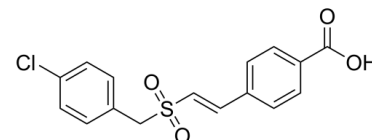


## Data Sheet

<b>Product Name:</b>	Recilisib
<b>Cat. No.:</b>	CS-6712
<b>CAS No.:</b>	334969-03-8
<b>Molecular Formula:</b>	C <sub>16</sub> H <sub>13</sub> ClO <sub>4</sub> S
<b>Molecular Weight:</b>	336.79
<b>Target:</b>	Akt; PI3K
<b>Pathway:</b>	PI3K/Akt/mTOR
<b>Solubility:</b>	DMSO : 35.71 mg/mL (ultrasonic)



### BIOLOGICAL ACTIVITY:

Recilisib (ON 01210) is a radioprotectant, which can activate **AKT**, **PI3K** activities in cells<sup>[1]</sup>. *In Vitro*: Recilisib (up to 50  $\mu$ M) shows a normal distribution of cells throughout the cell cycle, with a slight reduction in the number of cells in S-phase at 50  $\mu$ M. Continuous exposure of Recilisib (100  $\mu$ M) does not result in cell death. Recilisib does not inhibit the ability of human bone marrow to form colonies in methylcellulose at either timepoint. 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 GSK3 $\alpha/\beta$  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<sup>[1]</sup>. *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<sup>[1]</sup>.

### PROTOCOL (Extracted from published papers and Only for reference)

**Kinase Assay:** <sup>[1]</sup>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-PI3K 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 MgCl<sub>2</sub>, 2 mM DTT, 0.01% NP-40). L- $\alpha$ -Phosphatidylinositol (12.5 mM) and ATP (10  $\mu$ M) are added to the kinase buffer (60  $\mu$ L per sample) and incubated at 30°C for 30 minutes. The reaction is stopped by addition of 100  $\mu$ L of 1N HCl and extracted by addition of 200  $\mu$ L CHCl<sub>3</sub>/CH<sub>3</sub>OH (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  $\mu$ L of PI-4-P standard (0.5 mL CHCl<sub>3</sub>, 0.5 mL CH<sub>3</sub>OH, 2.5  $\mu$ L HCl) and spotted on TLC plates (VWR). The spotted plate is subjected to thin layer chromatography in CHCl<sub>3</sub>/CH<sub>3</sub>OH/NH<sub>4</sub>OH (40:40:15). The TLC plate is dried and subjected to autoradiography. **Cell Assay:** <sup>[1]</sup>For cytotoxicity assays, cells (1.0 $\times$ 10<sup>5</sup> 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.

## 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.

## CAIndexNames:

Benzoic acid, 4-[(1E)-2-[[[4-chlorophenyl)methyl]sulfonyl]ethenyl]-

## SMILES:

O=C(O)C1=CC=C(/C=C/S(=O)(CC2=CC=C(Cl)C=C2)=O)C=C1

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

Tel: 610-426-3128

Fax: 888-484-5008

E-mail: [sales@ChemScene.com](mailto:sales@ChemScene.com)

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA