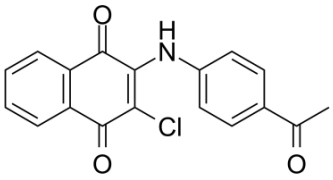
NQ301

Cat. No.:	HY-101054
CAS No.:	130089-98-4
Molecular Formula:	$C_{18}H_{12}ClNO_3$
Molecular Weight:	325.75
Target:	Thrombin
Pathway:	Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 29 mg/mL (89.03 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.0698 mL	15.3492 mL	30.6984 mL
5 mM	0.6140 mL	3.0698 mL	6.1397 mL
10 mM	0.3070 mL	1.5349 mL	3.0698 mL

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

BIOLOGICAL ACTIVITY

Description

NQ301 is an antithrombotic agent; inhibits collagen-challenged rabbit platelet aggregation with an IC_{50} of 10 mg/mL.

IC_{50} & Target

IC50: 0.60±0.02 μM (collagen-challenged rabbit platelet aggregation), 0.58±0.04 μM (U46619-challenged rabbit platelet aggregation), 0.78±0.04 μM (arachidonic acid-challenged rabbit platelet aggregation)[1]

In Vitro

NQ301 concentration-dependently inhibits collagen (10 mg/mL)-, U46619 (1 mg/mL)- and arachidonic acid (100 mg/mL)-challenged rabbit platelet aggregation, with IC_{50} values of 0.60±0.02, 0.58±0.04 and 0.78±0.04 μM, respectively. NQ301 potently suppresses thromboxane B_2 formation by platelets that are exposed to arachidonic acid in a concentration-dependent manner, but had no effect on the production of prostaglandin D_2, indicating an inhibitory effect on thromboxane A_2 synthase. NQ301 has a potential to inhibit thromboxane A_2 synthase activity with thromboxane A_2/prostaglandin H_2 receptor blockade, and modulate arachidonic acid liberation as well as 12-hydroxy-5,8,10,14-eicosatetraenoic acid formation in platelets[1]. NQ301 inhibits platelet aggregation by suppression of the intracellular pathway, rather than by direct inhibition of fibrinogen-GPIIb/IIIa complex binding. NQ301 significantly inhibits the increase of cytosolic Ca^{2+} concentration and ATP secretion, and also significantly increases platelet cAMP levels in the activated platelets. The

antiplatelet activity of NQ301 may be mediated by inhibition of cytosolic Ca^{2+} mobilization, enhancement of cAMP production and inhibition of ATP secretion in activated platelets[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]

ished rabbit platelet suspension is challenged by addition of collagen (10 mg/mL), arachidonic acid (100 μM) or U46619 (1 μ M). Concentration- response relationship is determined in the absence or presence of a range of concentrations of NQ301 (0, 0.25, 0.5, 0.75, 1 μM); aspirin-treated platelets (50 μM for 5 min) are used to prevent any possible contribution of endogenous arachidonic acid metabolites to platelet aggregation. The resulting aggregation, measured as the change in light transmission, is recorded for 5 min. The extent of platelet aggregation is expressed as % of the control[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Jin YR, et al. An antithrombotic agent, NQ301, inhibits thromboxane A2 receptor and synthase activity in rabbit platelets. Basic Clin Pharmacol Toxicol. 2005 Sep;97(3):162-7.

[2]. Zhang YH, et al. Antiplatelet effect of 2-chloro-3-(4-acetophenyl)-amino-1,4-naphthoquinone (NQ301): a possible mechanism through inhibition of intracellular Ca2+ mobilization. Biol Pharm Bull. 2001 Jun;24(6):618-22.

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