

Product Data Sheet

Product Name: Chlorfenapyr

Cat. No.: GC60106

Chemical Properties

Cas. No. 122453-73-0

SMILES N#CC1=C(C2=CC=C(Cl)C=C2)N(COCC)C(C(F)(F)F)=C1Br

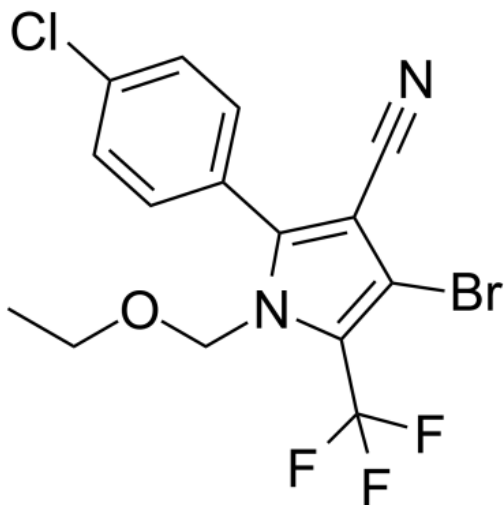
Formula $C_{15}H_{11}BrClF_3N_2O$ M.Wt 407.61

Solubility DMSO: 250 mg/mL (613.33 mM) 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 sizes: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

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Cell lines	A549 cells
Preparation Method	Dulbecco's modified eagle medium (DMEM) supplemented with 1 % antibiotics (streptomycin and penicillin) and 10 % fetal bovine serum was used to prepare the medium. The freshly prepared medium was used to culture A549 cells, which were incubated in the incubator at 37 °C, 5 % CO₂. The cytotoxic effect of Chlorfenapyr on the viability of A549 cells was measured by MTT assay. The treated concentrations of Chlorfenapyr for A549 cells were 0, 25, 37.5, 50, 75 and 100µg/mL for 24h. Chlorfenapyr was dissolved in dimethyl sulfoxide (DMSO, analytical reagent) and diluted 1000 times with the culture medium before use. The color developed was measured at 570nm using a multi-plates spectrophotometer reader. Percent cell viability inhibition was calculated by the following equation: Cell inhibition (%) = (OD_{control}-OD_{treatment})/OD_{control} × 100%.
Reaction Conditions	0, 25, 37.5, 50, 75 and 100µg/mL; 24h
Applications	Chlorfenapyr dose-dependently inhibited the viability of A549 cells.
Animal experiment [2]:	
Animal models	Adult wild-type zebrafish

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Preparation Method

Zebrafish were exposed to 1, 2, 3, 4, 5, 6, 7, 8, 9 and 10ug/L Chlorfenapyr for 21 days. Then the fish were transferred to a tank containing Chlorfenapyr-free standard water for 9 days. The selected exposure concentrations were half and five times of environmental concentrations (2.03ug/L), respectively. The solvent (acetone) control was included. The final content of acetone was <0.01% in control and Chlorfenapyr treatment groups, and the exposure media were renewed every 24h. The exposure external condition (temperature, photoperiod, dissolved oxygen) was the same as the culture environment. For enzyme activity, bioaccumulation and gene transcription analysis, each test concentration was run in triplicate in three glass tanks (30L), with 20L of working solution and 52 zebrafish in each tank. For the metabolomic analysis, there were six replicates per concentration in six 5 L beakers (each beaker contained 4L exposure solution and 10 zebrafish). During the test, the zebrafish were fed twice a day with live brine shrimp (equivalent to 2% of bodyweight), except for 24h before killing. During the experiment, two whole zebrafish were withdrawn from each tank for Chlorfenapyr content analysis on days 1, 2, 3, 5, 7, 10, 14, 21, 23, 27 and 30. The sampled zebrafish were rinsed five times with dechlorinated tap water, blotted dry on absorbent paper, weighed, and then stored at -20 °C until analysis. At day 21 and day 30, the zebrafish for each replicate were divided into four samples as follows: five zebrafish for histopathology analysis, four zebrafish for gene expression analysis, and five zebrafish for metabolomic analysis.

Dosage form

1, 2, 3, 4, 5, 6, 7, 8, 9 and 10ug/L for 21 days; water environment

Applications

High concentrations of Chlorfenapyr led to a significant decrease in the antioxidant enzyme activity in the liver and brain of zebrafish, resulting in oxidative damage to the liver and necrosis of neuronal cells, and significantly disrupting the metabolic profiles of the zebrafish liver and brain.

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References:

- [1] Wang L, Qu Z, Xu Y, et al. Insecticide chlorfenapyr confers induced toxicity in human cells through mitochondria-dependent pathways of apoptosis[J]. *Ecotoxicology and Environmental Safety*, 2025, 289: 117502.
- [2] Chen X, Zheng J, Teng M, et al. Bioaccumulation, metabolism and the toxic effects of chlorfenapyr in zebrafish (*Danio rerio*)[J]. *Journal of Agricultural and Food Chemistry*, 2021, 69(29): 8110-8119.

Background

Chlorfenapyr, a pyrrole insecticide that impairs mitochondrial activity^[1]. Oxidative removal of the N-ethoxymethyl group of Chlorfenapyr by mixed function oxidases results in a toxic form known as CL 303268 that acts to uncouple oxidative phosphorylation in the mitochondria, leading to a disruption of ATP production and loss of energy leading to cell dysfunction and ultimately death of the organism^[2]. Commercial applications of Chlorfenapyr include termite control and protection of crops from multiple insect and mite pests^[3]. Chlorfenapyr has moderate oral toxicity (LD₅₀ = 662mg/kg bw for mouse by mouth) and low transdermal toxicity in mammals (LD₅₀ > 2000mg/kg bw for rabbit by skin)^[4].

In vitro, Chlorfenapyr treatment for 24h can inhibit the viability of human A549 and L02 cells, with IC₅₀ values of 83.14µg/mL for A549 cells and 84.42µg/mL for L02 cells^[5]. Treatment with 10ng/ml Chlorfenapyr for 24 hours induced cell death in HepG2 cells, decreased the levels of SOD1 and GSH, and enhanced oxidative stress^[6]. At a concentration of 4.08µM, Chlorfenapyr treatment for 24 hours led to chromosomal aberrations in CHO_{K1} cells, and significantly increased the frequency of micronuclei^[7].

In vivo, exposure to 10µg/L Chlorfenapyr for 21 days caused zebrafish liver and brain oxidative damage, disturbing the metabolism profiles^[8].

References:

- [1] Yunta C, Ooi J M F, Oladepo F, et al. Chlorfenapyr metabolism by mosquito P450s associated with pyrethroid resistance identifies potential activation markers[J]. *Scientific Reports*, 2023, 13(1):

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[2] Raghavendra K, Barik T K, Sharma P, et al. Chlorfenapyr: a new insecticide with novel mode of action can control pyrethroid resistant malaria vectors[J]. Malaria journal, 2011, 10(1): 16.

[3] Baek B H, Kim S K, Yoon W, et al. Chlorfenapyr-induced toxic leukoencephalopathy with radiologic reversibility: a case report and literature review[J]. Korean journal of radiology, 2016, 17(2): 277-280.

[4] Huang P, Yan X, Yu B, et al. A comprehensive review of the current knowledge of chlorfenapyr: synthesis, mode of action, resistance, and environmental toxicology[J]. Molecules, 2023, 28(22): 7673.

[5] Wang L, Qu Z, Xu Y, et al. Insecticide chlorfenapyr confers induced toxicity in human cells through mitochondria-dependent pathways of apoptosis[J]. Ecotoxicology and Environmental Safety, 2025, 289: 117502.

[6] Elalfy M, Abomosallam M S, Sleem F R, et al. The Cytotoxic Combined Effects of Mixtures of Copper Oxychloride and Chlorfenapyr in HepG2 Cells and Postnatal Model of Toxicity in Female Sprague Dawleyrats and its Pups[J]. International Journal of Zoology and Animal Biology, 2020, 3(3): 1-10.

[7] Al-Sarar A S, Abobakr Y, Bayoumi A E, et al. Cytotoxic and genotoxic effects of abamectin, chlorfenapyr, and imidacloprid on CHOK1 cells[J]. Environmental Science and Pollution Research, 2015, 22(21): 17041-17052.

[8] Chen X, Zheng J, Teng M, et al. Bioaccumulation, metabolism and the toxic effects of chlorfenapyr in zebrafish (Danio rerio)[J]. Journal of Agricultural and Food Chemistry, 2021, 69(29): 8110-8119.

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