



Baricitinib (phosphate)

Catalog No: tcsc1378

Available Sizes
Size: 5mg
Size: 10mg
Size: 50mg
Size: 100mg
Specifications
CAS No: 1187595-84-1
Formula: $C_{16}^{H}_{20}^{N}_{7}^{O}_{6}^{PS}$
Pathway: Epigenetics;Stem Cell/Wnt;JAK/STAT Signaling
Target: JAK;JAK;JAK
Purity / Grade: >98%
Solubility: DMSO : ≥ 4.7 mg/mL (10.01 mM)
Alternative Names: INCB028050;LY3009104
Observed Molecular Weight: 469.41



Product Description

Baricitinib phosphate is a selective orally bioavailable **JAK1/JAK2** inhibitor with IC_{50} of 5.9 nM and 5.7 nM, respectively.

IC50 & Target: IC50: 5.9 nM (JAK1), 5.7 nM (JAK2), >400 nM (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 IC $_{50}$ values of 44 nM and 40 nM, respectively. In isolated naive T-cells, INCB028050 also inhibits pSTAT3 stimulated by IL-23 (IC $_{50}$ =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 IC $_{50}$ 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 μ M $^{[1]}$.

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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