Data Sheet (Cat.No.T206271)

TargetMol

Nrf2 activator 19

Chemical Properties

CAS No. :

Formula:

Molecular Weight:

Storage: Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Nrf2 activator 19 is a compound capable of crossing the blood-brain barrier and acts as an NRF2/HO-1 activator, offering potent antioxidant and neuroprotective effects. It effectively reduces brain damage and minimizes the accumulation of Reactive Oxygen Species (ROS). Additionally, Nrf2 activator 19 inhibits neuronal apoptosis, aiding in the recovery of neural function and motor skills. It has demonstrated significant potential in ischemic stroke research.
Targets(IC50)	Apoptosis,Reactive Oxygen Species,Nrf2,ROS
In vitro	Nrf2 activator 19 (Compound 6p) at a concentration of 40 μM for 24 hours demonstrates no cytotoxicity in PC12 and RAW cells, maintaining over 90% cell viability under t-BHP-induced damage, indicating significant protective effects. In t-BHP-induced oxidative damage in PC12 cells, it offers strong protective benefits at concentrations of 2.5 μM to 20 μM over 24 hours. At concentrations ranging from 2.5 μM to 10 μM for 24 hours in PC12 cells, it effectively reduces ROS levels and decreases malondialdehyde (MDA) levels while increasing superoxide dismutase (SOD) activity. Nrf2 activator 19 elevates the expression of NRF2 and HO-1 proteins in a concentration-dependent manner in PC12 cells over 24 hours, combating oxidative damage by activating the NRF2 pathway. At 10 μM for 3 hours in PC12 cells, it reduces Keap1 expression, implying a direct interaction with KEAP1. It exhibits moderate metabolic stability in rat liver microsomes at 1 μM for 1 hour. The compound demonstrates a permeability of 7.64% in a Transwell-based blood-brain barrier (BBB) model using bEnd.3 cells at 10 μM for 24 hours, suggesting its capability to cross the BBB.
In vivo	Nrf2 activator 19, administered intravenously at a dose of 5-20 mg/kg for 7 consecutive days, enhances neurological recovery in a rat model of cerebral ischemia-reperfusion injury (MCAO) by counteracting the damage through activation of the NRF2 pathway.

Tel:781-999-4286 E_mail:info@targetmol.com Address:34 Washington Street,Wellesley Hills,MA 02481