COH-SR4

Cat. No.: HY-124822

CAS No.: 73439-19-7

Molecular Formula: $C_{13}H_8Cl_4N_2O$

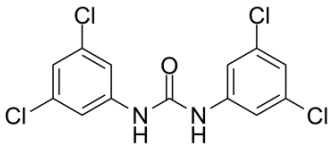
Molecular Weight: 350.03

Target: AMPK

Pathway: Epigenetics; PI3K/Akt/mTOR

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 : 125 mg/mL (357.11 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.8569 mL	14.2845 mL	28.5690 mL
5 mM	0.5714 mL	2.8569 mL	5.7138 mL
10 mM	0.2857 mL	1.4284 mL	2.8569 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: 2.08 mg/mL (5.94 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (5.94 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

COH-SR4 is an AMPK activator. COH-SR4 shows potent anti-proliferative activities against leukemia, melanoma, breast and lung cancers. COH-SR4 inhibits adipocyte differentiation via AMPK activation, and can be used for the research of obesity and related metabolic disorders[1].

IC_{50} & Target

AMPK[1]

In Vitro

COH-SR4 (1-5 μM; 24 hours) results in a dose-dependent increase in the phosphorylation of AMPK and its substrate ACC in 3T3-L1 preadipocytes, as well as in cancer cells such as HL-60, HeLa, MCF-7[1].
COH-SR4 (3-5 μM; 7 days) significantly inhibits 3T3-L1 adipocyte differentiation in a dose-dependent manner[1].
COH-SR4 (1-5 μM; 24 hours) promotes cell G1 cycle arrest[1].

COH-SR4 significantly reduces intracellular lipid accumulation and downregulated the expression of key adipogenesis-related transcription factors and lipogenic proteins[1].

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

Western Blot Analysis[1]

Cell Line:	3T3-L1 preadipocytes, HL-60 cells, HeLa cells, MCF-7 cells
Concentration:	1 μM, 3 μM, 5 μM
Incubation Time:	24 hours
Result:	Indirectly activated AMPK.

Cell Cycle Analysis[1]

Cell Line:	3T3-L1 cells
Concentration:	1 μM, 3 μM, 5 μM
Incubation Time:	24 hours
Result:	Modulated the level of proteins active during S and G2 phases of the cell cycle.

In Vivo

COH-SR4 (5 mg/kg; i.g.; 3x/week; for 6 weeks) reduces body weight and fat mass in high fat diet (HFD) obese mice without affecting food intake[2].

COH-SR4 improves glycemic control and dyslipidemia in HFD obese mice[2].

COH-SR4 decreases adipose tissue hypertrophy and affects circulating adipokine levels in HFD obese mice[2].

COH-SR4 prevents hepatic lipid accumulation and fatty liver in HFD obese mice[2].

COH-SR4 regulates expression of genes involved in lipid and glucose metabolism[2].

COH-SR4 inhibits hepatic lipogenic and gluconeogenic pathways[2].

COH-SR4 indirectly activates AMPK independent of calcium signaling and LKB1 in vitro[2].

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

Animal Model:	Nine-week old male C57BL/6J mice[2]
Dosage:	5 mg/kg
Administration:	Oral gavage, three times a week, for 6 weeks
Result:	Decreased body weight and fat mass in HFD obese mice.

REFERENCES

[1]. James L Figarola, et al. Small□molecule COH-SR4 inhibits adipocyte differentiation via AMPK activation. Int J Mol Med. 2013 May;31(5):1166-76.

[2]. James Lester Figarola, et al. COH-SR4 Reduces Body Weight, Improves Glycemic Control and Prevents Hepatic Steatosis in High Fat Diet-Induced Obese Mice. PLoS One. 2013; 8(12): e83801.

Caution: Product has not been fully validated for medical applications. For research use only.

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