Karanjin

Cat. No.: HY-N2534

CAS No.: 521-88-0

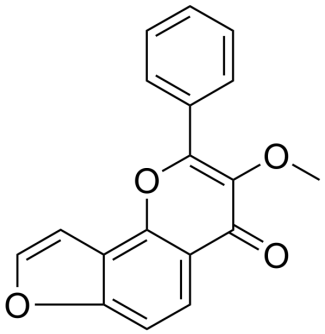
Molecular Formula: $C_{18}H_{12}O_4$

Molecular Weight: 292.29

Target: AMPK; Apoptosis; Cytochrome P450; Phosphatase; NF-κB

Pathway: Epigenetics; PI3K/Akt/mTOR; Apoptosis; Metabolic Enzyme/Protease; NF-κB

Storage: 4°C, sealed storage, away from moisture and light
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (85.53 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.4213 mL	17.1063 mL	34.2126 mL
	5 mM	0.6843 mL	3.4213 mL	6.8425 mL
	10 mM	0.3421 mL	1.7106 mL	3.4213 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (8.55 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Karanjin is an orally active furanoflavonoid which can be isolated from several Leguminosae. Karanjin exhibits evident anti-diabetic, anti-cancer, anti-inflammatory, antioxidant, anticolitis, anti-ulcer, anti-Alzheimer properties and multiple insect repellent/insecticidal, acaricide properties, suggesting the potential of Karanjin to be applied to relevant research[1].

In Vitro

Karanjin (10-25 μM, 16 h) enhances glucose uptake in L6-GLUT4 myc cells through stimulates translocation of GLUT4 to plasma membrane-associated with activation of AMPK pathway and inhibits the activity of PTPase in a dose-dependent manner. Shows no significant effect on cell viability[1].

Karanjin (4-8 μM, 72 h) blocks the cell cycle of A549 and HL-60 markedly as well as induces significant cell apoptosis[1].

Karanjin (80, 160 μM, 30 min) decreases ROS level by inhibition of I-κB degradation resulting in restriction of NF-κB nuclear translocation in Hela cells[1].

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

Cell Cycle Analysis[1]

Cell Line:	A549, HL-60
Concentration:	4, 6, 8 μM for A549, 2, 4, 6 μM for HL-60
Incubation Time:	72 h
Result:	Blocked the cell cycle markedly at G2/M phase of A549 (>60% for 8 μM) and HL-60 (>40% for 6 μM)

In Vivo

Karanjin (50, 100 mg/kg, p.o., 6 h) reduces the blood glucose level of Streptozotocin (HY-13753)-induced diabetic rats[1].
Karanjin (20 mg/kg/d, p.o., 21 d) reduces joint injury and cartilage damage along with edema and erythema in the AIA model rats[1].
Karanjin (10, 20 mg/kg/d, p.o., 14 d) significantly decreases serum ACP, ALP and TNFα levels in the AIA model rats [1].
Karanjin (25 or 50 mg/kg/d, p.o. or i.g., 8 d) improved learning and memory in Diazepam-induced amnesia mice[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Streptozotocin-induced diabetic rats[1]
Dosage:	50, 100 mg/kg
Administration:	Oral gavage (p.o.), wait for 6h
Result:	Lowered the blood glucose level by 11.7% and 20.7% at 50 and 100 mg/kg gavage.

Animal Model:	Adjuvant induced arthritis model (AIA) Wistar strain male albino rats[1]
Dosage:	10, 20 mg/kg/d
Administration:	Oral gavage (p.o.), 14 d for serum assay, 21 d for histological examination
Result:	Reduced articular cartilage damage, cellular infiltration in the articular cartilage and spongy bone damage significantly. Decreased serum acid phosphatase, alkaline phosphatase levels and TNFα level markedly.

Animal Model:	Diazepam-induced male swiss albino mice [1]
Dosage:	25, 50 mg/kg/d
Administration:	Oral gavage (p.o.) or Intraperitoneal injection (i.p.) for 8 d
Result:	Decreased the transfer latency on all the observation days, significantly reversed diazepam-induced amnesia, indicating improved learning and memory in treated mice.

REFERENCES

[1]. Singh A, et al. Karanjin. Phytochemistry. 2021 183:112641.

[2]. Guo JR, et al. Effects of karanjin on cell cycle arrest and apoptosis in human A549, HepG2 and HL-60 cancercells. Biol Res. 2015 Jul 26;48:40.

[3]. Jaiswal N, et al.Karanjin from Pongamia pinnata induces GLUT4 translocation in skeletal muscle cells in a phosphatidylinositol-3-kinase-independent manner. Eur J Pharmacol. 2011 Nov 16;670(1):22-8.

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

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