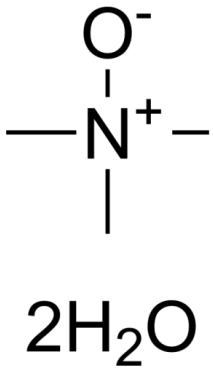
Data Sheet

Product Name:	Trimethylamine N-oxide (dihydrate)
Cat. No.:	CS-0031514
CAS No.:	62637-93-8
Molecular Formula:	$C_3H_{13}NO_3$
Molecular Weight:	111.14
Target:	Endogenous Metabolite; NOD-like Receptor (NLR); Reactive Oxygen Species; TGF-beta/Smad
Pathway:	Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB; Stem Cell/Wnt; TGF-beta/Smad
Solubility:	H2O : 100 mg/mL (899.77 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

Trimethylamine N-oxide dihydrate is a gut microbe-dependent metabolite of dietary choline and other trimethylamine-containing nutrients. Trimethylamine N-oxide dihydrate induces inflammation by activating the **ROS/NLRP3 inflammasome**. Trimethylamine N-oxide dihydrate also accelerates fibroblast-myofibroblast differentiation and induces cardiac fibrosis by activating the **TGF-β/smad2** signaling pathway[1][2][3]. ***In Vitro:*** The size and migration of fibroblasts are increased after Trimethylamine N-oxide (TMAO) dihydrate treatment compared with non-treated fibroblasts in vitro. Trimethylamine N-oxide dihydrate 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-β RI is decreased in neonatal mouse fibroblasts after Trimethylamine N-oxide dihydrate treatment. Trimethylamine N-oxide dihydrate 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]. ***In Vivo:*** Trimethylamine N-oxide (TMAO) dihydrate 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 dihydrate 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].

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.

CAIndexNames:

Methanamine, N,N-dimethyl-, N-oxide, dihydrate (9CI)

SMILES:

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

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