

Product Data Sheet

Product Name: T7 Peptide TFA

Cat. No.: GC79141

Chemical Properties

Cas. No.

Formula $C_{137}H_{189}N_{33}O_{39}S_3 \cdot xC_2HF_3O_2$ M.Wt 3018.36

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

Thr-Met-Pro-Phe-Leu-Phe-Cys-Asn-Val-
Asn-Asp-Val-Cys-Asn-Phe-Ala-Ser-Arg-
Asn-Asp-Tyr-Ser-Tyr-Trp-Leu (Disulfide
bridge: Cys7-Cys13) (TFA salt)

Background

T7 Peptide TFA is a protein synthesis inhibitor and anti-angiogenic agent, with a K_d of 10 nM for human transferrin receptor. T7 Peptide TFA inhibits the phosphorylation of focal adhesion kinase, the activation of phosphatidylinositol 3-kinase and Akt, the kinase activity of mTOR, as well as the phosphorylation of 4E-BP1 in endothelial cells. T7 Peptide TFA induces G0/G1 cell cycle arrest, apoptosis and protective autophagy in hepatocellular carcinoma cells, and suppresses tumor growth in mouse models. T7 Peptide TFA is applicable to research related to cancer, glioblastoma,

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hepatocellular carcinoma and glioma [1] [2] [3] [4].

In Vivo, T7 peptide TFA (2.5-5 mg/kg; i.p.; every other day) dose-dependently suppresses Hep3B xenograft tumor growth in nude mice, achieving a ~27% tumor cell apoptotic rate at the 5 mg/kg dose via pathways involving reduced Akt/mTOR phosphorylation, increased Bax expression, and decreased Bcl-2 expression^[3]. T7 peptide TFA (4% molar ratio; 0.5 mg/kg; intravenous tail vein injection) conjugation to LDL particles enhances accumulation in intracranial glioma tissue in female ICR mice bearing C6 glioma^[4].

In Vitro, T7 peptide TFA (4.5 μ M; 21 h) specifically inhibits cap-dependent translation in C-PAE cells without affecting cap-independent translation^[1]. T7 peptide (4.5 μ M) inhibits total protein synthesis in MLEC-wt cells^[1]. T7 peptide TFA (4.5 μ M) does not inhibit total protein synthesis in MLEC- β 3 null cells, indicating dependence on α V β 3 integrin for this activity^[1]. T7 peptide TFA (4.5 μ M) does not inhibit total protein synthesis in MEF-wt cells, indicating α V β 3 integrin expression is essential but not sufficient for this activity^[1]. T7 peptide TFA (4.5 μ M) does not inhibit total protein synthesis in non-endothelial cell lines, demonstrating endothelial cell specificity for this activity^[1]. The T7 peptide TFA (0.25-2 μ M; 6-24 h) significantly reduces viability of Huh-7 and Hep3B human HCC cells in a concentration- and time-dependent manner, but has minimal effect on normal human liver L-02 cells^[3]. The T7 peptide TFA (0.25-1 μ M; 24 h) induces significant apoptosis (both early and late) in Huh-7 and Hep3B human HCC cells^[3]. The T7 peptide TFA (0.25-1 μ M; 24 h) induces G0/G1 cell cycle arrest in Huh-7 and Hep3B human HCC cells^[3]. The T7 peptide TFA (0.5-1 μ M; 24 h) upregulates pro-apoptotic Bax, Fas, and FasL and downregulates anti-apoptotic Bcl-2 in a concentration-dependent manner in Huh-7 and Hep3B human HCC cells^[3]. The T7 peptide TFA (0.5-1 μ M; 24 h) induces autophagy in Huh-7 and Hep3B human HCC cells by upregulating Beclin-1, Atg5, and LC3-II in a concentration-dependent manner^[3]. The T7 peptide TFA (1 μ M; 24 h) has enhanced apoptotic effect in Huh-7 and Hep3B human HCC cells when autophagy is inhibited^[3]. The T7 peptide TFA (0.5-1 μ M; 24 h) induces autophagy in Huh-7 and Hep3B human HCC cells via inhibition of the Akt/mTOR signaling pathway, and co-treatment with Akt/mTOR inhibitors enhances this autophagic response^[3]. T7 peptide (5 μ M; 2 h at 37 °C) modified LDL shows enhanced cellular uptake in bEnd.3 and C6 cells via synergistic mediation by TfR and LDLR^[4]. T7 peptide TFA (0.5-

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6% molar ratio; 4 h at 37 °C) modification enhances BBB penetration and subsequent C6 glioma cell uptake, with optimal efficacy observed at a 4% molar ratio of T7 to LDL^[4].

References:

- [1]. Maeshima Y, et al. Tumstatin, an endothelial cell-specific inhibitor of protein synthesis. Science. 2002 Jan 4;295(5552):140-3.
- [2]. Liang M, et al. Enhanced blood-brain barrier penetration and glioma therapy mediated by T7 peptide-modified low-density lipoprotein particles. Drug Deliv. 2018;25(1):1652-1663.
- [3]. Liu F, et al. T7 peptide cytotoxicity in human hepatocellular carcinoma cells is mediated by suppression of autophagy. Int J Mol Med. 2019;44(2):523-534.
- [4]. Kim G, et al. Systemic delivery of microRNA-21 antisense oligonucleotides to the brain using T7-peptide decorated exosomes. J Control Release. 2020;317:273-281.

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