Trimethylamine N-oxide

Cat. No.: HY-116084

CAS No.: 1184-78-7

Molecular Formula: C_3H_9NO

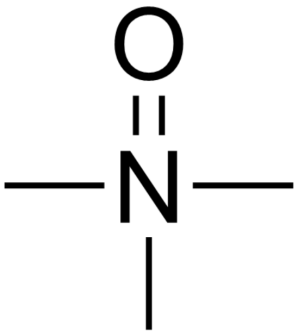
Molecular Weight: 75.11

Target: Endogenous Metabolite; NOD-like Receptor (NLR); Reactive Oxygen Species; TGF-beta/Smad

Pathway: Metabolic Enzyme/Protease; Immunology/Inflammation; NF-κB; Stem Cell/Wnt; TGF-beta/Smad

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (1331.38 mM; Need ultrasonic)
DMSO : 100 mg/mL (1331.38 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	13.3138 mL	66.5690 mL	133.1381 mL
	5 mM	2.6628 mL	13.3138 mL	26.6276 mL
	10 mM	1.3314 mL	6.6569 mL	13.3138 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 (1331.38 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (33.28 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (33.28 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (33.28 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Trimethylamine N-oxide is a gut microbe-dependent metabolite of dietary choline and other trimethylamine-containing nutrients. Trimethylamine N-oxide induces inflammation by activating the ROS/NLRP3 inflammasome. Trimethylamine N-

	oxide also accelerates fibroblast-myofibroblast differentiation and induces cardiac fibrosis by activating the TGF-β/smad2 signaling pathway[1][2][3].		
IC_{50} & Target	NLRP3	Microbial Metabolite	Human Endogenous Metabolite
In Vitro	The size and migration of fibroblasts are increased after Trimethylamine N-oxide (TMAO) treatment compared with non-treated fibroblasts in vitro. Trimethylamine N-oxide increases TGF-β receptor I expression, which promotes the phosphorylation of Smad2 and up-regulates the expression of α-SMA and collagen I. The ubiquitination of TGF-βRI is decreased in neonatal mouse fibroblasts after Trimethylamine N-oxide treatment. Trimethylamine N-oxide also inhibits the expression of smurf2[2]. Trimethylamine N-oxide is frequently found in the tissues of a variety of marine organisms that protects against the adverse effects of temperature, salinity, high urea and hydrostatic pressure[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.		
In Vivo	Trimethylamine N-oxide can be used in animal modeling to construct models of cardiac fibrosis. Trimethylamine N-oxide (TMAO) contributes to cardiovascular diseases by promoting inflammatory responses. C57BL/6 mice are fed a normal diet, high-choline diet and/or 3-dimethyl-1-butanol (DMB) diet. The levels of Trimethylamine N-oxide and choline are increased in choline-fed mice. Left ventricular hypertrophy, pulmonary congestion, and diastolic dysfunction are markedly exacerbated in heart failure with preserved ejection fraction (HFpEF) mice fed high-choline diets compared with mice fed the control diet. Myocardial fibrosis and inflammation were markedly increased in HFpEF mice fed high-choline diets compared with animals fed the control diet[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.		

CUSTOMER VALIDATION

- Phytomedicine. 2022 Mar 21;100:154067.
- Clin Chim Acta. 2023 Dec 16:117726.

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REFERENCES

[1]. Wei Shuai, et al. High-choline Diet Exacerbates Cardiac Dysfunction, Fibrosis, and Inflammation in a Mouse Model of Heart Failure With Preserved Ejection Fraction. J Card Fail. 2020 May 14;S1071-9164(19)31802-0.

[2]. Wenlong Yang, et al. Gut Microbe-Derived Metabolite Trimethylamine N-oxide Accelerates Fibroblast-Myofibroblast Differentiation and Induces Cardiac Fibrosis. J Mol Cell Cardiol. 2019 Sep;134:119-130.

[3]. Manuel T Velasquez, et al. Trimethylamine N-Oxide: The Good, the Bad and the Unknown. Toxins (Basel). 2016 Nov 8;8(11):326.

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