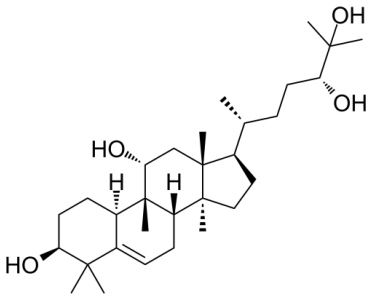
Data Sheet

Product Name:	Mogrol
Cat. No.:	CS-6010
CAS No.:	88930-15-8
Molecular Formula:	$C_{30}H_{52}O_4$
Molecular Weight:	476.73
Target:	ERK; STAT
Pathway:	JAK/STAT Signaling; MAPK/ERK Pathway; Stem Cell/Wnt
Solubility:	DMSO : 50 mg/mL (104.88 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

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. **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].

PROTOCOL (Extracted from published papers and Only for reference)

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.

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

CAIndexNames:

19-Norlanost-5-ene-3,11,24,25-tetrol, 9-methyl-, (3β,9β,10α,11α,24R)-

SMILES:

CC1(C)[C@@H](O)CC[C@@]2([H])[C@]3(C)[C@H](O)C[C@]4(C)[C@@H]([C@H](C)CC[C@@H](O)C(C)(O)C)CC[C@](C)4[C@]3([H])CC=C12

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

Tel: 610-426-3128 Fax: 888-484-5008 E-mail: sales@ChemScene.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA