Triclosan

Cat. No.: HY-B1119

CAS No.: 3380-34-5

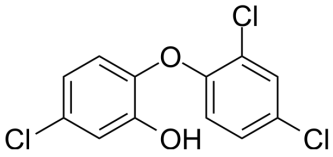
Molecular Formula: $C_{12}H_7Cl_3O_2$

Molecular Weight: 290

Target: Bacterial; Fungal; Antibiotic; Apoptosis

Pathway: Anti-infection; Apoptosis

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (344.83 mM)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 80°C) (insoluble)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.4483 mL	17.2414 mL	34.4828 mL
	5 mM	0.6897 mL	3.4483 mL	6.8966 mL
	10 mM	0.3448 mL	1.7241 mL	3.4483 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.62 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.62 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Triclosan is a broad-spectrum antibacterial agent that inhibits bacterial fatty acid synthesis at the enoyl-acyl carrier protein reductase (FabI) step. Triclosan inhibits E. coli enoyl-acyl carrier protein reductase (FabI) and FabI containing a glycine-to-valine substitution at position 93 (FabIG93V) with IC_{50}s of 2 μM and 10 μM, respectively. Triclosan causes apoptotic effect in cultured rat neural stem cells (NSC). Triclosan exacerbates colitis and colitis-associated colorectal tumorigenesis in animal models[1][2][3].

In Vitro

Triclosan (1-100 μM ; 24 h) decreases in cell viability in dose and time dependent manners with 50 and 100 μM. Triclosan with 50 μM significantly increases cleaved caspase3 and Bax proteins and decreases Bcl-2[2].
Triclosan (50 μM ; 1-3 h) induces the increased expressions of both phosphorylated p38 and JNK proteins[2].

Triclosan (10-50 μM ; 3 h) with 50 μM decreases the GSH activity and increases the ROS generation to about 40% in cultured NSCs[2].

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

Cell Viability Assay[2]

Cell Line:	Neural stem wells
Concentration:	1, 10, 20, 30, 50 and 100 μM
Incubation Time:	24 h
Result:	Initiated the decreases in cell viability in dose and time dependent manners with 50 and 100 μM.

Western Blot Analysis[2]

Cell Line:	Neural stem wells
Concentration:	50 μM
Incubation Time:	1, 3 h
Result:	Did not affect the expressions of MAPK signaling proteins per se. Differentially induced the increased expressions of both phosphorylated p38 and JNK proteins.

In Vivo

Triclosan (5, 50, 500 mg/kg; oral gavage, five days a week for a total of four weeks) causes an increase in the production of anti‐Der f IgE, IL‐4, and IL‐13[3].

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

Animal Model:	Wild type BALB/cJ mice[3]
Dosage:	5, 50, 500 mg/kg
Administration:	Oral gavage, five days a week for a total of four weeks
Result:	Caused an increase in the production of anti- dermatophagoides farinae (Der f) IgE, IL‐4, and IL‐13, and this resulted in the aggravation of airway hyperresponsiveness in aeroallergen‐exposed wild type mice.

CUSTOMER VALIDATION

- Chemosphere. 2019 Jun;225:378-387.
- Anal Chem. 2020 Mar 17;92(6):4419-4426.
- Ecotoxicol Environ Saf. 2023 Mar 1;253:114711.
- Viruses. 2019 Apr 25;11(4):385.
- ACS Omega. 2020 Nov 15;5(46):29935-29942.

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REFERENCES

[1]. Jianan Zhang, et al. Microbial enzymes induce colitis by reactivating triclosan in the mouse gastrointestinal tract. Nat Commun. 2022 Jan 10;13(1):136.

[2]. R J Heath, et al. Mechanism of triclosan inhibition of bacterial fatty acid synthesis. J Biol Chem. 1999 Apr 16;274(16):11110-4.

[3]. Bo Kyung Park, et al. Effects of Triclosan on Neural Stem Cell Viability and Survival. Biomol Ther (Seoul). 2016 Jan;24(1):99-107.

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