PFI-1

Cat. No.: HY-16586

CAS No.: 1403764-72-6

Molecular Formula: $C_{16}H_{17}N_3O_4S$

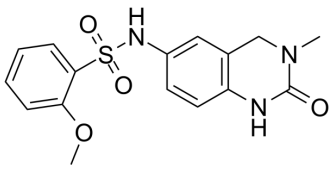
Molecular Weight: 347.39

Target: Epigenetic Reader Domain; Autophagy; Apoptosis

Pathway: Epigenetics; Autophagy; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 33.33 mg/mL (95.94 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8786 mL	14.3930 mL	28.7861 mL
	5 mM	0.5757 mL	2.8786 mL	5.7572 mL
	10 mM	0.2879 mL	1.4393 mL	2.8786 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.20 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.20 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

PFI-1 is a selective BET (bromodomain-containing protein) inhibitor for BRD4 with IC_{50} of 0.22 μM in a cell-free assay.

IC_{50} & Target

IC50: 0.22 μM (BRD4)

In Vitro

PFI-1 has antiproliferative effects on leukemic cell lines and efficiently abrogates their clonogenic growth. Exposure of sensitive cell lines with PFI-1 results in G1 cell-cycle arrest, downregulation of MYC expression, as well as induction of apoptosis and induces differentiation of primary leukemic blasts. Cells exposed to PFI-1 show significant downregulation of Aurora B kinase, thus attenuating phosphorylation of the Aurora substrate H3S10, providing an alternative strategy for the specific inhibition of this well-established oncology target[1]. PFI-1 binds to with cyclic AMP response binding protein with K_d of 49 μM. PFI-1 has an EC_{50} of 1.89 μM for the inhibition of IL6 production from human blood mononuclear cells stimulated

	by LPS[2]. PFI-1 induces dose-dependent reduction of cell viability in T4302 $CD133^+$ cells[3]. PFI-1 inhibits the proliferating of three NET cell lines (Bon-1 derived from a pancreatic NET, and H727 and H720 derived from lung NETs)[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	PFI-1 administrated (1 mg/kg, i.v.) in the rat results in the volume of distribution of 1 L/kg, the plasma clearance of 18 mL/min/kg and half-life of 1 hour. PFI-1 oral dosed (2 mg/kg) in the rat results in the oral bioavailability as low as 32%. PFI-1 administrated (2 mg/kg, s.c.) in the mouse results in a Cmax of 58 ng/mL with a Tmax of 1 h and a half-life of approximately 2 hours[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Proc Natl Acad Sci U S A. 2019 Feb 19;116(8):2961-2966.
- Patent. US20180263995A1.

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REFERENCES

[1]. Picaud S, et al. PFI-1, a Highly Selective Protein Interaction Inhibitor, Targeting BET Bromodomains. Cancer Res. 2013 May 21. [Epub ahead of print]

[2]. Fish PV, et al. Identification of a chemical probe for bromo and extra C-terminal bromodomain inhibition through optimization of a fragment-derived hit. J Med Chem. 2012 Nov 26;55(22):9831-7.

[3]. Cheng Z, et al. Inhibition of BET bromodomain targets genetically diverse glioblastoma. Clin Cancer Res. 2013 Apr 1;19(7):1748-59.

[4]. Kate E Lines, et al. Epigenetic modifiers reduce proliferation of human neuroendocrine tumour cell lines. Endocrine Abstracts (2013) 31 P149

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA