

Product Data Sheet

Product Name: SMER 28

Cat. No.: GC14272

Chemical Properties

Cas. No. 307538-42-7

Chemical Name N-allyl-6-bromoquinazolin-4-amine

SMILES BrC1=CC=C2N=CN=C(C2=C1)NCC=C

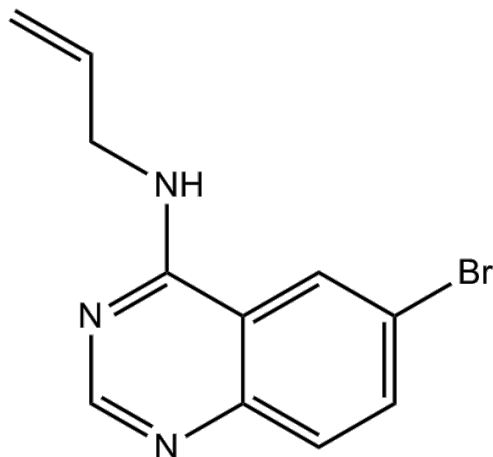
Formula $C_{11}H_{10}BrN_3$ M.Wt 264.12

Solubility $\geq 101.4\text{mg/mL}$ in DMSO 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

IC50: N/A

SMER 28 is a new small-molecule enhancer of the cytostatic effects. The target of rapamycin proteins has been reported to be able to regulate various cellular processes including autophagy, which may play a protective role in some neurodegenerative and infectious diseases.

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

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In vitro: SMER 28 independently induced autophagy of rapamycin in mammalian cells, enhancing the clearance of autophagy substrates such as A53T α -synuclein and mutant huntingtin, which were associated with Huntington's and Parkinson's disease. SMER 28, which seemed to act either independently or downstream of the rapamycin target, was found to attenuate the mutant huntingtin-fragment toxicity in Huntington's disease cells [1].

In vivo: Previous study confirmed that the reduction of EGFP-HDQ74 aggregation occurred through autophagy using autophagy-competent mouse embryonic fibroblasts (MEFs) (Atg5^{+/+}). EGFP-HDQ74 aggregation was increased significantly in untreated Atg5^{-/-} (autophagy-deficient) cells when compared with untreated Atg5^{+/+} cells. SMER 28 reduced EGFP-HDQ74 aggregation in Atg5^{+/+} cells significantly, but not in Atg5^{-/-} cells). Therefore, SMER 28 could only reduce mutant huntingtin aggregation in autophagy-competent cells [1].

Clinical trial: N/A

Reference:

[1] Sarkar S,Perlstein EO,Imarisio S,Pineau S,Cordenier A,Maglathlin RL,Webster JA,Lewis TA,O'Kane CJ,Schreiber SL,Rubinsztein DC. Small molecules enhance autophagy and reduce toxicity in Huntington's disease models. Nat Chem Biol.2007 Jun;3(6):331-8.

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