

Product Data Sheet

Product Name: EED226
Cat. No.: GC19130

Chemical Properties

Cas. No. 2083627-02-3

SMILES O=S(C1=CC=C(C2=CN=C(NCC3=CC=CO3)N4C2=NN=C4)C=C1)(C)=O

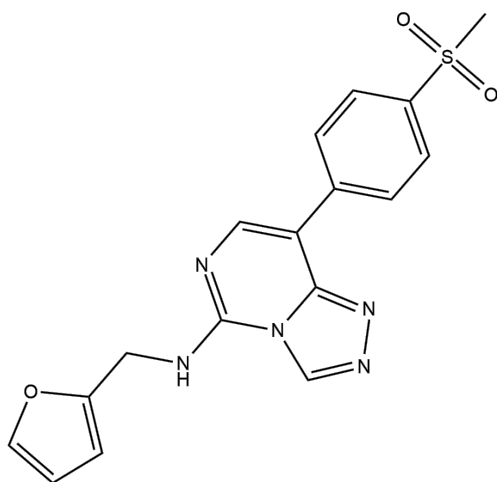
Formula $C_{17}H_{15}N_5O_3S$ M.Wt 369.4

Solubility DMSO : ≥ 29 mg/mL (78.51 mM); Water : < 0.1 mg/mL (insoluble) Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines IMR32 cells

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

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Preparation Method IMR32 cells were cultured in RPMI1640 medium supplemented with 10% fetal bovine serum (FBS) and 50µg/ml penicillin in a humidified atmosphere containing 5% CO₂ at 37°C. IMR32 cells were seeded in 96-well plates at a density of 10³ cells/well. Cells were treated with different concentrations of EED226 (0, 1, 2, 5, and 10µM) for 5 days. Then, the cell viability was analyzed.

Reaction Conditions 0, 1, 2, 5, and 10µM; 5 days

Applications EED226 treatment significantly reduced the cell viability of IMR32 cells in a dose-dependent manner.

Animal experiment [2]:

Animal models C57BL/6J mice

Preparation Method C57BL/6J mice (20-23g) were kept in temperature-controlled (21°C-25°C) and humidity-maintained (40%-70%) rooms with a 12-h light/dark cycle. Water and food were provided freely. To establish a mouse model of cisplatin-induced AKI, the mice were injected intraperitoneally with cisplatin (25mg/kg) in saline. EED226 (40mg/kg) dissolved in corn oil and DMSO was administered intragastrically immediately after cisplatin injection, and then administered twice a day for 2 days. The control group received the same amount of vehicle. Mice were euthanized and kidney samples were collected for histological examination.

Dosage form 40mg/kg; twice a day for 2 days; p.o.

Applications EED226 treatment improved renal function and attenuated renal tubular cell injury in the murine model of cisplatin-induced AKI.

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References:

- [1] Shaliman D, Takenobu H, Sugino R P, et al. The PRC2 molecule EED is a target of epigenetic therapy for neuroblastoma[J]. European Journal of Cell Biology, 2022, 101(3): 151238.
- [2] Yu C, Li T, Li J, et al. Inhibition of polycomb repressive complex 2 by targeting EED protects against cisplatin-induced acute kidney injury[J]. Journal of Cellular and Molecular Medicine, 2022, 26(14): 4061-4075.

Background

EED226 is a potent inhibitor of Polycomb repressive complex 2 (PRC2) with an IC₅₀ value of 22.3nM [1]. EED226 binds to the H3K27Me3 binding pocket of EED through a allosteric mechanism, thereby inhibiting the function of PRC2. It effectively suppresses the level of H3K27Me3 in the cell and regulates the expression of target genes. [2]. EED226 has been widely used in cell and animal models to inhibit tumor progression[3].

In vitro, EED226 treatment for 5 days significantly inhibited the proliferation of NGP and IMR32 cells, with IC₅₀ values of 2.64μM and 2.05μM, respectively [4]. EED226 treatment at 5μM for 48 hours significantly enhanced the proliferation of female germline stem cells (FGSCs), promoted the expression of OCT4, and inhibited the expression of P53 and P63[5].

In vivo, EED226 treatment via daily intragastric administration (40mg/kg) twice for 2 days improved renal function and alleviated renal injury in the mouse model of acute kidney injury (AKI) induced by cisplatin[6]. Oral administration of EED226 twice daily (40mg/kg) for 32 days can inhibit tumor growth in the Karpas422-xenograft mouse models[7]. Oral administration of EED226 twice daily (40mg/kg) for 30 days effectively alleviated the symptoms of experimental autoimmune encephalomyelitis (EAE) in mice and improved inflammatory infiltration[8].

References:

- [1] Cook N, Chen J, Zhou J, et al. Embryonic ectoderm development (EED) as a novel target for cancer treatment[J]. Current topics in medicinal chemistry, 2021, 21(31): 2771-2777.
- [2] Liu K L, Zhu K, Zhang H. An overview of the development of EED inhibitors to disable the PRC2 function[J]. RSC Medicinal Chemistry, 2022, 13(1): 39-53.

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- [3] Atadja P W. Abstract IA19: Targeting the PRC2 complex through EED for anti-cancer therapy[J]. Cancer Research, 2017, 77(22_Supplement): IA19-IA19.
- [4] Shaliman D, Takenobu H, Sugino R P, et al. The PRC2 molecule EED is a target of epigenetic therapy for neuroblastoma[J]. European Journal of Cell Biology, 2022, 101(3): 151238.
- [5] Wang J, Fang J, Feng M, et al. Inhibition of EED activity enhances cell survival of female germline stem cell and improves the oocytes production during oogenesis in vitro[J]. Open Biology, 2023, 13(1).
- [6] Yu C, Li T, Li J, et al. Inhibition of polycomb repressive complex 2 by targeting EED protects against cisplatin-induced acute kidney injury[J]. Journal of Cellular and Molecular Medicine, 2022, 26(14): 4061-4075.
- [7] Qi W, Zhao K, Gu J, et al. An allosteric PRC2 inhibitor targeting the H3K27me3 binding pocket of EED[J]. Nature chemical biology, 2017, 13(4): 381-388.
- [8] Hong W, Ma H, Li Z, et al. Inhibition of EED-mediated histone methylation alleviates neuroinflammation by suppressing WNT-mediated dendritic cell migration[J]. Journal of Neuroinflammation, 2025, 22(1): 97.

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