Dexanabinol

Cat. No.: HY-106387

CAS No.: 112924-45-5

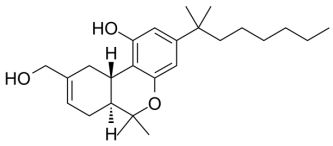
Molecular Formula: $C_{25}H_{38}O_3$

Molecular Weight: 386.57

Target: Reactive Oxygen Species; TNF Receptor; iGluR

Pathway: Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Apoptosis; Membrane Transporter/Ion Channel; Neuronal Signaling

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



BIOLOGICAL ACTIVITY

Description

Dexanabinol (HU-211) is a synthetic cannabinoid derivative (non-psychoactive cannabinoid). Dexanabinol effectively inhibits the production of TNF-α and NO and has NMDA antagonistic activity. Dexanabinol is also a neuroprotectant that effectively scavenges peroxygen free radicals and protects neurons from the toxic effects of ROS. Dexanabinol can be used in studies of traumatic brain injury, septic shock, stroke and cancer[1][2][3].

In Vitro

Dexanabinol (HU-211) (10.4, 20.8 μM; 6 h) markedly suppresses TNF-α production in macrophages and RAW 264.7 cells[1].
Dexanabinol (2.6, 13 μM; 2 h) completely inhibits LPS-induced TNF-α gene expression in rat macrophage NR 8383 cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	RAW 264.7 cells, macrophages
Concentration:	10.4, 20.8 μM
Incubation Time:	6 h
Result:	Led to 92% and 59% suppression of TNF-α when at concentration of 20.8 and 10.4 μM in macrophages, respectively. Suppressed TNF-α production by 84% and 41% at concentration of 20.8 and 10.4 μM in RAW 264.7 cells, respectively.

Cell Viability Assay[1]

Cell Line:	Rat macrophage NR 8383 cells (LPS-induced)
Concentration:	2.6, 13 μM
Incubation Time:	2 h
Result:	Completely inhibited LPS-induced TNF-α gene expression.

Cell Viability Assay[1]

Cell Line:	Rat macrophage NR 8383 cells (LPS-induced)

Concentration:	2.6, 12.9 mM
Incubation Time:	18 h
Result:	Suppressed LPS-induced nitrite accumulation by 86 and 74%, respectively, and under basal conditions, by 57 and 29%, respectively.

In Vivo

Dexanabinol (HU-211) (10 mg/kg; i.p.; single) protects mice in a model of endotoxic shock[1].
Dexanabinol (HU-211) (5 mg/kg; i.v.; single) inhibits TNF-α production in the brain following CHI (traumatic brain injury model)[2].
Dexanabinol (5 mg/kg; i.v.; single) protects the brain against disruption of the BBB during the first 24 h post injury[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	BALB/c male mice (LPS-stimulated; endotoxic shock model)[1].
Dosage:	10 mg/kg
Administration:	Intraperitoneal injection; single (30 min before LPS).
Result:	Reduced mortality to 9 and 67% at 24 and 48 h, respectively.

Animal Model:	BALB/c mice (traumatic brain injury model)[2].
Dosage:	5 mg/kg
Administration:	Intravenous injection; single.
Result:	Inhibited the surge in rat brain TNF-α bioactivity at 4 h. Reduced brain edema formation after CHI.

REFERENCES

[1]. Gallily R, et al. Protection against septic shock and suppression of tumor necrosis factor alpha and nitric oxide production by dexanabinol (HU-211), a nonpsychotropic cannabinoid. J Pharmacol Exp Ther. 1997 Nov;283(2):918-24.

[2]. Shohami E, et al. Cytokine production in the brain following closed head injury: dexanabinol (HU-211) is a novel TNF-alpha inhibitor and an effective neuroprotectant. J Neuroimmunol. 1997 Feb;72(2):169-77.

[3]. Shohami E, et al. Dexanabinol (HU-211): A nonpsychotropic cannabinoid with neuroprotective properties. Drug development research, 2000, 50(3-4): 211-215.