

Product Data Sheet

Product Name: SCR130

Cat. No.: GC62693

Chemical Properties

Cas. No. 2377858-38-1

Formula $C_{19}H_{13}Cl_2N_3O_2S$

M.Wt 418.3

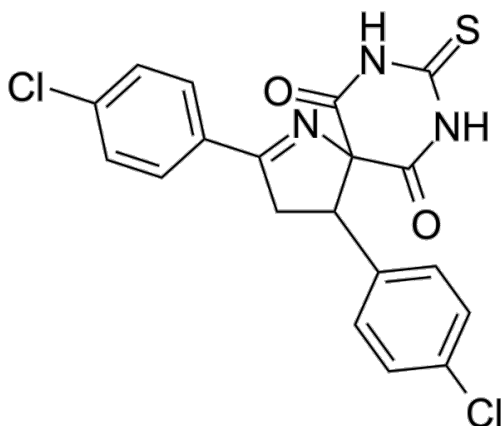
Solubility

Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Background

SCR130 is a SCR7-based DNA nonhomologous end-joining (NHEJ) inhibitor. SCR130 inhibits the end-joining of DNA in a Ligase IV-dependent manner. SCR130 is specific to Ligase IV, and shows minimal or no effect on Ligase III and Ligase I mediated joining. SCR130 induces cell apoptosis and has anticancer activity[1].

SCR130 (7-21 μM; 48 hours) increase in the number of late and early apoptotic cells. SCR130 induces apoptosis by both intrinsic and extrinsic pathways. SCR130 increases the expression of p-p53, BCL2 and MCL1, and CYTOCHROME C, BAX, and BAK also increased. The activation of

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Tel: (909) 407-4943 Fax: (626) 353-8530 E-mail: tech@glpbio.com

Address: 10292 Central Ave. #205, Montclair, CA, USA

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caspase 8, increase in expression of FAS and SMAC-DIABLO proteins are also observed[1].SCR130 (48 hours) exhibits cytotoxicity in Reh, HeLa, CEM, Nalm6, and N114 cells with IC50 values of 14.1 μ M, 5.9 μ M, 6.5 μ M, 2.2 μ M, and 11 μ M, respectively[1].SCR130 can potentiate the effect of radiation (0.5 and 1 Gy) by inducing enhanced cell death upon coadministration in Reh and Nalm6 cell lines[1].SCR130 blocks the endogenous NHEJ leading to accumulation of unrepaired DNA breaks. Treatment with SCR130 leads to inhibition of endogenous NHEJ, resulting in the accumulation of DNA double-strand breaks (DSBs) and cell death by activating apoptotic pathways[1].

[1]. Ujjayinee Ray, et al. Identification and characterization of novel SCR7-based small-molecule inhibitor of DNA end-joining, SCR130 and its relevance in cancer therapeutics. Mol Carcinog. 2020 Jun;59(6):618-628.

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