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Calenduloside E

Cat. No.: HY-N6850

CAS No.: 26020-14-4

Molecular Formula: $C_{36}H_{56}O_9$

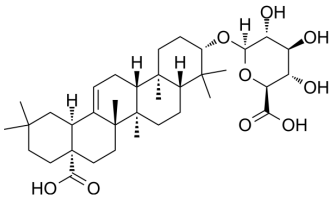
Molecular Weight: 632.82

Target: Apoptosis; Pyroptosis; AMPK; Bcl-2 Family; JAK; STAT; Calcium Channel; Interleukin Related; TNF Receptor; SOD; Reactive Oxygen Species; PPAR

Pathway: Apoptosis; Immunology/Inflammation; Epigenetics; PI3K/Akt/mTOR; JAK/STAT Signaling; Protein Tyrosine Kinase/RTK; Stem Cell/Wnt; Membrane Transporter/Ion Channel; Neuronal Signaling; Metabolic Enzyme/Protease; NF-κB; Cell Cycle/DNA Damage; Vitamin D Related/Nuclear Receptor

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 : 100 mg/mL (158.02 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.5802 mL	7.9011 mL	15.8023 mL
	5 mM	0.3160 mL	1.5802 mL	3.1605 mL
	10 mM	0.1580 mL	0.7901 mL	1.5802 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.95 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.95 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Calenduloside E is a pentacyclic triterpenoid saponin that can be extracted from the bark and roots of Aralia ovata, and has anti-inflammatory and anti-apoptotic activities. Calenduloside E alleviates atherosclerosis by regulating macrophage polarization, improves mitochondrial function by regulating the AMPK-SIRT3 pathway, and alleviates acute liver injury. In addition, Calenduloside E promotes the interaction between L-type calcium channels and Bcl-2 related apoptosis genes, inhibits calcium overload, and alleviates myocardial ischemia/reperfusion injury. Calenduloside E also improves non-alcoholic fatty liver disease by regulating heat shock-dependent pathways, and inhibits ROS mediated JAK1-STAT3

pathways to reduce cellular inflammatory responses[1][2][3][4][5][6].

In Vitro

Calenduloside E (1.25 μg/mL; 24 h) inhibits glycolysis-mediated M1 macrophage polarization[2].
Calenduloside E (1 μM; 2 h) alleviates LPS (HY-D1056)/D-galn-induced AML12 and LX2 cell damage and AMPK-SIRT3 signaling pathway protein expression[3].
Calenduloside E (0-16 μM; 24 h) inhibits inflammasome activation and pyroptosis in AML-12 cells stimulated by lipid mixture [5].

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

Western Blot Analysis[2]

Cell Line:	ox-LDL-induced M1 macrophages
Concentration:	1.25 μg/mL
Incubation Time:	24 h
Result:	Reduced the levels of IL-1 β, IL-6, PFKFB3, GLUT1 and LDHA.

Western Blot Analysis[3]

Cell Line:	LPS/ d-galn-induced AML12 and LX2 cells
Concentration:	1 μM
Incubation Time:	2 h
Result:	Reduced ROS and JC-1 levels, as well as cell apoptosis.

Immunofluorescence[5]

Cell Line:	AML-12 cells stimulated with lipid mixture
Concentration:	2, 4, 8 and 16 μM
Incubation Time:	24 h
Result:	Inhibited the increased expression of NLRP3, Caspase-1 p20, and IL-1β.

In Vivo

Calenduloside E (11 mg/kg; i.g.; once daily for 16 weeks) reduces atherosclerotic plaque size, enhanced plaque stability, and reduced inflammatory responses in ApoE-/- mice[2].
Calenduloside E (15 and 30 mg/kg; i.g.; once daily for 7 days) inhibits hepatocyte apoptosis and reduced oxidative stress and immune inflammation in mice with acute liver injury induced by LPS (HY-D1056) and D-GalN[3].
Calenduloside E (7.5-30 mg/kg; i.g.; once daily for 3 days) restores sarcomere contraction and calcium transients in adult rat ventricular myocytes (ARVMs)[4].
Calenduloside E (5 and 10 mg/kg; i.g.; once daily for 4 weeks) improves liver injury, lipid accumulation, and profibrotic phenotypes in nonalcoholic fatty liver disease model mice and inhibits inflammasome activation and pyroptosis in the liver of mice[5].

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

Animal Model:	HFD-fed ApoE-/- mice[2]
Dosage:	11 mg/kg
Administration:	i.g.、Once a day for 16 weeks
Result:	Reduced the levels of IL-1β, IL-6, TNF-α, and monocyte chemoattractant protein-1 (MCP-1)

	in the serum of ApoE-/- mice.

Animal Model:	LPS (HY-D1056)/dGalN-induced acute liver injury in mice[3]
Dosage:	15 and 30 mg/kg
Administration:	i.g.; 1 time per day for 7 consecutive days
Result:	Improved hepatocyte infiltration and reduced hepatocyte necrosis and shrinkage. Reduced hepatocyte ROS levels and serum MDA levels, and increased GSH-Px and SOD levels.

Animal Model:	Rat Model of MI/R Injury[4]
Dosage:	7.5, 15 and 30 mg/kg
Administration:	i.g.; 1 time per day for 3 consecutive days
Result:	Myocardial infarction area/risk area decreased to 53%, 38% and 43% respectively. Restored the expression of calcium-regulating proteins, including calcium transporters (SERCA, a1C, RyR2, and NCX) to normal levels.

Animal Model:	Establishment of NAFLD model in apoE-/- mice by western diet[5]
Dosage:	5 and 10 mg/kg
Administration:	i.g.; 1 time per day for 4 weeks
Result:	Reduced the expression of TNF-α, MCP-1, CCL2, Ly6c and cd68 in the liver. Reversed the upregulation of lipogenic genes FASN, Srebpf, ACC and PPARγ and lipid uptake gene cd36. Reduced the expression of NLRP3, pNLRC4, NLRC4, cleaved GSDMD, cleaved Caspase1 and IL-1β.

CUSTOMER VALIDATION

- J Mol Histol. 2022 Jul 12.

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REFERENCES

[1]. Lanfang Li, et al. "Calenduloside e modulates macrophage polarization via KLF2-regulated glycolysis, contributing to attenuates atherosclerosis." International Immunopharmacology 117 (2023): 109730.

[2]. Pengli Guo, et al. "Isolation of Calenduloside E from achyranthes bidentata blume and its effects on LPS/D-GalN-induced acute liver injury in mice by regulating the AMPK-SIRT3 signaling pathway." Phytomedicine 125 (2024): 155353.

[3]. Ruiying Wang, et al. "Calenduloside E suppresses calcium overload by promoting the interaction between L-type calcium channels and Bcl2-associated athanogene 3 to alleviate myocardial ischemia/reperfusion injury." Journal of Advanced Research 34 (2021): 173-186.

[4]. Yifei Le, et al. "Calenduloside E ameliorates non-alcoholic fatty liver disease via modulating a pyroptosis-dependent pathway." Journal of Ethnopharmacology 319 (2024): 117239.

[5]. Min Wang, et al. "Calenduloside E ameliorates myocardial ischemia-reperfusion injury through regulation of AMPK and mitochondrial OPA1." Oxidative Medicine and Cellular Longevity 2020.1 (2020): 2415269.

[6]. Tian Y, et al. The clickable activity-based probe of anti-apoptotic calenduloside E. Pharm Biol. 2019 Dec;57(1):133-139.

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