Web: www.taiclone.com
Tel: +886-2-2735-9682
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Palomid 529

Catalog No: tcsc0258

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
914913-88-5

Formula:
$C_{24}H_{22}O_6$

Pathway:
PI3K/Akt/mTOR

Target:
mTOR

Purity / Grade:
>98%

Solubility:
DMSO : 20.5 mg/mL (50.44 mM; Need ultrasonic and warming)

Alternative Names:
P529

Observed Molecular Weight:
406.43

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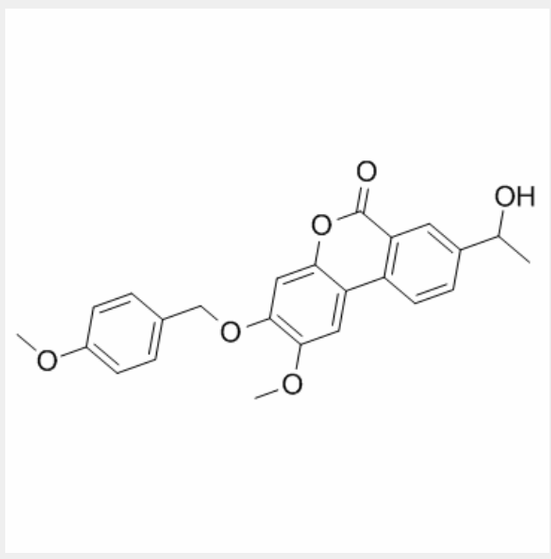
Product Description

Palomid 529 is a potent inhibitor of **mTORC1** and **mTORC2** complexes.

IC50 & Target: TORC1/TORC2[1]

In Vitro: Palomid 529 (P529) inhibits both VEGF-driven (IC_{50}, 20 nM) and bFGF-driven (IC_{50}, 30 nM) endothelial cell proliferation and retained the ability to induce endothelial cell apoptosis[1]. Palomid 529 (RES-529) is a PI3K/AKT/mTOR pathway inhibitor that interferes with the pathway through both mTOR complex 1 (mTORC1) and mTOR complex 2 (mTORC2) dissociation. Palomid 529 inhibits mTORC1/mTORC2 activity in various cancer cell lines, as noted by decreased phosphorylation of substrates including ribosomal protein S6, 4E-BP1, and AKT, leading to cell growth inhibition and death, with activity generally in the range of 5-15 μM. At 10 μM concentrations, Palomid 529 reduces the binding of 0.5 nM [^{3}H]estradiol to estrogen receptor (ER)α and ERβ by 3% or less. Palomid 529 inhibits both VEGF-stimulated and β fibroblast growth factor-stimulated HUVEC cell proliferation with IC_{50} of ~10 and 30 nM, respectively. Treatment of HUVEC cells with Palomid 529 also results in a four-fold induction of apoptosis on the basis of DNA fragmentation. Growth inhibition is observed with Palomid 529 treatment in various cancer cell lines from the National Cancer Institute-60 (NCI-60) tumor panel, with IC_{50} ranges of 5-15 μM for central nervous system cancer cells and 5-30 μM for prostate cancer cells[2]. Palomid 529 (P529) results in a dose- and time-dependent decrease in Akt activity in PC3, LnCaP, and 22rv1 cells as evidenced by a reduced phosphorylation of Akt (Ser^{473}). Similar results are observed in all PCa cells with similar enzymatic IC_{50}s of about 0.2 μM. Palomid 529 inhibits the cell proliferation of neoplastic cells at different extent (IC_{50}s ranged from 5 to 28 μM), whereas very few effects are observed in non-neoplastic BPH1 and EPN cells. Treatment with Palomid 529 results in a concentration-dependent reduction in viable/proliferating tumor cells compared with non-neoplastic BPH1 and EPN cells. IC_{50}s range from 5 to 28 μM[3].

In Vivo: Palomid 529 (200 mg/kg/2d) inhibits C6V10 glioma tumor growth in nude mice following i.p. dosing. Analysis of signaling within the tumor lysates reveals that Palomid 529 (P529) also reduces AktS473 but not AktT308 signaling[1]. Palomid 529 (RES-529) has shown antitumor activity in a variety of mouse models, including those for glioblastoma, and prostate and breast cancer. In a C6V10 glioblastoma subcutaneous xenograft model, mice pretreated with Palomid 529 (200 mg/kg/2 days, intraperitoneal) 1 week before and for 3 weeks after a tumor cell injection showed an ~70% decrease in tumor volume compared with the control. In another glioblastoma tumor model using human U87 cells, mice treated with micronized Palomid 529 3 days after a tumor cell injection showed a reduction in tumor growth by ~78 and 29% with 50 and 25 mg/kg/2 days, intraperitoneal, Palomid 529, respectively, after 24 days compared with the control[2]. Palomid 529 (P529) is able to reduce tumor growth in a dose-dependent manner both in PC3 and 22rv1 xenografts. A 10, 47.6, and 59.3% reduction of tumor mass is demonstrated in mice bearing PC3 xenografts receiving 50, 100, and 200 mg/kg Palomid 529 respectively and a 9, 38.7, and 51.5% reduction of tumor mass in mice bearing 22rv1 xenografts receiving 50, 100, and 200 mg/kg Palomid 529 respectively[3].



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