Mogrol

Cat. No.: HY-N2312

CAS No.: 88930-15-8

Molecular Formula: $C_{30}H_{52}O_4$

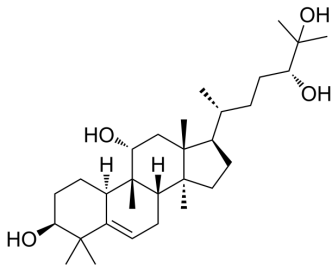
Molecular Weight: 476.73

Target: ERK; STAT

Pathway: MAPK/ERK Pathway; Stem Cell/Wnt; JAK/STAT Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (104.88 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0976 mL	10.4881 mL	20.9762 mL
5 mM	0.4195 mL	2.0976 mL	4.1952 mL
10 mM	0.2098 mL	1.0488 mL	2.0976 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: ≥ 2.5 mg/mL (5.24 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.24 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.24 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Mogrol is a biometabolite of mogrosides, and acts via inhibition of the ERK1/2 and STAT3 pathways, or reducing CREB activation and activating AMPK signaling.

IC_{50} & Target	ERK1	ERK2	STAT3

In Vitro

Mogrol (0-250 μM) significantly and dose- and time-dependently inhibits K562 cell growth and increases the number of

apoptotic cells. Mogrol (0, 10, 100, and 250 μM) induces G1 phase cell cycle arrest in K562 cells. Treatment with mogrol significantly decreases ERK phosphorylation as compared to control cells, whereas total ERK protein is not affected. Mogrol dose-dependently induces growth arrest in G0/G1 phase of the cell cycle. Mogrol significantly and dose-dependently enhances p21 protein expression in K562 cells[1]. Mogrol significantly represses the increase in cellular TG levels induced by differentiation stimuli, and suppresses TG accumulation at micromolar levels, with a statistically significant suppression observed above 10 μM. Mogrol suppresses adipogenesis in 3T3-L1 cells at concentrations that does not affect cell viability. Mogrol suppresses adipogenesis through at least two different mechanisms, increasing AMPK phosphorylation and repressing the activation of CREB[2].

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

PROTOCOL

Cell Assay [1]

Cell viability is determined with a MTT assay. Leukemia cells are plated in triplicate into a 96-well plate. After overnight incubation, they are treated with various concentrations of mogrol (0, 0.1, 1, 10, 100, 200 and 250 μM) for 24 h and 48 h. The percentage of viable cells is calculated as the ratio (A490) of treated cells over control cells. Triplicate experiments are performed.

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

CUSTOMER VALIDATION

- Nat Commun. 2023 Jul 17;14(1):4267.

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REFERENCES

[1]. Liu C, et al. Mogrol represents a novel leukemia therapeutic, via ERK and STAT3 inhibition. Am J Cancer Res. 2015 Mar 15;5(4):1308-18.

[2]. Naoki Harada, et al. Mogrol Derived from Siraitia grosvenorii Mogrosides Suppresses 3T3-L1 Adipocyte Differentiation by Reducing cAMP-Response Element-Binding Protein Phosphorylation and Increasing AMP-Activated Protein Kinase Phosphorylation. PLoS One. 2

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