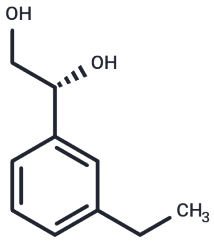
Data Sheet (Cat.No.T210940)

TargetMol

AH001

Chemical Properties



CAS No. : 1456769-95-1

Formula: C10H14O2

Molecular Weight: 166.22

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	AH001 is an orally active compound that binds to a concealed pocket near GDP within RhoA, exhibiting a binding affinity of 73.16 nM. By interacting with GDP, AH001 stabilizes the interaction between RhoA and its endogenous inhibitor, RhoGDIα. This compound reduces nuclear translocation of downstream MRTFA and downregulates fibrosis/hypertrophy-related proteins. AH001 alleviates myocardial remodeling in various heart failure animal models and 3D cardiac tissue models. Its cardioprotective effects are mediated through the RhoA-RhoGDIα axis, effectively inhibiting downstream RhoA activation signals.
Targets(IC50)	Ras
In vitro	AH001 at a concentration of 20 μM reduces the levels of RhoA-GTP in HEK 293F cells. The compound exhibits a concentration-dependent effect on the RhoA GTP/GDP exchange ratio in HEK 293F cells, with an IC50 value of 25.72 nM for concentrations between 7.8-1000 nM. Further, AH001 at 20 μM interacts with RhoA-GDP in HEK 293F cells, stabilizing the RhoA-RhoGDIα complex and thereby restricting RhoA to its GDP-bound state. In the range of 10-40 μM over 24 hours, AH001 inhibits downstream signaling of RhoA activation by decreasing RhoA-GTP levels (20 μM), F-actin formation, and MRTFA nuclear translocation. Additionally, AH001 at 10-40 μM for 24 hours lowers the levels of fibrosis-related proteins, including FN1 and COL3, in fibroblasts, effectively suppressing fibroblast proliferation. This proliferation inhibition leads to a dose-dependent reduction in pathological myocardial cell contraction within a 3D heart tissue model. Moreover, at 20 μM over 24 hours, AH001 exerts antifibrotic effects in a RhoGDIα-dependent manner by stabilizing the RhoA-RhoGDIα complex to inhibit fibroblast proliferation and activation, enhance cardiomyocyte survival, and block profibrotic MRTFA signaling in the 3D cardiac tissue model.
In vivo	AH001 has demonstrated effectiveness in mitigating myocardial remodeling across various heart failure animal models. At concentrations of 15.16-166.64 μM, AH001 reduces myocardial remodeling in 2-day-old zebrafish models induced by isoproterenol (ISO). In 8-week-old mice models, administration of AH001 at 10 mg/kg (i.g., for 4 weeks) alleviates Angiotensin II (Ang II)-induced myocardial remodeling. Dose ranges of 3-30 mg/kg (i.g., for 1 week) reduce myocardial remodeling in 8-week-old LAD models of mice and rats. Moreover, AH001, given at doses of 1-50 mg/kg (i.g., for 12 days), mitigates myocardial remodeling in 8-week-old mice models with doxorubicin (Dox)-induced cardiotoxicity. Additionally, at a dose of 10 mg/kg (i.g., for 1 week), AH001

In vivo	provides cardioprotection in LAD mice by acting through the RhoA-RhoGDIα axis and effectively suppressing downstream RhoA signaling activation.

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	6.0161 mL	30.0806 mL	60.1612 mL
5 mM	1.2032 mL	6.0161 mL	12.0322 mL
10 mM	0.6016 mL	3.0081 mL	6.0161 mL
50 mM	0.1203 mL	0.6016 mL	1.2032 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.