Delmitide

Cat. No.:	HY-106359
CAS No.:	287096-87-1
Molecular Formula:	$C_{59}H_{105}N_{17}O_{11}$
Molecular Weight:	1228.57
Sequence:	d(Arg-{Nle}-{Nle}-{Nle}-Arg-{Nle}-{Nle}-{Nle}-Gly-Tyr-NH2)
Sequence Shortening:	d(R-{Nle}-{Nle}-{Nle}-R-{Nle}-{Nle}-{Nle}-GY-NH2)
Target:	TNF Receptor; IFNAR; Reactive Oxygen Species
Pathway:	Apoptosis; Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description

Delmitide (RDP58) is an orally active d-isomer decapeptide with potent anti-inflammatory activity. Delmitide inhibits production of TNF-α, IFN-γ, and interleukin (IL)-12, and up-regulates heme oxygenase 1 activity. Delmitide can be used for the research of ulcerative colitis[1][2].

In Vivo

Delmitide (oral; 2.5, 5, 10 mg/kg; daily) significantly reduced CPT-11induced diarrhea, mucosal inflammation, and mortality in mice by suppressing the overproduction of proinflammatory cytokines TNF-a, IFN-y, and IL-12 in vivo[2].

Delmitide (oral; 2.5, 5, 10 mg/kg; daily) generates an enhanced tumor response and prolongation of time to relapse without concomitant Gl toxicity in mice[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	BALB/c mice (female, 9-10-week)[2]
Dosage:	2.5, 5, 10 mg/kg or 0.2 mL, 10 mg/kg
Administration:	Oral, daily
Result:	Reduced the incidence of diarrhea and attenuated CPT-11-associated GI toxicity and mortality in a dose-dependent manner. Had protective effect against chemotherapy-induced GI side-effects and reduced CPT-11-induced overexpression of TNF-α, IFN-γ, and IL-12 in vivo. Preserved the intestinal mucosa morphology by maintaining villus and crypt structure and inhibited TNF-α-mediated apoptosis in the crypt compartment, thereby protecting intestinal mucosa integrity in mice. Protected mice from CPT-11-induced GI toxicity and mortality and enhanced animal survival in tumor-bearing mice. Significantly reduced the incidence and overall tumor burden in a spontaneously metastatic model.

REFERENCES

[1]. Arthur Kaser, et al. Novel therapeutic targets in the treatment of IBD. Kaser, Arthur; Tilg, Herbert (2008). Expert Opinion on Therapeutic Targets, 12(5), 553–563.

[2]. Jingsong Zhao, et al. Oral RDP58 allows CPT-11 dose intensification for enhanced tumor response by decreasing gastrointestinal toxicity. Clin Cancer Res. 2004 Apr 15;10(8):2851-9.

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