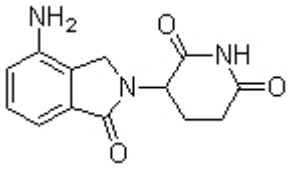
Product Introduction

Lenalidomide (CC-5013)

Lenalidomide (CC-5013) is a **TNF-α** secretion inhibitor with **IC50** of 13 nM.

Technical Data:

Molecular Weight (MW):	259.26	
Formula:	$C_{13}H_{13}N_3O_3$	
Solubility (25°C) * <1 mg/ml means slightly soluble or insoluble:	DMSO 52 mg/mL	
	Water <1 mg/mL	
	Ethanol <1 mg/mL	
Purity:	>98%	
Storage:	3 years -20℃Powder	
	6 months-80℃in DMSO	
CAS No.:	191732-72-6	

Biological Activity

Lenalidomide strongly induces IL-2 and sIL-2R production. Lenalidomide-induced tyrosine phosphorylation of CD28 on T cells is followed by a down-stream activation of NF-κB. [2] Lenalidomide and pomalidomide inhibits autoubiquitination of CRBN in HEK293 T cells expressing thalidomide-binding competent wild-type CRBN, but not thalidomide-binding defective CRBN(YW/AA). Overexpression of CRBN wild-type protein, but not CRBN(YW/AA) mutant protein, in KMS12 myeloma cells, amplifies pomalidomide-mediated reductions in c-myc and IRF4 expression and increases in p21(WAF-1) expression. Long-term selection for Lenalidomide resistance in H929 myeloma cell lines is accompanied by a reduction in CRBN, while in

DF15R myeloma cells resistant to both pomalidomide and Lenalidomide, CRBN protein is undetectable. [3] Lenalidomide prevents induction of defects by down-regulating tumor cell inhibitory molecule expression. Lenalidomide prevents induction of tumor-induced T cell lytic synapse dysfunction. Lenalidomide treatment blocks CLL cell-induced T cell actin synapse dysfunction, mimicks antibody blockade, and down-regulates expression of CLL inhibitory ligands and their receptors on T cells. Lenalidomide treatment prevents tumor-induced immune suppression in FL, DLBCL, HL, MM, SCC, and OC and down-regulates immunosuppressive ligand expression on all tumor cells examined. CTL killing function significantly increases following antibody blockade of CLL inhibitory ligands or Lenalidomide treatment compared to control treatments. Treatment of autologous CLL-T cell co-cultures with Lenalidomide reverses impaired $CD8^+$ T cell lytic synapse formation and granzyme B trafficking. [4]

The induction of angiogenesis by bFGF is significantly inhibited by oral treatment of Lenalidomide in a dose-dependent manner. Lenalidomide significantly decreases the percentage of vascularized area from 5.16% (control group) to 2.58% (50 mg/kg). Lenalidomide significantly reduces the calculated total MVL from 21.07 (control) to 8.11 (50 mg/kg). Lenalidomide significantly inhibites HUVEC migration through the fibronectin-coated membranes towards 0.1 ng/mL of bFGF at 100 μM, 1 ng/mL of VEGF at concentrations of 10 μM and 100 μM. [5]

References

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