ReACp53

Cat. No.:	HY-P0121
Molecular Formula:	$C_{108}H_{206}N_{52}O_{24}$
Molecular Weight:	2617.13
Sequence:	Arg-Arg-Arg-Arg-Arg-Arg-Arg-Arg-Arg-Arg-Pro-Ile-Leu-Thr-Arg-Ile-Thr-Leu-Glu
Sequence Shortening:	RRRRRRRRRRPILTRITLE
Target:	MDM-2/p53
Pathway:	Apoptosis
Storage:	Sealed storage, away from moisture Powder -80°C 2 years -20°C 1 year * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)

RRRRRRRRRRPILTRITLE

SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (38.21 mM; Need ultrasonic)
H_2O : 25 mg/mL (9.55 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	0.3821 mL	1.9105 mL	3.8210 mL
5 mM	0.0764 mL	0.3821 mL	0.7642 mL
10 mM	0.0382 mL	0.1910 mL	0.3821 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 50 mg/mL (19.10 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (0.96 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (0.96 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (0.96 mM); Clear solution

BIOLOGICAL ACTIVITY

Description	ReACp53 could inhibit p53 amyloid formation and rescue p53 function in cancer cell lines.
IC_{50} & Target	p53 amyloid formation[1].

In Vitro	ReACp53 penetrates into HGSOC primary cancer cells and converts mutant p53 from a punctate state into soluble WT-like p53. ReACp53 also induces cancer cell death, cell cycle arrest and results in p53 degradation. ReACp53 specifically affects cell viability and proliferation of cancer cells bearing mutant p53 but not wild type when grown as organoids[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Only mutant p53-bearing tumors in the ReACp53-treated mice cohorts are 80-90% smaller in weight than the control cohort, confirming the ability of ReACp53 to limit tumor proliferation and shrink tumors. A significant reduction of Ki67 positive cells is evident in ReACp53-treated OVCAR3 xenografts, indicative of a reduced proliferative index. Similar results are observed in the minimal residual disease model. In the paradigm, administration of ReACp53 results in a significant increase in p21 and MDM2 transcription in OVCAR3 but not MCF7 xenografts. A significantly increased population is also found in G0/G1 phase, supporting proliferative arrest upon ReACp53 administration in vivo[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1]	Mice[1] In the minimal residual disease model, three cohorts of mice (n=3) are injected with a matrigel/OVCAR3 (p53 mutant) suspension on one flank and with a matrigel/MCF7 (WT p53) suspension on the other flank. Treatment is started the same day. In both models, the treatment phase consist of three weeks of daily IP injections with 15 mg/kg of ReACp53, sequence-scrambled control peptide or vehicle alone[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- iScience. 2023 May 26.

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REFERENCES

[1]. Soragni A et al. A Designed Inhibitor of p53 Aggregation Rescues p53 Tumor Suppression in Ovarian Carcinomas.Cancer Cell. 2016 Jan 11;29(1):90-103.

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