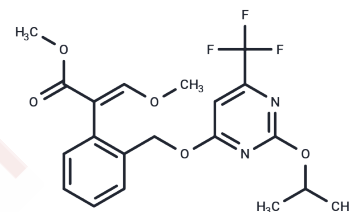


Fluacrypyrim

Chemical Properties

CAS No. :	229977-93-9
Formula:	C ₂₀ H ₂₁ F ₃ N ₂ O ₅
Molecular Weight:	426.39
Storage:	Powder: -20°C for 3 years In solvent: -80°C for 1 year Actual storage temperature shall be subject to the COA.



Biological Description

Description	Fluacrypyrim is an ester-based acaricide and functions as a STAT3 inhibitor. It significantly enhances the activity of protein tyrosine phosphatases (PTPs) and suppresses leukemia cell growth by inducing substantial G1 phase arrest and significantly reducing the levels of cyclin D1 protein and mRNA. Fluacrypyrim selectively inhibits the STAT3 signaling pathway, leading to growth arrest and apoptosis in STAT3-dependent cancer cells. It mainly alleviates radiation-induced hematopoietic system damage by preventing apoptosis in hematopoietic stem cells (HSCs). Additionally, fluacrypyrim exhibits notable analgesic and anti-inflammatory effects by inhibiting uterine smooth muscle contraction and inflammatory responses.
Targets(IC50)	Apoptosis,STAT,Parasite,Phosphatase,MDM-2/p53
In vitro	Fluacrypyrim decreases the apoptosis rate of bone marrow nucleated cells (BMNCs) and their subpopulations (Lin ⁻ cells, Lin ⁻ c-Kit ⁺ cells, LK cells, and LSK cells) following 6.5 Gy irradiation when applied for 3-12 hours. At a concentration of 5 μM for 4-10 hours, fluacrypyrim reduces BMNCs apoptosis through modulating the p53-PUMA pathway. The compound exhibits strong concentration and time-dependent inhibitory effects on HL-60 cell growth, with an IC ₅₀ of 3.8 μM, when applied in the range of 0.1-12 μM for 0-72 hours. Fluacrypyrim induces G1 phase arrest in HL-60 cells by downregulating cyclin-D1 at concentrations of 1.5-12 μM for 6-36 hours. In HL-60 cells, fluacrypyrim at 0.75-12 μM for 6-36 hours significantly inhibits phosphorylation of STAT3 (tyr705), an effect reversible by sodium pervanadate treatment. Additionally, fluacrypyrim at 0.75-12 μM for 12 hours induces a dose-dependent increase in tyrosine phosphatase activity in HL-60 cells. The compound, at 3-12 μM for 8-24 hours, inhibits STAT3-dependent luciferase activity in IL-6-stimulated HepG-2 cells and c-Src transfected NIH 3T3 cells. Fluacrypyrim at 0.75-12 μM for 24 hours suppresses constitutively activated STAT3, thus blocking cyclin D1 and c-Myc expression in HL-60 cells. It preferentially inhibits the growth of cancer cells with constitutively activated STAT3, showing IC ₅₀ values of 3.8 μM (HL-60 cells), 6.0 μM (K562 cells), 8.2 μM (XG-7 cells), and 12.3 μM (Jurkat-T cells). In breast cancer cells (MDA-MB-231), fluacrypyrim at 6-24 μM for 24 hours induces caspase-dependent apoptosis. Moreover, fluacrypyrim significantly inhibits uterine contractions induced by Dinoprost (PGF ₂ α), Oxytocin, acetylcholine (Ach), and KCL in a dose-dependent manner (pD ₂ ranging from 5.72 to 5.92) when used at 0.62-10 μM for 60 minutes, and also inhibits PGF ₂ α-induced MLC20 phosphorylation at concentrations of 2.5-10 μM.

In vivo	Fluacrypyrim, administered intraperitoneally at 20-75 mg/kg 3-48 hours before irradiation, mitigates radiation-induced hematopoietic system damage in C57BL/6j (CD45 .2) and B6.SJL/BoyJ (CD45 .1) mice. At doses of 50-200 mg/kg, given intraperitoneally one hour before acetic acid injection, it reduces acetic acid-induced writhing responses in mice. Additionally, at 100-200 mg/kg, administered intraperitoneally one hour prior to PGF 2 α injection, fluacrypyrim alleviates inflammatory activity and pain responses in mouse and rat edema models.
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Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	2.3453 mL	11.7264 mL	23.4527 mL
5 mM	0.4691 mL	2.3453 mL	4.6905 mL
10 mM	0.2345 mL	1.1726 mL	2.3453 mL
50 mM	0.0469 mL	0.2345 mL	0.4691 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Inhibitor · Natural Compounds · Compound Libraries · Recombinant Proteins

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