

Product Datasheet

PTC596

Catalog No. **BP-01904** · CAS **1610964-64-1** · Form **free base** · Physical state **solid** · Color **White to off-white** · Version **1.0** · Revision **2026-09-04**

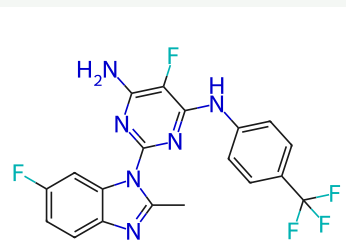
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Target BMI1; Apoptosis	Research area Cancer
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Molecular formula **C₁₉H₁₃F₅N₆**

Molecular weight **420.3387**

STRUCTURE



DESCRIPTION

PTC596 is an orally active and selective B-cell-specific Moloney murine leukemia virus integration site 1 (BMI-1) inhibitor. PTC596 targets BMI1 expressed by both tumor cells and cancer stem cells (CSCs), and induces hyperphosphorylation of BMI1, leading to its degradation.

BIOLOGICAL ACTIVITY

In Vitro

Unesbulin (PTC596; 20-200 nM; for 48 hours) induces apoptosis in AML cells in a p53-independent manner. BMI-1 overexpression desensitizes AML cells to PTC596-induced apoptosis.

Unesbulin (200 nM; for 10 hours) leads to an accumulation of cells in G2/M phase.

Unesbulin (0.012-1 μM; for 20 hours) significantly reduces protein levels of BMI-1.

Unesbulin inhibits APC/CCDC20 activity resulting in the persistent activation of CDK1 and CDK2 which mediate the hyperphosphorylation of BMI1.

In Vivo

Unesbulin (PTC596; 5 mg/kg; oral gavage; every 3 days for 13 days) significantly prolongs mouse survival.

Unesbulin (20 mg/kg; oral gavage; once a week for 15 days) causes tumor volume significantly smaller than that of control SCID mice with K562 cells.

Unesbulin (10 or 12.5 mg/kg; oral gavage; twice a week until death) causes the survival significantly longer than the vehicle-treated group in NOD-SCID mice with HL-60 cells.

SOLUBILITY

Soluble in DMSO

REFERENCES

[1]. Nishida Y, et al. The novel BMI-1 inhibitor PTC596 downregulates MCL-1 and induces p53-independent mitochondrial apoptosis in acute myeloid leukemia progenitor cells. Blood Cancer J. 2017 Feb 17;7(2):e527.

[2]. BMI1 inhibitor PTC596

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Lot CoA and SDS are separate documents.