

Baricitinib

Catalog No: tcsc0724



Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Size: 200mg

Size: 500mg



Specifications

CAS No:

1187594-09-7

Formula:

$C_{16}H_{17}N_7O_2S$

Pathway:

Epigenetics;Stem Cell/Wnt;JAK/STAT Signaling

Target:

JAK;JAK;JAK

Purity / Grade:

>98%

Solubility:

DMSO : 25 mg/mL (67.31 mM; Need ultrasonic and warming)

Alternative Names:

INCB028050;LY3009104

Observed Molecular Weight:

371.42

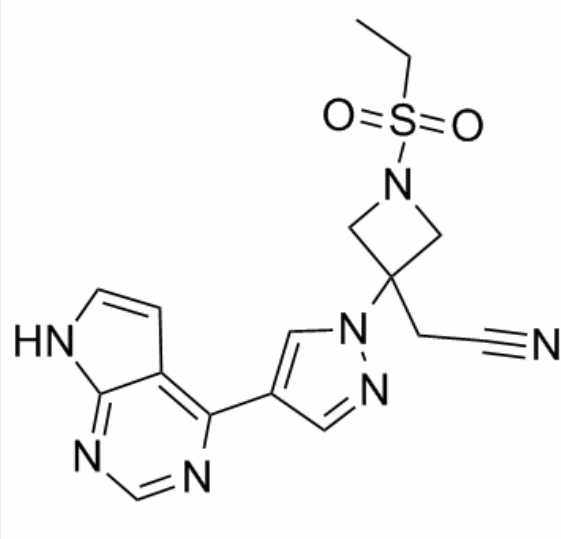
Product Description

Baricitinib is a selective orally bioavailable **JAK1/JAK2** inhibitor with **IC₅₀** of 5.9 nM and 5.7 nM, respectively.

IC50 & Target: IC50: 5.9 nM (JAK1), 5.7 nM (JAK2), >400 (JAK3), 53 nM (Tyk2)^[1]

In Vitro: In cell-based assays, Baricitinib (INCB028050) proves to be a potent inhibitor of JAK signaling and function. In PBMCs, Baricitinib inhibits IL-6-stimulated phosphorylation of the canonical substrate STAT3 (pSTAT3) and subsequent production of the chemokine MCP-1 with IC₅₀ values of 44 nM and 40 nM, respectively. In isolated naive T-cells, INCB028050 also inhibits pSTAT3 stimulated by IL-23 (IC₅₀=20 nM). Importantly, this inhibition prevented the production of two pathogenic cytokines (IL-17 and IL-22) produced by Th17 cells-a subtype of helper T cells with demonstrable inflammatory and pathogenic properties-with an IC₅₀ value of 50 nM. In stark contrast, the structurally similar but ineffective JAK1/2 inhibitors INCB027753 and INCB029843 has no significant effect in any of these assays systems when tested at concentrations up to 10 μM^[1].

In Vivo: Baricitinib (INCB028050) treatment, compares with vehicle, inhibits the increase in hind paw volumes during the 2 wk of treatment by 50% at a dose of 1 mg/kg and >95% at doses of 3 or 10 mg/kg. Because baseline paw volume measurements are taken on treatment day 0-in animals with significant signs of disease-it is possible to have >100% inhibition in animals showing marked improvement in swelling^[1]. Baricitinib (0.7 mg/day) treated mice exhibits substantially reduced inflammation as assessed by H&E staining, reduced CD8 infiltration, and reduced MHC class I and class II expression when compared with vehicle-control treated mice. CD8⁺NKG2D⁺ cells, critical effectors of disease in murine and human alopecia areata (AA), are greatly diminished in Baricitinib treated mice compare with vehicle control treated mice^[2].



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