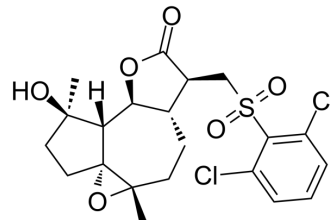


PD-L1-IN-6

Cat. No.:	HY-162399
Molecular Formula:	C ₂₁ H ₂₄ Cl ₂ O ₆ S
Molecular Weight:	475.38
Target:	PD-1/PD-L1; STAT
Pathway:	Immunology/Inflammation; JAK/STAT Signaling; Stem Cell/Wnt
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	PD-L1-IN-6 (Compound 16) is an orally active inhibitor for PD-L1 expression in neutrophil by targeting STAT3 with K _D of 90.5 nmol/L. PD-L1-IN-6 promotes neutrophil-mediated antifungal immunoresponse ^[1] .																																			
In Vitro	<p>PD-L1-IN-6 (40 μM) inhibits 80.17% PD-L1 expression at 40 μM without significant cytotoxicity in neutrophil^[1]. PD-L1-IN-6 is slight permeable for blood brain barrier, with a p_e of 1.22×10⁻⁶ cm/s^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <p>Cell Viability Assay^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>Murine neutrophil</td> </tr> <tr> <td>Concentration:</td> <td>40 μM</td> </tr> <tr> <td>Incubation Time:</td> <td></td> </tr> <tr> <td>Result:</td> <td>Maintained 81.52% cell viability at 40 μM.</td> </tr> </table>	Cell Line:	Murine neutrophil	Concentration:	40 μM	Incubation Time:		Result:	Maintained 81.52% cell viability at 40 μM.																											
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In Vivo	<p>PD-L1-IN-6 (50-100 mg/kg, p.o. for 3 days) inhibits β-glucaninduced secretion of CXCL1 and CXCL2, induces the neutrophil mobilization, enhances neutrophil-mediated antifungal immunity in <i>C. albicans</i> infected C57BL/6 mice^[1].</p> <p>Pharmacokinetic Analysis of PD-L1-IN-6 in Sprague-Dawley rats^[1]</p> <table border="1"> <thead> <tr> <th>route</th> <th>Dose (mg/kg)</th> <th>C_{max} (ng/mL)</th> <th>T_{1/2} (h)</th> <th>T_{max} (h)</th> <th>AUC_{0-t} (ng·h/mL)</th> <th>AUC_{0-inf} (ng·h/mL)</th> <th>CL (mL/min/kg)</th> <th>MRT_{0-t} (h)</th> <th>V_{ss} (L/kg)</th> <th>F (%)</th> </tr> </thead> <tbody> <tr> <td>po</td> <td>10</td> <td>113.52</td> <td>1.46</td> <td>0.33</td> <td>166.57</td> <td>182.25</td> <td>-</td> <td>1.74</td> <td>-</td> <td>45.1</td> </tr> <tr> <td>iv</td> <td>2</td> <td>174.78</td> <td>0.17</td> <td>0.14</td> <td>73.87</td> <td>75.44</td> <td>453.10</td> <td>0.25</td> <td>7.46</td> <td>-</td> </tr> </tbody> </table> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1"> <tr> <td>Animal Model:</td> <td><i>C. albicans</i> infected C57BL/6 mice^[1]</td> </tr> </table>	route	Dose (mg/kg)	C _{max} (ng/mL)	T _{1/2} (h)	T _{max} (h)	AUC _{0-t} (ng·h/mL)	AUC _{0-inf} (ng·h/mL)	CL (mL/min/kg)	MRT _{0-t} (h)	V _{ss} (L/kg)	F (%)	po	10	113.52	1.46	0.33	166.57	182.25	-	1.74	-	45.1	iv	2	174.78	0.17	0.14	73.87	75.44	453.10	0.25	7.46	-	Animal Model:	<i>C. albicans</i> infected C57BL/6 mice ^[1]
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Dosage:	50-100 mg/kg
Administration:	p.o. for 3 days
Result:	Inhibited secretion of CXCL1 and CXCL2, and accumulation of neutrophils in bone marrow. Caused reduced fungal burden and increased infiltration rate of neutrophils in kidney.

REFERENCES

[1]. Lu X, et al., Drug Repurposing of ACT001 to Discover Novel Promising Sulfide Prodrugs with Improved Safety and Potent Activity for Neutrophil-Mediated Antifungal Immunotherapy. J Med Chem. 2024 Mar 25.

Caution: Product has not been fully validated for medical applications. For research use only.

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