

Product Data Sheet

Product Name: GSK3368715 dihydrochloride

Cat. No.: GC36192

Chemical Properties

Cas. No. 1628925-77-8

SMILES CNCCN(CC1=CN=C1C2CCC(COCC)(COCC)CC2)C.Cl.Cl

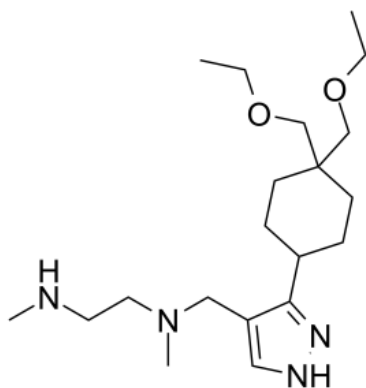
Formula $C_{20}H_{40}Cl_2N_4O_2$ M.Wt 439.46

Solubility DMSO: 250 mg/mL (568.88 mM); Storage Store at 4°C, stored under nitrogen
water : 100mg/ml(227.55 mM)

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



H-Cl H-Cl

Protocol

Cell experiment [1]:

Cell lines HEK293T cells

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

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Preparation Method HEK293T cells were treated with MS023 (6μM) or GSK3368715 (6μM) for 24 hours, and the cGAS methylation level was detected.

Reaction Conditions 6μM; 24h

Applications 6μM GSK3368715 and MS023 blocked cGAS methylation.

Animal experiment [1]:

Animal models Six-weeks old BALB/c female mice

Preparation Method Six-weeks old BALB/c female mice were treated with GSK3368715 (80mg/kg body weight, by gastric gavage), or respective vehicle, one dose daily for 14 days. GSK3368715 was dissolved in ddH₂O. Then, tissues and organs were collected and analyzed the expression of mPD-L1.

Dosage form 80mg/kg/d; 14d; p.o.

Applications mPD-L1 expression was elevated in multiple mouse organs/tissues in mice treated with GSK3368715, like ovary, spleen, colon...

References:

[1] Liu J, Bu X, Chu C, et al. PRMT1 mediated methylation of cGAS suppresses anti-tumor immunity. *Nat Commun.* 2023;14(1):2806.

Background

GSK3368715 dihydrochloride is a potent, reversible type I PRMT inhibitor with anti-tumor effects in human cancer models^[1]. GSK3368715 decrease the PRMT1-specific histone mark H4R3me2a without affecting the methylation of H3R17me2a (by CARM1 and PRMT6) or PABP1 (by CARM1)^[3].

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In vitro, treatment with 6 μ M GSK3368715 for 24 hours inhibited cGAS methylation levels in HEK293T cells^[1]. Treatment with 6 μ M GSK3368715 for 48 hours increased the phosphorylation level of IRF3 in HeLa cells expressing wild-type cGAS^[1]. The growth half-maximal inhibitory concentration (gIC50) values of GSK3368715 against MM cell lines ranged from 0.021 μ M to 14.405 μ M^[2]. GSK3368715 at concentrations ranging from 0.12 to 1 μ M reduced colony growth in a dose-dependent manner when MDA-MB-468 cells were cultured on plastic for 9 days^[3].

In vivo, oral administration of GSK3368715 at 80mg/kg once daily for 14 days resulted in elevated mPD-L1 expression across multiple organs and tissues in six-weeks old BALB/c female mice^[1]. Treatment with GSK3368715 (80mg/kg; orally; once daily for 5 days) in nude mice bearing MDA-MB-468–derived xenografts led to increased global monomethylation, decreased H4R3me2a levels in tumors, and a significant delay in tumor growth^[3].

References:

- [1] Liu J, Bu X, Chu C, et al. PRMT1 mediated methylation of cGAS suppresses anti-tumor immunity. *Nat Commun*. 2023;14(1):2806.
- [2] Hussain T, Awasthi S, Shahid F, Yi SS, Sahni N, Aldaz CM. Therapeutic potential of PRMT1 as a critical survival dependency target in multiple myeloma. *BMC Cancer*. 2025;25(1):1704.
- [3] Suresh S, Huard S, Brisson A, et al. PRMT1 Regulates EGFR and Wnt Signaling Pathways and Is a Promising Target for Combinatorial Treatment of Breast Cancer. *Cancers (Basel)*. 2022;14(2):306.

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