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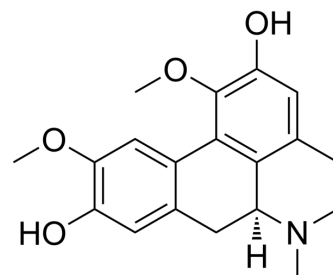
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Boldine

Cat. No.:	HY-N6973
CAS No.:	476-70-0
Molecular Formula:	C ₁₉ H ₂₁ NO ₄
Molecular Weight:	327.37
Target:	RANKL/RANK; Apoptosis
Pathway:	NF-κB; Apoptosis
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 : 100 mg/mL (305.46 mM; Need ultrasonic)				
	Preparing Stock Solutions	Solvent Concentration Mass	1 mg	5 mg	10 mg
		1 mM	3.0546 mL	15.2732 mL	30.5465 mL
		5 mM	0.6109 mL	3.0546 mL	6.1093 mL
		10 mM	0.3055 mL	1.5273 mL	3.0546 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 (7.64 mM); Clear solution				
	2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (7.64 mM); Clear solution				
	3. Add each solvent one by one: 10% DMSO >> 90% corn oil				
	Solubility: ≥ 2.5 mg/mL (7.64 mM); Clear solution				

BIOLOGICAL ACTIVITY

Description	Boldine is an apomorphine isoquinoline alkaloid extracted from the root of the pheasant pepper (<i>Litsea cubeba</i>). Boldine is an oral effective antioxidant, anti-inflammatory, antitumor agent, and can inhibit osteoclast formation. Boldine induces apoptosis of human bladder cancer cells by regulating ERK, AKT and GSK-3β. Boldine ameliorates bone destruction by down-regulating the OPG/RANKL/RANK signaling pathway. It can be used in rheumatoid arthritis research ^{[1][2][3]} .
IC ₅₀ & Target	OPG/RANKL/RANK ^[1]

In Vitro

Boldine (10-500 μM , 24 h) inhibits the growth of bladder cancer cells in T24 patients[2].

Boldine (200-500 μM , 24 h) induces cycle arrest and apoptosis of bladder cancer cells in T24 by regulating ERK, AKT and GSK-3 β [2].

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

Cell Viability Assay^[2]

Cell Line:	T24 and RT4
Concentration:	10, 50, 100, 200, 300, 400, 500 μM
Incubation Time:	24 h
Result:	Inhibited cell growth.

Apoptosis Analysis^[2]

Cell Line:	T24
Concentration:	200, 400 μM
Incubation Time:	24 h
Result:	Caused a G2/M-phase arrest and a marked increase in cell death

Western Blot Analysis^[2]

Cell Line:	T24
Concentration:	200, 400 μM
Incubation Time:	1,, 3, 24 h
Result:	Decreased the level of p-ERK after 24 hours with 200 μM and after 3 and 24 hours with 400 μM . The anti-p-cdc2 antibody did not demonstrate any difference in the phosphorylation status of cdc2. Decreased the phosphorylation of GSK-3 β after 24 hours at a concentration of 400 μM .

In Vivo

Boldine (50 mg/kg, p.o, for 7 days) attenuates Dextran Sulfate Sodium (DSS) -induced colitis in mice via p65-NF- κB and STAT3 signaling pathways^[3].

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

Animal Model:	DSS-induced mice model ^[3]
Dosage:	50 mg/kg
Administration:	p.o.
Result:	Reduced the expression of CD 68+ and activity of myeloperoxidase (MPO) Attenuated Pro-Inflammatory Cytokines and the level of malondialdehyde (MDA) Increased the Activities of Enzymic Antioxidants and Inhibited p65-NF- κB and STAT3 Activity.

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- Phytother Res. 2024 Apr 22.

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REFERENCES

- [1]. Gerhardt D, et al. Boldine induces cell cycle arrest and apoptosis in T24 human bladder cancer cell line via regulation of ERK, AKT, and GSK-3 β . Urol Oncol. 2014 Jan;32(1):36.e1-9.
- [2]. Pandurangan AK, et al. Boldine suppresses dextran sulfate sodium-induced mouse experimental colitis: NF- κ B and IL-6/STAT3 as potential targets. Biofactors. 2016 May;42(3):247-58.
- [3]. Zhao H, et al. Boldine isolated from Litsea cubeba inhibits bone resorption by suppressing the osteoclast differentiation in collagen-induced arthritis. Int Immunopharmacol. 2017 Oct;51:114-123.
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Caution: Product has not been fully validated for medical applications. For research use only.

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