

Torkinib

Catalog No: tcsc0247



Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Size: 200mg

Size: 500mg

Size: 1g



Specifications

CAS No:

1092351-67-1

Formula:

$C_{16}H_{16}N_6O$

Pathway:

PI3K/Akt/mTOR;Autophagy;Autophagy

Target:

mTOR;Autophagy;Mitophagy

Purity / Grade:

>98%

Alternative Names:

PP 242

Observed Molecular Weight:

308.34

Product Description

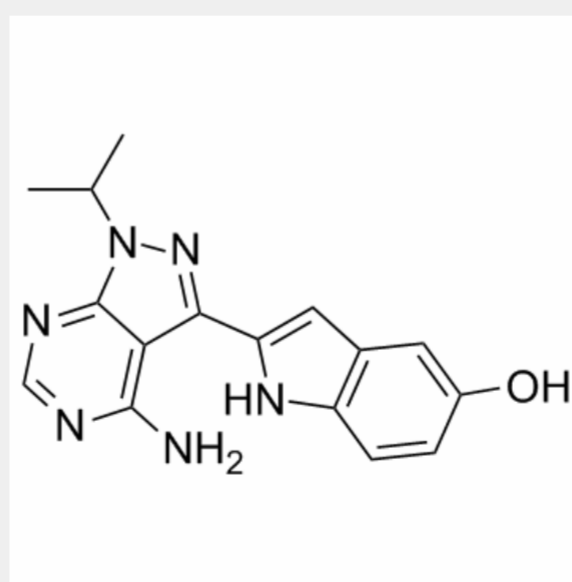
Torkinib (PP 242) is a selective and ATP-competitive **mTOR** inhibitor with an **IC₅₀** of 8 nM. PP242 inhibits both **mTORC1** and **mTORC2** with **IC₅₀**s of 30 nM and 58 nM, respectively.

IC50 & Target: IC50: 8 nM (mTOR), 100 nM (p110δ), 410 nM (DNA-PK), 410 nM (PDGFR)^[1]

IC50: 30 nM (mTORC1), 58 nM (mTORC2)^[2]

In Vitro: Torkinib (PP 242) potently inhibits mTOR (IC₅₀=8 nM) but is much less active against other PI3K family members. Testing of Torkinib (PP 242) against 219 protein kinases reveals remarkable selectivity relative to the protein kinome: at a concentration 100-fold above its IC₅₀ for mTOR, Torkinib (PP 242) inhibits only one kinase by more than 90% (Ret) and only three by more than 75% (PKCα, PKCβII and JAK2^{V617F})^[1]. Torkinib (PP 242) has a dose-dependent effect on proliferation and at higher doses is much more effective than Rapamycin at blocking cell proliferation. The ability of Torkinib (PP 242) to block cell proliferation more efficiently than Rapamycin could be a result of its ability to inhibit mTORC1 and mTORC2, because Rapamycin can only inhibit mTORC1. In SIN1^{-/-} mouse embryonic fibroblasts (MEFs), Rapamycin is also less effective at blocking cell proliferation than Torkinib. That Torkinib (PP 242) and Rapamycin exhibit very different anti-proliferative effects in SIN1^{-/-} MEFs suggests that the two compounds differentially affect mTORC1^[2].

In Vivo: In fat and liver, Torkinib (PP 242) is able to completely inhibit the phosphorylation of Akt at S473 and T308, consistent with its effect on these phosphorylation sites observed in cell culture. Surprisingly, Torkinib (PP 242) is only partially able to inhibit the phosphorylation of Akt in skeletal muscle and is more effective at inhibiting the phosphorylation of T308 than S473, despite its ability to fully inhibit the phosphorylation of 4EBP1 and S6. These results will be confirmed by in vivo dose-response experiments, but, consistent with the partial effect of Torkinib (PP 242) on pAkt in skeletal muscle, a muscle-specific knockout of the integral mTORC2 component rictor resulted in only a partial loss of Akt phosphorylation at S473. These results suggest that a kinase other than mTOR, such as DNA-PK, may contribute to phosphorylation of Akt in muscle^[2].



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