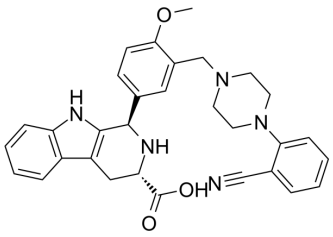
Ned-K

Cat. No.:	HY-131041
CAS No.:	2250019-90-8
Molecular Formula:	$C_{31}H_{31}N_5O_3$
Molecular Weight:	521.61
Target:	Calcium Channel
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Ned-K is a nicotinic acid adenine dinucleotide phosphate (NAADP) antagonist. Ned-K is effective at dampening simulated ischaemia and reperfusion (sIR)-induced Ca^{2+} oscillations in cardiomyocytes[1].

In Vitro

Ned-K suppresses Ca^{2+} oscillations and dramatically protects cardiomyocytes from cell death in vitro after ischaemia and reoxygenation, preventing opening of the mitochondrial permeability transition pore. Ned-K (10 μM) almost completely eliminates $[Ca^{2+}]_c$ oscillations, and Ned-K (0.1 μM) is effective at suppressing $[Ca^{2+}]_c$ levels[1].

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

Cell Viability Assay[1]

Cell Line:	Dead primary adult cardiomyocytes
Concentration:	0.1 and 10 μM
Incubation Time:	
Result:	Treatment with 0.1 μM caused a slight decrease in cardiomyocyte death (34±6%). Treatment with 10 μM at reoxygenation significantly decreased cell death after sIR to 16±1%.

In Vivo

Injection of Ned-K causes a significant reduction in infarct size in mice. Ned-K (administered i.v. to mice 5 min before reperfusion) significantly decreases myocardial infarct size relative to area at risk[1].

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REFERENCES

[1]. Sean M Davidson, et al. Inhibition of NAADP signalling on reperfusion protects the heart by preventing lethal calcium oscillations via two-pore channel 1 and opening of the mitochondrial permeability transition pore. Cardiovasc Res. 2015 Dec 1;108(3):357-66.

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

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