

Product Data Sheet

Product Name: KDS12025

Cat. No.: GC81841

Chemical Properties

Cas. No. 2769053-56-5

Formula $C_{16}H_{20}N_2O$

M.Wt 256.34

Solubility Storage Store at 4°C, sealed storage, away from moisture

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

KDS12025 is a blood-brain barrier-permeable, orally active H₂O₂-decomposing peroxidase enhancer. KDS12025 enhances the H₂O₂-decomposing pseudoperoxidase activity of Hb without altering oxygen transport function, and reduces intracellular H₂O₂ load. KDS12025 reduces

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

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abnormal H₂O₂ levels and inhibits the production of COL1. KDS12025 restores cerebral blood flow, maintains neuronal excitability, membrane properties, synaptic connections and corticospinal tract fibers, reduces cerebral edema and exerts neuroprotective effects. KDS12025 improves motor function and overall neurological function. KDS12025 can be used in the research of ischemic stroke, Alzheimer's disease, Parkinson's disease, amyotrophic lateral sclerosis, aging-related neurodegenerative diseases and rheumatoid arthritis [1] [2].

In Vivo, KDS12025 (0.001-10 mg/kg/day; i.p., oral ad libitum; once daily; 3 consecutive days) exhibits potent neuroprotective effects in a mouse ischemic stroke model, with an IC₅₀ of 0.0027 mg/kg/day for motor function recovery, reduces pathological remodeling, preserves neuronal function, and retains efficacy when administered up to 2 days post-stroke^[1]. KDS12025 (1 mg/kg/day; p.o.; ad libitum in drinking water; 4 weeks) fully restores motor function and reverses Parkinson's disease-related pathology in A53T α -synuclein mice, with efficacy dependent on both astrocytic and neuronal Hb β ^[2]. KDS12025 (0.1-1 mg/kg/day; p.o.; ad libitum in drinking water; 22 months) increases median survival by ~14%, restores locomotor function, prevents neuronal loss, reverses astrocytic atrophy, and restores astrocytic Hb β levels in aging C57BL/6J mice^[2]. KDS12025 (0.1-10 mg/kg/day; i.p., p.o.; daily, ad libitum in drinking water; 7 days, 2 weeks, 16 days) potently reverses Alzheimer's disease-related memory impairment, neurodegeneration, astrogliosis, oxidative stress, and neuronal dysfunction in APP/PS1 mice, with efficacy dependent on astrocytic Hb β ^[2]. KDS12025 (1 mg/kg/day) mitigates rheumatoid arthritis pathology and systemic inflammation in collagen-induced arthritis mice^[2].

In Vitro, KDS12025 significantly protects primary cortical neurons against astrocyte-mediated in vitro neurotoxicity induced by H₂O₂^[1]. KDS12025 (1 μ M; 30 min) potently enhances the H₂O₂-decomposing pseudoperoxidase activity of purified hemoglobin, with an EC₅₀ of 0.04 μ M, and effectively achieves H₂O₂ decomposition even at extremely low Hb concentrations^[2]. KDS12025 (10 μ M; 24 h) effectively reduces H₂O₂ levels induced by A β , putrescine and 6-OHDA in primary cultured hippocampal astrocytes, with an EC₅₀ of 0.5 μ M, and its antioxidant effect depends on Hb β in astrocytes^[2]. KDS12025 binds strongly to the proximal heme site of the hemoglobin β subunit with a binding energy of -16.5 kcal/mol, thereby enhancing the pseudoperoxidase activity of Hb β ^[2].

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References:

- [1]. Lee JH, et al. Oxidative stress-induced astrocytic collagen biosynthesis drives glial barrier formation and neuronal death in ischemic stroke. *Cell metabolism*. 2026 Jul 07;38(7):1404-1424.e8.
- [2]. Won W, et al. Hemoglobin as a pseudoperoxidase and drug target for oxidative stress-related diseases. *Signal transduction and targeted therapy*. 2025 Aug 22;10(1):270.

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