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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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Cilengitide

Cat. No.: HY-16141

CAS No.: 188968-51-6

Molecular Formula: $C_{27}H_{40}N_8O_7$

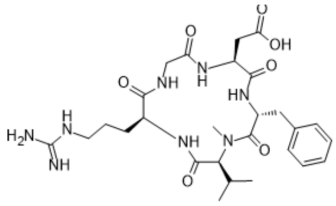
Molecular Weight: 588.66

Target: Integrin; Autophagy; Apoptosis; STAT; PD-1/PD-L1

Pathway: Cytoskeleton; Autophagy; Apoptosis; JAK/STAT Signaling; Stem Cell/Wnt; Immunology/Inflammation

Storage:

Powder	-20°C	3 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (169.88 mM; Need ultrasonic)

DMSO : ≥ 44 mg/mL (74.75 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.6988 mL	8.4939 mL	16.9877 mL
5 mM	0.3398 mL	1.6988 mL	3.3975 mL
10 mM	0.1699 mL	0.8494 mL	1.6988 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (169.88 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Cilengitide (EMD 121974) is a potent integrins antagonist with IC_{50}s of 0.61 nM ($\alpha_v\beta_3$), 8.4 nM ($\alpha_v\beta_5$) and 14.9 nM ($\alpha_5\beta_1$), respectively. Cilengitide inhibits the binding of $\alpha_v\beta_3$ and $\alpha_v\beta_5$ to Vitronectin with IC_{50}s of 4 nM and 79 nM, respectively. Cilengitide inhibits TGF-β/Smad signaling, mediates PD-L1 expression. Cilengitide also induces apoptosis, shows antiangiogenic effect in the research against glioblastoma and other cancers[1][2][3].

IC_{50} & Target

αvβ3 4 nM (IC_{50}, αvβ3-Vitronectin interaction[2])	αvβ5 79 nM (IC_{50}, αvβ5-Vitronectin interaction[2])	αvβ3 0.61 nM (IC_{50}, [1])	αvβ5 8.4 nM (IC_{50}, [1])
α5β1	STAT3		

	14.9 nM (IC_{50}, [1])	([3])

In Vitro

Cilengitide is a cyclized RGD (Arg-Gly-Asp motif)-containing pentapeptide. Cilengitide blocks integrin αvβ3- and αvβ5-mediated endothelial cell attachment and migration[2].
Cilengitide inhibits integrin-mediated binding to Vitronectin with IC_{50}s of 0.4 and 0.4 μM in cell adhesion studies assessing the human melanoma M21 or UCLA-P3 human lung carcinoma cell lines[2].
Cilengitide inhibits the attachment of human umbilical vein endothelial cells to Vitronectin with an IC_{50} of 2 μM[2].
Cilengitide (0-1 mg/mL; 24-72 h) inhibits cell viability of melanoma cells in vitro and (5 μg/mL; 12 h) induces B16 and A375 cells apoptosis[3].
Cilengitide (5 μg/mL, 10 μg/mL; 2 weeks) inhibits colony formation of B16 and A375 cells[3].
Cilengitide (0-20 μg/mL; 12 h) inhibits STAT3 phosphorylation to decrease the expression of PD-L1[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[3]

Cell Line:	B16 and A375 cells
Concentration:	0, 5, 10, and 20 μg/mL
Incubation Time:	12 hours
Result:	Suppressed PD-L1 expression and STAT3 phosphorylation at concentrations greater than 5 μg/mL.

Apoptosis Analysis[3]

Cell Line:	B16 and A375 cells
Concentration:	5 μg/mL
Incubation Time:	12 hours
Result:	Resulted apoptosis rates in B16 and A375 cells of 15.27% and 14.89%, respectively.

In Vivo

Cilengitide (i.p. at 10, 50, and 250 μg three times per week) inhibits M21-L melanoma tumors growth in nude mice[2].
Cilengitide (50 mg/kg; i.p.; daily) enhances the function of CD8+ T cells and promotes anti-PD1 efficacy with Anti-PD1 monoclonal antibody in B16 murine melanoma model[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Nude mice bearing M21-L melanoma tumors[2]
Dosage:	10, 50, and 250 μg
Administration:	Dosed i.p. three times per week
Result:	Demonstrated inhibition of tumor growth with a reduction in both tumor volume (55%, 75%, and 89%, respectively) and tumor weight (23%, 38%, and 61%, respectively), when compared to controls.

Animal Model:	Female C57BL/6 mice (6-8 weeks old) with B16 cells s.c.[3]
Dosage:	50 mg/kg; with or without 10 mg/kg Anti-PD1 monoclonal antibody or isotype control i.p. every 3 days;
Administration:	Intraperitoneal injection; daily

Result:	Downregulated the expression of PD-L1 via STAT3 pathway and decreased the expression of PD-L1.

CUSTOMER VALIDATION

- Cell. 2020 Aug 6;182(3):545-562.e23.
- Cancer Cell. 2021 Sep 28;S1535-6108(21)00492-X.
- Nat Cell Biol. 2020 Mar;22(3):289-296.
- Engineering. 8 October 2020.
- J Immunother Cancer. 2020 Mar;8(1):e000111.

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REFERENCES

[1]. Kapp TG, et al. A Comprehensive Evaluation of the Activity and Selectivity Profile of Ligands for RGD-binding Integrins. Sci Rep. 2017 Jan 11;7:39805.

[2]. Pan X, et al. Cilengitide, an αvβ3-integrin inhibitor, enhances the efficacy of anti-programmed cell death-1 therapy in a murine melanoma model. Bioengineered. 2022 Feb;13(2):4557-4572.

[3]. Hariharan S, et al. Assessment of the biological and pharmacological effects of the alpha nu beta3 and alpha nu beta5 integrinreceptor antagonist, Cilengitide (EMD 121974), in patients with advanced solid tumors. Ann Oncol. 2007 Aug;18(8):1400-7.

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