

Product Data Sheet

Product Name: SK-129
Cat. No.: GC81610

Chemical Properties

Cas. No. 1919889-97-6

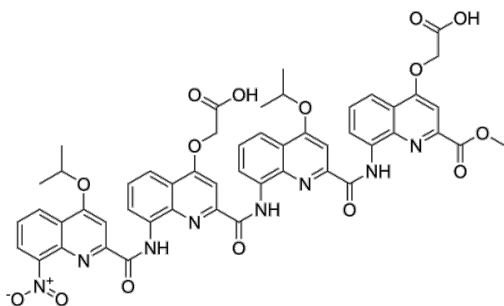
Formula $C_{51}H_{42}N_8O_{15}$ M.Wt 1006.92

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

SK-129 is a blood-brain barrier-permeable inhibitor of α -synuclein (α S) oligomers with a K_d of 221 nM. SK-129 preferentially binds to neurotoxic α S oligomers over physiological α S monomers, inhibits α S aggregation, blocks the interaction and co-aggregation of α S with tau protein, and prevents the maturation of α S-tau condensates into amyloid aggregates. SK-129 reduces ROS production, rescues dopaminergic neuron degeneration, improves motor function, restores endogenous dopamine synthesis, increases the number of Tyrosine Hydroxylase-positive neurons, prevents brain histopathological changes, alleviates neuroinflammation, and improves survival rates in relevant models. SK-129 can be used in research related to Parkinson's disease (PD) and Lewy body

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

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dementia (LBD) [1].

In Vivo, SK-129 (25-50 μ M; administered on days 2 and 4, day 5) rescues DA neuron degeneration, motor deficits, oxidative stress, and behavioral deficits in both early and postdisease UA196 C. elegans PD models in a dose-dependent manner^[1]. SK-129 (20 mg/kg; i.v.; every other day; 21 days) confers 100% survival up to 270 days, prevents PD-related weight loss, eliminates α -synuclein and α -synuclein-tau pathology, reduces neuroinflammation, and shows no systemic toxicity in the M83(A) mouse PD model^[1].

In Vitro, SK-129 (SK-129F; 100 nM; until signal saturation) preferentially binds pathological α S oligomers and fibrils over physiological α S monomers, with a Kd value of 221 nM for α S oligomers^[1]. SK-129 (10 μ M; 12 h pre- or post-treatment) potently inhibits α S aggregation in α SA53T-YFP-expressing HEK293T cells mediated by NDE derived from Parkinson's disease (PD)^[1]. SK-129 (1 μ M) binds to α S-tau liquid condensates and inhibits their transition to toxic amyloid-like co-aggregates, instead inducing the formation of non-amyloid gel-like aggregates^[1].

References:

[1]. Dohoney RA, et al. Foldamers rescue synucleinopathy phenotypes in multiple in vitro and in vivo models. Sci Transl Med. 2026;18(843):eadu1050.

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