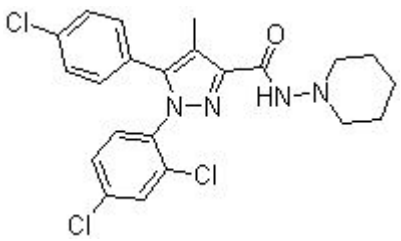
Product Introduction

Rimonabant

Rimonabant is a selective antagonist of **CB1** with **IC50** of 13.6 nM and **EC50** of 17.3 nM in hCB1 transfected HEK 293 membrane.

Technical Data:

Molecular Weight (MW):	463.79	
Formula:	$C_{22}H_{21}Cl_3N_4O$	
Solubility (25°C) * <1 mg/ml means slightly soluble or insoluble:	DMSO 25 mg/mL	
	Water <1 mg/mL	
	Ethanol 2 mg/mL	
Purity:	>98%	
Storage:	3 years -20℃Powder	
	6 months-80℃in DMSO	
CAS No.:	168273-06-1	

Biological Activity

Rimonabant dose-dependently reduces ACAT activity in Raw264.7macrophages with IC50 of 2.9 μM and isolated peritoneal macrophages. Rimonabant inhibits ACATactivity in intact CHO-ACAT1 and CHO-ACAT2 cells and in cell-free assays with approximately equal efficiency with IC50 of 1.5 μM and 2.2 μM for CHO-ACAT1 and CHO-ACAT2, respectively. Consistent with ACAT inhibition, Rimonabant treatment blocks ACAT dependent processes in macrophages, oxysterol-induced apoptosis and acetylated-LDL induced foam cell formation. [2] Rimonabant antagonizes the inhibitory effects of cannabinoid receptor agonists on

both mouse vas deferens contractions and adenylyl cyclase activity in rat brain membranes in a concentration-dependent manner. [3] Rimonabant significantly reduces cell growth and induces cell death of human colorectal cancer cells (DLD-1, CaCo-2 and SW620). Rimonabant is able to alter cell cycle distribution in all the cell lines tested. Particularly, Rimonabant produces a G2/M cell cycle arrest in DLD-1 cells without inducing apoptosis or necrosis. [4]

Rimonabant is administered intraperitoneally or orally potently and dose-dependently antagonize classical pharmacological and behavioural effectos of cannabinoid receptor agonists. [3] In the mouse model of azoxymethane-induced colon carcinogenesis, Rimonabant significantly decreased aberrant crypt foci (ACF) formation, which precedes colorectal cancer. [4] Rimonabant (10 mg/kg by gavage) is fed for 2 weeks to 3-month-old male obese Zucker rats as an impaired glucose tolerance model and for 10 weeks to 6-month-old male obese Zucker rats as a model of the metabolic syndrome. RANTES (Regulated upon Activation, Normal T cell Expressed, and Secreted) and MCP-1 (monocyte chemotactic protein-1) serum levels are increased in obese vs lean Zucker rats and significantly reduced by long-term treatment with Rimonabant, which slowes weight gain in rats with the metabolic syndrome. Neutrophils and monocytes are significantly increased in young and old obese vs lean Zucker rats and lowered by Rimonabant. Platelet-bound fibrinogen is significantly enhanced in obese vs lean Zucker rats of both age, and is reduced by Rimonabant. Platelets from obese rats are more sensitive to thrombin-induced aggregation and adhesion to fibrinogen, which are both attenuated by Rimonabant therapy. [5]

Efficacious to induce weight reduction and improvements in cardiometabolic risk factors, however was withdrawn in 2009 due to severe depressive disorder and anxiety.

References

[1] Chu CM, et al, Org Biomol Chem, 2008, 6(18), 3399-3407

[2] Netherland C, et al, Biochem Biophys Res Commun, 2010, 398(4), 671-676.

[3] Rinaldi-Carmona M, et al, FEBS Lett, 1994, 350(2-3), 240-244.

[4] Santoro A, et al, Int J Cancer, 2009, 125(5), 996-1003.

[5] Schafer A, et al, Br J Pharmacol, 2008, 154(5), 1047-1054.

[6] Wise LE, et al, Neuropsychopharmacology, 2007, 32(8), 1805-1812.

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