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SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien T. +43(0)1 489 3961-0 F. +43(0)1 489 3961-7 <u>mail@szabo-scandic.com</u> www.szabo-scandic.com

Data Sheet (Cat.No.T0065)

TargetM**Ò**I

Acetaminophen

Chemical Propert	ies	
CAS No. :	103-90-2	сн _з I
Formula:	C8H9NO2	0 NH
Molecular Weight:	151.16	
Appearance:	no data available	
Storage:	Powder: -20°C for 3 years In solvent: -80°C for 1 year	 ОН

Biological Description

Description	Acetaminophen (APAP) is a COX inhibitor that inhibits COX-1 and COX-2 (IC50=113.7/25. 8 μ M). Acetaminophen has antipyretic and analgesic activity as well as weak anti-inflammatory activity.
Targets(IC50)	COX,Endogenous Metabolite,Histone Acetyltransferase
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

subs	sequently. The degree of COX-1 or COX-2 inhibition is calculated as the percentage
char	nge of plasma eicosanoid (TxB2 for COX-1 and PGE2 for COX-2).Concentration
resp	onse curves are fitted by a sigmoidal regression with variable slope for both
enzy	matic assays, and the 50% inhibitory concentration (IC50) values are derived by
usin	g of PRISM Version 3.0.
Cell Research Cells	s are exposed to Acetaminophen for 48 hours. Cell viability is determined by the
trypo	an blue exclusion method. Intracellular GSH is measured by recording the disulfide,
GS-1	INB and 5-thio-nitrobenzoic acid (TNB), the yellow colored compound formed by the
reac	tion between GSH with DTNB.(Only for Reference)

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Solubility	DMSO: 60 mg/mL (396.93 mM),	
	H2O: 20 mg/mL (132.3 mM),Sonication is recommended.	
	Ethanol: 15.1 mg/mL (100 mM),	
	(< 1 mg/ml refers to the product slightly soluble or insoluble)	

Preparing Stock Solutions

1 mM 6.6155 mL 33.	0//5 mL 66	5.1551 mL
5 mM 1.3231 mL 6.6	5155 mL 1	3.231 mL
10 mM 0.6616 mL 3.3	3078 mL 6.	.6155 mL
50 mM 0.1323 mL 0.6	5616 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.

Inhibitor · Natural Compounds · Compound Libraries · Recombinant Proteins

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Tel:781-999-4286 E_mail:info@targetmol.com Address:36 Washington Street,Wellesley Hills,MA 02481