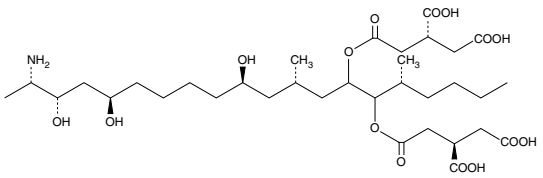
Product Information

Fumonisin B_1

Item No. 62580

CAS Registry No.: 116355-83-0
Formal Name: 1,2,3-propanetricarboxylic acid, 1,1'-[1-(12-amino-4,9,11-trihydroxy-2-methyltridecyl)-2-(1-methylpentyl)-1,2-ethanediyl] ester
MF: $C_{34}H_{59}NO_{15}$
FW: 721.8
Purity: ≥98%
Stability: ≥2 yearS at -20°C
Supplied as: A crystalline solid



Laboratory Procedures

For long term storage, we suggest that fumonisin B_1 be stored as supplied at -20°C. It should be stable for at least two years.

Fumonisin B_1 is supplied as a crystalline solid. A stock solution may be made by dissolving the Fumonisin B_1 in an organic solvent purged with an inert gas. Fumonisin B_1 is soluble in solvents such as methanol and acetonitrile. The solubility of fumonisin B_1 in these solvents is approximately 10 mg/ml.

Further dilutions of the stock solution into aqueous buffers or isotonic saline should be made prior to performing biological experiments. Ensure that the residual amount of organic solvent is insignificant since organic solvents may have physiological effects at low concentrations. If an organic solvent-free solution of fumonisin B_1 is needed, it can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of fumonisin B_1 in PBS (pH 7.2) is approximately 18.5 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Fumonisin B_1 is a mycotoxin produced by *F. moniliforme*, a prevalent fungus of corn and other grains. Outbreaks of food poisoning in livestock and humans following the consumption of *Fusarium* infested corn are caused by fumonisins.[1] It functions as an inhibitor of ceramide synthase (sphingosine N-acyltransferase).[2] Fumonisin B_1 attenuates the response of P388D1 cells to PAF and LPS by inhibiting ceramide formation.[3] It also blocks the apoptotic response of HaCaT cells to the antiproliferative drug hexadecylphosphocholine, again through inhibition of ceramide production.[4] Incubation of Swiss 3T3 cells with fumonisin B_1 results in both an altered cell morphology due to disruption of axonal growth and a decrease in cell proliferation.[5]

References

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2. Wang, E., Norred, W.P., Bacon, C.W., *et al.* Inhibition of sphingolipid biosynthesis by fumonisins. Implications for diseases associated with *Fusarium moniliforme*. *J. Biol. Chem.* **266**, 14486-14490 (1991).
3. Balsinde, J., Balboa, M.A., and Dennis, E.A. Inflammatory activation of arachidonic acid signaling in murine P388D1 macrophages via sphingomyelin synthesis. *J. Biol. Chem.* **272**, 20373-20377 (1997).
4. Wieder, T., Orfanos, C.E., and Geilen, C.C. Induction of ceramide-mediated apoptosis by the anticancer phospholipid analog, hexadecylphosphocholine. *J. Biol. Chem.* **273**, 11025-11031 (1998).
5. Meivar-Levy, I., Sbanay, H., Bershadsky, A.D., *et al.* The role of sphingolipids in the maintenance of fibroblast morphology. The inhibition of protrusional activity, cell spreading, and cytokinesis induced by fumonisin B_1 can be reversed by ganglioside GM_3. *J. Biol. Chem.* **272**, 1558-1564 (1997).

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