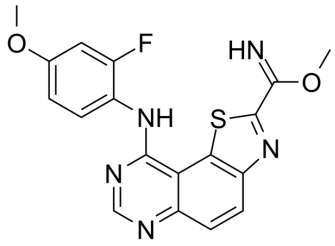
EHT 1610

Cat. No.:	HY-111380
CAS No.:	1425945-60-3
Molecular Formula:	$C_{18}H_{14}FN_5O_2S$
Molecular Weight:	383.4
Target:	DYRK
Pathway:	Protein Tyrosine Kinase/RTK
Storage:	-20°C, protect from light, stored under nitrogen * The compound is unstable in solutions, freshly prepared is recommended.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 5 mg/mL (13.04 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6082 mL	13.0412 mL	26.0824 mL
	5 mM	0.5216 mL	2.6082 mL	5.2165 mL
	10 mM	0.2608 mL	1.3041 mL	2.6082 mL

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

BIOLOGICAL ACTIVITY

Description

EHT 1610 is a potent inhibitor of DYRK, with IC_{50}s of 0.36 nM (DYRK1A), 0.59 nM (DYRK1B), respectively. EHT 1610 exhibits antileukemia effect, regulates cell cycle and induces cell apoptosis[1]-[4].

IC_{50} & Target

IC50: 0.36 nM (DYRK1A), 0.59 nM (DYRK1B)[1]

In Vitro

EHT 1610 induces apoptosis of primary ALL cells that were resistant to cytarabine[2].
EHT 1610 dose-dependently induces apoptosis in B- and T-cell lines and primary human pediatric[2].
EHT 1610 (; 72 h) inhibits DYRK1A, results loss of DYRK1A-mediated FOXO1 and STAT3 signaling, leading to preferential cell death in leukemic B cells[3].
EHT 1610 (2.5-10 μM; 4-5 h) inhibits phosphorylation of FOXO1, STAT3 and cyclin D3, thus regulates late cell-cycle progression, mitochondrial ROS and DNA damage, respectively[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[3]

Cell Line:	MHH-CALL-4 cells

Concentration:	0, 2.5, 5, 10 μM
Incubation Time:	4, 5 hours
Result:	Reduced p-cyclin D3 (Thr283), and p-FOXO1 protein level in a dose-dependent manner.

In Vivo

EHT 1610 (20 mg/kg/d; i.p.; twice a day; 3 weeks) shows antileukemia activity against in leukemic aggressive model in mice [3].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Xenograft models of B-ALL in mice (12-14 weeks old)[3]
Dosage:	20 mg/kg
Administration:	Intraperitoneal injection; twice a day, 5 days on, 2 days off; 3 weeks
Result:	Reduced leukemic burden by approximately 8% and conferred a modest survival advantage.

CUSTOMER VALIDATION

- Gene. 2020 Oct 20;758:144960.

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REFERENCES

[1]. Thompson B J, et al. The Chromosome 21 Kinase DYRK1A Controls Cell Cycle Exit and Survival During Lymphoid Development and Is a Novel Therapeutic Target In Acute Lymphoblastic Leukemia[C]// Ash Meeting & Exposition. 2013. l

[2]. Bhansali RS, et al. DYRK1A regulates B cell acute lymphoblastic leukemia through phosphorylation of FOXO1 and STAT3. J Clin Invest. 2021 Jan 4;131(1):e135937.

[3]. Foucourt A, et al. Design and synthesis of thiazolo[5,4-f]quinazolines as DYRK1A inhibitors, part II. Molecules. 2014 Sep 26;19(10):15411-39.

[4]. Chaikuad A, et al. An Unusual Binding Model of the Methyl 9-Anilinothiazolo[5,4-f] quinazoline-2-carbimidates (EHT 1610 and EHT 5372) Confers High Selectivity for Dual-Specificity Tyrosine Phosphorylation-Regulated Kinases. J Med Chem. 2016 Nov 23;59(22):

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