Data Sheet (Cat.No.T0065)



Acetaminophen

Chemical Properties

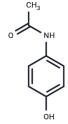
CAS No.: 103-90-2

Formula: C8H9NO2

Molecular Weight: 151.16

Appearance: no data available

Storage: Powder: -20°C for 3 years



Biological Description

Description	Acetaminophen (APAP) is a COX inhibitor that inhibits COX-1 and COX-2 (IC50=113.7/25. $8 \mu M$). Acetaminophen has antipyretic and analgesic activity as well as weak anti-inflammatory activity.			
Targets(IC50)	Endogenous Metabolite,Histone Acetyltransferase,COX			
In vitro	METHODS: Non-melanoma and melanoma cell lines were treated with Acetaminophen (100 μM) for 48 h and cell viability was measured using MTT Assay. RESULTS: Acetaminophen showed considerable toxicity in melanoma cell lines SK-MEL-5, MeWo, B16-F0 and B16-F10, resulting in 40±3%, 45±7%, 66±8% and 60±5% cytotoxicity, respectively. Acetaminophen showed negligible toxicity at 100 μM in the non-melanoma cell lines PC-3, BJ, Saos-2 and SW-620 cells. [1] METHODS: Neuroblastoma cells SH-SY5Y were treated with Acetaminophen (2 mM) for 24-48 h, and the expression levels of target proteins were detected using Western Blot. RESULTS: Acetaminophen induced cytochrome c release from mitochondria in a time-dependent manner, reaching a maximum level after 48 h of treatment. In addition, immunoblot analysis of cytoplasmic and mitochondrial fractions showed that Acetaminophen was able to induce the accumulation of Bax into mitochondria at 24 h after treatment. [2]			
In vivo	METHODS: To detect hepatotoxicity in vivo, Acetaminophen was administered intraperitoneally to mice (300 mg/kg) and rats (1 g/kg). RESULTS: Extensive liver necrosis was observed in mice, but little damage was observed in rat samples. The rats were highly resistant to Acetaminophen-induced liver injury. [3] METHODS: To test for in vivo hepatotoxicity, Acetaminophen was administered to aged and weakened mice acutely (300 mg/kg by gavage), chronically (100 mg/kg by diet once daily for six weeks), or subacutely (250 mg/kg by gavage three times daily for three days). RESULTS: There was no overall increase in Acetaminophen hepatotoxicity with age or frailty in mice, despite changes in certain pathways that would be expected to affect susceptibility to Acetaminophen toxicity. [4]			
Kinase Assay	Effect of inhibition of Acetaminophen on COX-1 and COX-2 activity in human whole blood: For COX-1 assay, aliquots of human whole blood drawn from healthy volunteers without anticoagulant are transferred to glass tubes containing Acetaminophen or DMSO, serum is separated by centrifugation after clotting, and serum TxB2 levels are determined. For COX-2 assay, aliquots of heparinized whole blood are incubated with			

	LPS (10 µg/mL) and aspirin (10 µg/mL), plus Acetaminophen or DMSO for 24 hours at 37 °C, plasma is separated by centrifugation, and PGE2 levels are determined subsequently. The degree of COX-1 or COX-2 inhibition is calculated as the percentage change of plasma eicosanoid (TxB2 for COX-1 and PGE2 for COX-2). Concentration response curves are fitted by a sigmoidal regression with variable slope for both enzymatic assays, and the 50% inhibitory concentration (IC50) values are derived by using of PRISM Version 3.0.
Cell Research	Cells are exposed to Acetaminophen for 48 hours. Cell viability is determined by the trypan blue exclusion method. Intracellular GSH is measured by recording the disulfide, GS-TNB and 5-thio-nitrobenzoic acid (TNB), the yellow colored compound formed by the reaction between GSH with DTNB.(Only for Reference)

Solubility Information

Solubility	DMSO: 60 mg/mL (396.93 mM), Sonication is recommended.
	H2O: 20 mg/mL (132.31 mM), Sonication is recommended. The compound is unstable in
	solution, please use soon.
	Ethanol: 15.1 mg/mL (99.89 mM), Sonication is recommended.
	(< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	6.6155 mL	33.0775 mL	66.1551 mL
5 mM	1.3231 mL	6.6155 mL	13.231 mL
10 mM	0.6616 mL	3.3078 mL	6.6155 mL
50 mM	0.1323 mL	0.6616 mL	1.3231 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.

Reference

Vad NM, et al. Biochemical mechanism of acetaminophen (APAP) induced toxicity in melanoma cell lines. J Pharm Sci. 2009 Apr;98(4):1409-25.

Ma R, Fang L, Chen L, et al. Ferroptotic stress promotes macrophages against intracellular bacteria. Theranostics. 2022, 12(5): 2266

Guo J, Xu Y, Chen L J, et al. Gut Microbiota and Host Cyp450s Co-contribute to Pharmacokinetic Variability in Mice With Non-Alcoholic Steatohepatitis: Vary From Drug to Drug. Journal of Advanced Research. 2021

Posadas I, et al. Acetaminophen induces human neuroblastoma cell death through NFKB activation. PLoS One. 2012;7(11):e50160.

McGill MR, et al. Acetaminophen-induced liver injury in rats and mice: comparison of protein adducts, mitochondrial dysfunction, and oxidative stress in the mechanism of toxicity. Toxicol Appl Pharmacol. 2012 Nov 1; 264(3):387-94.

Kane AE, et al. Acetaminophen hepatotoxicity in mice: Effect of age, frailty and exposure type. Exp Gerontol. 2016 Jan;73:95-106.

Gardner CR, et al. Hepatology, 1998, 27(3), 748-754.

Dini M, et al. Lactobacillus fermentum Postbiotic-induced Autophagy as Potential Approach for Treatment of Acetaminophen Hepatotoxicity. Front Microbiol. 2017 Apr 6;8:594.

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

Page 2 of 3 www.targetmol.com

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