Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

PF-06651600

Catalog No: tcsc0020243

Available Sizes

Size: 5mg

Size: 10mg

Size: 25mg

Size: 50mg

Specifications

CAS No:
1792180-81-4

Formula:
$C_{15}H_{19}N_5O$

Pathway:
Epigenetics;Stem Cell/Wnt;JAK/STAT Signaling

Target:
JAK;JAK;JAK

Purity / Grade:
>98%

Solubility:
DMSO : 150 mg/mL (525.69 mM; Need ultrasonic and warming)

Observed Molecular Weight:
285.34

Product Description

PF-06651600 is a potent **JAK3**-selective inhibitor with an **IC_{50}** of 33.1 nM.

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IC50 & Target: IC50: 33.1 nM (JAK3)[1]

In Vitro: PF-06651600 is a potent JAK3-selective inhibitor which can inhibit the JAK3 kinase activity with an IC_{50} of 33.1 nM but without activity (IC_{50}>10 000 nM) against JAK1, JAK2, and TYK2. PF-06651600 inhibits the phosphorylation of STAT5 elicited by IL-2, IL-4, IL-7, and IL-15 with IC_{50} values of 244, 340, 407, and 266 nM, respectively. PF-06651600 also inhibits the phosphorylation of STAT3 elicited by IL-21 with an IC_{50} of 355 nM. Functional assessment in T-cell differentiation assays demonstrate that PF-06651600 suppresses Th1 and Th17 differentiation as measured by IFNγ, after 5 days under Th1 conditions, and IL-17 production, after 6 days under Th17 conditions, with IC_{50} values of 30 nM and 167 nM, respectively. PF-06651600 also suppresses Th1 and Th17 function as measured by the inhibition of IFNγ production (IC_{50}=48 nM) and IL-17 production (IC_{50}=269 nM) in cells that have been previously differentiated and rested before being treated with PF-06651600[1].

In Vivo: In the rat adjuvant-induced arthritis (AIA) model, PF-06651600 reduces paw swelling with an unbound EC_{50} of 169 nM. Similarly, PF-06651600 significantly reduces disease severity in the experimental autoimmune encephalomyelitis (EAE) mouse model when dosed either therapeutically at 30 or 100 mg/kg or prophylactically at 20 and 60 mg/kg. The efficacy of PF-06651600 in these two rodent models of inflammatory and autoimmune diseases illustrates that JAK3-selective inhibition can be sufficient to have disease modifying effects in human diseases[1].