Rimonabant
Item No. 9000484

CAS Registry No.: 168273-06-1
Formal Name: 5-(4-chlorophenyl)-1-(2,4-
dichlorophenyl)-4-methyl-N-
1-piperidinyl-1H-pyrazole-3-
carboxamide
Synonym: SR141716
MF: C_{22}H_{21}Cl_{3}N_{4}O
FW: 463.8
Purity: ≥98%
Stability: ≥2 years at -20°C
Supplied as: A crystalline solid

Laboratory Procedures

For long term storage, we suggest that rimonabant be stored as supplied at -20°C. It should be stable for at least two years.

Rimonabant is supplied as a crystalline solid. A stock solution may be made by dissolving the rimonabant in an organic solvent purged with an inert gas. Rimonabant is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide. The solubility of rimonabant in these solvents is approximately 20 mg/ml.

Rimonabant is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, rimonabant should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. Rimonabant has a solubility of approximately 0.3 mg/ml in a 1:2 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Rimonabant, also known as SR141716, was the first selective central cannabinoid (CB₁) receptor inverse agonist (Kᵢ = 1.8 nM) to be developed as an appetite suppressant, anti-obesity drug.² It is widely used as a tool to investigate CB receptor properties and the mechanisms by which CB agonists exert their pharmacological effects. In rodent models and clinical trials, rimonabant effectively induces lipolysis, reduces hepatomegaly, decreases body weight, and improves dyslipidemia by reducing triglyceride, free fatty acid, and total cholesterol levels and by increasing HDL/LDL ratios.² However, rimonabant reportedly produces adverse psychiatric and neurological effects (e.g., depression or anxiety) and therefore is not approved by the FDA for use as a weight control medication.² Rimonabant elicits anti-proliferative and immunomodulatory effects (e.g., cell cycle arrest, increased expression of IκB and phosphorylated Akt, and decreased expression of NF-κB, phosphorylated ERK1/2, COX-2, and iNOS) in vitro.³

References