

Product Data Sheet

Product Name: FP802 dihydrochloride

Cat. No.: GC79534

Chemical Properties

Cas. No. 2490401-57-3

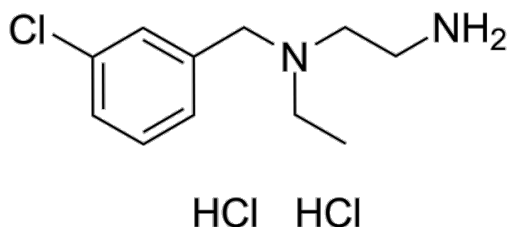
Formula $C_{11}H_{19}Cl_3N_2$ M.Wt 285.64

Solubility H_2O : 116.67 mg/mL (408.45 mM; Storage Store at $-20^{\circ}C$
Need ultrasonic)

General tips For obtaining a higher solubility, please warm the tube at $37^{\circ}C$ and shake it in the ultrasonic bath for a while. Stock solution can be stored below $-20^{\circ}C$ 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

FP802 dihydrochloride is an orally active potent TwinF interface inhibitor that disrupts and detoxifies the NMDAR/TRPM4 death complex. FP802 dihydrochloride exerts powerful neuroprotective effects in the 5xFAD mouse model of Alzheimer's disease (AD) by preventing cognitive decline, preserving neuronal structural integrity, reducing amyloid- β plaque formation, and mitigating mitochondrial

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

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pathology [1]. FP802 dihydrochloride stops loss of motor neurons, reduces serum neurofilament light chain (NfL) levels, improves motor performance, and extends life in a mouse model of amyotrophic lateral sclerosis (ALS) [2]. FP802 dihydrochloride can be used for AD and ALS research [1] [2].

In Vivo, FP802 (10 and 40 mg/kg, p.o., daily for 4 months) dihydrochloride rescues cognitive function, prevents neuronal structure degeneration, and reduces amyloid pathology in 5xFAD mice [1]. FP802 (40 mg/kg, s.c., daily from ~week 15 for 4 weeks) dihydrochloride safely halts motor neuron degeneration and extends survival in ALS mice by targeting the NMDAR/TRPM4 complex [2].

In Vitro, FP802 (8 μ M, 24-72 h) hydrochloride disrupts the NMDAR/TRPM4 complex and provides neuroprotection in cellular models, without directly affecting neurite outgrowth [1]. FP802 (10 μ M, 30 min) hydrochloride exhibits potent neuroprotection against glutamate (20 μ M)-mediated toxicity (IC_{50} = 8.7 μ M) and restores NMDA-suppressed immediate-early gene expression to physiological levels [2]. FP802 hydrochloride shows no antagonistic activity against NMDARs in HEK293 cells (IC_{50} > 250 μ M for both, GluN1/GluN2A and GluN1/GluN2B) [2]. FP802 (30 min) hydrochloride dose-dependently blocks NMDA-induced death of post-mitotic neurons in forebrain organoids derived from sporadic ALS patient induced pluripotent stem cells (iPSCs) [2].

References:

- [1]. Yan J, et al. The NMDAR/TRPM4 death complex is a major promoter of disease progression in the 5xFAD mouse model of Alzheimer's disease. *Mol Psychiatry*. 2025 Aug 26.
- [2]. Yan J, et al. TwinF interface inhibitor FP802 stops loss of motor neurons and mitigates disease progression in a mouse model of ALS. *Cell Rep Med*. 2024 Feb 20;5(2):101413.

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