Product Datasheet

BBI-2779

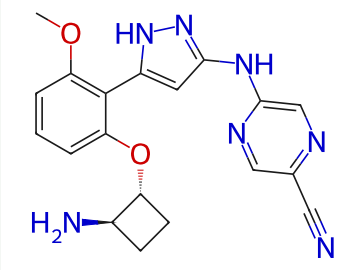
Catalog No. **BP-02275** · CAS **2871057-47-3** · Form **free base** · Physical state **solid** · Color **White to off-white** · Version **1.0** · Revision **2026-08-31**

FOR RESEARCH USE ONLY. NOT FOR HUMAN OR VETERINARY USE.

Target	Research area
Checkpoint Kinase (Chk)	**Cancer**

Molecular formula	$C_{19}H_{19}N_7O_2$
Molecular weight	**377.3999**

STRUCTURE



DESCRIPTION

BBI-2779 is a potent, selective and orally bioavailable inhibitor of CHK1 with biochemical IC50 of 0.5 nM and cellular IC50 of 3 nM (pCHK1-S345 activity in HT29 cells), 160-fold selective for CHK1 over CHK2.

BIOLOGICAL ACTIVITY

In Vitro

BBI-2779 selectively eliminates ecDNA-positive tumor cells by exploiting transcription-replication conflicts to induce synthetic lethality, and blocks ecDNA-mediated acquired resistance to targeted therapies in in vitro models[1].

BBI-2779 potently and selectively inhibits CHK1 in a cell-free biochemical assay with an IC50 of 0.3 nM and 160-fold selectivity over CHK2; it inhibited CHK1 activity, with an IC50 of 3 nM in HT29 cells[2].

BBI-2779 (3-12 nM; 24 h) induces dose-dependent replication stress and DNA damage, measured via pCHK1-S345, pRPA2-S8, and γH2AX expression, preferentially in ecDNA-positive COLO320DM colorectal cancer cells compared to chromosomal amplification-positive COLO320HSR cells[2].

In Vivo

BBI-2779 (30 mg/kg; p.o.; every other day; 27 days) alone drives significant tumour growth inhibition in ecDNA-positive gastric cancer xenografts, and in combination with Infigratinib (HY-13311) (15 mg/kg; p.o.; once daily; 27 days) induces potent tumour regression and prevents ecDNA-mediated acquired resistance to FGFR inhibition[2].

SOLUBILITY

Soluble in DMSO

REFERENCES

[1]. Zhu X, Qian Y, Tang Q, Li J, Geng Y, Gong S, Jin C. Research Advances of Extrachromosomal Circular DNA in Breast Cancer. Cancer Med. 2025 Oct;14(19):e71285. doi: 10.1002/cam4.71285. PMID: 41051137; PMCID: PMC12498277.

[2]. Tang J, Weiser NE, Wang G, Chowdhry S, Curtis EJ, Zhao Y, Wong IT, Marinov GK, Li R, Hanoian P, Tse E, Mojica SG, Hansen R, Plum J, Steffy A, Milutinovic S, Meyer ST, Luebeck J, Wang Y, Zhang S, Altemose N, Curtis C, Greenleaf WJ, Bafna V, Benkovic SJ, Pinkerton AB, Kasibhatla S, Hassig CA, Mischel PS, Chang HY. Enhancing transcription-replication conflict targets ecDNA-positive cancers. Nature. 2024 Nov;635(8037):210-218. doi: 10.1038/s41586-024-07802-5. Epub 2024 Nov 6. PMID: 39506153; PMCID: PMC11540844.

[3]. Anthony B. Pinkerton, et al. Checkpoint kinase 1 (chk1) inhibitors and uses thereof. WO2022251502A1.

Tel: +86 20 31130026 · Fax: +86 20 36822386 · tech@biochemprobe.com · www.biochemprobe.com