QNZ

Cat. No.: HY-13812

CAS No.: 545380-34-5

Molecular Formula: $C_{22}H_{20}N_4O$

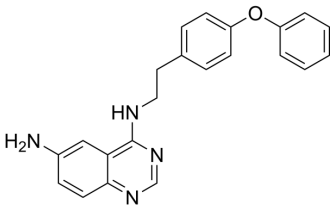
Molecular Weight: 356.42

Target: NF-κB; TNF Receptor

Pathway: NF-κB; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 37 mg/mL (103.81 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.8057 mL	14.0284 mL	28.0568 mL
5 mM	0.5611 mL	2.8057 mL	5.6114 mL
10 mM	0.2806 mL	1.4028 mL	2.8057 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.01 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.01 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.01 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

QNZ (EVP4593) shows strong inhibitory effects on NF-κB transcriptional activation and TNF-α production with IC_{50}s of 11 and 7 nM, respectively. QNZ (EVP4593) is a neuroprotective inhibitor of SOC channel.

IC_{50} & Target

NF-κB

11 nM (IC_{50}, in human Jurkat cells transfected with pNFκB-Luc)

In Vitro	QNZ (Compound 11q) has a suppressing effect of the NF-κB mediated-inflammatory response. QNZ inhibits edema formation dose-dependently[1]. QNZ (EVP4593) reduces the number of lysosomes/autophagosomes and store-operated channel (SOC) currents in Huntington's disease (HD). Normalization of calcium transport within neurons in response to QNZ is expect to reduce pathology manifestation. A number of lysosomes/autophagosomes are evaluated in HD and WT neurons treated with QNZ using transmission electron microscopy (TEM). Incubation with QNZ reduces the number of lysosomes/autophagosomes in HD GABAergic medium spiny (GABA MS)-like neurons (GMSLNs) by almost two-fold (from 0.41±0.04 to 0.23±0.04; $p<0.05$), while WT neurons are not affected. This observation is confirmed by examining lysosome content by flow cytometry (FC) analysis. The median fluorescence intensity is reduced by 34±6 % in HD GMSLNs upon QNZ treatment ($p<0.05$)[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	iPSHD22 cells are cultured in K-4 medium in a 96-well black plates with clear flat bottom. Next, cells are treated with chemical compounds (e.g., QNZ 100 nM) for 24 h prior to analysis. Fluorescent assay MultiTox-Fluor Multiplex Cytotoxicity Assay is used to measure simultaneously the relative number of live (viability) and dead (cytotoxicity) cells in each well. Fluorescence is detected by DTX 880 Multimode Microplate Reader. To evaluate the level of cell death (LoCD), the following equation is employed: ([cytotoxicity in a well with cells]-[cytotoxicity in a well without cells])/([viability in a well with cells]-[viability in a well without cells])[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Adv Sci (Weinh). 2023 Mar 8;e2201164.
- Cell Death Differ. 2023 Aug 15.
- Haematologica. 2020 Jan 23;haematol.2019.236927.
- Cancer Lett. 2022 Jun 7;215781.
- Biomed Pharmacother. 2023 Nov 18:169:115896.

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REFERENCES

[1]. Tobe M, et al. Discovery of quinazolines as a novel structural class of potent inhibitors of NF-kappa B activation.

[2]. Nekrasov ED, et al. Manifestation of Huntington's disease pathology in human induced pluripotent stem cell-derived neurons. Mol Neurodegener. 2016 Apr 14;11:27.

[3]. Wu J, et al. Enhanced Store-Operated Calcium Entry Leads to Striatal Synaptic Loss in a Huntington's Disease Mouse Model. J Neurosci. 2016 Jan 6;36(1):125-41.

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