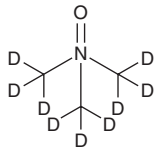
PRODUCT INFORMATION

Trimethylamine-d_9 N-oxide

Item No. 31449

CAS Registry No.: 1161070-49-0
Formal Name: N,N-di(methyl-d_3)-methan-d_3-amine, N-oxide
Synonym: TMAO-d_9
MF: C_3D_9NO
FW: 84.2
Chemical Purity: ≥98% (Trimethylamine N-oxide)
Deuterium Incorporation: ≥99% deuterated forms (d_1-d_9); ≤1% d_0
Supplied as: A solid
Storage: -20°C
Stability: ≥2 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Trimethylamine-d_9 N-oxide is intended for use as an internal standard for the quantification of trimethylamine N-oxide (Item No. 17354) by GC- or LC-MS. The accuracy of the sample weight in this vial is between 5% over and 2% under the amount shown on the vial. If better precision is required, the deuterated standard should be quantitated against a more precisely weighed unlabeled standard by constructing a standard curve of peak intensity ratios (deuterated *versus* unlabeled).

Trimethylamine-d_9 N-oxide is supplied as a solid. A stock solution may be made by dissolving the trimethylamine-d_9 N-oxide in the solvent of choice, which should be purged with an inert gas. Trimethylamine-d_9 N-oxide is slightly soluble in methanol.

Description

Trimethylamine N-oxide (TMAO) is a metabolite of choline, phosphatidylcholine, and L-carnitine (Item No. 21489).[1] It is formed by gut microbiota-mediated metabolism of choline, phosphatidylcholine, and L-carnitine to TMA followed by oxidation of TMA by flavin-containing monooxygenase 3 (FMO3) in the liver.[1-3] Dietary administration of TMAO (0.12% w/w) increases renal tubulointerstitial fibrosis, collagen deposition, and Smad3 phosphorylation in mice and increases aortic lesion area in atherosclerosis-prone *ApoE*$^{-/-}$ mice.[1,4] Plasma levels of TMAO are elevated in patients with chronic kidney disease and decreased in patients with active, compared with inactive, ulcerative colitis.[1,2] Elevated plasma levels of TMAO are associated with increased risk of cardiovascular disease.[4]

References

1. Tang, W.H.W., Wang, Z., Kennedy, D.J., *et al.* Gut microbiota-dependent trimethylamine *N*-oxide (TMAO) pathway contributes to both development of renal insufficiency and mortality risk in chronic kidney disease. *Circ. Res.* **116(3)**, 448-455 (2015).
2. Wilson, A., Teft, W.A., Morse, B.L., *et al.* Trimethylamine-*N*-oxide: A novel biomarker for the identification of inflammatory bowel disease. *Dig. Dis. Sci.* **60(12)**, 3620-3630 (2015).
3. Zhang, L.S. and Davies, S.S. Microbial metabolism of dietary components to bioactive metabolites: Opportunities for new therapeutic interventions. *Genome Med.* **8(1)**, 46 (2016).
4. Wang, Z., Klipfell, E., Bennett, B.J., *et al.* Gut flora metabolism of phosphatidylcholine promotes cardiovascular disease. *Nature* **472(7341)**, 57-63 (2011).

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