

Product Data Sheet

Product Name: Fusaric Acid

Cat. No.: GC18061

Chemical Properties

Cas. No. 536-69-6

Chemical Name 5-butyl-2-pyridinecarboxylic acid

SMILES O=C(O)C1=CC=C(CCCC)C=N1

Formula $C_{10}H_{13}NO_2$

M.Wt 179.2

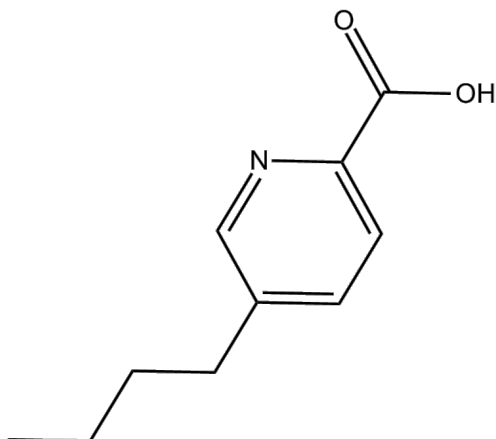
Solubility DMF: 30 mg/ml, DMSO: 30 mg/ml, Ethanol: 30 mg/ml, PBS (pH 7.2): 10 mg/ml

Storage Store at -20°C

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

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

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Cell experiment [1]:	
Cell lines	Thp-1 cells
Preparation Method	Thp-1 cells were cultured in RPMI-1640 medium, supplemented with 10% fetal bovine serum (FBS), 1% L-glutamine, 1mM sodium pyruvate, 0.05mM β -mecaptoethanol, and 1% penicillin/streptomycin at 37°C in an incubator with 5% CO ₂ . Cells were seeded in a 96-well plate at a density of 2×10^4 cells/well overnight and incubated with different concentrations of Fusaric Acid (0, 30, 60, 90, 120, 150, and 300 μ g/ml) for 24h, the cell proliferation was tested.
Reaction Conditions	0, 30, 60, 90, 120, 150, and 300 μ g/ml; 24h
Applications	Fusaric Acid treatment inhibited cell proliferation of Thp-1 cells in a dose-dependent manner.
Animal experiment [2]:	
Animal models	Male C57BL/6 mice
Preparation Method	Male C57BL/6 mice (6 weeks old; 18-22g) were housed under standard conditions [temperature (22-24°C) and humidity (40-60%), a 12h light/dark cycle] with ad libitum access to food and normal drinking water. Fusaric Acid was administered orally via oral gavage once daily at a dose of 50mg/kg. Control groups received phosphate buffer saline (0.1M PBS; 0.25ml) (by oral gavage). At the end of the experimental periods (10 days), mice were euthanized by gas inhalation anaesthesia. Brain tissues were surgically removed for analysis.

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Dosage form	50mg/kg/day; 10 days; p.o.
Applications	Fusaric Acid treatment dampened PI3K/Akt and AMPK signalling and increased the expression of GLUT receptors (1 and 4) in the mouse brain.
References: [1] Dhani S, Nagiah S, Naidoo D B, et al. Fusaric Acid immunotoxicity and MAPK activation in normal peripheral blood mononuclear cells and Thp-1 cells[J]. Scientific reports, 2017, 7(1): 3051. [2] Dhani S, Ghazi T, Nagiah S, et al. Fusaric acid alters Akt and ampk signalling in c57bl/6 mice brain tissue[J]. Food and Chemical Toxicology, 2020, 138: 111252.	

Background

Fusaric Acid, a picolinic acid derivative isolated from *Fusarium heterosporium*, is a fungal phytotoxin^[1]. Fusaric Acid induces chloroplast and mitochondrial dysfunction, lipid peroxidation, protein damage and DNA fragmentation, causing wilting, necrotic spots on leaves, and reduction in root and root hair growth^[2]. Fusaric Acid has been widely used in plant toxicity research to induce oxidative stress damage models in plant cells^[3].

In vitro, Fusaric Acid treatment for 24 hours significantly inhibited the viability of human peripheral blood mononuclear cells (PBMCs) and Thp-1 cells, with IC₅₀ values of 240.8µg/ml and 107.7µg/ml, respectively^[4]. 200µg/ml Fusaric Acid incubation for 24 hours promoted cell death in HepG2 cells, decreased intracellular ATP levels, and downregulated the expression of mitochondrial stress response proteins SIRT3 and LON^[5]. Incubation with 200µg/ml Fusaric Acid for 24 hours induced apoptosis in SNO cells, accompanied by increased Bax expression and decreased Bcl-2 expression^[6].

In vivo, Fusaric acid treatment via oral administration at a dose of 100mg/kg/day for 14 days inhibited isoproterenol-induced cardiac hypertrophy and dysfunction, and alleviated cardiac fibrosis in mice^[7].

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Oral administration of Fusaric acid at a dose of 50mg/kg daily for 10 days reduced ATP levels, decreased the Akt signaling pathway, and increased the expression of glucose receptors in the brain tissues of mice^[8].

References:

- [1] López-Díaz C, Rahjoo V, Sulyok M, et al. Fusaric acid contributes to virulence of Fusarium oxysporum on plant and mammalian hosts[J]. Molecular Plant Pathology, 2018, 19(2): 440-453.
- [2] Iqbal N, Czékus Z, Ördög A, et al. Fusaric acid-evoked oxidative stress affects plant defence system by inducing biochemical changes at subcellular level[J]. Plant Cell Reports, 2024, 43(1): 2.
- [3] Singh V K, Upadhyay R S. Fusaric acid induced cell death and changes in oxidative metabolism of Solanum lycopersicum L[J]. Botanical studies, 2014, 55(1): 66.
- [4] Dhani S, Nagiah S, Naidoo D B, et al. Fusaric Acid immunotoxicity and MAPK activation in normal peripheral blood mononuclear cells and Thp-1 cells[J]. Scientific reports, 2017, 7(1): 3051.
- [5] Abdul N S, Nagiah S, Chuturgoon A A. Fusaric acid induces mitochondrial stress in human hepatocellular carcinoma (HepG2) cells[J]. Toxicon, 2016, 119: 336-344.
- [6] Devnarain N, Tiloke C, Nagiah S, et al. Fusaric acid induces oxidative stress and apoptosis in human cancerous oesophageal SNO cells[J]. Toxicon, 2017, 126: 4-11.
- [7] Li X, Zhang Z L, Wang H F. Fusaric acid (FA) protects heart failure induced by isoproterenol (ISP) in mice through fibrosis prevention via TGF- β 1/SMADs and PI3K/AKT signaling pathways[J]. Biomedicine & Pharmacotherapy, 2017, 93: 130-145.
- [8] Dhani S, Ghazi T, Nagiah S, et al. Fusaric acid alters Akt and ampk signalling in c57bl/6 mice brain tissue[J]. Food and Chemical Toxicology, 2020, 138: 111252.

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