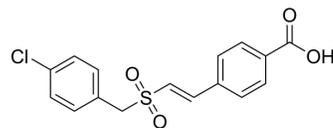


Recilisib

Cat. No.:	HY-101625		
CAS No.:	334969-03-8		
Molecular Formula:	C ₁₆ H ₁₃ ClO ₄ S		
Molecular Weight:	336.79		
Target:	Akt; 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 : 35.71 mg/mL (106.03 mM; Need ultrasonic)				
		Solvent Concentration	Mass 1 mg	5 mg	10 mg
	Preparing Stock Solutions	1 mM	2.9692 mL	14.8460 mL	29.6921 mL
		5 mM	0.5938 mL	2.9692 mL	5.9384 mL
10 mM		0.2969 mL	1.4846 mL	2.9692 mL	
Please refer to the solubility information to select the appropriate solvent.					
In Vivo	1. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.08 mg/mL (6.18 mM); Clear solution				

BIOLOGICAL ACTIVITY

Description	Recilisib (ON 01210) is a radioprotectant, which can activate AKT, PI3K activities in cells ^[1] .
IC ₅₀ & Target	PI3K
In Vitro	Recilisib (up to 50 μM) shows a normal distribution of cells throughout the cell cycle, with a slight reduction in the number of cells in S-phase at 50 μM. Continuous exposure of Recilisib (100 μM) does not result in cell death. Recilisib treatment does not inhibit the colony forming potential of human bone marrow cells. Recilisib provides dose dependent protection of human bone marrow cells at all three doses of IR. Recilisib activates the phosphorylation of AKT and GSK3α/β in HFL cells. Recilisib increases PI3K activity in HFL-1 cells and murine bone marrow cells in response to radiation exposure. Recilisib treatment in combination with radiation alters the MAPK signaling pathway ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Recilisib (500 mg/kg) significantly increases the rate of recovery and differentiation of primitive bone marrow myeloid progenitor cells in mice. Recilisib in combination with radiation reduces CFU numbers in mice, but the Recilisib-treated mice consistently retain a capability to form differentiated colonies. Recilisib treated mice have a progenitor cell population that is never completely depleted by radiation exposure^[1].

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

PROTOCOL

Kinase Assay ^[1]

PI3-kinase assays are performed using exponentially growing HFL-1 or freshly harvested murine bone marrow cells that are treated with increasing concentrations of Recilisib Sodium for 2 hours and then irradiated with 10 Gy IR. These cells are then returned to the incubator for 2 to 24 hours and lysed in HEPES pH 7.5 lysis buffer. PI-3K is immunoprecipitated using an anti-PI3 Kinase polyclonal antibody for 2 hours at 4°C. Protein A/G PLUS-Agarose is incubated with immunoprecipitates for 8-16 hours at 4°C and the resulting immunoprecipitates washed with twice HEPES pH 7.5 lysis buffer and once with the kinase buffer (20 mM Tris pH 7.5, 1mM EGTA, 10mM MgCl₂, 2 mM DTT, 0.01% NP-40). L- α -Phosphatidylinositol (12.5 mM) and ATP (10 μ M) are added to the kinase buffer (60 μ L per sample) and incubated at 30°C for 30 minutes. The reaction is stopped by addition of 100 μ L of 1N HCl and extracted by addition of 200 μ L CHCl₃/CH₃OH (1:1). The extracted samples are vortexed, centrifuged and the lower organic phases containing phospholipids are dried at 27°C for 2 hours. The dried samples are resuspended in 10 μ L of PI-4-P standard (0.5 mL CHCl₃, 0.5 mL CH₃OH, 2.5 μ L HCl) and spotted on TLC plates (VWR). The spotted plate is subjected to thin layer chromatography in CHCl₃/CH₃OH/NH₄OH (40:40:15). The TLC plate is dried and subjected to autoradiography.

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Cell Assay ^[1]

For cytotoxicity assays, cells (1.0 \times 10⁵ cells/mL HPGM) are treated with various concentrations of Recilisib Sodium or vehicle without radiation treatment for 2 or 24 hours. The cells are washed and plated into methocult using gridded dishes. The total number of colony forming units (CFUs) is determined 14 days post-plating by microscopic observation using an Olympus IMT-2 microscope.

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

CUSTOMER VALIDATION

- J Pharm Anal. 2023 Mar 24.
- Int J Biol Macromol. 2020 Mar 15;147:79-88.
- Phytomedicine. 2023 Jan 31;112:154684.
- Phytomedicine. 6 July 2022, 154323.
- Int Immunopharmacol. 2023 Jan 10;115:109677.

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REFERENCES

[1]. Kang AD, et al. ON01210.Na (Ex-RAD) mitigates radiation damage through activation of the AKT pathway. PLoS One. 2013;8(3):e58355.

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

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