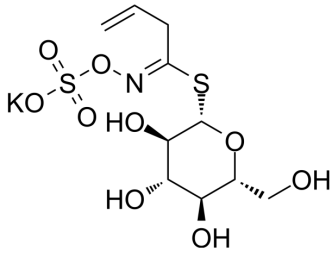
Sinigrin

Cat. No.:	HY-N0404
CAS No.:	3952-98-5
Molecular Formula:	$C_{10}H_{16}KNO_9S_2$
Molecular Weight:	397.46
Target:	p38 MAPK; AMPK
Pathway:	MAPK/ERK Pathway; Epigenetics; PI3K/Akt/mTOR
Storage:	4°C, sealed storage, away from moisture and light * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 12.5 mg/mL (31.45 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.5160 mL	12.5799 mL	25.1598 mL
	5 mM	0.5032 mL	2.5160 mL	5.0320 mL
	10 mM	0.2516 mL	1.2580 mL	2.5160 mL

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

BIOLOGICAL ACTIVITY

Description

Sinigrin is a major glucosinolate present in plants of the Brassicaceae family. Sinigrin inhibits early-stage adipogenesis of 3T3-L1 adipocytes through the AMPK and MAPK signaling pathways. Sinigrin has potent anti-oxidant, anti-tumor and anti-inflammatory effects[1].

In Vitro

Sinigrin (100 μg/mL; 24 H) arrestsd cells in the G0/G1phase of the cell cycle and increased the expression of p21 and p27[1].
Sinigrin (1-100 μg/mL; 8 days) remarkably inhibits the accumulation of lipid droplets and adipogenesis by downregulating the expression of C/EBPα, PPARγ, leptin and aP2. Sinigrin increases the phosphorylation of AMPK, MAPK and acetyl-CoA carboxylase (ACC) in the early stage of adipocyte differentiation, suggesting that sinigrin has anti-adipogenic effects through AMPK, MAPK and ACC activation[1].
Sinigrin (1-100 μg/mL; 8 days) inhibits the production of pro-inflammatory cytokines including TNF-α and IL-6, IL-1β and IL-18[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Cycle Analysis[1]

Cell Line:	3T3-L1 adipocytes

Concentration:	100 μg/mL
Incubation Time:	24 h
Result:	Arrested cells in the G0/G1phase of the cell cycle and increased the expression of p21 and p27.

Western Blot Analysis[1]

Cell Line:	3T3-L1 adipocytes
Concentration:	1, 10, and 100 μg/mL
Incubation Time:	8 days
Result:	Remarkably inhibited the accumulation of lipid droplets and adipogenesis by downregulating the expression of CCAAT-enhancer-binding protein α (C/EBPα), PPARγ, leptin and aP2.

RT-PCR[1]

Cell Line:	3T3-L1 adipocytes
Concentration:	1, 10, and 100 μg/mL
Incubation Time:	8 days
Result:	Inhibited the production of pro-inflammatory cytokines including TNF-α and IL-6, IL-1β and IL-18.

In Vivo

Sinigrin (15-30 mg/kg; Oral administration; for 12 days) exerts protective and therapeutic effects on DSS‐induced colitis, by enhancing the anti-oxidant enzymes and suppressing the intestinal inflammatory cascade of markers by regulating the MAPK pathway[2].

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

Animal Model:	A dextran sulfate sodium (DSS)-induced mouse model[2]
Dosage:	15 mg/kg or 30 mg/kg
Administration:	Oral administration; for 12 days
Result:	Significantly mitigated the DSS-induced body weight loss, attenuated the colon length shrinkage, and improved the disease index score. Successfully abrogated the DSS-induced IL-17 levels and improved the colonic barrier in colon tissues.

CUSTOMER VALIDATION

- Plant Physiol Biochem. 2023 Dec 27:206:108304.

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REFERENCES

[1]. Rama Satya Sri Kotipalli, et al. Sinigrin Attenuates the Dextran Sulfate Sodium-induced Colitis in Mice by Modulating the MAPK Pathway. Inflammation. 2023 Jun;46(3):787-807.

[2]. Lee HW, et al. Inhibitory effect of sinigrin on adipocyte differentiation in 3T3-L1 cells: Involvement of AMPK and MAPK pathways. Biomed Pharmacother. 2018 Jun;102:670-680.

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