula:** $C_{15}H_{15}N_7O$

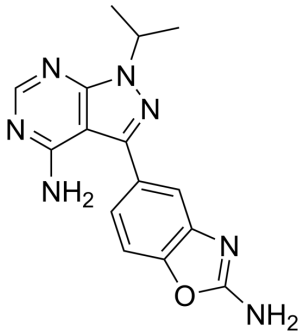
**Molecular Weight:** 309.33

**Target:** mTOR; Autophagy

**Pathway:** PI3K/Akt/mTOR; Autophagy

**Storage:**

| | | |
|---|---|---|
| Powder | -20°C | 3 years |
| | 4°C | 2 years |
| In solvent | -80°C | 6 months |
| | -20°C | 1 month |



## SOLVENT & SOLUBILITY

**In Vitro**

DMSO : 55 mg/mL (177.80 mM; Need ultrasonic)

| Preparing Stock Solutions | Solvent / Mass / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 3.2328 mL | 16.1640 mL | 32.3279 mL |
| | 5 mM | 0.6466 mL | 3.2328 mL | 6.4656 mL |
| | 10 mM | 0.3233 mL | 1.6164 mL | 3.2328 mL |

Please refer to the solubility information to select the appropriate solvent.

**In Vivo**

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (6.72 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (6.72 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (6.72 mM); Clear solution

## BIOLOGICAL ACTIVITY

**Description**

Sapanisertib (INK-128; MLN0128; TAK-228) is an orally available, ATP-dependent mTOR1/2 inhibitor with an $IC_{50}$ of 1 nM for mTOR kinase.

**$IC_{50}$ & Target**

| | | | |
|---|---|---|---|
| mTOR<br>1 nM ($IC_{50}$) | mTORC1 | mTORC2 | PI3Kα<br>219 nM ($IC_{50}$) |
| PI3Kγ<br>221 nM ($IC_{50}$) | PI3Kδ<br>230 nM ($IC_{50}$) | PI3Kβ<br>5.293 μM ($IC_{50}$) | Autophagy |

| | |
|---|---|
| **In Vitro** | Sapanisertib (INK-128) exhibits an enzymatic inhibition activity against mTOR and more than 100-fold selectivity to PI3K kinases[1].<br>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].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **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].<br>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].<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## PROTOCOL

| | |
|---|---|
| **Cell Assay** [2] | PC3 cells are treated with the appropriate drug for 48 h, and proliferation is measured using CellTiter-Glo Luminescent reagent. The concentration of Sapanisertib (INK-128) necessary to achieve inhibition of cell growth by 50% ($IC_{50}$) is calculated using concentrations ranging from 20.0 μM to 0.1 nM (12-point curve).<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |
| **Animal Administration** [2] | Nude mice are inoculated subcutaneously in the right subscapular region with $5\times10^6$ MDA-MB-361 cells. After tumours reach a size of 150-200 $mm^3$, mice are randomLy assigned into vehicle control or treatment groups. Sapanisertib (INK-128) is formulated in 5% polyvinylpropyline, 15% NMP, 80% water and administered by oral gavage at 0.3 mg/kg and 1 mg/kg daily.<br>MCE has not independently confirmed the accuracy of these methods. They are for reference only. |

## CUSTOMER VALIDATION

- Nature. 2016 Dec 1;540(7631):119-123.
- Cell Stem Cell. 2020 Sep 3;27(3):441-458.e10.
- Cell Stem Cell. 2018 Mar 1;22(3):369-383.e8.
- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.
- Nat Commun. 2017 Jun 8;8:15617.

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## REFERENCES

[1]. Liu A, et al. mTOR Mediated Anti-Cancer Drug Discovery. Drug Discovery Today: Therapeutic Strategies. 2009, 6(2), 47-55.

[2]. Hsieh AC, et al. The translational landscape of mTOR signalling steers cancer initiation and metastasis. Nature. 2012 Feb 22;485(7396):55-61.

**Caution: Product has not been fully validated for medical applications. For research use only.**

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