

Product Datasheet

MK-1775

Catalog No. **BP-02302** · CAS **955365-80-7** · Form **free base** · Physical state **solid** · Color **Light yellow to yellow** · Version **1.0** · Revision **2026-09-10**

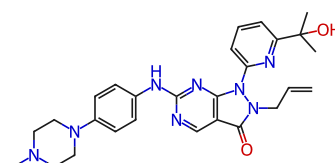
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Target Wee1	Research area Cancer
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Molecular formula **C₂₇H₃₂N₈O₂**

Molecular weight **500.5954**

STRUCTURE



DESCRIPTION

Adavosertib (MK-1775, AZD1775) is a potent and selective Wee1 inhibitor with IC₅₀ of 5.2 nM in a cell-free assay; hinders G2 DNA damage checkpoint.

BIOLOGICAL ACTIVITY

In Vitro

Adavosertib (MK-1775) enhances the cytotoxic effects of 5-FU in p53-deficient human colon cancer cells.

Adavosertib (MK-1775) inhibits CDC2 Y15 phosphorylation in cells, abrogates DNA damaged checkpoints induced by 5-FU treatment, and causes premature entry of mitosis determined by induction of Histone H3 phosphorylation.

Adavosertib (MK-1775) abrogates the radiation-induced G2 block in p53-defective cells but not in p53 wild-type lines.

The combination of NSC 613327 with Adavosertib (MK-1775) produces robust anti-tumor activity and remarkably enhances tumor regression response (4.01 fold) compared to NSC 613327 treatment in p53-deficient tumors.

In Vivo

In vivo, Adavosertib (MK-1775) potentiates the anti-tumor efficacy of 5-FU at tolerable doses.

Adavosertib (MK-1775) (60 mg/kg twice daily, p.o.) enhances H1299 xenograft tumor response to fractionated radiotherapy.

Adavosertib (MK-1775) (30 mg/kg. p.o.) regresses tumor growth in PANC198, PANC215 and PANC185 as compared to GEM treated mice.

SOLUBILITY

Soluble in DMSO

REFERENCES

[1]. Hirai H, et al. Small-molecule inhibition of Wee1 kinase by MK-1775 selectively sensitizes p53-deficient tumor cells to DNA-damaging agents. *Mol Cancer Ther*, 2009, 8(11), 2992-3000.

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