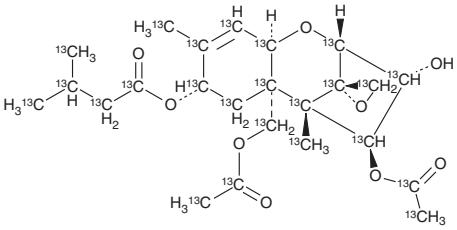
PRODUCT INFORMATION

T-2 Toxin-$^{13}C_{24}$

Item No. 31300

Formal Name: (2S,2'R,3'R,4'S,5'S,5a'R,7'S,9a'R)-4'-(acetoxy-$^{13}C_2$)-5a'-((acetoxy-$^{13}C_2$)methyl-^{13}C)-3'-hydroxy-5',8'-di(methyl-^{13}C)-2',3',4',5',5a',6',7',9a'-octahydrospiro[oxirane-2,10'-[2,5]methanobenzo[b]oxepin]-7'-yl-2,2',3,3',4',5',5a',6',7',8',9',9a'-$^{13}C_{12}$ 3-(methyl-^{13}C)butanoate-1,2,3,4-$^{13}C_4$



Synonyms: Fusariotoxin T-2-$^{13}C_{24}$, Insariotoxin-$^{13}C_{24}$, Mycotoxin T-2-$^{13}C_{24}$

MF: $[^{13}C]_{24}H_{34}O_9$

FW: 490.3

Purity: ≥98%

Supplied as: A 25 µg/ml solution in acetonitrile

Storage: -20°C

Stability: ≥2 years

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

Description

T-2 toxin-$^{13}C_{24}$ is intended for use as an internal standard for the quantification of T-2 toxin (Item No. 11444) by GC- or LC-MS. T-2 toxin is a trichothecene mycotoxin that has been found in *Fusarium*.[1] It binds to and inhibits peptidyltransferase in the 60S ribosomal subunit, inducing a ribotoxic stress response that triggers JNK and p38 MAPK signaling. T-2 toxin (3 nM) decreases toll-like receptor expression and LPS-induced production of IL-1β, TNF-α, and nitric oxide (NO) in, and is cytotoxic to (IC_{50} = 19.47), primary pig alveolar macrophages (PAMs).[2] *In vivo*, T-2 toxin induces production of reactive oxygen species (ROS), lipid peroxidation, and glutathione (GSH) depletion in mouse brain and is lethal to mice (LD_{50}s = 1.54-5.94 mg/kg).[1] It also induces hepatocyte apoptosis and dyslipidemias in mice. T-2 toxin has been found in *Fusarium*-infected wheat, barley, and rice crops both in fields and in storage.

References

1. Doi, K. and Uetsuka, K. Mechanisms of mycotoxin-induced neurotoxicity through oxidative stress-associated pathways. *Int. J. Mol. Sci.* **12(8)**, 5213-5237 (2011).
2. Seeboth, J., Solinhac, R., Oswald, I.P., *et al.* The fungal T-2 toxin alters the activation of primary macrophages induced by TLR-agonists resulting in a decrease of the inflammatory response in the pig. *Vet. Res.* **43(1)**, 1-18 (2012).

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