Acetaminophen

Cat. No.: HY-66005

CAS No.: 103-90-2

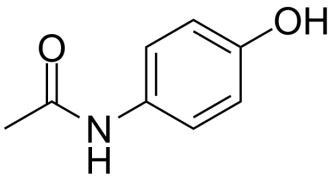
Molecular Formula: $C_8H_9NO_2$

Molecular Weight: 151.16

Target: COX; Endogenous Metabolite; Histone Acetyltransferase; Bacterial; Parasite; Ferroptosis

Pathway: Immunology/Inflammation; Metabolic Enzyme/Protease; Epigenetics; Anti-infection; Apoptosis

Storage: Store at room temperature 3 years
* The compound is unstable in solutions, freshly prepared is recommended.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 250 mg/mL (1653.88 mM; Need ultrasonic)
H_2O : 10 mg/mL (66.16 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	6.6155 mL	33.0775 mL	66.1551 mL
	5 mM	1.3231 mL	6.6155 mL	13.2310 mL
	10 mM	0.6616 mL	3.3078 mL	6.6155 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 50 mg/mL (330.78 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 10 mg/mL (66.16 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 0.5% Tween-80 >> 99.5% Saline
   Solubility: 10 mg/mL (66.16 mM); Clear solution; Need ultrasonic
4. Add each solvent one by one: PBS
   Solubility: 6.67 mg/mL (44.13 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Acetaminophen (Paracetamol) is a selective cyclooxygenase-2 (COX-2) inhibitor with an IC_{50} of 25.8 μM; is a widely used antipyretic and analgesic agent.[1][2][3]. Acetaminophen is a potent hepatic N-acetyltransferase 2 (NAT2) inhibitor[4]. Acetaminophen induces ferroptosis and leads to acute liver injury in mice model[5].

IC_{50} & Target	COX-2 25.8 μM (IC_{50})	COX-1 113.7 μM (IC_{50})

In Vitro

In vitro, acetaminophen elicites a 4.4-fold selectivity toward COX-2 inhibition (IC_{50} 113.7 μM for COX-1; IC_{50} 25.8 μM for COX-2). Following oral administration of the drug, maximal ex vivo inhibitions are 56% (COX-1) and 83% (COX-2). Acetaminophen plasma concentrations remaine above the in vitro IC_{50} for COX-2 for at least 5 h postadministration. Ex vivo IC_{50} values (COX-1: 105.2 μM; COX-2: 26.3 μM) of acetaminophen compared favorably with its in vitro IC_{50} values. In contrast to previous concepts, acetaminophen inhibited COX-2 by more than 80%, i.e., to a degree comparable to nonsteroidal antiinflammatory drugs (NSAIDs) and selective COX-2 inhibitors. However, a >95% COX-1 blockade relevant for suppression of platelet function is not achieved[1]. MTT assay shows that Acetaminophen (APAP) in a dose of 50 mM significantly ($p<0.001$) reduces cell viability to 61.5±6.65%. Interestingly, the significant ($p<0.01$) increase in cell viability to 79.7±2.47% is observed in the Acetaminophen/HV110 co-treated cells, compared to Acetaminophen treated cells[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Administering Acetaminophen (250 mg/kg, orally) to the mice causes significant ($p<0.001$) liver damage and necrosis of cells as evidenced by the elevated serum hepatic enzymes alanine aminotransferase (ALT), aminotransferase (AST), alkaline phosphatase (ALP), and gamma-glutamyl transferase (γGT) compared with normal group. Conversely, effects of pretreatment with different doses of citral (125, 250, and 500 mg/kg) exhibited a significant ($p<0.05$) decrease in serum activities of ALT (91.79%, 93.07%, and 95.61%, resp.), AST (93.40%, 91.89%, and 96.52%, resp.), ALP (39.29%, 37.07%, and 59.80%, resp.), and γGT (92.83%, 91.59%, and 93.0%, resp.), when compared to the Acetaminophen group. Similar results were found in pretreatment with SLM on the activity of ALT (95.90%), AST (95.03%), ALP (70.52%), and γGT (92.69%)[3]. Acetaminophen (300 mg/kg, i.p., single dose) induces ferroptosis, by enhancing levels of Fe^{2+} and malondialdehyde (MDA), and decreasing levels of glutathione (GSH) and glutathione peroxidase 4 (GPX4), and induces acut eliver injury in C57BL/6J mice model[5].

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Animal Model:	Acetaminophen induced liver injury in C57BL/6J mice[5]
Dosage:	300 mg/kg
Administration:	i.p., single dose
Result:	Increased levels of MDA and Fe^{2+}, decreased levels of GSH and GPX4. Destoryed the boundary plate, disordered the arrangement of hepatic cords, caused liver cells edema, tissue necrosis and inflammatory cells infiltration

CUSTOMER VALIDATION

- Cell. 2023 Dec 7;186(25):5500-5516.e21.
- Cell Res. 2025 Jan 29.
- Cell Discov. 2025 Feb 18;11(1):15.
- Nat Commun. 2025 Jan 31;16(1):1233.
- Nat Commun. 2021 Sep 20;12(1):5548.

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REFERENCES

[1]. An Y, et al., Abietic acid inhibits acetaminophen-induced liver injury by alleviating inflammation and ferroptosis through regulating Nrf2/HO-1 axis. Int

Immunopharmacol. 2023 May;118:110029.

[2]. Hinz, B, et al. Acetaminophen (paracetamol) is a selective cyclooxygenase-2 inhibitor in man. FASEB J, 2008. 22(2): p. 383-90.

[3]. Miroslav Dinić, et al. Lactobacillus fermentum Postbiotic-induced Autophagy as Potential Approach for Treatment ofAcetaminophen Hepatotoxicity. Front Microbiol. 2017 Apr 6;8:594.

[4]. Uchida NS, et al. Hepatoprotective Effect of Citral on Acetaminophen-Induced Liver Toxicity in Mice. Evid Based Complement Alternat Med. 2017;2017:1796209.

[5]. Rothen JP, et al. Acetaminophen is an inhibitor of hepatic N-acetyltransferase 2 in vitro and in vivo. Pharmacogenetics. 1998 Dec;8(6):553-9.

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