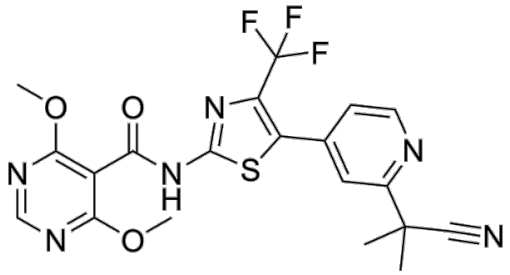
Product Data Sheet

Product Name: Trpvicin
Cat. No.: GC79530

Chemical Properties

Cas. No.	2019994-90-0		
Formula	$C_{20}H_{17}F_3N_6O_3S$	M.Wt	478.45
Solubility	DMSO: 25 mg/mL (52.25 mM; Need ultrasonic)	Storage	Store at -20°C
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

Trpvicin is a potent and subtype-selective TRPV3 inhibitor with IC_{50} s of 0.41 and 0.22 μM for hTRPV3-WT and hTRPV3-G573S mutant, respectively. Trpvicin exhibits minimal effects on other TRP family members (such as TRPV1/2/4/5/6, TRPA1 and TRPM8). Trpvicin inhibits the TRPV3

channel by stabilizing it in a closed state via VSLD-PD binding. Trpvicin accesses additional binding sites inside the central cavity of the G573S mutant to remodel symmetry and block the channel. Trpvicin inhibits itch and hair loss in mouse models. Trpvicin can be used for study of inflammation and immunology [1].

In Vivo, Trpvicin (10 and 100 μM, i.d., 30 min pre-SLIGRL) significantly reduces acute scratching in mice induced by SLIGRL [1]. Trpvicin (30 and 100 mg/kg, p.o., daily starting from 5 days before MC903 for 12 days) alleviates chronic scratching and ear swelling in an MC903 -induced dermatitis mice model[1]. Trpvicin (1 wt%, topically, daily from postnatal day 50 for 16 days) effectively rescues hair loss in both male and female Trpv3+/G568V knock-in mice[1].

In Vitro, Trpvicin (0-100 μM) dose-dependently inhibits the currents of TRPV3-WT and hTRPV3-G573S expressed in HEK293T cells, achieving nearly complete inhibition at 10 μM, with potency comparable to Ruthenium Red [1]. Trpvicin (10 μM, 24 h) rescues the cell viability of HEK293T cells expressing the cytotoxic hTRPV3-G573S mutant, demonstrating its functional blockade of the constitutively active channel[1]. Trpvicin (0-100 μM) dose-dependently inhibits the currents of hTRPV3-G568V and mTRPV3-G568V heterologously expressed in HEK293T cells[1]. Trpvicin potently inhibits both wild-type mouse TRPV3 (mTRPV3-WT) and the gain-of-function mutant mTRPV3-G568V heterologously expressed in HEK293T cells, with IC_{50} values of 0.38 μM and 0.42 μM, respectively[1]. Trpvicin exhibits a dramatic loss in potency (158-, 162-, and 208-fold) against the hTRPV3 A556V, A560T, and F601A mutants compared to the wild-type channel, suggesting that these residues are the key determinants for Trpvicin binding[1]. Trpvicin exhibits a 17-fold, 12-fold, and 16-fold decrease in potency against hTRPV3-G573S-F666A, hTRPV3-G573S-F666Y and hTRPV3-G573S-T665A double mutants, respectively, suggesting that these residues are functionally important for drug binding in the central cavity, beyond its primary VSLD-PD binding site[1].

References:
[1]. Fan J, et al. Structural basis of TRPV3 inhibition by an antagonist. Nat Chem Biol. 2023 Jan;19(1):81-90.