Lenalidomide (Revlimid) Datasheet

**Technical Data**

<table>
<thead>
<tr>
<th>Molecular Weight (MW)</th>
<th>259.26</th>
</tr>
</thead>
<tbody>
<tr>
<td>Formula</td>
<td>C_{13}H_{13}N_{2}O_{3}</td>
</tr>
<tr>
<td>CAS No.</td>
<td>191732-72-6, 1243329-97-6 (HCl)</td>
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<tr>
<td>Synonyms</td>
<td>CC-5013</td>
</tr>
<tr>
<td>Solubility (25°C)</td>
<td>DMSO 52 mg/mL Water &lt;1 mg/mL Ethanol &lt;1 mg/mL</td>
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<tr>
<td>Storage</td>
<td>2 years -20°C Powder 2 weeks 4°C in DMSO 6 months -80°C in DMSO</td>
</tr>
</tbody>
</table>

**Biological Activity**

**Description**

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

**Targets**

TNF-α

**IC50**

13 nM

**In vitro**

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. Lenalidomide and pomalidomide inhibits autoubiquitination of CRBN in HEK293 T cells expressing thalidomide-binding competent wild-type CRBN, but not thalidomide-binding defective CRBN(W21A). Overexpression of CRBN wild-type protein, but not CRBN(W21A) 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. 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.

**In vivo**

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 inhibits HUVEC migration from 5.16% (control group) to 2.58% (50 mg/kg). Lenalidomide significantly reduces the calculated total 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.

**Clinical Trials**

Lenalidomide has entered in a Phase II clinical trial in the treatment of chronic lymphocytic leukemia.

**Features**

**Protocol (Only for Reference)**

**Kinase Assay:**

**Assay for inhibition of TNF synthesis in human PBMCs**

Human PBMCs from normal donors are obtained by Ficoll–Hypaque density centrifugation. Cells (10^6 cells/mL) are cultured in RPMI supplemented with 10% AB+ serum, 2 mM l-glutamine, 100 U/mL penicillin, and 100 μg/mL streptomycin. Lenalidomide is dissolved in DMSO at 20 mg/mL; further dilution is done with culture medium. The final DMSO concentration in all assays including the controls is 0.25%. Lenalidomide is added to cells 1 hour prior to the addition of LPS. PBMCs (10^6 cells/mL) are stimulated with 1 μg/mL of LPS from Salmonella minnesota R595. Cells, in triplicate, are incubated with LPS for 18-20 hours at 37 °C in 5% CO2. Supernatants are then harvested and assayed for cytokine levels. In some experiments, supernatants are kept frozen at -70 °C until use. Cell viability is assayed by Trypan blue exclusion dye method. The concentration of TNFs in the culture supernatants is determined by ELISA. Lenalidomide is assayed in a minimum of three separate experiments. Percent inhibition is determined as 100 × [1 – (cytokine (experimental)/cytokine (control))].

**Animal Study**

**Animal Models**

Adult male Sprague-Dawley rats bearing HUVECs cells

**Formulation**

0.5% DMSO

**Doses**

50 mg/kg and 250 mg/kg

**Administration**

Administered via i.p.

**References**

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Lenalidomide (Revlimid) Chemical Structure

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www.selleckchem.com/datasheet/Lenalidomide-DataSheet.html
Customer Reviews

Data independently produced by Harvard Medical School.

Lenalidomide (Revlimid) purchased from Selleck

MM.1S were treated with AT9283 (0.125 μM), lenalidomide (2 μM) or combined therapy for 72 hours. Annexin/PI staining show increased apoptosis associated with caspase 8 and PARP cleavage after 18 and 36 hours of exposure. B) MM.1S cells were treated with AT9283 (0.125 μM), lenalidomide (2 μM) or combined therapy for 4 hours. Whole lysates were immunoblotted with indicated antibodies.

LENALIDOMIDE (REVIMALD)

PLEASE KEEP THE PRODUCT UNDER -20°C FOR LONG-TERM STORAGE.

NOT FOR HUMAN, VETERINARY DIAGNOSTIC OR THERAPEUTIC USE

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www.selleckchem.com/datasheet/Lenalidomide-DataSheet.html