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

Trilaciclib

Catalog No: tcsc0021431

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
1374743-00-6

Formula:
$C_{24}H_{30}N_8O$

Pathway:
Cell Cycle/DNA Damage

Target:
CDK

Purity / Grade:
>98%

Solubility:
10 mM in DMSO

Alternative Names:
G1T28

Observed Molecular Weight:
446.55

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

Product Description

Trilaciclib is a **CDK4/6** inhibitor with **IC_{50}**s of 1 nM and 4 nM for CDK4 and CDK6, respectively.

IC50 & Target: IC50: 1 nM (CDK4), 4 nM (CDK6)[1]

In Vitro: Incubation with Trilaciclib (G1T28) for 24 hours induces a robust G_1 cell-cycle arrest (time=0). By 16 hours after Trilaciclib hydrochloride washout, cells have reentered the cell cycle and demonstrate cell-cycle kinetics similar to untreated control cells. These results demonstrate that Trilaciclib causes a transient, and reversible G_1 arrest. A transient Trilaciclib-mediated G_1 cell-cycle arrest in CDK4/6-sensitive cells decreases the *in vitro* toxicity of a variety of commonly used cytotoxic chemotherapy agents associated with myelosuppression[1].

In Vivo: Trilaciclib (G1T28) treatment results in a robust and dose-dependent suppression of proliferation in HSPCs at 12 hours, with EdU incorporation returning near baseline levels in a dose-dependent manner by 24 hours after administration. These data demonstrate that a single oral dose of Trilaciclib can produce reversible cell-cycle arrest in HSPCs in a dose-dependent manner *in vivo*. Mice given 100 mg/kg Trilaciclib 30 minutes prior to etoposide treatment, exhibits only background levels of caspase-3/7 activity. These data demonstrate that Trilaciclib can protect the bone marrow from chemotherapy-induced apoptosis *in vivo*. The data demonstrate that treatment with Trilaciclib prior to 5-FU likely decreases 5-FU-induced damage by chemotherapy in HSPCs, thus accelerating blood count recovery after chemotherapy[1].