

# 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

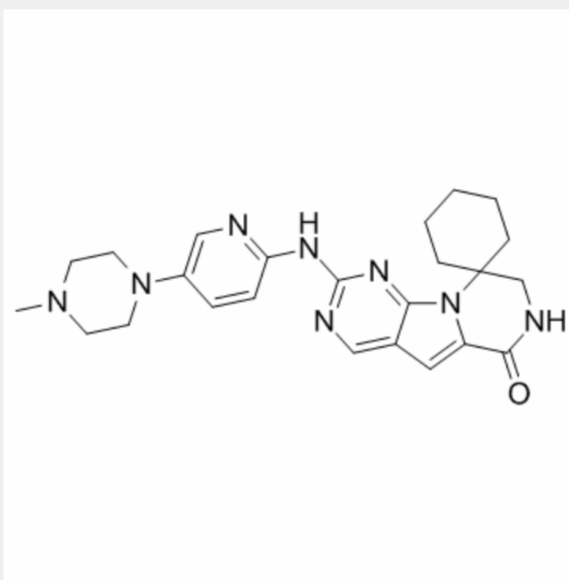
## Product Description

Trilaciclib is a **CDK4/6** inhibitor with **IC<sub>50</sub>s** of 1 nM and 4 nM for CDK4 and CDK6, respectively.

IC50 & Target: IC50: 1 nM (CDK4), 4 nM (CDK6)<sup>[1]</sup>

**In Vitro:** Incubation with Trilaciclib (G1T28) for 24 hours induces a robust G<sub>1</sub> 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<sub>1</sub> arrest. A transient Trilaciclib-mediated G<sub>1</sub> 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<sup>[1]</sup>.

**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<sup>[1]</sup>.



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