Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Edaravone

Catalog No: tcsc1832

Available Sizes

Size: 5g

Specifications

CAS No:
89-25-8

Formula:
$C_{10}H_{10}N_2O$

Pathway:
Metabolic Enzyme/Protease

Target:
MMP

Purity / Grade:
>98%

Solubility:
DMSO : 125 mg/mL (717.57 mM; Need ultrasonic)

Alternative Names:
MCI-186

Observed Molecular Weight:
174.2

Product Description

Edaravone is a strong novel free radical scavenger, and inhibits **MMP**-9-related brain hemorrhage in rats treated with tissue plasminogen activator.

In Vitro: Edaravone performs both preventative and therapeutic effects against toxicity of glutamate. Pretreatment of edaravone reduces the toxicity of glutamate towards SGNs. Edaravone reduces apoptosis and necrosis caused by glutamate. Pretreatment of

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edaravone (500 μM) reverses these changes to approximately normal levels. The protective effect of edaravone on SGNs against glutamate-induced apoptosis is associated with PI3K/Akt pathway and Bcl-2 protein family[4].

In Vivo: Edaravone exerts neuroprotective effects by inhibiting endothelial injury and by ameliorating neuronal damage in brain ischemia. Edaravone provides the desirable features of NOS: it increases eNOS (beneficial NOS for rescuing ischemic stroke) and decreases nNOS and iNOS (detrimental NOS). Post-reperfusion brain edema and hemorrhagic events induced by thrombolytic therapy may be reduced by edaravone pretreatment[1]. Edaravone significantly decreases infarct volume, with the average infarct volume in the edaravone-treated rats (227.6 mm^3) being significantly lower than that in the control rats (264.0 mm^3). Edaravone treatment also decreases the postischemic hemorrhage volumes (53.4 mm^3 in edaravone-treated rats vs 176.4 mm^3 in controls). In addition, the ratio of hemorrhage volume to infarct volume is lower in the edaravone-treated rats (23.5%) than in the untreated rats (63.2%)[2]. In edaravone (20 mg/kg)-treated rats, astrocyte activity (glial fibrillary acidic protein) and apoptotic cells (caspase-3) are decreased on the corpus callosum, germinal matrix, and cerebral cortex[3].