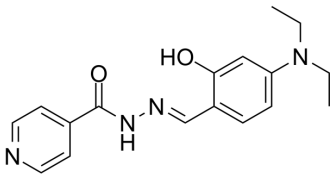
RSVA405

Cat. No.:	HY-103238
CAS No.:	140405-36-3
Molecular Formula:	$C_{17}H_{20}N_4O_2$
Molecular Weight:	312.37
Target:	AMPK; STAT; Autophagy
Pathway:	Epigenetics; PI3K/Akt/mTOR; JAK/STAT Signaling; Stem Cell/Wnt; Autophagy
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (400.17 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.2013 mL	16.0067 mL	32.0133 mL
	5 mM	0.6403 mL	3.2013 mL	6.4027 mL
	10 mM	0.3201 mL	1.6007 mL	3.2013 mL

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

BIOLOGICAL ACTIVITY

Description

RSVA405 is a potent, orally active activator of AMPK, with an EC_{50} of 1 μM. RSVA405 facilitates CaMKKβ-dependent activation of AMPK, inhibits mTOR, and promotes autophagy to increase Aβ degradation. RSVA405 has anti-inflammatory effects through the inhibition of STAT3 function. RSVA405 can also be used for the research of obesity[1][2][3][4].

IC_{50} & Target

AMPK
1 μM (EC50, in cell-based assays)

In Vitro

RSVA405 (0.2-2 μM; 24 h) inhibits adipocyte differentiation[2].
RSVA405 (0.2-2 μM; 24 h) significantly inhibits the expression of peroxisome proliferator-activated receptor (PPAR)-γ, fatty acid synthase (FAS) and fatty acid binding protein 4 (aP2) in 3T3-L1 cells[2].
RSVA405 (1-3 μM; 16 h) inhibits LPS-induced STAT3 activity, intracellular signaling, and cytokine response in activated RAW 264.7 macrophages[3].
RSVA405 (1-3 μM; 24 h) inhibits mTOR, induces autophagy, and facilitates the lysosomal degradation of Aβ, with an EC_{50} of ~1 μM in APP-HEK293 cells[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[2]

Cell Line:	3T3-L1 preadipocytes
Concentration:	0.2, 0.5, 1, 2 μM
Incubation Time:	24 h
Result:	Increased the phosphorylation of AMPK and its substrate acetyl-CoA carboxylase (ACC). Inhibited the accumulation of lipid droplets in a dose-dependent manner, with an IC_{50} of 0.5 μM.

In Vivo

RSVA405 (3 mg/kg; i.p.) attenuates renal injury and protects renal function after ischemia-reperfusion (I/R) in rats[1].
RSVA405 (20-100 mg/kg/d; p.o. for 11 weeks) significantly reduces the body weight gain of mice fed a high-fat diet[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley rats (300-350 g) are induced I/R injury[1]
Dosage:	3 mg/kg
Administration:	I.p. one hour before inducing I/R injury
Result:	Decreased the levels of creatinine and blood urea nitrogen (BUN), by 35.8% and 44.3% in serum, respectively. Decreased the levels of aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) by 33.0% and 59.8% in serum, respectively.

REFERENCES

[1]. Khader A, et, al. Novel resveratrol analogues attenuate renal ischemic injury in rats. J Surg Res. 2015 Feb;193(2):807-15.

[2]. Vingtdeux V, et, al. Small-molecule activators of AMP-activated protein kinase (AMPK), RSVA314 and RSVA405, inhibit adipogenesis. Mol Med. Sep-Oct 2011;17(9-10):1022-30.

[3]. Capiralla H, et, al. Identification of potent small-molecule inhibitors of STAT3 with anti-inflammatory properties in RAW 264.7 macrophages. FEBS J. 2012 Oct;279(20):3791-9.

[4]. Vingtdeux V, et, al. Novel synthetic small-molecule activators of AMPK as enhancers of autophagy and amyloid-β peptide degradation. FASEB J. 2011 Jan;25(1):219-31.

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