Visnagin

Cat. No.:	HY-N1082		
CAS No.:	82-57-5		
Molecular Formula:	C ₁₃ H ₁₀ O ₄		
Molecular Weight:	230.22		
Target:	Others		
Pathway:	Others		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month

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SOLVENT & SOLUBILITY

	e,	(ultrasonic;warming;heat to 60°C) (in rasonic;warming;heat to 60°C) (insol	,				
	Solvent Mass Concentration	1 mg	5 mg	10 mg			
	Preparing Stock Solutions	1 mM	4.3437 mL	21.7184 mL	43.4367 mL		
		5 mM	0.8687 mL	4.3437 mL	8.6873 mL		
		10 mM	0.4344 mL	2.1718 mL	4.3437 mL		
	Please refer to the so	Please refer to the solubility information to select the appropriate solvent.					
In Vivo		one by one: 10% DMSO >> 40% PEC ng/mL (10.86 mM); Clear solution	G300 >> 5% Tween-8	0 >> 45% saline			

BIOLOGICAL ACTIVITY				
Description	Visnagin, an antioxidant furanocoumarin derivative, possess anti-inflammatory and analgesic properties. Visnagin has substantial potential to prevent Cerulein induced acute pancreatitis (AP). Visnagin possess promising vasodilator effects in vascular smooth muscles ^{[1][2]} .			
In Vitro	Visnagin (10 μM; for 4, 8, 16, 24 h) induces CYP1A1 transcription in HepG2 cells ^[1] . Visnagin (10 μM; for 16 h) elevates CYP1B1 gene expression in an aryl hydrocarbon receptor (AHR)-dependent manner, whereas MNF (3'-methoxy-4'-nitroflavone; 20 μM; pre-treated for 1 h) successfully counteracted this induction. Visnagin also enhances PAI-2 transcription in an AHR-dependent manner ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.			

Product Data Sheet

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In Vivo

Visnagin (10, 30, 60 mg/kg; ip; for 7 days) is effective in reducing plasma amylase and lipase levels and reduces Cerulein (50 μ g/kg, six, hourly i.p. injections) induced oxidative stress in male Swiss albino mice (age: 6-8 weeks, weighing 20-25 g)^[1]. Visnagin dose dependently decreases the expression of IL-1β, IL-6, TNF-α and IL-17. It attenuates the levels of nuclear p65-NFκB. Visnagin improves the antioxidant defence by improving Nrf2 expression and halts pancreatic inflammation by suppressing NFκB and nitrotyrosine expression in the acinar cells^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Radim Vrzal, et al. Khellin and visnagin differentially modulate AHR signaling and downstream CYP1A activity in human liver cells. PLoS One. 2013 Sep 19;8(9):e74917.

[2]. Lakshmi Priya Pasari, et al. Visnagin attenuates acute pancreatitis via Nrf2/NFkB pathway and abrogates associated multiple organ dysfunction. Biomed Pharmacother. 2019 Apr;112:108629.

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

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