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- Mindermengenzuschlag
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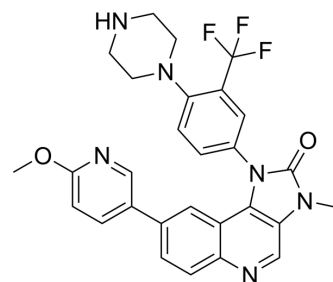
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BGT226

Cat. No.:	HY-13334A		
CAS No.:	915020-55-2		
Molecular Formula:	C ₂₈ H ₂₅ F ₃ N ₆ O ₂		
Molecular Weight:	534.53		
Target:	PI3K; mTOR; Autophagy		
Pathway:	PI3K/Akt/mTOR; Autophagy		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	6 months
		-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro	DMSO : 10 mg/mL (18.71 mM; ultrasonic and warming and heat to 60°C)					
	Preparing Stock Solutions	Solvent Concentration	Mass	1 mg	5 mg	10 mg
		1 mM		1.8708 mL	9.3540 mL	18.7080 mL
		5 mM		0.3742 mL	1.8708 mL	3.7416 mL
		10 mM		0.1871 mL	0.9354 mL	1.8708 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: ≥ 1 mg/mL (1.87 mM); Clear solution					
	2. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 1 mg/mL (1.87 mM); Clear solution					

BIOLOGICAL ACTIVITY

Description	BGT226 (NVP-BGT226) is a PI3K (with IC ₅₀ s of 4 nM, 63 nM and 38 nM for PI3Kα, PI3Kβ and PI3Kγ)/mTOR dual inhibitor which displays potent growth-inhibitory activity against human head and neck cancer cells ^{[1][2]} .			
IC ₅₀ & Target	PI3Kα 4 nM (IC ₅₀)	PI3Kβ 63 nM (IC ₅₀)	PI3Kγ 38 nM (IC ₅₀)	mTOR
	Autophagy			
In Vitro	BGT226 shows significant growth inhibition or signal blockage profiles compared with LY294002 and Rapamycin. BGT226 (10-10000 nM) inhibits FaDu and OECM1 cells growth with IC ₅₀ s of 23.1±7.4 and 12.5±5.1 nM, respectively ^[2] .			

The expression levels of p-mTOR Ser2481 are decreased in BGT226-treated cell lines (200 nM; 24 hours) and both p-AKT Ser473 and p-mTOR Ser2448 are also decreased in BGT226-treated cell lines^[2].

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

Cell Viability Assay^[2]

Cell Line:	FaDu cells; OECM1 cells
Concentration:	10, 100, 1000, 10000 nM
Incubation Time:	
Result:	Inhibited FaDu and OECM1 cells growth with IC ₅₀ s of 23.1±7.4 and 12.5±5.1 nM, respectively.

Western Blot Analysis^[2]

Cell Line:	FaDu cells; OECM1 cells
Concentration:	200 nM
Incubation Time:	24 hours
Result:	p-mTOR Ser2481 expression levels decreased, and both p-AKT Ser473 and p-mTOR Ser2448 expression levels also decreased.

In Vivo

BGT226 (2.5 and 5 mg/kg; oral administration for 21 days in male athymic mice) causes 34.7% and 76.1% reduction of the tumor growth on day 21 compared with control^[2].

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

Animal Model:	Male athymic mice (strain BALB/cAnN.Cg-Foxn1nu/CrINarl) with FaDu cell xenografted mouse model ^[2]
Dosage:	2.5 and 5 mg/kg
Administration:	Oral administration; 21 days
Result:	Caused 34.7% and 76.1% reduction of the tumor growth.

CUSTOMER VALIDATION

- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.
- Front Pharmacol. 2020 Nov 11;11:580407.
- Molecules. 2020 Apr 23;25(8):1980.
- Research Square Print. 2023 Mar 9.
- Harvard Medical School LINCS LIBRARY

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REFERENCES

[1]. Markman B, et al. Phase I safety, pharmacokinetic, and pharmacodynamic study of the oral phosphatidylinositol-3-kinase and mTOR inhibitor BGT226 in patients with

advanced solid tumors. Ann Oncol. 2012 Sep;23(9):2399-408.

[2]. Chang KY, et al. Novel phosphoinositide 3-kinase/mTOR dual inhibitor, NVP-BGT226, displays potent growth-inhibitory activity against human head and neck cancer cells in vitro and in vivo. Clin Cancer Res. 2011 Nov 15;17(22):7116-26.

Caution: Product has not been fully validated for medical applications. For research use only.

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