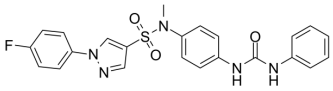
BTT-3033

Cat. No.:	HY-110112
CAS No.:	1259028-99-3
Molecular Formula:	$C_{23}H_{20}FN_5O_3S$
Molecular Weight:	465.5
Target:	Integrin; Apoptosis
Pathway:	Cytoskeleton; Apoptosis
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (537.06 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1482 mL	10.7411 mL	21.4823 mL
	5 mM	0.4296 mL	2.1482 mL	4.2965 mL
	10 mM	0.2148 mL	1.0741 mL	2.1482 mL

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

BIOLOGICAL ACTIVITY

Description

BTT-3033 is an orally active conformation-selective inhibitor of α2β1 (EC50: 130 nM) by binding to the α2I domain. BTT-3033 inhibits platelet binding to collagen Ⅰ and cell proliferation, and induces cell apoptosis. BTT-3033 can be used in the research of prostate cancer, inflammation and cardiovascular disease[1][2][4].

IC_{50} & Target

α2β1
130 nM (EC50)

In Vitro

BTT-3033 (1 nM-100 μM, 2 h) inhibits CHO-α2wt cell adhesion to rat tail collagen Ⅰ (EC_{50}: 130 nM), exhibits selectivity for α2β1 over α3β1, α4β1, α5β1 and αv[1].

BTT-3033 (10 μM, 5 min) inhibits human platelet binding to collagenⅠcoated capillaries under flow, with the EC_{50} value for mouse whole blood to be 6 μM[1].

BTT-3033 (10 μM, 5 min) inhibits binding of α2-expressing CHO cells to collagen Ⅰ under shear stress conditions[1].

BTT-3033 (1 μM, 60 min) inhibits of neurogenic and thromboxane A2Ⅰinduced human prostate smooth muscle contraction [3].

BTT-3033 (25 and 50 μM, 48 h) inhibits cell viability and proliferation by inducing G1 cell cycle arrest in LNcapⅠFGC, and DUⅠ145 cells[4].

BTT-3033 (50 μM, 48 h) induces apoptosis through the activation of ROS, Bax protein upregulation, caspase‐3 activation, and depletion of ΔΨm[4].
BTT-3033 (10 μM, 15/28 days) suppresses MMP13 expression, increases the expression of MMP1 and MT-MMP1 in human articular cartilage‐derived chondrocytes[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[4]

Cell Line:	LNcap‐FGC, and DU‐145 cells
Concentration:	0.05, 0.5 5, 25, and 50 μM
Incubation Time:	48 h
Result:	Decreased the cell viability at 25 μM and 50 μM.

Cell Viability Assay[4]

Cell Line:	LNcap‐FGC, and DU‐145 cells
Concentration:	5, 25, and 50 μM
Incubation Time:	48 h
Result:	Induced cell apoptosis about 20%, 32%, and 47% (LNcap‐FGC) and 26%, 41%, and 59% (DU‐145) at 5, 25, and 50 μM.

Western Blot Analysis[4]

Cell Line:	LNcap‐FGC, and DU‐145 cells
Concentration:	25 μM
Incubation Time:	48 h
Result:	Resulted in down-regulation of N‐cadherin and upregulation of E‐cadherin (EMT‐associated proteins).

In Vivo

BTT-3033 (oral administration, 10 mg/kg, at 24 h and 2 h before PAF induction) shows anti-inflammatory effects in mouse air pouch model[2].
BTT-3033 (oral administration, 10 mg/kg, at 48 ,24 and 2 h before ear swelling) shows anti-inflammatory effects in arachidonic acid-induced ear edema model[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	PAF (platelet-activating factor)-induced mouse air pouch model[2]
Dosage:	1, 10 mg/kg at 24 h and 2 h before PAF induction
Administration:	Oral administration
Result:	Reduced the infiltration of leukocytes by about 50% at 10 mg/kg.

Animal Model:	Male DBA/1 mice (Pharmacokinetic assay)[2]
Dosage:	10 mg/kg for a single dose
Administration:	Oral administration

Result:	Plasma levels: about 1 ng/mL at 24 h post-dose.

REFERENCES

[1]. Liisa Nissinen, et al. Novel α2β1 integrin inhibitors reveal that integrin binding to collagen under shear stress conditions does not require receptor preactivation. J Biol Chem. 2012 Dec 28;287(53):44694-702.

[2]. Liisa Nissinen, et al. Sulfonamide inhibitors of α2β1 integrin reveal the essential role of collagen receptors in in vivo models of inflammation. Pharmacol Res Perspect. 2015 Jun;3(3):e00146.

[3]. Bingsheng Li, et al. Inhibition of neurogenic and thromboxane A 2 -induced human prostate smooth muscle contraction by the integrin α2β1 inhibitor BTT-3033 and the integrin-linked kinase inhibitor Cpd22. Prostate. 2020 Aug;80(11):831-849.

[4]. Zahra Salemi, et al. Integrin α2β1 inhibition attenuates prostate cancer cell proliferation by cell cycle arrest, promoting apoptosis and reducing epithelial-mesenchymal transition. J Cell Physiol. 2021 Jul;236(7):4954-4965.

[5]. Takashi Kanamoto, et al. Integrin α2β1 plays an important role in the interaction between human articular cartilage-derived chondrocytes and atelocollagen gel. Sci Rep. 2021 Jan 19;11(1):1757.

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