(-)-ZK 216348

Cat. No.: HY-123352A

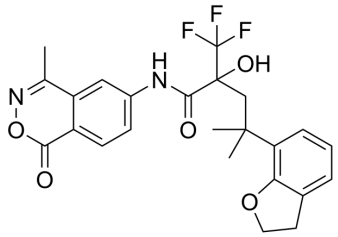
Molecular Formula: $C_{24}H_{23}F_3N_2O_5$

Molecular Weight: 476.45

Target: Glucocorticoid Receptor

Pathway: Immunology/Inflammation; Vitamin D Related/Nuclear Receptor

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 : 230 mg/mL (482.74 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.0989 mL	10.4943 mL	20.9886 mL
	5 mM	0.4198 mL	2.0989 mL	4.1977 mL
	10 mM	0.2099 mL	1.0494 mL	2.0989 mL

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

BIOLOGICAL ACTIVITY

Description

(-)-ZK 216348 is the enantiomer of (+)-ZK 216348 (HY-123352). (+)-ZK 216348 is a nonsteroidal selective glucocorticoid receptor agonist with an IC_{50} of 20.3 nM. ZK 216348 also binds to Progesterone and mineralocorticoid receptors with IC_{50}s of 20.4 nM and 79.9 nM, respectively. ZK 216348 has antiinflammatory activity similar to Prednisolone and induces less transactivation-mediated side effects[1][2].

IC_{50} & Target

IC50: 20.3 nM (Glucocorticoid recepto), 20.4 nM (Progesterone receptor) and 79.9 nM (mineralocorticoid receptor)[1]

In Vitro

In human peripheral blood mononuclear cells (PBMCs), ZK 216348 inhibits TNF-α and IL-12 with IC_{50} of 89 nM and 52 nM, respectively[1].
Participation of an active GR in the antiinflammatory response of ZK 216348 is further investigated in Caco-2 cells, where the TNF-α-induced expression of the proinflammatory cytokine IL-8 is suppressed in the presence of ZK 216348[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

ZK 216348 (1-30 mg/kg; subcutaneous injection; for 24 hours; NMRI mice and Wistar rats) treatment inhibits ear edema in both mice and rats. A markedly superior side-effect profile is found in ZK 216348 with regard to increases in blood glucose, spleen involution, and, to a lesser extent, skin atrophy[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Schäcke H, et al. Dissociation of transactivation from transrepression by a selective glucocorticoid receptor agonist leads to separation of therapeutic effects from side effects. Proc Natl Acad Sci U S A. 2004 Jan 6;101(1):227-32.

[2]. Reuter KC, et al. Selective glucocorticoid receptor agonists for the treatment of inflammatory bowel disease: studies in mice with acute trinitrobenzene sulfonic acid colitis. J Pharmacol Exp Ther. 2012 Apr;341(1):68-80.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA