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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
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3,5,7,3′,4′-Pentamethoxyflavone

Cat. No.: HY-N7690

CAS No.: 1247-97-8

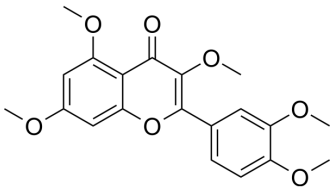
Molecular Formula: $C_{20}H_{20}O_7$

Molecular Weight: 372.37

Target: Calcium Channel

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (67.14 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6855 mL	13.4275 mL	26.8550 mL
	5 mM	0.5371 mL	2.6855 mL	5.3710 mL
	10 mM	0.2686 mL	1.3428 mL	2.6855 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 (6.71 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.71 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

3,5,7,3’,4’-Pentamethoxyflavone is a Ca^{2+} channel inhibitor. 3,5,7,3’,4’-Pentamethoxyflavone can protect DNA from oxidative damage. 3,5,7,3’,4’-Pentamethoxyflavone can induce relaxation of the human corpus cavernosum through calcium mobilization-related mechanisms. 3,5,7,3’,4’-Pentamethoxyflavone can promote the expression of eNOS and cystathionine gamma lyase CSE proteins in middle-aged male rats and regulate vascular function. 3,5,7,3’,4’-Pentamethoxyflavone can be used in research related to diabetes and cardiovascular diseases[1][2][4].

In Vitro

3,5,7,3’,4’-Pentamethoxyflavone (25-100 μg/mL) can protect pUC19 plasmid DNA from oxidative damage[1].
3,5,7,3’,4’-Pentamethoxyflavone (25-100 μg/mL) can reduce oxidative DNA damage in peripheral blood mononuclear cells of mice[1].
3,5,7,3’,4’-Pentamethoxyflavone induces relaxation of the human corpus cavernosum through mechanisms related to voltage dependent Ca^{2+}channels and calcium mobilization[2].

3,5,7,3’,4’-Pentamethoxyflavone (100 μM, 48 h) can increase the expression of tight junction (TJ) protein in human intestinal Caco-2 cells and exert an in vitro intestinal barrier promoting effect[3].

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

Western Blot Analysis[3].

Cell Line:	Human intestinal Caco-2 cells
Concentration:	100 μM
Incubation Time:	48 h
Result:	Increased the cytoskeletal association of TJ proteins, such as ZO1, ZO2, occludin,claudin-1, claudin-3, and claudin-4, in the cells.

In Vivo

3,5,7,3’,4’-Pentamethoxyflavone (22 mg/kg; twice daily; 6 weeks; p.o.) can promote the expression of eNOS and CSE proteins in middle-aged male rats, and regulate vascular function by antagonizing the contraction of phenylephrine and enhancing the vasodilation of acetylcholine[4].

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

Animal Model:	Middle-aged (12–14 month old) Wistar male rats[4].
Dosage:	22 mg/kg
Administration:	Oral gavage (p.o.); twice daily; 6 weeks
Result:	Caused a decrease in plasma glucose and an increase in plasma HDL cholesterol levels of the rats.

REFERENCES

[1]. Jakhar R, et al. 3,5,7,3',4'-pentamethoxyflavone, a quercetin derivative protects DNA from oxidative challenges: potential mechanism of action. J Photochem Photobiol B. 2014 Feb 5;131:96-103.

[2]. Jansakul C, et al. Relaxant mechanisms of 3, 5, 7, 3', 4'-pentamethoxyflavone on isolated human cavernosum. Eur J Pharmacol. 2012 Sep 15;691(1-3):235-44.

[3]. Mayangsari Y, et al. 3,5,7,3',4'-Pentamethoxyflavone Enhances the Barrier Function through Transcriptional Regulation of the Tight Junction in Human Intestinal Caco-2 Cells. J Agric Food Chem. 2021 Sep 8;69(35):10174-10183.

[4]. Yorsin S, et al. Increased vascular eNOS and cystathionine-γ-lyase protein after 6 weeks oral administration of 3, 5, 7, 3', 4'-pentamethoxyflavone to middle-aged male rats. Naunyn Schmiedebergs Arch Pharmacol. 2016 Nov;389(11):1183-1194.

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