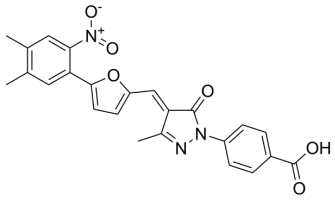
C646

Cat. No.:	HY-13823
CAS No.:	328968-36-1
Molecular Formula:	$C_{24}H_{19}N_3O_6$
Molecular Weight:	445.42
Target:	Histone Acetyltransferase; Autophagy; Epigenetic Reader Domain; Apoptosis
Pathway:	Epigenetics; Autophagy; Apoptosis
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 13.89 mg/mL (31.18 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2451 mL	11.2254 mL	22.4507 mL
5 mM	0.4490 mL	2.2451 mL	4.4901 mL
10 mM	0.2245 mL	1.1225 mL	2.2451 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: 1.67 mg/mL (3.75 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 1.67 mg/mL (3.75 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

C646 is a selective and competitive histone acetyltransferase p300 inhibitor with K_i of 400 nM, and is less potent for other acetyltransferases[1].

IC_{50} & Target

CBP/p300

In Vitro

C646 is a linear competitive inhibitor of p300 versus acetyl-CoA with a K_i?of 400 nM. C646 shows a noncompetitive pattern of p300 inhibition versus the H4-15 peptide substrate. C646 treatment reduces histone H3 and H4 acetylation levels and abrogates TSA-induced acetylation in cells. C646 has a more potent effect on cell growth than Lys-CoA-Tat does[1].
C646 enhances mitotic catastrophe after IR and suppresses phosphorylation of CHK1 after IRin A549 cells[2]. C646 attenuates the increased acetylation of GATA1 and the increased transcriptional activity of GATA1 induced by EDAG[3].

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

In Vivo

Suppression of P300 by c646 (intraperitoneally injected, 30 nmol/g/d for 2 weeks) dramatically reduces the level of blood glucose in db/db mice[4].

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

Animal Model:	Fourteen-week-old male db/db mice and normal m/m mice[4]
Dosage:	30 nmol/g
Administration:	Intraperitoneally injected; daily; 2 weeks
Result:	The db/db mice showed greater body masses and higher levels of fasting blood glucose than the m/m mice.

CUSTOMER VALIDATION

- Cell Res. 2023 Jul 13.
- Immunity. 2024 Feb 13;57(2):364-378.e9.
- Nat Microbiol. 2021 Jul;6(7):932-945.
- Adv Funct Mater. 2023 Dec 21.
- Nucleic Acids Res. 2019 Mar 18;47(5):2455-2471.

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REFERENCES

[1]. Bowers EM, et al. Virtual ligand screening of the p300/CBP histone acetyltransferase: identification of a selective small molecule inhibitor. Chem Biol. 2010 May 28;17(5):471-82.

[2]. Oike T, et al. C646, a selective small molecule inhibitor of histone acetyltransferase p300, radiosensitizes lung cancer cells by enhancing mitotic catastrophe. Radiother Oncol. 2014 May;111(2):222-7.

[3]. Zheng WW, et al. EDAG positively regulates erythroid differentiation and modifies GATA1 acetylation through recruiting p300. Stem Cells. 2014 Aug;32(8):2278-89.

[4]. Zhen Fan, et al. Type 2 diabetes-induced overactivation of P300 contributes to skeletal muscle atrophy by inhibiting autophagic flux. Life Sci. 2020 Aug 10;258:118243.

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