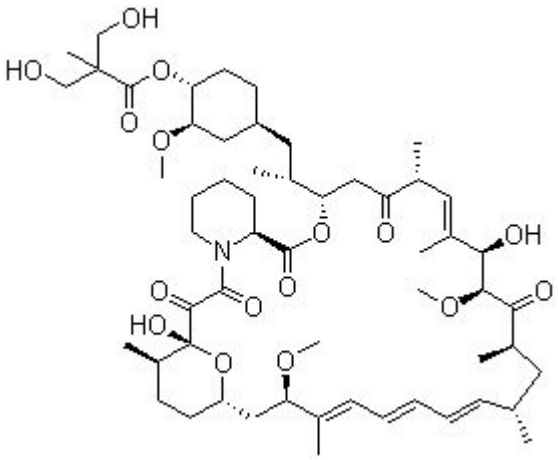
Product Introduction

Temsirolimus (CCI-779, NSC 683864)

Temsirolimus (CCI-779) is a specific **mTOR** inhibitor with **IC50** of 1.76 μM.

Technical Data:

Molecular Weight (MW):	1030.29	
Formula:	$C_{56}H_{87}NO_{16}$	
Solubility (25°C) * <1 mg/ml means slightly soluble or insoluble:	DMSO 75 mg/mL	
	Water <1 mg/mL	
	Ethanol 75 mg/mL	
Purity:	>98%	
Storage:	3 years -20℃Powder	
	6 months-80℃in DMSO	
CAS No.:	162635-04-3	

Biological Activity

In the absence of FKBP12, Temsirolimus potently inhibits mTOR kinase activity with IC50 of 1.76 μM, similar to that of rapamycin with IC50 of 1.74 μM. Temsirolimus treatment at nanomolar concentrations (10 nM to <5 μM) displays a modest and selective antiproliferative activity via FKBP12-dependent mechanism, but can completely inhibit the proliferation of a broad panel of tumor cells at low micromolar concentrations (5-15 μM), involving FKBP12-independent suppression of mTOR signaling. Temsirolimus treatment at micromolar but not nanomolar concentrations (20 μM) causes a marked decline in global protein synthesis and disassembly of polyribosomes, accompanied by rapid increase in the

Note： Products protected by valid patents are not offered for sale in countries where the sale of such products constitutes a patent infringement and its liability is at buyer's risk. This item is only for R&D purpose not for commercial business in kilos. Buyers should overview the patent issue in their countries.

phosphorylation of translation elongation factor eEF2 and the translation initiation factor eIF2A. [1] Temsirolimus inhibits the phosphorylation of ribosomal protein S6, more potently in PTEN-positive DU145 cells than in PTEN-negative PC-3 cells, and inhibits cell growth and clonogenic survival of both cells in a concentration-dependent manner. [2] Temsirolimus (100 ng/mL) potently inhibits proliferation and induces apoptosis in primary human lymphoblastic leukemia (ALL) cells. [3]

In the NOD/SCID xenograft models with human ALL, Temsirolimus treatment at 10 mg/kg/day produces a decrease in peripheral blood blasts and in splenomegaly [3] Administration of Temsirolimus (20 mg/kg i.p. 5 days/week) significantly delays the growth of DAOY xenografts by 160% after 1 week and 240% after 2 weeks, compared with controls. Single high-dose of Temsirolimus (100 mg/kg i.p) treatment induces 37% regression of tumor volume within 1 week. Temsirolimus treatment for 2 weeks also delays the growth of rapamycin-resistant U251 xenografts by 148%. [4] Inhibition of mTOR by Temsirolimus improves performance on four different behavioral tasks and decreases aggregate formation in a mouse model of Huntington disease. [5] Administration of Temsirolimus induces significant dose-dependent, antitumor responses against subcutaneous growth of 8226, OPM-2, and U266 xenografts with ED50 of 20 mg/kg and 2 mg/kg for 8226 and OPM-2, respectively, which are associated with inhibited proliferation and angiogenesis, induction of apoptosis, and reduction in tumor cell size. [6]

References

[1] Shor B, et al. Cancer Res, 2008, 68(8), 2934-2943.
[2] Wu L, et al. Cancer Res, 2005, 65(7), 2825-2831.
[3] Teachey DT, et al. Blood, 2006, 107(3), 1149-1155.
[4] Geoerger B, et al. Cancer Res, 2001, 61(4), 1527-1532.
[5] Ravikumar B, et al. Nat Genet, 2004, 36(6), 585-595.
[6] Frost P, et al. Blood, 2004, 104(13), 4181-4187.

Note：Products protected by valid patents are not offered for sale in countries where the sale of such products constitutes a patent infringement and its liability is at buyer’s risk. This item is only for R&D purpose not for commercial business in kilos. Buyers should overview the patent issue in their countries.