

Product Data Sheet

Product Name: Mdivi 1
Cat. No.: GC10200

Chemical Properties

Cas. No. 338967-87-6

Chemical Name 3-(2,4-dichloro-5-methoxyphenyl)-2-sulfanylidene-1H-quinazolin-4-one

SMILES COC1=C(C=C(C(=C1)N2C(=O)C3=CC=CC=C3NC2=S)Cl)Cl

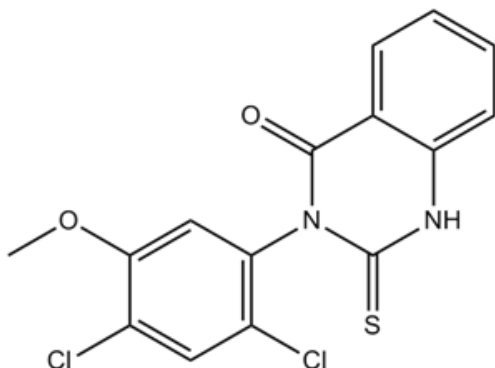
Formula $C_{15}H_{10}Cl_2N_2O_2S$ M.Wt 353.22

Solubility $\geq 17.65\text{mg/mL}$ in DMSO 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



Protocol

Cell experiment [1]:

Cell lines Primary cortical neuron(PCN)

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

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Preparation Method PCNs were treated with Mdivi-1 (from 0.001 nM to 1 mM) dissolved in 0.1% DMSO 1 h before scratch cell injury. The release of lactate dehydrogenase(LDH)into the culture media was measured 6 h after scratch cell injury.

Reaction Conditions 0.001 nM to 1 mM for 1hour

Applications Using LDH assay, exposing PCNs to scratch cell injury with Mdivi-1 (100 μ M and 1 mM) resulted in significant dose-dependent inhibition of cell death. Hence the range of the optimum concentration was from 10 μ M to 100 μ M.

Animal experiment [2]:

Animal models male ICR mice

Preparation Method For the mice treated with Mdivi-1 (3 mg/kg), dynamin-related protein 1 (Drp1) inhibitor, was administered by intraperitoneal injection 10 min after TBI. Mdivi-1 was dissolved in 0.01 M PBS. Vehicle animals received an intraperitoneal injection of 0.01 M PBS.

Dosage form Intraperitoneal injection, 3 mg/kg

Applications Treatment with Mdivi-1 accelerated the recovery of motor functional outcome on days 3-5 post-TBI. After injury, animals subjected to Mdivi-1 treatment demonstrated a significant decrease in the latencies, relative to vehicle mice on days 15 and 16, thereby Mdivi-1 treatment could result in cognitive functional recovery.

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References:

- [1]: Q. Wu, C. Gao, H. Wang, X. Zhang, et al. Mdivi-1 alleviates blood-brain barrier disruption and cell death in experimental traumatic brain injury by mitigating autophagy dysfunction and mitophagy activation. *Int. J. Biochem. Cell Biol.*, 94 (2018), pp. 44-55, 10.1016/j.biocel.2017.11.007
- [2]: Q. Wu, S.X. Xia, Q.Q. Li, et al. Mitochondrial division inhibitor 1 (Mdivi-1) offers neuroprotection through diminishing cell death and improving functional outcome in a mouse model of traumatic brain injury. *Brain Res.*, 1630 (2016), pp. 134-143

Background

Mdivi-1 (Mitochondrial division inhibitor 1) inhibits traumatic brain injury (TBI)-induced dynamin-related protein 1 (Drp1) up-regulation, autophagy dysfunction and mitophagy activation. Mdivi-1 decreased TBI-induced cell death and lesion volume [1,2].

Mdivi-1 is a selective Drp 1 inhibitor with IC50 value as 10 μ M [3]. Mdivi-1 significantly alleviated the scratch injury-induced cell death, loss of mitochondrial membrane potential, reactive oxygen species (ROS) production and ATP reduction in primary cortical neurons (PCNs). Additionally, the lysosome inhibitor chloroquine (CQ) abrogated the Mdivi-1-induced decrease in autophagosomes accumulation and cell death at 24 h both in the basal state and under the conditions of scratch cell injury [1].

Mdivi-1 could significantly rescue neurons from death induced by seizures in a dose-dependent manner. In addition, the seizures increase Drp1 expression in hippocampus. These suggested that the up-regulation of Drp1 expression could be partially responsible for seizure-induced neuronal death. Moreover, CytC release, AIF translocation and caspase-3 activation may be involved in the protective mechanisms of mdivi-1 against seizure-induced neuronal death [4].

References:

- [1]. Q. Wu, C. Gao, H. Wang, X. Zhang, et al. Mdivi-1 alleviates blood-brain barrier disruption and cell death in experimental traumatic brain injury by mitigating autophagy dysfunction and mitophagy activation. *Int. J. Biochem. Cell Biol.*, 94 (2018), pp. 44-55, 10.1016/j.biocel.2017.11.007
- [2]. Q. Wu, S.X. Xia, Q.Q. Li, et al. Mitochondrial division inhibitor 1 (Mdivi-1) offers neuroprotection through diminishing cell death and improving functional outcome in a mouse model of traumatic brain

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injury. Brain Res., 1630 (2016), pp. 134-143

[3]. D. Wu, A. Dasgupta, K.H. Chen, M. Neuber-Hess, J. Patel, T.E. Hurst, J.D. Mewburn, P.D.A. Lima, E. Alizadeh, A. Martin, M. Wells, V. Snieckus, S.L. Archer. Identification of novel dynamin-related protein 1 (Drp1) GTPase inhibitors: therapeutic potential of Drpitor1 and Drpitor1a in cancer and cardiac ischemia-reperfusion injury. FASEB J., 34 (2020), pp. 1447-1464

[4]. Xie N, Wang C, Lian Y, Zhang H, Wu C, Zhang Q. A selective inhibitor of Drp1, mdivi-1, protects against cell death of hippocampal neurons in pilocarpine-induced seizures in rats. (2013b) Neurosci Lett 545: 64 -68.

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