Rotenone

Cat. No.: HY-B1756

CAS No.: 83-79-4

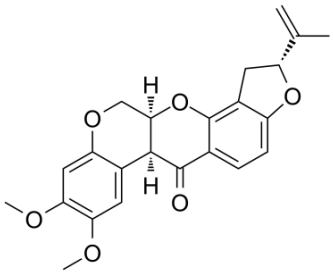
Molecular Formula: $C_{23}H_{22}O_6$

Molecular Weight: 394.42

Target: Mitochondrial Metabolism; Autophagy; Apoptosis

Pathway: Metabolic Enzyme/Protease; Autophagy; Apoptosis

Storage: 4°C, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (126.77 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5354 mL	12.6768 mL	25.3537 mL
5 mM	0.5071 mL	2.5354 mL	5.0707 mL
10 mM	0.2535 mL	1.2677 mL	2.5354 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 (6.34 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.34 mM); Clear solution
3. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (6.34 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Rotenone is an mitochondrial electron transport chain complex I inhibitor. Rotenone induces apoptosis through enhancing mitochondrial reactive oxygen species production.

In Vitro

Mitogen Activated Protein Kinase (MAPK), Toll-like receptor, Wnt, and Ras signaling pathways are intensively involved in the effect of rotenone on the ENS[2]. Rotenone-induced cell death is reduced by MCE treatment as measured by decline in the levels of pro-apoptotic proteins. Moreover, MCE treatment significantly augments the levels of anti-apoptotic Bcl2 and blocks the release of cytochrome c, thereby alleviating the rotenone-induced dopaminergic neuronal loss, as evidenced by tyrosine hydroxylase (TH) immunostaining in the striatum[3].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Rotenone causes a significant increase in the excitatory amino acid neurotransmitters; glutamate and aspartate together with a significant decrease in the inhibitory amino acids, GABA, glycine and taurine are observed in the cerebellum of rat model of PD[1]. Rotenone (1.5, 2, or 2.5 mg/kg) causes a dose-dependent increase in α-synuclein in the substantia nigra. Furthermore, at 2 and 2.5 mg/kg, rotenone causes a significant decrease in the number of tyrosine hydroxylase-immunoreactive neurons in the substantia nigra, and dopamine in the striatum in rats[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Environ Pollut. 2019 Mar 3;248:857-864.
- J Cell Physiol. 2019 Aug;234(10):18849-18857.
- Oxid Med Cell Longev. 2019 Dec 2;2019:6170936.
- Aging. 2019 Aug 19;11(16):6490-6502.
- Sci Rep. 2017 Aug 29;7(1):9873.

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REFERENCES

[1]. Khadrawy YA, et al. Cerebellar neurochemical and histopathological changes in rat model of Parkinson's disease induced by intrastriatal injection of rotenone. Gen Physiol Biophys. 2016 Nov 30.

[2]. Guan Q, et al. RNA-Seq Expression Analysis of Enteric Neuron Cells with Rotenone Treatment and Prediction of Regulated Pathways. Neurochem Res. 2016 Nov 30.

[3]. Kishore Kumar SN, et al. Morinda citrifolia mitigates rotenone-induced striatal neuronal loss in male Sprague-Dawley rats by preventing mitochondrial pathway of intrinsic apoptosis. Redox Rep. 2016 Nov 24:1-12.

[4]. Zhang ZN, et al. Subcutaneous rotenone rat model of Parkinson's disease: dose exploration study. Brain Res. 2016 Nov 19. pii: S0006-8993(16)30776-4.

[5]. Li N, et al. Mitochondrial complex I inhibitor rotenone induces apoptosis through enhancing mitochondrial reactive oxygen species production. J Biol Chem. 2003 Mar 7;278(10):8516-25.

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