Small Molecules

SMER28

Enhances mammalian autophagy

Catalog # 74202 5 mg
74204 10 mg

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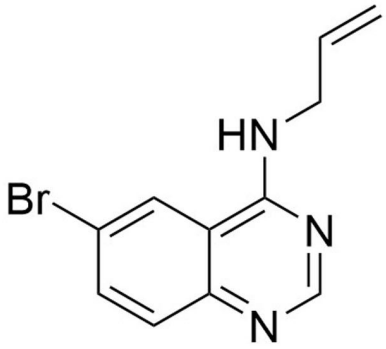
Product Description

SMER28, a bromo-substituted quinazoline, was first identified as a small-molecule enhancer of rapamycin (SMER; Sarkar et al.). SMER28 induces autophagy by increasing autophagosome synthesis and enhancing the clearance of model autophagy substrates (Renna et al.).

Molecular Name:	SMER28
Alternative Names:	Not applicable
CAS Number:	307538-42-7
Chemical Formula:	$C_{11}H_{10}BrN_3$
Molecular Weight:	264.1 g/mol
Purity:	≥ 98%
Chemical Name:	6-bromo-N-prop-2-enylquinazolin-4-amine
Structure:	



Properties

Physical Appearance:	A crystalline solid
Storage:	Product stable at -20°C as supplied. Protect product from prolonged exposure to light. For long-term storage, store with a desiccant. Stable as supplied for 12 months from date of receipt.
Solubility:	· DMSO ≤ 110 mM · Absolute ethanol ≤ 110 mM For example, to prepare a 10 mM stock solution in DMSO, resuspend 1 mg in 379 µL of DMSO. Prepare stock solution fresh before use. Information regarding stability of small molecules in solution has rarely been reported, however, as a general guide we recommend storage in DMSO at -20°C. Aliquot into working volumes to avoid repeated freeze-thaw cycles. The effect of storage of stock solution on compound performance should be tested for each application. Compound has low solubility in aqueous media. For use as a cell culture supplement, stock solution should be diluted into culture medium immediately before use. Avoid final DMSO concentration above 0.1% due to potential cell toxicity.

Published Applications

REPROGRAMMING
· As a component of a neural reprogramming cocktail, SMER28 reprograms mouse fibroblasts into induced neural stem cell-like cells (Zhang et al.).
DISEASE MODELING
· Enhances clearance of autophagy substrates including mutant huntingtin in Huntington's disease and A53T α-synuclein in familial Parkinson's disease (Sarkar et al.).
· Through autophagy factor ATG5, stimulates erythropoiesis and up-regulates expression of globin genes in both in vitro and in vivo models of Diamond-Blackfan anemia (Doulatov et al.).
· Decreases the level of indicators of Alzheimer's disease (amyloid-β peptide and the amyloid precursor protein [APP]-derived fragment) via the autophagy-related protein 5-dependent pathway (Tian et al.).

References

Doulatov S et al. (2017) Drug discovery for Diamond-Blackfan anemia using reprogrammed hematopoietic progenitors. Sci Transl Med 9(376): eaah5645.
Renna M et al. (2010) Chemical inducers of autophagy that enhance the clearance of mutant proteins in neurodegenerative diseases. J Biol Chem 285(15): 11061–7.
Sarkar S et al. (2007) Small molecules enhance autophagy and reduce toxicity in Huntington's disease models. Nat Chem Biol 3(6): 331–8.
Tian Y et al. (2011) A small-molecule enhancer of autophagy decreases levels of Aβ and APP-CTF via Atg5-dependent autophagy pathway. FASEB J 25(6): 1934–42.
Zhang M et al. (2016) Pharmacological reprogramming of fibroblasts into neural stem cells by signaling-directed transcriptional activation. Cell Stem Cell 18(5): 653–67.

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This product is hazardous. Please refer to the Safety Data Sheet (SDS).