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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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PDE1-IN-7

Cat. No.: HY-161506

CAS No.: 3027833-49-1

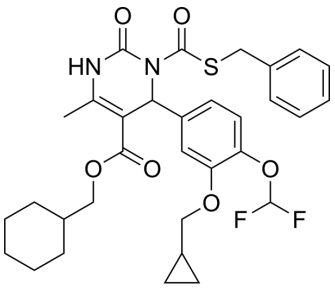
Molecular Formula: $C_{32}H_{36}F_2N_2O_6S$

Molecular Weight: 614.7

Target: Phosphodiesterase (PDE)

Pathway: Metabolic Enzyme/Protease

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

PDE1-IN-7 (Compound 13h) is a selective inhibitor of bPDE1 (IC_{50}= 10 nM). PDE1-IN-7 exhibits significant anti-fibrotic effects in a BDL-induced liver fibrosis rat model. PDE1-IN-7 can be used for research in liver fibrosis[1].

IC_{50} & Target

PDEI

10 nM (IC_{50})

In Vitro

PDE1-IN-7 (2.5-20 μM; 48 h) effectively inhibits TGF-β-induced myofibroblast differentiation and proliferation in LX-2 cells[1].

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

Western Blot Analysis[1]

Cell Line:	human stellate cell LX-2
Concentration:	2.5, 5, 10, 20 μM
Incubation Time:	48 h
Result:	Dose-dependently reduced the elevated expression levels of fibronectin, collagen I, and α-SMA induced by TGF-β in LX-2 cells.

In Vivo

PDE1-IN-7 (i.p.; 2.5 mg/kg; once daily for 21 days) shows significant antifibrotic effects in a rat model of bile duct ligation-induced hepatic fibrosis[1].

Pharmacokinetic Analysis in SD rats[1]

Route	Dose (mg/kg)	$t_{1/2}$ (h)	C_{max} (ng/mL)	AUC_{∞} (h)(ng·/mL)	Cl_{obs} (mL/min/kg)	MRT (h)	V_{ss_obs} (mL/kg)
i.v.	2.5	7.51 ± 0.64	25,006 ± 3082	6317 ± 839	6.56 ± 0.81	1.77 ± 0.07	698 ± 104

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

Animal Model:	BDL-induced hepatic fibrosis rats[1]
Dosage:	2.5 mg/kg
Administration:	i.p.; once daily for 21 days
Result:	Significantly reduced alanine transaminase (ALT), aspartate transaminase (AST) and total bile acids (TBA) levels. Reduced structural damage to liver tissue, decreased fibrotic foci, and lowered collagen deposition levels. Significantly reduced protein expression levels at α-SMA and collagen I levels. Significantly increased cAMP levels.

REFERENCES

[1]. Zhao ZJ, et al. Design, Synthesis, and Evaluation of Dihydropyrimidine Derivatives as Selective PDE1 Inhibitors for the Treatment of Liver Fibrosis. J Med Chem. 2024 Apr 26.

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