

Product Data Sheet

Product Name: NXPZ-2
Cat. No.: GC71072

Chemical Properties

Cas. No. 2254492-08-3

Formula $C_{27}H_{27}N_5O_7S_2$ M.Wt 597.66

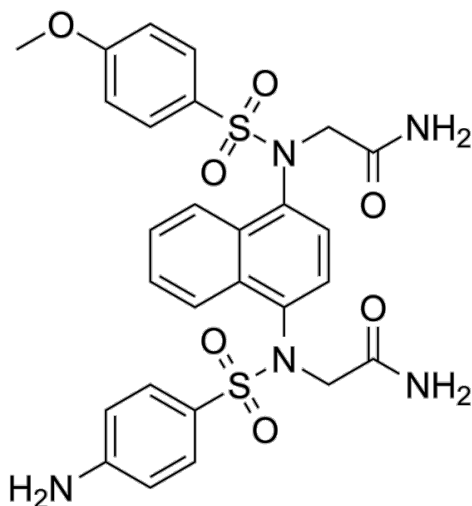
Solubility DMSO : 100 mg/mL (167.32 mM); ultrasonic and warming and heat to 60°C; Hygroscopic DMSO has a significant impact on the solubility of product, please use newly opened DMSO)

Storage -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

NXPZ-2 is an orally active Keap1-Nrf2 protein–protein interaction (PPI) inhibitor with a K_i value of 95 nM, EC_{50} value of 120 and 170 nM. NXPZ-2 can dose-dependently ameliorate A β [1-42]-Induced

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

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cognitive dysfunction, improve brain tissue pathological changes in Alzheimer's disease (AD) mouse by increasing neuron quantity and function. NXPZ-2 can inhibit oxidative stress by increasing Nrf2 expression levels and promoting its cytoplasm to nuclear translocation, which is helpful for Keap1-Nrf2 PPI inhibitors and AD associated disease research.

NXPZ-2 (0-200 μ M, 7 days) has no obvious toxicity on primary cortical neuron [1].

NXPZ-2 (Male ICR mice, 52.5/105/210 mg/kg, p.o., once daily for 7 days) improves AD mice learning and memorizing function including increased spontaneous alternation, increases number of active avoidance times, shortened escape latency, increased the time spent in the target quadrant and number of platform crossing [1].

NXPZ-2 (Male ICR mice, 52.5/105/210 mg/kg, p.o., once daily for 7 days) rescues the brain structure damage and lowers dead neuron numbers of AD mice with no obvious toxicity on mouse organs [1].

NXPZ-2 (Male ICR mice, 52.5/105/210 mg/kg, p.o., once daily for 7 days) alleviates oxidative stress by increasing Nrf2 expression levels, and promotes Nrf2's cytoplasm to nuclear translocation, and improves cognitive dysfunction by elevating Nrf2 in both the central nervous system and peripheral blood [1].

References:

[1]. Yi Sun, et al. Direct inhibition of Keap1-Nrf2 Protein-Protein interaction as a potential therapeutic strategy for Alzheimer's disease. *Bioorg Chem.* 2020 Oct;103:104172.

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