

DATASHEET

NBQX disodium salt

Product overview

Name	NBQX disodium salt
Cat No	HB0443
Alternative names	FG9202
Biological action	Antagonist
Purity	>99%
Customer comments	<i>High quality and affordable! We use this compound routinely in the lab for neuronal recordings. Verified customer, The University of Montana</i>

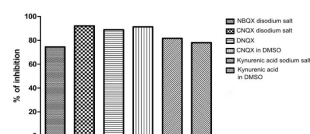
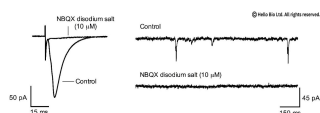
*Worked just as it should, results indistinguishable from our previous product but at a significant cost reduction! **Verified customer, The University of Toronto***

*Good quality and great price! **Verified customer, The University of Newcastle***

*NBQX disodium salt produced by Hello Bio produced a very potent and "clean" block of synaptic AMPA currents, with no effect on other GABAA or NMDA receptors. **Verified customer, The University of Edinburgh***

Description Potent, selective, competitive AMPA receptor antagonist. Disodium salt.

Images



Biological Data

Biological description

Potent, selective and competitive AMPA receptor antagonist. Also kainate receptor antagonist. Water soluble, disodium salt. Blocks the induction of excitatory post synaptic currents. Shows neuroprotective, antinociceptive and anticonvulsive actions. **NBQX** also available.

Application notes

The AMPA receptor antagonist NBQX disodium salt inhibits the actions of glutamate by acting at AMPARs and is commonly used at 10 μ M. NBQX disodium salt from Hello Bio inhibits spontaneous and evoked excitatory post synaptic currents (EPSCs) (see Fig 1 above). Complete AMPA receptor blockade was achieved at 10 μ M and NBQX disodium salt was also effective at reducing these currents at 1 μ M.

#Protocol 1: Evoked and spontaneous excitatory post synaptic currents (EPSCs)

- Whole cell voltage clamp recordings were obtained from layer V neurons of the

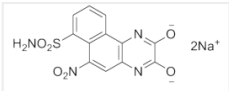
mouse prelimbic cortex brain slice.

- EPSCs were evoked via a stimulating electrode placed in layers II/III delivering a single square (150 μ s) pulse every 10 sec at an intensity that gave a reliable EPSC.
- Neurons were held at -70 to -60 mV (the reversal potential of GABA currents). EPSCs were continuously stimulated and recorded in response to 5 min applications of varying concentrations of NBQX disodium salt until complete receptor inhibition.
- Spontaneous EPSCs were recorded before and after addition of NBQX disodium salt by holding the neuron at -70 mV and recording for 10 sec.
- Recordings for EPSCs were made in the absence of GABA_A-R antagonists.

Solubility & Handling

Storage instructions	-20°C
Solubility overview	Soluble in water (100mM)
Important	This product is for RESEARCH USE ONLY and is not intended for therapeutic or diagnostic use. Not for human or veterinary use.

Chemical Data

Chemical name	2,3-Dioxo-6-nitro-1,2,3,4-tetrahydrobenzo[f]quinoxaline-7-sulfonamide disodium salt
Molecular Weight	380.24
Chemical structure	
Molecular Formula	C ₁₂ H ₆ N ₄ Na ₂ O ₆ S
CAS Number	479347-86-9
PubChem identifier	3272523
SMILES	[Na+].[Na+].NS(=O)(=O)c3cccc2c3c(cc1nc([O-])c([O-])nc12)[N+][([O-])=O
Source	Synthetic
InChi	InChI=1S/C12H8N4O6S.2Na/c13-23(21,22)8-3-1-2-5-9(8)7(16(19)20)4-6-10(5)15-12(18)11(17)14-6;;/h1-4H,(H,14,17)(H,15,18)(H2,13,21,22);;/q;2*+1/p-2
InChiKey	SVJKYIUJRJEABK-UHFFFAOYSA-L
MDL number	MFCD12910445
Appearance	Orange solid

References

It is AMPA receptor, not kainate receptor, that contributes to the NBQX-induced antinociception in the spinal cord of rats.

Kong LL *et al* (2006) Brain Res 1100(1)

PubMedID [16777075](#)

Competitive inhibition by NBQX of kainate/AMPA receptor currents and excitatory synaptic potentials: importance of 6-nitro substitution.

Randle JC *et al* (1992) Eur J Pharmacol 215(2-3)

PubMedID [1382998](#)

Both MK801 and NBQX reduce the neuronal damage after impact-acceleration brain injury.

Goda M *et al* (2002) J Neurotrauma 19(11)

PubMedID [12490009](#)

Antiepileptogenic and anticonvulsant effects of NBQX, a selective AMPA receptor antagonist, in the rat kindling model of epilepsy.

Namba T *et al* (1994) Brain Res 638(1-2)

PubMedID [8199874](#)

Pharmacological characterization of glutamatergic agonists and antagonists at recombinant human homomeric and heteromeric kainate receptors in vitro.

Alt *et al* (2004) Neuropharmacology 46(6)

