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Produktinformation

- Forschungsprodukte & Biochemikalien
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- Laborgeräte & Service

Weitere Information auf den folgenden Seiten!
See the following pages for more information!

Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

T. +43(0)1 489 3961-0

F. +43(0)1 489 3961-7

mail@szabo-scandic.com

www.szabo-scandic.com

linkedin.com/company/szaboscandic

Chloranil

Cat. No.: HY-Y0278

CAS No.: 118-75-2

Molecular Formula: $C_6Cl_4O_2$

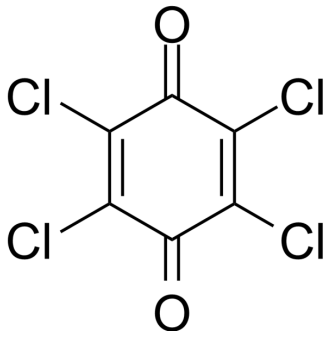
Molecular Weight: 245.88

Target: Toll-like Receptor (TLR); MyD88; Apoptosis; Ferroptosis; JNK; Fungal; Reactive Oxygen Species

Pathway: Immunology/Inflammation; Apoptosis; MAPK/ERK Pathway; Anti-infection; Metabolic Enzyme/Protease; NF-κB

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 5 mg/mL (20.34 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	4.0670 mL	20.3351 mL	40.6702 mL
	5 mM	0.8134 mL	4.0670 mL	8.1340 mL
	10 mM	0.4067 mL	2.0335 mL	4.0670 mL

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

BIOLOGICAL ACTIVITY

Description

Chloranil (Tetrachloro-p-benzoquinone), an orally active metabolite of pentachlorophenol and hexachlorobenzene, is a widely used fungicide. Chloranil can induce ROS production. Chloranil induces neutrophil extracellular traps through the ROS-JNK-NOX2 pathway. Chloranil induces ferroptosis and neuroinflammation. Chloranil induces apoptosis of mouse embryonic stem cells[1][2][3][4][5] .

IC_{50} & Target

TLR4

In Vitro

Chloranil (20 μM; 12-24 h) induces ferroptosis in PC12 cells[1].

Chloranil (0-50 μM; 24 h) induces apoptosis in mouse embryonic stem cells in a dose-dependent manner[2].

Chloranil (10 μM; 1.5-3 h) induces neutrophil extracellular traps by ROS-JNK-NOX2 signaling pathway in mouse neutrophils [3].

Chloranil (25 μM; 6 h) decreases the viability of PC12 cells, increases the expression and interaction of TLR4 and MyD88, and up-regulates the expression and interaction of CD14 and MD2[4].

Chloranil (25 μM; 6 h) stimulates the expression of inflammatory factors and activates MAPK signaling pathway in PC12 cells [4].

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

Western Blot Analysis[3]

Cell Line:	Mouse neutrophil
Concentration:	10 μM
Incubation Time:	1.5 h
Result:	Significantly increased the phosphorylation level of JNK. Increased the level of NOX2.

Western Blot Analysis[4]

Cell Line:	PC12 cells
Concentration:	25 μM
Incubation Time:	6 h
Result:	Enhanced the expression levels of TLR4 and MyD88. Enhanced the expression levels of CD14 and MD2. Enhanced the expression levels of TNF-a, IL-1β and IL-6. Enhanced the expression levels of c-fos, c-jun and AP-1. Increased the phosphorylation levels of p38, JNK and ERK.

In Vivo

Chloranil (10 mg/kg; Oral administration; 2 weeks) induces nerve damage in mice[4].

Chloranil (1 mg/kg; Intraperitoneal injection; 3 days) has a strong hepatotoxic effect in mice, but can be alleviated by chlorogenic acid (HY-N0055)[5].

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

Animal Model:	Male C57BL/6 TLR4 wild-type mice (5-8 weeks old)[4]
Dosage:	10 mg/kg
Administration:	Oral administration (p.o.); 2 weeks
Result:	Caused severe coagulation necrosis, nuclear pyknosis, anachromasis and shrunken neuronal bodies in both cortical and the hippocampus region. Caused cortical damage. Decreased NeuN-positive neurons and increased highly activated microglia in cortex.

Animal Model:	Male Kunming mice (22 ± 2 g)[5]
Dosage:	1 mg/kg
Administration:	Intraperitoneal injection (i.p.); 3 days
Result:	Caused marked liver cell necrosis and inflammation but not apoptosis, and this damage was alleviated by Chlorogenic acid (HY-N0055). Enhanced serum ALT, AST activities, TBIL content, hepatic oxidative stress and lipid peroxidation, decreased GSH content and inhibited the activities of antioxidant enzymes. Up-regulated HO-1 and NQO1 expression.

REFERENCES

[1]. Fu J, et al.The acute exposure of tetrachloro-p-benzoquinone (a.k.a. chloranil) triggers inflammation and neurological dysfunction via Toll-like receptor 4 signaling: The protective role of melatonin preconditioning. Toxicology. 2017 Apr 15;381:39-50.

[2]. Liu Z, et al. Tetrachlorobenzoquinone exposure triggers ferroptosis contributing to its neurotoxicity. Chemosphere. 2021 Feb;264(Pt 1):128413.

[3]. Zuehlke A, et al. Elevated 5-hydroxymethycytosine and cell apoptosis induced by tetrachloro-1,4-benzoquinone in mouse embryonic stem cells. J Environ Sci (China). 2017 Jan;51:1-4.

[4]. Lv X, et al. Tetrachlorobenzoquinone exhibits immunotoxicity by inducing neutrophil extracellular traps through a mechanism involving ROS-JNK-NOX2 positive feedback loop. Environ Pollut. 2021 Jan 1;268(Pt B):115921.

[5]. Xu D, et al. Tetrachlorobenzoquinone induces acute liver injury, up-regulates HO-1 and NQO1 expression in mice model: the protective role of chlorogenic acid. Environ Toxicol Pharmacol. 2014 May;37(3):1212-20.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA