N-Acetylneuraminic acid

Cat. No.: HY-I0400

CAS No.: 131-48-6

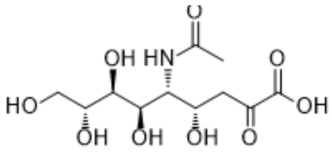
Molecular Formula: $C_{11}H_{19}NO_9$

Molecular Weight: 309.27

Target: Endogenous Metabolite; Influenza Virus; Tyrosinase; Ras

Pathway: Metabolic Enzyme/Protease; Anti-infection; GPCR/G Protein; MAPK/ERK Pathway

Storage: -20°C, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 125 mg/mL (404.18 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.2334 mL	16.1671 mL	32.3342 mL
5 mM	0.6467 mL	3.2334 mL	6.4668 mL
10 mM	0.3233 mL	1.6167 mL	3.2334 mL

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

In Vivo

1. Add each solvent one by one: PBS
Solubility: 100 mg/mL (323.34 mM); Clear solution; Need ultrasonic and warming and heat to 60°C

BIOLOGICAL ACTIVITY

Description

N-Acetylneuraminic acid (NANA; Lactaminic acid), a nonphenolic structure, is the predominant form of sialic from Collocalia esculenta. N-Acetylneuraminic acid plays a biological role in myocardial injury, melanoma and viral or bacterial infection. N-Acetylneuraminic acid inhibits melanogenesis by reducing tyrosinase activity and triggers myocardial injury in vitro and in vivo by activation of the Rho/Rho-associated signaling pathway through binding to RhoA and Cdc42. N-Acetylneuraminic acid may prevent high fat diet (HFD)-induced inflammation and oxidative stress, thereby prevents hyperlipidemia-associated inflammation and oxidative stress. N-Acetylneuraminic acid is promising for research in the field of melanoma, coronary artery, obesity-related diseases and hyperlipidemia[1][2][3][4][5].

IC_{50} & Target

Microbial Metabolite	Human Endogenous Metabolite

In Vitro

N-Acetylneuraminic acid (0 - 2 mg/mL, 2 days) has no effect on cell proliferation, but significantly decreases tyrosinase protein level, the number of melanosomes, p62 protein level and increases CCT3, CCT5, LC3-II protein level mRNA levels in B16 melanoma cells[1].

N-Acetylneuraminic acid (0 - 500 μM, 12 h) specifically binds to RhoA and Cdc42 and activates Rho/ROCK-JNK/ERK signaling

in cardiomyocytes[2].

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

Immunofluorescence[1]

Cell Line:	B16 melanoma cells
Concentration:	0 - 2 mg/mL
Incubation Time:	2 days
Result:	Was colocalized in the presence of tyrosinase, LAMP-1 and cathepsin L2 at 1 and 2 mg/mL concentrations and reduced p62 levels in B16 melanoma cells.

Real Time qPCR[1]

Cell Line:	B16 melanoma cells
Concentration:	0 - 2 mg/mL
Incubation Time:	2 days
Result:	Significantly decreased tyrosinase protein level in a concentration-dependent manner and increased CCT3 and CCT5 mRNA levels in B16 melanoma cells.

Western Blot Analysis[1]

Cell Line:	B16 melanoma cells
Concentration:	0 - 2 mg/mL
Incubation Time:	2 days
Result:	Significantly increased the LC3-II protein level and decreased the p62 protein level at 1 and 2 mg/mL in B16 melanoma cells.

Cell Viability Assay[2]

Cell Line:	Neonatal rat ventricular myocytes (NRVMs)
Concentration:	0.05 - 5 mM
Incubation Time:	12 h
Result:	Decreased the NRVMs cells viability and increased the release of lactate dehydrogenase and creatine kinase-MB.

Western Blot Analysis[2]

Cell Line:	NRVMs cells
Concentration:	0 - 500 μM
Incubation Time:	12 h
Result:	Induced significant increase and activation in RhoA and Cdc42, also enhanced the phosphorylation of JNK and ERK in NRVMs cells.

In Vivo

N-Acetylneuraminic acid (0 - 20 mg/kg, i.p., twice a day for 6 weeks) triggers myocardial injury in healthy rats[2].

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

Animal Model:	Healthy rats[2]
Dosage:	0 - 20 mg/kg
Administration:	i.p., twice a day for 6 weeks
Result:	Increased left ventricular end-diastolic pressure, -dP/dtmax and dp/dtmax, but showed no significant effects on left ventricular systolic pressure in rats.

CUSTOMER VALIDATION

- Nano Today. 2024 Aug.
- Int Immunopharmacol. 2023 Jun 2;120:110410.
- IUBMB Life. 2023 Oct 11.

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REFERENCES

[1]. Yoshikawa, K., et al. N-Acetylneuraminic Acid Inhibits Melanogenesis via Induction of Autophagy[J]. Cosmetics 2024, 11, 82.

[2]. Zhang L, et al. Functional Metabolomics Characterizes a Key Role for N-Acetylneuraminic Acid in Coronary Artery Diseases[J]. Circulation. 2018 Mar 27;137(13):1374-1390.

[3]. Yida Z, et al. High fat diet-induced inflammation and oxidative stress are attenuated by N-acetylneuraminic acid in rats[J]. J Biomed Sci. 2015 Oct 24;22:96.

[4]. Kiefel MJ, et al. Synthesis and biological evaluation of N-acetylneuraminic acid-based rotavirus inhibitors[J]. J Med Chem. 1996 Mar 15;39(6):1314-20.

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