Wedelolactone

Cat. No.: HY-N0551

CAS No.: 524-12-9

Molecular Formula: $C_{16}H_{10}O_7$

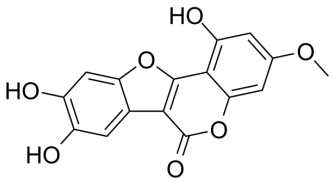
Molecular Weight: 314.25

Target: Caspase; Lipoxygenase; Apoptosis

Pathway: Apoptosis; Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (397.77 mM; Need ultrasonic)

Preparing Stock Solutions	Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
	1 mM	3.1822 mL	15.9109 mL	31.8218 mL
	5 mM	0.6364 mL	3.1822 mL	6.3644 mL
	10 mM	0.3182 mL	1.5911 mL	3.1822 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: ≥ 2.08 mg/mL (6.62 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (6.62 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Wedelolactone suppresses LPS-induced caspase-11 expression by directly inhibits the IKK Complex. Wedelolactone also inhibits 5-lipoxygenase (5-Lox) with an IC_{50} of 2.5 μM. Wedelolactone induces caspase-dependent apoptosis in prostate cancer cells via downregulation of PKCε without inhibiting Akt. Wedelolactone can extract from Wedelia chinensis and Eclipta alba, and it can be used for the research of cancer[1][2][3].

IC_{50} & Target

Caspase-11	5-LOX 2.5 μM (IC_{50})	Apoptosis

In Vitro

Wedelolactone (0-5 μg/mL; 0-21 d) enhances bone marrow mesenchymal stem cells (BMSC) differentiation towards osteoblasts[3].
Wedelolactone (0-6 μg/mL; 0-9 d) inhibits GSK3β activity and increases β-catenin and runx2 nuclear accumulation in BMSC,

and inhibits the effect of RANKL[3].
Wedelolactone (0-5 μg/ml; 60 min) inhibits GSK3β activity and proves GSK3β is the target of wedelolactone[3].
Wedelolactone (0-5 μg/ml; 6 d) inhibits c-src, c-fos and cathepsin k expression level[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Differentiation Assay[3]

Cell Line:	Mouse BMSC
Concentration:	0-5 μg/mL
Incubation Time:	0, 6, 9, 12 and 21 days
Result:	Increased Mouse BMSC into osteoblastic cells and dose-dependently increased the activity of alkaline phosphatase at incubation for 9 days.

Western Blot Analysis[3]

Cell Line:	Mouse BMSC and RAW264.7 cells
Concentration:	0-5 μg/mL
Incubation Time:	0-9 days
Result:	Decreased GSK3β expression level and up-regulated GSK3β phosphorylation, nuclear accumulation of β-catenin and runx2 in BMSC. Inhibited RANKL-induced phosphorylation of NF-κB/p65 and the expression level of c-fos and c-Src.

Cell Viability Assay[3]

Cell Line:	Mouse BMSC
Concentration:	0.1, 1.25, 2.5, 5 μg/ml
Incubation Time:	60 min
Result:	Inhibited GSK3β activity with an IC_{50} of 21.7 μM weeker than staurosporin which is a GSK3 β inhibitor and proved GSK3β is a target.

RT-PCR[3]

Cell Line:	RAW264.7 cells
Concentration:	0, 0.6, 1.25, 2.5 and 5 μg/mL
Incubation Time:	6 days
Result:	Inhibited the expression of osteoclast differentiation related marker genes c-src, c-fos and cathepsin in RAW264.7 cells.

In Vivo

Wedelolactone (10 mg/kg; i.p. every 2 days for 4 weeks) decreases bone volumn and trabecular number at the femur after ovarietomy, and prevents the VOX-induced bone loss[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Ovariectomized 9-week-old mice[3]
Dosage:	10 mg/kg

Administration:	Intraperitoneal injection; 10 mg/kg every 2 days; for 4 weeks
Result:	Inhibited osteoclast activity and stimulated osteoblast differentiation to achieved osteoprotective effect.

CUSTOMER VALIDATION

- Cell Rep. 2021 Sep 21;36(12):109750.
- Acta Pharm Sin B. 15 October 2021.
- Eur J Pharmacol. 2022 Feb 16;920:174830.
- Front Neurol. 2021; 12: 621555.
- Drug Dev Res. 2020 Nov;81(7):859-866.

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REFERENCES

[1]. Kobori M, et al. Wedelolactone suppresses LPS-induced caspase-11 expression by directly inhibiting the IKK complex. Cell Death Differ. 2004 Jan;11(1):123-30.

[2]. Sarveswaran S, et al. Wedelolactone, a medicinal plant-derived coumestan, induces caspase-dependent apoptosis in prostate cancer cells via downregulation of PKCε without inhibiting Akt. Int J Oncol. 2012 Dec;41(6):2191-9.

[3]. Liu YQ, et al. Wedelolactone enhances osteoblastogenesis by regulating Wnt/β-catenin signaling pathway but suppresses osteoclastogenesis by NF-κB/c-fos/NFATc1 pathway. Sci Rep. 2016 Aug 25;6:32260.

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