

Product Data Sheet

Product Name: NECA
Cat. No.: GC15304

Chemical Properties

Cas. No. 35920-39-9

Chemical Name (2S,3S,4R,5S)-5-(6-amino-9H-purin-9-yl)-N-ethyl-3,4-dihydroxytetrahydrofuran-2-carboxamide

SMILES O[C@H]1[C@@H](N2C3=NC=NC(N)=C3N=C2)O[C@H](C(NCC)=O)[C@H]1O

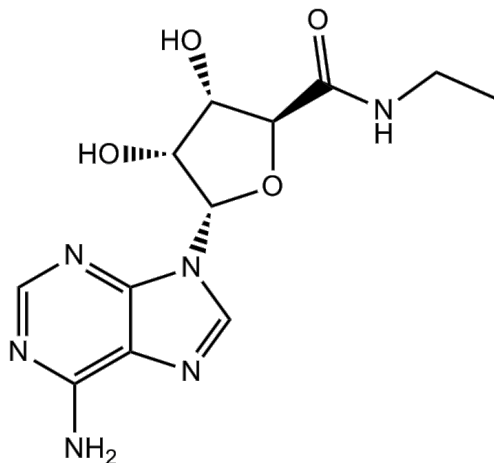
Formula $C_{12}H_{16}N_6O_4$ M.Wt 308.3

Solubility $\geq 15.35\text{mg/mL}$ 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

Cell experiment [1]:

Cell lines H9c2 cells

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

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Preparation Method	Cells were washed twice with phosphate-buffered saline (PBS) and then incubated in Tyrode solution for 2h before experiments. To examine the effect of NECA on mitochondrial membrane potential, GSK-3 β phosphorylation, and GRP94, cells were exposed to 800 μ M H ₂ O ₂ for 20 minutes to cause oxidant damage. A range concentrations of NECA (0.1–10 μ M) were given 10min before exposing to H ₂ O ₂ . In the study exploring roles of ERS and the cGMP/PKG signaling pathway in NECA-induced cardioprotection against oxidative damage, cells were exposed to 0.1 μ M NECA/20mM 2-DG/0.1 μ M KT5823 for 20min. The 2-DG and KT5823 were applied for 10min before the cells were exposed to NECA.
Reaction Conditions	0.1–10 μ M; 20min
Applications	NECA blocked H ₂ O ₂ -induced GSK-3 β phosphorylation and GRP94 expression.

Animal experiment [2]:

Animal models	Ex-hypoxic polycythemic mice (EHPCM) model
Preparation Method	This in vivo experiment used the Ex-hypoxic polycythemic mice (EHPCM) model, which induced erythrocytosis by exposing mice to a low-pressure hypoxic environment for a long time. On the 3rd to 6th day after the end of the hypoxic treatment, NECA was given intravenously or intraperitoneally, and finally a short-term hypoxia stimulation was given again. The blood, kidneys and livers of the mice were then collected, and the Epo (erythropoietin) content in the serum and tissues was detected to evaluate the in vivo regulatory effect of NECA on Epo production.
Dosage form	0.05, 0.1 μ mol/kg; iv; single injection

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Applications NECA significantly increased serum Epo levels in mice with hypoxic erythrocytosis.

References:

- [1]. Xing F, Han H, He Y, et al. Roles of endoplasmic reticulum stress in NECA-induced cardioprotection against ischemia/reperfusion injury[J]. *Oxidative Medicine and Cellular Longevity*, 2017, 2017(1): 2490501.
- [2]. Nakashima J, Ohigashi T, Brookins J W, et al. Effects of 5'-N-ethylcarboxamideadenosine (NECA) on erythropoietin production[J]. *Kidney international*, 1993, 44(4): 734-740.

Background

NECA is a non-selective agonist of adenosine receptors [1]. NECA regulates intracellular cAMP levels by activating adenosine receptors, thereby affecting many cellular physiological processes, such as anti-inflammation, vasodilation, inhibition of neuronal activity, cardioprotection, and immune regulation [2-3].

In H9c2 cells, NECA (0.1–10 μ M; 20min) blocked H₂O₂-induced GSK-3 β phosphorylation and GRP94 expression [4]. In skeletal muscle cells, NECA (100nM, 10 μ M; 19h) modulates CPT1B, stimulates LCAD, inhibits ACC β mRNA gene expression [5]. In HREC cells, NECA (10 μ M; 24-72h) induced a time-dependent increase in cell proliferation [6].

In ex-hypoxic polycythemic mice model, NECA (0.05, 0.1 μ mol/kg; iv; single injection) significantly increased serum erythropoietin levels in mice with hypoxic erythrocytosis [7]. In mice that were only immunized, intranasal NECA (50 μ L; 1mM; intranasal administration; single administration) administration caused a significant increase in bronchoalveolar lavage total cell count (TCC), neutrophils and eosinophils [8].

References:

- [1]. Knapp C M, Foye M M, Cottam N, et al. Adenosine agonists CGS 21680 and NECA inhibit the initiation of cocaine self-administration[J]. *Pharmacology Biochemistry and Behavior*, 2001, 68(4): 797-803.
- [2]. Elsherbiny N M, Abd El Galil K H, Gabr M M, et al. Reno-protective effect of NECA in diabetic

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- nephropathy: implication of IL-18 and ICAM-1[J]. European Cytokine Network, 2012, 23(3): 78-86.
- [3]. Li L, Chen J, Wang Z, et al. NECA alleviates inflammatory responses in diabetic retinopathy through dendritic cell toll-like receptor signaling pathway[J]. Frontiers in Immunology, 2024, 15: 1415004.
- [4]. Xing F, Han H, He Y, et al. Roles of endoplasmic reticulum stress in NECA-induced cardioprotection against ischemia/reperfusion injury[J]. Oxidative Medicine and Cellular Longevity, 2017, 2017(1): 2490501.
- [5]. Haddad M. The effect of NECA, CGS 21680, PSB 603 on fatty acid transport and oxidation in skeletal muscle cells[J]. International Journal of Pharmaceutical Sciences and Research, 2016, 7(12): 4827.
- [6]. Grant M B, Davis M I, Caballero S, et al. Proliferation, migration, and ERK activation in human retinal endothelial cells through A2B adenosine receptor stimulation[J]. Investigative ophthalmology & visual science, 2001, 42(9): 2068-2073.
- [7]. Nakashima J, Ohigashi T, Brookins J W, et al. Effects of 5'-N-ethylcarboxamideadenosine (NECA) on erythropoietin production[J]. Kidney international, 1993, 44(4): 734-740.
- [8]. El-Hashim A Z, Abduo H T, Rachid O M, et al. Intranasal administration of NECA can induce both anti-inflammatory and pro-inflammatory effects in BALB/c mice: Evidence for A2A receptor sub-type mediation of NECA-induced anti-inflammatory effects[J]. Pulmonary Pharmacology & Therapeutics, 2009, 22(3): 243-252.

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