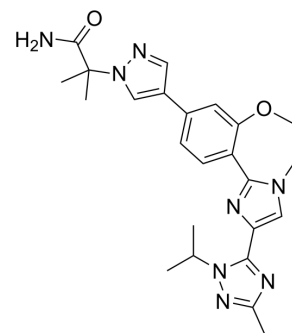


Taselisib

Cat. No.:	HY-13898		
CAS No.:	1282512-48-4		
Molecular Formula:	C ₂₄ H ₂₈ N ₈ O ₂		
Molecular Weight:	460.53		
Target:	PI3K		
Pathway:	PI3K/Akt/mTOR		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	1 year
		-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro	DMSO : 25 mg/mL (54.29 mM; ultrasonic and warming and heat to 60°C)				
		Solvent Concentration	Mass 1 mg	5 mg	10 mg
	Preparing Stock Solutions	1 mM	2.1714 mL	10.8571 mL	21.7141 mL
		5 mM	0.4343 mL	2.1714 mL	4.3428 mL
10 mM		0.2171 mL	1.0857 mL	2.1714 mL	
Please refer to the solubility information to select the appropriate solvent.					
In Vivo	<ol style="list-style-type: none"> Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.5 mg/mL (5.43 mM); Clear solution Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: ≥ 2.5 mg/mL (5.43 mM); Clear solution Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (5.43 mM); Clear solution 				

BIOLOGICAL ACTIVITY

Description	Taselisib (GDC-0032) is a potent PI3K inhibitor targets PIK3CA mutations, with K _i s of 0.12 nM, 0.29 nM, 0.97 nM, and 9.1 nM for PI3Kδ, PI3Kα, PI3Kγ and PI3Kβ, respectively.			
IC₅₀ & Target	PI3Kδ 0.12 nM (K _i)	PI3Kα 0.29 nM (K _i)	PI3Kγ 0.97 nM (K _i)	PI3Kβ 9.1 nM (K _i)
In Vitro	Taselisib (GDC-0032) (100 nM) inhibits AKT/mTOR signaling in PIK3CA mutant cell lines but not in cells with loss or mutation			

of PTEN; Tselisib (GDC-0032) enhances radiation-induced apoptosis and inhibits growth in head and neck cancer cell lines that are sensitive to its single-agent activity^[1]. Tselisib (GDC-0032) enhances the effects of MEK1/2 inhibition on both BRAF V600E/PTEN^{Null} human melanoma cells autochthonous mouse melanomas^[2].

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

In Vivo

Tselisib (GDC-0032) (5 mg/kg, p.o.) potently impairs PI3K signaling and enhances the efficacy of fractionated radiotherapy; Tselisib (GDC-0032) and radiation is more effective than either treatment alone in nude mice implanted with subcutaneous Cal-33 xenografts^[1]. The vehicle-treated BRAFV600E/PTEN^{Null} melanoma-bearing mice experiences initial tumor regression after treatment with Tselisib (GDC-0032) (22.5 mg/kg, p.o.)^[2].

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

PROTOCOL

Cell Assay ^[1]

Cells are seeded in replicates of 6 in 96-well plates with 500 to 5,000 cells/well overnight and then treated with Tselisib (GDC-0032). After 4 days, the media are removed and the cells are fixed with 4% glutaraldehyde for 30 minutes. Fixed cells are stained with 0.1% crystal violet for 2 minutes, then washed, and dissolved in 10% acetic acid.

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

Animal Administration ^[1]

Six-week-old Nu/Nu mice are injected bilaterally with 5×10^5 cells resuspended in 200 μ L of culture media and Matrigel mixed in a 1:1 ratio. After tumors reach approximately 100 to 200 cm^3 , mice are randomized into treatment arms with 8 to 10 tumors per group. Tselisib (GDC-0032) (5 mg/kg) is dissolved in a vehicle containing 0.5% methylcellulose with 0.2% TWEEN-80 and is administered via daily oral gavage.

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

CUSTOMER VALIDATION

- Nature. 2018 Aug;560(7719):499-503.
- Cell Discov. 2016 Sep 20;2:16030.
- Nat Cancer. 2024 Jul 11.
- J Clin Invest. 2021 Dec 15;131(24):e140436.
- Biosens Bioelectron. 2020 Aug 1;161:112240.

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REFERENCES

[1]. Zachary S. Zumsteg, et al. Tselisib (GDC-0032), a Potent β -Sparing Small Molecule Inhibitor of PI3K, Radiosensitizes Head and Neck Squamous Carcinomas Containing Activating PIK3CA Alterations. Clin Cancer Res. 2016 Apr 15; 22(8): 2009–2019.

[2]. Marian M. Deuker, et al. PI3'-Kinase Inhibition Forestalls the Onset of MEK1/2 Inhibitor Resistance in BRAF-Mutated Melanoma. Cancer Discov. 2015 Feb; 5(2): 143–153.

[3]. Ndubaku CO, et al. Discovery of 2-[3-[2-(1-isopropyl-3-methyl-1H-1,2,4-triazol-5-yl)-5,6-dihydrobenzo[f]imidazo[1,2-d][1,4]oxazepin-9-yl]-1H-pyrazol-1-yl]-2-methylpropanamide (GDC-0032): a β -sparing phosphoinositide 3-kinase inhibitor with high unbound exposure and robust in vivo antitumor activity. J Med Chem. 2013 Jun 13;56(11):4597-610.

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

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