Product Data Sheet

Product Name: Retrocyclin-101 TFA
Cat. No.: GC78903

Chemical Properties

Cas. No.			
Formula	$C_{74}H_{130}N_{28}O_{19}S_6 \cdot xC_2HF_3O_2$	M.Wt	1908.39
Solubility	DMSO: 100 mg/mL (Need ultrasonic)	Storage	Sealed storage, away from moisture
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		
Structure			

Background

Retrocyclin-101 (RC-101) TFA is an artificially synthesized, cyclic-structured θ-defensin, a broad-spectrum agent with antimicrobial (covering viruses, bacteria, and fungi) activity and anti-inflammatory activity. Retrocyclin-101 TFA can inhibit the serine protease activity of ZIKV NS2B-NS3, with an IC_{50} of 7.20 μM. Retrocyclin-101 TFA has significant inhibitory activity against HIV, SARS-CoV-2, flaviviruses, influenza viruses, HSV-1/2, Staphylococcus aureus, etc. Retrocyclin-101 TFA

inhibits the signal transduction mediated by TLR4 and TLR2, reducing the expression of pro-inflammatory cytokines [1] [2] [3] [4] [5].

In Vivo, Retrocyclin-101 (vaginal; 8 days) TFA maintains stability and bioactivity for at least 8 days following in vivo vaginal administration in pigtailed macaques[4]. Retrocyclin-101 (100 μg/mouse; i.v.; daily; days 2-6 post-infection) TFA confers significant protection against influenza A virus (PR8) infection in mice, increasing survival and reducing clinical severity when administered therapeutically starting 2 days post-infection[5].

In Vitro, Retrocyclin-101 (50 μM; 1 h) TFA inhibits the infection of H1299 cells by SARS-CoV-2 spike protein-pseudotyped lentivirus, but this effect is only observed when it is pre-incubated with viral particles (no such effect occurs when pre-incubated with cells), and serum abrogates this effect[1]. Retrocyclin-101 (0.5-5 μM; 1 h) TFA inhibits the binding of recombinant prefusion-stabilized SARS-CoV-2 spike protein to ACE2 in a concentration-dependent manner, and reduces the binding rate to approximately 25% of that in the control group at 5 μM after pre-incubation at 37°C[1]. Retrocyclin-101 (3-50 μM; 1-24 h) TFA inhibits natural SARS-CoV-2 infection in Vero E6 cells; its IC_{50} is 29 μM without pre-incubation with the virus; when pre-incubated with the virus for 1 h under serum-free conditions, it exerts strong inhibitory activity at a concentration of 50 μM[1]. Retrocyclin-101 (0-50 μM; 47 h) TFA potently inhibits ZIKV PRVABC59 infection in Vero cells, with an IC_{50} of 7.033 μM[2]. Retrocyclin-101 (0-50 μM; 71 h) TFA inhibits ZIKV MR766 infection in Vero cells, with an IC_{50} of 15.58 μM[2]. Retrocyclin-101 (0-100 μM) TFA significantly reduces the viral titers of ZIKV PRVABC59 and MR766 in Vero cells and U251 glioma cells in a dose-dependent manner[2]. Retrocyclin-101 (40 μM; 48-72 h) TFA inhibits infections by ZIKV PRVABC59 and MR766 in Vero cells during the viral entry and replication stages, but does not act through direct virucidal activity or blocking cellular receptors[2]. Retrocyclin-101 (25-50 μM; 50 h) TFA inhibits ZIKV replication in BHK-21 cells but has no effect on the translation of initial replicon RNA[2]. Retrocyclin-101 (0-50 μM; 24 h) TFA inhibits the activity of JEV NS2B-NS3 serine protease in BHK-21 cells, reduces the expression level of NS3 and increases the content of unprocessed polyprotein precursors at lower concentrations[2]. Retrocyclin-101 (0-50 μM; 24 h) TFA inhibits JEV AT31 infection in BHK-21 and U251 cells, with an IC_{50} of 10.67 μM[2]. Retrocyclin-101 (50 μM; 24 h) TFA inhibits wild-type (WT), N154A, H144A, and Q258A strains

of JEV in BHK-21 cells, whereas the DE mutant and T410A JEV strains are resistant to RC-101[2]. Retrocyclin-101 TFA can be cleaved from its GFP fusion partner in vitro by Factor Xa protease; partial cleavage occurs naturally in chloroplast extracts of plastid-transformed tobacco due to endogenous Factor Xa-like activity[3]. Retrocyclin-101 (2.5-20 μM; 16 h) TFA potently inhibits the growth of Staphylococcus aureus nasal colonizing strains D20-7, D535-6, D30 and clinical strain USA300 in a concentration-dependent manner; specifically, at a concentration of 20 μM, the growth inhibition duration of nasal colonizing strains exceeds 10 h, and that of USA300 reaches 5 h[4]. Retrocyclin-101 (2.5-10 μM; 0.25-9 h) TFA is a rapidly acting, concentration-dependent bactericide active against Staphylococcus aureus nasal carriage strains D20-7, D535-6, D30 and clinical strain USA300; it achieves complete growth inhibition within 30 min at 10 μM and retains activity even against high bacterial inocula[4]. Retrocyclin-101 (1-10 μg/tissue; 9 h) TFA inhibits the adhesion and survival of Staphylococcus aureus nasal colonization strains D20-7, D535-6, D30 and clinical strain USA300 on human nasal epithelial cells (RPMI 2650), with 10 μg/tissue completely blocking bacterial adhesion[4]. Retrocyclin-101 (4-20 μg/tissue; 9 h) TFA completely inhibits the adhesion of S. aureus nasal colonization strain D20-7 to organotypic human airway epithelium at a concentration of 20 μg/tissue[4]. Retrocyclin-101 (30 μg/mL; 30-60 min) TFA inhibits TRIF- and MyD88-dependent TLR4-mediated proinflammatory cytokine gene expression in human monocyte-derived macrophages[5].

References:

[1]. Kudryashova E, et al. Inhibition of SARS-CoV-2 Infection by Human Defensin HNP1 and Retrocyclin RC-101. J Mol Biol. 2022;434(6):167225.
[2]. Jia X, et al. Mechanism through Which Retrocyclin Targets Flavivirus Multiplication. J Virol. 2021;95(15):e0056021.
[3]. Lee SB, et al. Expression and characterization of antimicrobial peptides Retrocyclin-101 and Protegrin-1 in chloroplasts to control viral and bacterial infections. Plant Biotechnol J. 2011;9(1):100-115.
[4]. Lamers RP, et al. Characterization of the retrocyclin analogue RC-101 as a preventative of Staphylococcus aureus nasal colonization. Antimicrob Agents Chemother. 2011;55(11):5338-5346.
[5]. Prantner D, et al. The θ-defensin retrocyclin 101 inhibits TLR4- and TLR2-dependent signaling and protects mice against influenza infection. J Leukoc Biol. 2017 Oct;102(4):1103-1113.