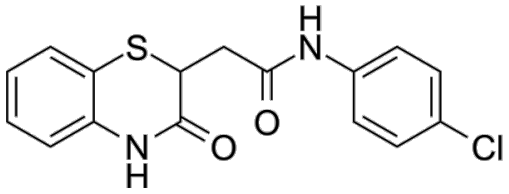
Product Data Sheet

Product Name: YHV98-4
Cat. No.: GC81609

Chemical Properties

Cas. No.	132858-26-5		
Formula	$C_{16}H_{13}ClN_2O_2S$	M.Wt	332.8
Solubility	DMSO: 100 mg/mL (300.48 mM; Need ultrasonic)	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

YHV98-4 is a selective, blood-brain barrier-permeable Hv1 inhibitor with an IC_{50} of 0.7 μM. YHV98-4 reduces ROS production and restores the SHP-1-pAKT signaling pathway. YHV98-4 decreases the spread of hyperphosphorylated Tau protein. YHV98-4 alleviates inflammation and produces

analgesic effects. YHV98-4 improves cognitive function in mouse models of Alzheimer's disease. YHV98-4 can be used in research related to inflammatory pain, neuropathic pain and Alzheimer's disease [1] [2] [3].

In Vivo, YHV98-4 (10 mg/kg; i.p.; daily; 30 days) restores microglial homeostasis, reduces tau pathology and oxidative stress, protects neurons, and improves cognitive function in 3×Tg Alzheimer's disease mice[2]. YHV98-4 (10 mg/kg; i.p.) restores the impaired SHP-1-pAKT inflammatory signaling pathway in sensory neurons of CFA -treated mice[3].

In Vitro, YHV98-4 specifically inhibits Hv1 with a half-maximal inhibitory concentration of 1 μM, without inhibiting other ion channels[1]. YHV98-4 (0.1-10 μM) potently inhibits the Hv1 channel expressed in HEK-293T cells, with an IC_{50} of 0.7 μM[3]. YHV98-4 (20 μM) exhibits no inhibitory activity against hERG, KCNQ2, BK, Nav1.7, TRPV1, TRPA1 or VRAC channels, demonstrating high selectivity for Hv1[3]. YHV98-4 (20 μM) alleviates mitochondrial oxidative stress, restores energy metabolism, rescues defects in mitophagy and autophagy, reduces mtDNA leakage, and enhances the phagocytosis of BV2 microglia stimulated by PHF[2].

References:
[1]. Furutani K, et al. Targeting Hv1 proton channel for pain control. Cell Res. 2022 May;32(5):419-420.
[2]. Lin J, et al. Hv1 inhibition rescues AD pathology by restoring microglial mitochondrial function and enhancing mitochondrial transfer. Experimental & molecular medicine. 2025 Dec;57(12):2833-2851.
[3]. Zhang Q, et al. Inhibiting Hv1 channel in peripheral sensory neurons attenuates chronic inflammatory pain and opioid side effects. Cell research. 2022 May;32(5):461-476.