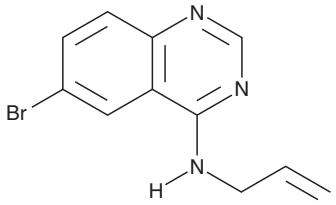
PRODUCT INFORMATION

SMER28

Item No. 17768

CAS Registry No.:	307538-42-7
Formal Name:	6-bromo-N-2-propen-1-yl-4-quinazolinamine
MF:	$C_{11}H_{10}BrN_3$
FW:	264.1
Purity:	≥98%
UV/Vis.:	λ_{max}: 213, 296, 325, 339 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

SMER28 is supplied as a crystalline solid. A stock solution may be made by dissolving the SMER28 in the solvent of choice, which should be purged with an inert gas. SMER28 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of SMER28 in ethanol and DMSO is approximately 30 mg/ml and approximately 1 mg/ml in DMF.

SMER28 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, SMER28 should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. SMER28 has a solubility of approximately 0.09 mg/ml in a 1:10 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

SMER28 is a small-molecule enhancer (SMER) of autophagy that increases autophagosome synthesis and enhances clearance of aggregate-prone substrates, including those relevant to Huntington's, Parkinson's, and Alzheimer's diseases.[1-3] SMER28 induces clearance of amyloid-β (Aβ) peptides and the amyloid precursor protein c-terminal fragment (APP-CTF) in mouse embryonic fibroblasts (MEFs) isolated from wild-type, but not autophagy-related protein 5 knockout ($Atg5^{-/-}$), mouse embryos in a concentration-dependent manner.[4] It induces autophagy independently of rapamycin (Item No. 13346) in mammalian cells at a concentration of 47 µM.[1] SMER28 also promotes reprogramming of fibroblasts into neural stem cells when used in combination with other chemical reagents.[5]

References

1. Sarkar, S., Perlstein, E.O., Imarisio, S., *et al.* Small molecules enhance autophagy and reduce toxicity in Huntington's disease models. *Nat. Chem. Biol.* **3(6)**, 331-338 (2007).
2. Renna, M., Jiminez-Sanchez, M., Sarkar, S., *et al.* Chemical inducers of autophagy that enhance the clearance of mutant proteins in neurodegenerative diseases. *J. Biol. Chem.* **285(15)**, 11061-11067 (2010).
3. Shen, D., Coleman, J., Chan, E., *et al.* Novel cell- and tissue-based assays for detecting misfolded and aggregated protein accumulation within aggresomes and inclusion bodies. *Cell Biochem. Biophys.* **60(3)**, 173-185 (2011).
4. Tian, Y., Bustos, V., Flajolet, M., *et al.* A small-molecule enhancer of autophagy decreases levels of Abeta and APP-CTF via Atg5-dependent autophagy pathway. *FASEB J.* **25(6)**, 1934-1942 (2011).
5. Zhang, M., Lin, Y.-H., Sun, Y.J., *et al.* Pharmacological reprogramming of fibroblasts into neural stem cells by signaling-directed transcriptional activation. *Cell Stem Cell Apr*, **18(5)**, 653-667 (2016).

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