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

Cerdulatinib

Catalog No: tcsc3329

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Size: 200mg

Specifications

CAS No:
1198300-79-6

Formula:
$C_{20}H_{27}N_7O_3S$

Pathway:
Epigenetics;Stem Cell/Wnt;JAK/STAT Signaling;Protein Tyrosine Kinase/RTK

Target:
JAK;JAK;JAK;Syk

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 30 mg/mL (67.33 mM)

Alternative Names:
PRT062070;PRT2070

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Observed Molecular Weight:
445.54

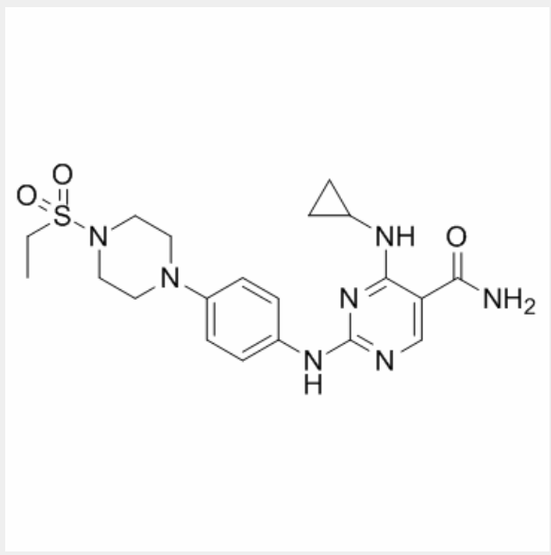
Product Description

Cerdulatinib is a novel, orally available, ATP-competitive **JAK** and **SYK** inhibitor that demonstrates selective inhibition of TYK2 and SYK with an **IC_{50}** of 0.5 nM and 32 nM.

IC50 & Target: IC50: 0.5 nM (SYK)[2]

In Vitro: Cerdulatinib shows inhibitory effect on 60 CLL with IC_{50} ranging from 0.37 to 10.02 μM. Cerdulatinib induces apoptosis in CLL in association with MCL-1 down-regulation and PARP cleavage. Cerdulatinib (2μM) is able to overcome the support of the microenvironment and induces CLL cell death. Cerdulatinib (250-500 nM) blocks proliferation of ibrutinib-sensitive and ibrutinib-resistant primary CLL cells. Cerdulatinib also blocks proliferation of both ibrutinib-sensitive and ibrutinib-resistant primary CLL cells as well as BTKC481S-transfected cell lines, and blocks BCR and JAK-STAT signaling pathways. Furthermore, inhibition of SYK and JAK by cerdulatinib translates to downstream inhibition of AKT and ERK. Cerdulatinib inhibits the activity of NF-kB pathway[1].
PRT062070 reduces the ability of stimulated B cells to upregulate cell-surface expression of the early activation marker CD69 (IC_{50} =0.11 μM). PRT062070 exhibits differential potency against cytokine JAK/STAT signaling pathways. PRT062070 (1 or 3 μM) induces apoptosis in BCR-signaling competent NHL cell lines[2]. Cerdulatinib demonstrates inhibitory activity against both ABC and GCB subtypes of DLBCL cells. Cerdulatinib also induces apoptosis in both GCB and ABC subtypes of DLBCL cell lines via caspase 3 and PARP cleavage. And cerdulatinib blocks cell cycle in both ABC and GCB subtypes of DLBCL via inhibition of RB phosphorylation and down-regulation of cyclin E. Cerdulatinib induces cell cycle arrest and apoptosis under the condition of BCR stimulation in all DLBCL cell lines. Besides, cerdulatinib blocks JAK/STAT and BCR signaling in both ABC and GCB DLBCL cell lines. Cerdulatinib induces cell death in primary human DLBCL samples[3]. Cerdulatinib inhibits BCR-induced signals in a dose-dependent manner and most strongly between 0.3 to 1 μM. and particularly in IGHV-unmutated samples with greater BCR signaling capacity and response to IL4, or samples expressing higher levels of sIgM, $CD49d^+$, or $ZAP70^+$. Cerdulatinib overcomes anti-IgM, IL4/CD40L, or NLC-mediated protection by preventing upregulation of MCL-1 and $BCL\text{-}X_L$; however, BCL-2 expression is unaffected. Furthermore, in samples treated with IL4/CD40L, cerdulatinib synergizes with venetoclax in vitro to induce greater apoptosis than either drug alone[4].

In Vivo: PRT062070 (0.5 mg/kg) results in a nonstatistically significant trend toward reduced ankle inflammation, whereas significant reductions in inflammation are achieved with the 1.5, 3, and 5 mg/kg doses. PRT062070 also affects anticollagen antibody formation. PRT062070 (15 mg/kg) suppresses upregulation of splenic B-cell surface CD80/86 and CD69, and inhibits BCR signaling and activation in the spleen after oral dosing in mice[2].



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