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Zuschläge

- Mindermengenzuschlag
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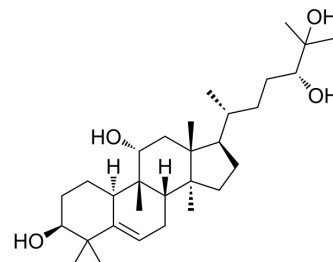
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Mogrol

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:	<div> <div>Powder</div> <div>-20°C 3 years</div> <div>4°C 2 years</div> </div> <div> <div>In solvent</div> <div>-80°C 2 years</div> <div>-20°C 1 year</div> </div>



SOLVENT & SOLUBILITY

In Vitro	DMSO : 50 mg/mL (104.88 mM; Need ultrasonic)				
	H ₂ O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)				
	Preparing Stock Solutions	<div><div>Solvent</div><div>Concentration</div></div>	Mass		
			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 μ M) 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 μ M. 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 μ M) 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

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

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