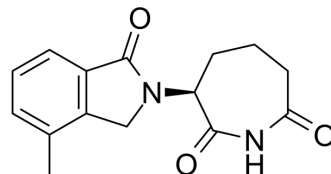


BTX161

Cat. No.:	HY-120084		
CAS No.:	2052301-24-1		
Molecular Formula:	C ₁₅ H ₁₆ N ₂ O ₃		
Molecular Weight:	272.3		
Target:	Casein Kinase		
Pathway:	Cell Cycle/DNA Damage; Stem Cell/Wnt		
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 : 25 mg/mL (91.81 mM); ultrasonic and warming and heat to 60°C)

Concentration	Solvent	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM	3.6724 mL	18.3621 mL	36.7242 mL
	5 mM	0.7345 mL	3.6724 mL	7.3448 mL
	10 mM	0.3672 mL	1.8362 mL	3.6724 mL

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (9.18 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 2.5 mg/mL (9.18 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (9.18 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

BTX161, a Thalidomide analog, is a potent CK1α degrader. BTX161 mediates degradation of CK1α better than Lenalidomide in human AML cells and activates DNA damage response (DDR) and p53, while stabilizing the p53 antagonist MDM2^[1].

In Vitro

BTX161 (25 μM; 4 hours; MV4-11 cells) upregulates all the Wnt targets including MYC and did not affect MDM2 mRNA expression^[1].
BTX161 (10 μM; 6 hours; MV4-11 cells), on its own, augmented p53 and MDM2 protein expression, yet in combination with THZ1, and particularly with both THZ1 and CDK9, further augmented p53 and induced maximal caspase 3 activation^[1].

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

REFERENCES

[1]. Minzel W, et al. Small Molecules Co-targeting CKI α and the Transcriptional Kinases CDK7/9 Control AML in Preclinical Models. Cell. 2018;175(1):171-185.e25.

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

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