Product Datasheet

GSK126

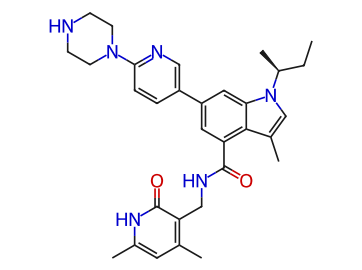
Catalog No. **BP-02305** · CAS **1346574-57-9** · Form **free base** · Physical state **solid** · Color **White to yellow** · Version **1.0** · Revision **2026-09-10**

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

Target	Research area
Histone Methyltransferase	**Cancer**

Molecular formula	$C_{31}H_{38}N_6O_2$
Molecular weight	**526.6724**

STRUCTURE



DESCRIPTION

GSK126 (GSK2816126A, GSK2816126) is a potent, highly selective EZH2 methyltransferase inhibitor with IC50 of 9.9 nM, >1000-fold selective for EZH2 over 20 other human methyltransferases.

BIOLOGICAL ACTIVITY

In Vitro

GSK126 potently inhibits both wild-type and mutant EZH2 methyltransferase activity with similar potencies (Ki=0.5-3 nM) independent of substrate used, and is competitive with S-adenosyl-methionine (SAM) and non-competitive with peptide substrates. GSK126 is highly selective against other methyltransferases and multiple other protein classes (EZH1, IC50=680 nM)[1]. Treatment of three SCLC cell lines with GSK126, induces growth inhibition. SCLC cell lines (Lu130, H209, and DMS53) are treated with 0.5, 2, and 8 μM GSK126, and growth curve is analyzed by WST-8 assay. Inhibition of cellular growth by GSK126 treatment is observed at 8 μM in all the three cell lines, while Lu130 and H209 are more sensitive to GSK126, even at lower doses.

In Vivo

GSK126 is administered intraperitoneally at a dose volume of 0.2 mL per 20 g body weight in female beige SCID mice. GSK126 effectively inhibits the proliferation of EZH2 mutant DLBCL cell lines and markedly inhibits the growth of EZH2 mutant DLBCL xenografts in mice.

SOLUBILITY

Soluble in DMSO; Ethanol: Insoluble

REFERENCES

[1]. McCabe, Michael T et al. “EZH2 inhibition as a therapeutic strategy for lymphoma with EZH2-activating mutations.” Nature vol. 492,7427 (2012): 108-12.

[2]. Ott, Heidi M et al. “A687V EZH2 is a driver of histone H3 lysine 27 (H3K27) hypertrimethylation.” Molecular cancer therapeutics vol. 13,12 (2014): 3062-73

[3]. Takeshima, Hideyuki et al. “Identification of coexistence of DNA methylation and H3K27me3 specifically in cancer cells as a promising target for epigenetic therapy.” Carcinogenesis vol. 36,2 (2015): 192-201.

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