

Product Data Sheet

Product Name: DPQ
Cat. No.: GC15294

Chemical Properties

Cas. No. 129075-73-6

Chemical Name 5-(4-(piperidin-1-yl)butoxy)-3,4-dihydroisoquinolin-1(2H)-one

SMILES O=C1NCCC2=C(C=CC=C21)OCCCN3CCCCC3

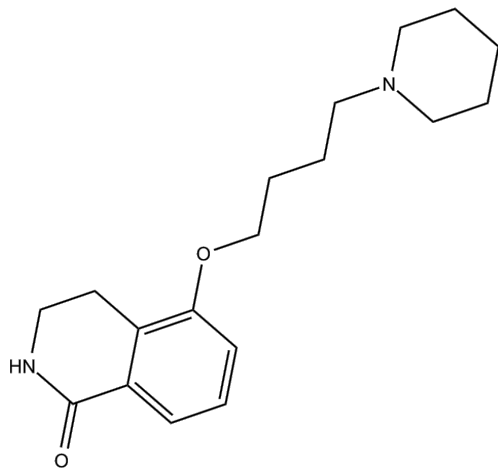
Formula $C_{18}H_{26}N_2O_2$ M.Wt 302.41

Solubility Soluble in DMSO Storage Store at -20°C

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

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

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Cell experiment [1]:

Cell lines A549 and H1299 (human non-small cell lung cancer cells), HeLa (human cervical cancer cells), MCF7 (human breast adenocarcinoma cells)

Preparation Method Cells were maintained in DMEM supplemented with 10% fetal bovine serum (FBS) at 37°C, 5% CO₂. Cells were treated with DPQ (1µM) 4 hours prior to carbon ion (¹²C) irradiation and cultured in the presence of DPQ post-irradiation.

Reaction Conditions 1µM; 4h pre-treatment followed by ¹²C irradiation (0.5–2Gy)

Applications DPQ significantly suppressed cell migration and wound healing in A549 and H1299 cells by inhibiting phosphorylation of EGFR, Akt, p38, and ERK, leading to inactivation of NF-κB. DPQ reduced MMP-2 and MMP-9 expression. DPQ also inhibited epithelial-mesenchymal transition (EMT) by downregulating N-cadherin, vimentin, and anillin while upregulating claudin-1 and claudin-2.

Animal experiment [2]:

Animal models C57BL/6 mice (8–10 weeks old, male)

Preparation Method Mice were intraperitoneally injected with LPS (7.5mg/kg) to induce acute lung injury. DPQ (1 or 10µg/kg) was administered intraperitoneally 30 minutes after LPS challenge. Mice were sacrificed 6 hours post-LPS exposure for lung tissue analysis.

Dosage form 10µg/kg; i.p.; Single injection.

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Applications DPQ treatment significantly attenuated LPS-induced acute lung injury by reducing neutrophil infiltration and inhibiting vascular permeability. DPQ suppressed mRNA expression of pro-inflammatory mediators (TNF- α , IL-1 β , IL-6, MIP-2, iNOS, and CXCL-1) in lung tissues and decreased apoptotic cell death.

References:

- [1] Chowdhury P, Dey P, Ghosh S, et al. Reduction of metastatic potential by inhibiting EGFR/Akt/p38/ERK signaling pathway and epithelial-mesenchymal transition after carbon ion exposure is potentiated by PARP-1 inhibition in non-small-cell lung cancer. BMC Cancer. 2019 Aug 22;19(1):829.
- [2] Wang G, Huang X, Li Y, et al. PARP-1 inhibitor, DPQ, attenuates LPS-induced acute lung injury through inhibiting NF- κ B-mediated inflammatory response. PLoS One. 2013 Nov 21;8(11):e79757.

Background

DPQ is a potent, blood-brain barrier-permeable poly(ADP-ribose) polymerase-1 (PARP-1) selective inhibitor used to study DNA damage repair and related cellular processes^[1-2]. DPQ can be applied to investigate inflammatory responses and cell death mechanisms in acute lung injury, myocardial ischemia-reperfusion injury, and neurodegenerative diseases, demonstrating neuroprotective and cardioprotective effects^[3-4].

In vitro, pretreatment of non-small cell lung cancer cells (A549 and H1299) with DPQ (1 μ M) for 4 hours, followed by carbon ion irradiation (0.5–2Gy) for 24 hours, significantly inhibited cell migration, wound healing capacity, and the activity and expression of matrix metalloproteinases (MMP-2 and MMP-9)^[5]. Pretreatment of HeLa cells with DPQ (20–150 μ M) for 1 hour, followed by stimulation with PMA (50nM) for 3 hours, significantly suppressed the upregulation of histamine H1 receptor (H1R) gene expression and reduced H1R promoter activity^[6].

In vivo, pretreatment of C57BL/6 mice with DPQ (15mg/kg) 24 hours before renal ischemia for 45 minutes/reperfusion for 6 hours reduced ischemia-reperfusion-related pathological damage, such as

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acute tubular necrosis (ATN), tubular cell vacuolization, and brush border loss, by inhibiting PARP-1 overactivation, while also ameliorating tubular epithelial cell shedding and cast formation^[7]. Pretreatment of C57BL/6 mice with LPS (7.5mg/kg) for 30 minutes, followed by intraperitoneal injection of DPQ (10µg/kg) for 6 hours, significantly inhibited neutrophil infiltration and myeloperoxidase (MPO) activity in lung tissue, while reducing the mRNA expression levels of pro-inflammatory factors such as TNF-α, IL-1β, and IL-6^[8].

References:

- [1] Meli E, Pangallo M, Picca R, et al. Differential role of poly(ADP-ribose) polymerase-1 in apoptotic and necrotic neuronal death induced by mild or intense NMDA exposure in vitro. *Mol Cell Neurosci*. 2004 Jan;25(1):172-80.
- [2] Wang J, Hao L, Wang Y, et al. Inhibition of poly (ADP-ribose) polymerase and inducible nitric oxide synthase protects against ischemic myocardial damage by reduction of apoptosis. *Mol Med Rep*. 2015 Mar;11(3):1768-76.
- [3] Czapski GA, Cakala M, Kopczuk D, et al. Effect of poly(ADP-ribose) polymerase inhibitors on oxidative stress evoked hydroxyl radical level and macromolecules oxidation in cell free system of rat brain cortex. *Neurosci Lett*. 2004 Feb 6;356(1):45-8.
- [4] Suh SW, Aoyama K, Alano CC, et al. Zinc inhibits astrocyte glutamate uptake by activation of poly(ADP-ribose) polymerase-1. *Mol Med*. 2007 Jul-Aug;13(7-8):344-9.
- [5] Chowdhury P, Dey P, Ghosh S, et al. Reduction of metastatic potential by inhibiting EGFR/Akt/p38/ERK signaling pathway and epithelial-mesenchymal transition after carbon ion exposure is potentiated by PARP-1 inhibition in non-small-cell lung cancer. *BMC Cancer*. 2019 Aug 22;19(1):829.
- [6] Mizuguchi H, Terao T, Kitai M, et al. Involvement of protein kinase Cdelta/extracellular signal-regulated kinase/poly(ADP-ribose) polymerase-1 (PARP-1) signaling pathway in histamine-induced up-regulation of histamine H1 receptor gene expression in HeLa cells. *J Biol Chem*. 2011 Sep 2;286(35):30542-30551.
- [7] del Moral RM, Gómez-Morales M, Hernández-Cortés P, et al. PARP inhibition attenuates histopathological lesion in ischemia/reperfusion renal mouse model after cold prolonged ischemia. *ScientificWorldJournal*. 2013 Nov 11;2013:486574.
- [8] Wang G, Huang X, Li Y, et al. PARP-1 inhibitor, DPQ, attenuates LPS-induced acute lung injury through inhibiting NF-κB-mediated inflammatory response. *PLoS One*. 2013 Nov 21;8(11):e79757.

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