Disitertide

Cat. No.:	HY-P0118
CAS No.:	272105-42-7
Molecular Formula:	$C_{68}H_{109}N_{17}O_{22}S_2$
Molecular Weight:	1580.82
Sequence Shortening:	TSLDASIIWAMMQN
Target:	TGF-beta/Smad; PI3K; Apoptosis
Pathway:	Stem Cell/Wnt; TGF-beta/Smad; PI3K/Akt/mTOR; Apoptosis
Storage:	Sealed storage, away from moisture and light, under nitrogen Powder -80°C 2 years -20°C 1 year * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light, under nitrogen)

BIOLOGICAL ACTIVITY

Description

Disitertide (P144) is a peptidic transforming growth factor-beta 1 (TGF-β1) inhibitor specifically designed to block the interaction with its receptor. Disitertide (P144) is also a PI3K inhibitor and an apoptosis inducer[1][2][3][4][5].

In Vitro

Disitertide (P144, 100 μg/mL) suppresses the protein expression levels of PI3K and p-Akt, and induce the protein expression of Bax in MC3T3-E1 cells[2].

Disitertide (TGF-β1 inhibitor) abrogates the MACC1- AS1 expression in GC cells, suggesting that targeting TGFβ signaling pathway may be a potential strategy to inhibit MSC-induced stemness and chemoresistance[3].

Disitertide (10 μg/mL to 200 μg/mL) affects proliferation, induces apoptosis as well as anoikis in A172 and U-87 MG GBM cell lines[5].

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

Western Blot Analysis[2]

Cell Line:	Mouse embryo osteoblast precursor MC3T3-E1 cells
Concentration:	100 μg/mL
Incubation Time:	4 h
Result:	Significantly suppressed the protein expression levels of PI3K and p-Akt, and induce the protein expression of Bax in MC3T3-E1 cells compared with the miR-590 group

In Vivo

Disitertide (Topical application, 300 μg/mL) may promote scar maturation and improvement of hypertrophic scar morphology features in an "in vivo" model in nude mice after two weeks of treatment[4].

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Animal Model:	Human hypertrophic scars were implanted in 60 nude mice[4].
Dosage:	300 μg/mL was added the Lipogel.

Administration:	Topical application daily administered.
Result:	Successful shedding was achieved in 83,3% of the xenografts.

CUSTOMER VALIDATION

- Cell Death Differ. 2021 Jan;28(1):219-232.
- J Exp Clin Cancer Res. 2021 Feb 9;40(1):62.
- Oncogene. 2019 Jun;38(23):4637-4654.
- Front Immunol. 2017 Feb 3;8:91.
- Cells. 2019 Jun 25;8(6):635.

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REFERENCES

[1]. Cindy Neuzillet, et al. Targeting the TGFβ pathway for cancer therapy. Pharmacol Ther. 2015 Mar;147:22-31.

[2]. Jun Yang, et al. Upregulation of microRNA‐590 in rheumatoid arthritis promotes apoptosis of bone cells through transforming growth factor‐β1/phosphoinositide 3‐kinase/Akt signaling. Int J Mol Med. 2019 May;43(5):2212-2220.

[3]. Wanming He, et al. MSC-regulated lncRNA MACC1-AS1 promotes stemness and chemoresistance through fatty acid oxidation in gastric cancer. Oncogene. 2019 Jun;38(23):4637-4654.

[4]. Shan Shan Qiu, et al. Effect of P144® (Anti-TGF-β) in an "In Vivo" Human Hypertrophic Scar Model in Nude Mice. PLoS One. 2015 Dec 31;10(12):e0144489.

[5]. Gabriel Gallo-Oller, et al. P144, a Transforming Growth Factor beta inhibitor peptide, generates antitumoral effects and modifies SMAD7 and SKI levels in human glioblastoma cell lines. Cancer Lett. 2016 Oct 10;381(1):67-75.

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