Product Data Sheet

Product Name: Phytic acid
Cat. No.: GN10554

Chemical Properties

Cas. No.	83-86-3		
Chemical Name	(2,3,4,5,6-pentaphosphonooxycyclohexyl) dihydrogen phosphate		
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Formula	$C_6H_{18}O_{24}P_6$	M.Wt	660.04
Solubility	Water : ≥ 250 mg/mL	Storage	Store at -20°C
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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	Phytic acid		

Protocol

Cell experiment [1]:

Cell lines	MC65 cells

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Preparation Method	Cell viability was determined using an MTS assay and a calcium ion assay. At 90% confluence, cells were harvested from TC flasks using 0.05% trypsin/EDTA, washed twice with PBS, and seeded at 15×10^3 cells/well in 96-well plates in OPTIMem medium containing (Tet+) or (Tet-) tetracycline (1μg/mL) and excipients, or phytic acid (100μM). Cells grown under Tet- conditions typically died after approximately 3–4 days of culture, accompanied by expression of endogenous A oligomers, whereas cells grown under Tet+ conditions remained viable during this period. Wells containing growth medium without cells served as background controls, and wells containing vehicle served as positive controls. Each treatment was replicated in 3–6 wells, and the treatment duration for each experiment was 72 hours. Absorbance was measured at a wavelength of 490nm using a Spectra Max PLUS microplate reader. The cell viability under different treatments was determined relative to the cell viability under Tet + treatment. The experiments were repeated at least three times.
Reaction Conditions	100μM; 4d
Applications	Cell survival rate was significantly improved after Phytic acid treatment.

Animal experiment [2]:

Animal models	Dextran sulfate sodium (DSS)-induced UC mice model

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Preparation Method	The C57BL/6 mice were randomly divided into four groups of five mice each. The specific groups were as follows: no treatment group (NT group), Phytic acid feeding group (Phytic acid group), DSS modeling group (DSS group), and Phytic acid treatment group (Phytic acid + DSS group). The mice in the Phytic acid and Phytic acid + DSS groups were gavaged with Phytic acid (0.25g/kg/day) for 21days. After receiving Phytic acid for 14days, mice in the DSS and Phytic acid + DSS groups were given 2.5% DSS aqueous solution for 7days to develop the mouse model of UC. The body weights of all mice were recorded from day 1 of the DSS feeding. Following the completion of the DSS induction period, all mice were euthanized, and colon samples were collected for subsequent studies.
Dosage form	0.25g/kg; ig; 14d
Applications	Phytic acid treatment improved weight loss, shortened colon length, improved clinical scores, and reduced the release of proinflammatory factors (Il-1β, Il-6, and TNF-α).

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References:
[1]. Anekonda T S, Wadsworth T L, Sabin R, et al. Phytic acid as a potential treatment for Alzheimer's pathology: evidence from animal and in vitro models[J]. Journal of Alzheimer’s disease, 2011, 23(1): 21-35.
[2]. Hou X, Sang Y, Dong L. The improved effect and its mechanism of phytic acid on DSS-induced UC mice[J]. Life sciences, 2022, 311: 121139.

Background

Phytic acid is an antinutrient found naturally in legume seeds[1]. Phytic acid and its

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Phytic acid is an antinutrient found naturally in legume seeds[1]. Phytic acid and its degradation products can scavenge free radicals and reduce oxidative stress [2]. Phytic acid chelation of metal ions (such as iron) can reduce metal-catalyzed oxidative reactions [3]. Phytic acid exhibits anticancer and antioxidant properties [4].

In MC65 cells, Cell survival rate was significantly improved after Phytic acid (100μM; 4d) treatment [5]. In HepG2 cells, Phytic acid (0.1-4mM; 24-48h) treatment inhibited cell growth [6].

In dextran sulfate sodium (DSS)-induced UC mice model, Phytic acid (0.25g/kg; ig; 14d) treatment improved weight loss, shortened colon length, improved clinical scores, and reduced the release of proinflammatory factors (Il-1β, Il-6, and TNF-α) [7]. In a high-fat diet mice model, Phytic acid (0.25-2mg/mL; po; 13 weeks) significantly inhibited obesity and alleviated hepatic steatosis [8].

References:
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[2]. Graf E, Empson K L, Eaton J W. Phytic acid. A natural antioxidant[J]. Journal of Biological Chemistry, 1987, 262(24): 11647-11650.
[3]. Lee B J, Hendricks D G. Metal-catalyzed oxidation of ascorbate, deoxyribose and linoleic acid as affected by phytic acid in a model system[J]. Journal of Food Science, 1997, 62(5): 935-984.
[4]. Bloot A P M, Kalschne D L, Amaral J A S, et al. A review of phytic acid sources, obtention, and applications[J]. Food Reviews International, 2023, 39(1): 73-92.
[5]. Anekonda T S, Wadsworth T L, Sabin R, et al. Phytic acid as a potential treatment for Alzheimer's pathology: evidence from animal and in vitro models[J]. Journal of Alzheimer’s disease, 2011, 23(1): 21-35.
[6]. Al-Fatlawi A A, Al-Fatlawi A A, Irshad M, et al. Rice bran phytic acid induced apoptosis through regulation of Bcl-2/Bax and p53 genes in HepG2 human hepatocellular carcinoma cells[J]. Asian Pacific Journal of Cancer Prevention, 2014, 15(8): 3731-3736.
[7]. Hou X, Sang Y, Dong L. The improved effect and its mechanism of phytic acid on DSS-induced UC mice[J]. Life sciences, 2022, 311: 121139.
[8]. Ran X, Hu G, He F, et al. Phytic acid improves hepatic steatosis, inflammation, and oxidative stress in high-fat diet (HFD)-fed mice by modulating the gut–liver axis[J].

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