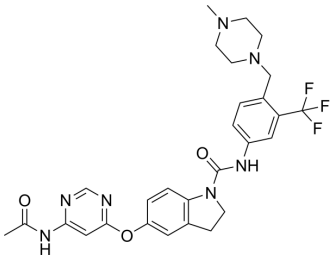
BBT594

Cat. No.:	HY-18840
CAS No.:	882405-89-2
Molecular Formula:	$C_{28}H_{30}F_3N_7O_3$
Molecular Weight:	569.58
Target:	RET
Pathway:	Protein Tyrosine Kinase/RTK
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 33 mg/mL (57.94 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	1.7557 mL	8.7784 mL	17.5568 mL
	5 mM	0.3511 mL	1.7557 mL	3.5114 mL
	10 mM	0.1756 mL	0.8778 mL	1.7557 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (4.39 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (4.39 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.39 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BBT594 is a potent receptor tyrosine kinase RET inhibitor, used for cancer treatment.

In Vitro

NVP-BBT594 blocks the GDNF-mediated enhancement of MCF7-LTED cell viability in 2D culture and 3D colony formation. The addition of 10 pM E2, to mimic the E2 level in post-menopausal patients that have relapsed on AI treatment and ceased AI therapy, increases 3D colony formation of both MCF7 and MCF7-LTED cells, and this effect is efficiently reverted by NVP-BBT594. Parental T47D cells cultured in presence of low level E2, GFRα1/GDNF stimulation results in increased 3D colony formation, which is significantly reverted by NVP-BBT594. NVP-BBT594 targets GDNF-RET signaling and sensitizes MCF7-2A

cells to letrozole treatment. NVP-BBT594 impairs GDNF-mediated RET downstream signaling and significantly enhances the antiproliferative effects of letrozole[1]. NVP-BBT594 shows the highest suppression of GDNF-induced RET signaling, as assessed by RET, ERK1/2, AKT and ER phosphorylation. NVP-AST487 and NVP-BBT594 have comparable RET inhibitory activity in wild-type MCF7 cells[2].

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

CUSTOMER VALIDATION

- Nat Metab. 2021 Apr;3(4):513-522.
- University of London. 2022 May.
- Nat Metab. 2021 Apr 12.

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REFERENCES

[1]. Morandi A, et al. GDNF-RET signaling in ER-positive breast cancers is a key determinant of response and resistance to aromatase inhibitors. Cancer Res. 2013 Jun 15;73(12):3783-95

[2]. Andreucci E, et al. Targeting the receptor tyrosine kinase RET in combination with aromatase inhibitors in ER positive breast cancer xenografts. Oncotarget. 2016 Sep 2. doi: 10.18632/oncotarget.11826. [Epub ahead of print]

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