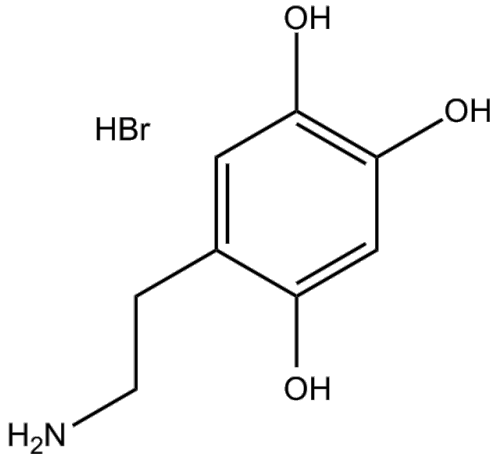
Product Data Sheet

Product Name: 6-Hydroxydopamine hydrobromide
Cat. No.: GC16267

Chemical Properties

Cas. No.	636-00-0		
Chemical Name	5-(2-aminoethyl)benzene-1,2,4-triol hydrobromide		
SMILES	OC(C(CCN)=C1)=CC(O)=C1O.Br		
Formula	$C_8H_{11}NO_3$.HBr	M.Wt	250.09
Solubility	≥ 25mg/mL in DMSO with ultrasonic	Storage	Store at -20°C,stored under nitrogen,unstable in solution, ready to use,away from light.
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]:

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Cell lines	Primary nigral cells (PNCs)
Preparation Method	The conditioned media were replaced with new media containing AS-IV at the final concentration indicated with or without 200 µM of 6-Hydroxydopamine hydrobromide.
Reaction Conditions	200µM for 24 hours
Applications	Compared with vehicle controls, exposure to 200 µM 6-Hydroxydopamine resulted in the loss of 75% of PNCs, as determined using the LDH assay.

Animal experiment [2]:

Animal models	Female Sprague-Dawley rats
Preparation Method	The animals received a single, intrastriatal injection of 6-Hydroxydopamine hydrobromide. Twenty-five micrograms of 6-Hydroxydopamine hydrobromide, which was dissolved in 1.5 µl of normal saline with 0.2% ascorbic acid, was injected at a depth of 5.6 mm ventral to the skull at the same anterior/posterior and medial/lateral coordinates at which the cells had been injected. The injection site was chosen to be midway between the two implant sites. 6-Hydroxydopamine hydrobromide was injected over 5 min, and the needle was left in place for an additional 5 min before withdrawal of the needle.
Dosage form	25 µg in 1.5 µl of normal saline with 0.2% ascorbic acid

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Applications	Induced neurodegeneration in the SNc by unilateral injection of 6-hydroxydopamine hydrobromide into mfb, act as 6-OHDA lesion model

References:
[1]: Chan W S, Durairajan S S K, Lu J H, et al. Neuroprotective effects of Astragaloside IV in 6-hydroxydopamine-treated primary nigral cell culture[J]. Neurochemistry International, 2009, 55(6): 414-422.
[2]: Shults C W, Ray J, Tsuboi K, et al. Fibroblast growth factor-2-producing fibroblasts protect the nigrostriatal dopaminergic system from 6-hydroxydopamine[J]. Brain research, 2000, 883(2): 192-204.

Background

6-Hydroxydopamine hydrobromide (6-OHDA) is a structural analogue of catecholamines, dopamine and noradrenaline, and exerts its toxic effects on catecholaminergic neurons. The neurotoxin 6-hydroxydopamine continues to constitute a valuable topical tool used chiefly in modeling Parkinson's disease (PD) in the rat [1].

The classical method of intracerebral infusion of 6-Hydroxydopamine hydrobromide, involving a massive destruction of nigrostriatal dopaminergic neurons, is largely used to investigate motor and biochemical dysfunctions in Parkinson's disease [1]

6-Hydroxydopamine hydrobromide undergoes robust auto-oxidation generating cytotoxic H2O2, reactive oxygen species (ROS) and catecholamine quinones which attack endocellular nucleophilic groups [2].

Neurotoxic effects of 6-Hydroxydopamine hydrobromide occur through a two-step mechanism involving accumulation of the toxin into catecholaminergic neurons, followed by alteration of cellular homeostasis and neuronal damage. Intracellular storage of 6-Hydroxydopamine hydrobromide is mediated by the dopamine or noradrenaline membrane transporters (DAT and NAT respectively), which recognize and uptake 6-Hydroxydopamine hydrobromide due to its structural similarity with endogenous catecholamines [3]. 6-Hydroxydopamine hydrobromide is infused unilaterally in the

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MFB, producing a functional imbalance between the dopaminergic nigrostriatal systems and resulting in motor slowness, indicative of parkinsonian-like akinesia, and typical rotational behaviour in response to dopaminomimetic agents [4].

References:

[1]. Simola N, Morelli M, Carta A R. The 6-hydroxydopamine model of Parkinson's disease[J]. Neurotoxicity research, 2007, 11(3): 151-167.
[2]. Palumbo A, A Napolitano, P Barone and M d'Ischia (1999) Nitrite- and peroxide-dependent oxidation pathways of dopamine: 6-nitrodopamine and 6-hydroxydopamine formation as potential contributory mechanisms of oxidative stress- and nitric oxide-induced neurotoxicity in neuronal degeneration. Chem. Res. Toxicol. 12, 1213-1222.
[3]. Luthman J, A Fredriksson, E Sundstrom, G Jonsson and T.Archer (1989) Selective lesion of central dopamine or noradrenaline neuron systems in the neonatal rat: motor behavior and monoamine alterations at adult stage. Behav. Brain.Res. 33, 267-277.
[4]. Ungerstedt U and G Arbuthnott (1970) Quantitative recording of rotational behavior in rats after 6-hydroxydopamine lesions of the nigrostriatal dopamine system. Brain Res. 24, 485-493.

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