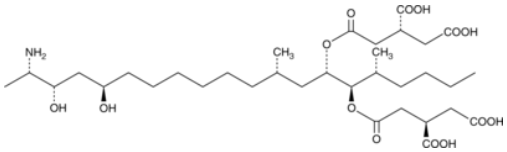
Product Data Sheet

Product Name: Fumonisin B2
Cat. No.: GC43709

Chemical Properties

Cas. No.	116355-84-1		
Chemical Name	(2R,2'R)-1,2,3-propanetricarboxylic acid, 1,1'-[(1S,2R)-1-[(2S,4R,9R,11S,12S)-12-amino-4,9,11-trihydroxy-2-methyltridecyl]-2-[(1R)-1-methylpentyl]-1,2-ethanediyl] ester		
SMILES	C[C@H](N)[C@@H](O)C[C@H](O)CCCCCC[C@H](C)C[C@H](OC(C[C@H](C(O)=O)CC(O)=O)=O)[C@H](OC(C[C@H](C(O)=O)CC(O)=O)=O)[C@H](C)CCCC		
Formula	$C_{34}H_{59}NO_{14}$	M.Wt	705.8
Solubility	10mg/ml in methanol & acetonitrile	Storage	Store at -20°C,protect from light
General tips	For obtaining a higher solubility , please warm the tube at 37 °C and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20°C for several months.		
Shipping Condition	Evaluation sample solution : ship with blue ice All other available size: ship with RT , or blue ice upon request.		

Structure



Protocol

Cell experiment [1]:

Cell lines HEK293 cells

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Preparation Method	HEK293 cells were cultured in 25cm^3 cell culture flasks using Dulbecco's minimum essential medium (DMEM) supplemented with 2.5mM HEPES, 10% foetal bovine serum, 1% pen-strep-fungizone, and 1% L-glutamine, maintained in a humidified incubator (37°C; 5% CO_2) until approximately 80% confluent. 2×10^4 cells/well were seeded and allowed to adhere overnight in a 96-well plate (37°C, 5% CO_2). Thereafter, cells were incubated for 24h with varying concentrations of Fumonisin B2 (0, 1, 5, 10, 50, 100, 250, and 500μM). Control wells contained DMEM only. Cell viability was measured.
Reaction Conditions	0, 1, 5, 10, 50, 100, 250, and 500μM; 24h
Applications	Fumonisin B2 treatment significantly decreased the cell viability of HEK293 cells in a dose-dependent manner.

Animal experiment [2]:

Animal models	Female BALB/c mice
Preparation Method	Female BALB/c mice (7 weeks old) were housed in standard cages at room temperature (20-26°C) and humidity (40-60%) under a 12/12h light/dark cycle with ad libitum access to autoclaved chow and water. 5% toluene-2,4-diisocyanate (TDI) dissolved in acetone was topically applied onto the depilated abdominal surface on day 1 through day 3, and re-sensitization with 0.5% acetone was performed 3 weeks after the first sensitization. One week after re-sensitization, 0.5% TDI was applied on both sides of the ear auricle as a challenge. Mice were orally administered vehicle alone. After oral administration of Fumonisin B2 at a dose of 1mg/kg/day for 3 consecutive days, the skin condition of mice was analyzed.
Dosage form	1mg/kg/day for 3 days; p.o.

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

Applications	Fumonisin B2 treatment considerably exacerbated skin thickness, transepidermal water loss, histological features, and proinflammatory cytokine production in mice.

References:
[1] Mohan J, Sheik Abdul N, Nagiah S, et al. Fumonisin B2 induces mitochondrial stress and mitophagy in human embryonic kidney (Hek293) cells—a preliminary study[J]. Toxins, 2022, 14(3): 171.
[2] Ando M, Yamaguchi H, Morimoto A, et al. Chronic oral exposure to low-concentration fumonisin B2 significantly exacerbates the inflammatory responses of allergies in mice via inhibition of IL-10 release by regulatory T cells in gut-associated lymphoid tissue[J]. Archives of Toxicology, 2023, 97(10): 2707-2719.

Background

Fumonisin B2 is a selective ceramide synthase inhibitor and carcinogenic mycotoxin [1]. Fumonisin B2 can induce an increase in the mRNA expression of the p21 gene and a decrease in the mRNA expression of the cyclin D1 gene, thereby altering the cell cycle progression[2]. Fumonisin B2 has been widely used to induce cytotoxicity and DNA fragmentation in turkey peripheral blood lymphocytes [3].

In vitro, Fumonisin B2 treatment for 96 hours significantly inhibited the viability of H4TG, FaO, and MDCK cells, with IC_{50} values of 2, 5, and 2μg/ml, respectively [4]. Treatment with 40μM Fumonisin B2 for 48 hours significantly inhibited the proliferation of GES-1 cells, increased the level of lactate dehydrogenase, and induced endoplasmic reticulum stress[5]. Treatment with 317.4μM Fumonisin B2 for 24 hours significantly induced cell death in HEK293 cells, promoted production of reactive oxygen species (ROS), and caused mitochondrial membrane depolarization [6].

In vivo, Fumonisin B2 treatment via oral administration at a dose of 1mg/kg/day for 3 days significantly exacerbated the inflammatory response in the atopic dermatitis (AD) mouse model, reduced epidermal water loss, and increased production of pro-inflammatory cytokines[7]. Oral administration of a 0.1mg/kg/day dose of Fumonisin B2 for 10 consecutive days significantly

exacerbated the pathological process of psoriasis induced by imiquimod in mice[8].

References:

[1] Frisvad J C, Smedsgaard J, Samson R A, et al. Fumonisin B2 production by Aspergillus niger[J]. Journal of agricultural and food chemistry, 2007, 55(23): 9727-9732.
[2] Bondy G S, Barker M G, Lombaert G A, et al. A comparison of clinical, histopathological and cell-cycle markers in rats receiving the fungal toxins fumonisin B1 or fumonisin B2 by intraperitoneal injection[J]. Food and chemical toxicology, 2000, 38(10): 873-886.
[3] Dombrink-Kurtzman M A. Fumonisin and beauvericin induce apoptosis in turkey peripheral blood lymphocytes[J]. Mycopathologia, 2003, 156(4): 357-364.
[4] Shier W T, Abbas H K, Mirocha C J. Toxicity of the mycotoxins fumonisins B1 and B2 and Alternaria alternata f. sp. lycopersici toxin (AAL) in cultured mammalian cells[J]. Mycopathologia, 1991, 116(2): 97-104.
[5] Yu S, Jia B, Liu N, et al. Evaluation of the individual and combined toxicity of fumonisin mycotoxins in human gastric epithelial cells[J]. International Journal of Molecular Sciences, 2020, 21(16): 5917.
[6] Mohan J, Sheik Abdul N, Nagiah S, et al. Fumonisin B2 induces mitochondrial stress and mitophagy in human embryonic kidney (Hek293) cells—a preliminary study[J]. Toxins, 2022, 14(3): 171.
[7] Ando M, Yamaguchi H, Morimoto A, et al. Chronic oral exposure to low-concentration fumonisin B2 significantly exacerbates the inflammatory responses of allergies in mice via inhibition of IL-10 release by regulatory T cells in gut-associated lymphoid tissue[J]. Archives of Toxicology, 2023, 97(10): 2707-2719.
[8] Ando M, Yamaguchi H, Iwashita N, et al. Oral Exposure to Low Concentration of Fumonisin B2, but Not Fumonisin B1, Significantly Exacerbates the Pathophysiology of Imiquimod-Induced Psoriasis in Mice[J]. International Journal of Molecular Sciences, 2024, 25(14): 7852.