

Produktinformation



Forschungsprodukte & Biochemikalien
Zellkultur & Verbrauchsmaterial
Diagnostik & molekulare Diagnostik
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

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4-tert-Octylphenol

Cat. No.:	HY-B1941		
CAS No.:	140-66-9		
Molecular Formula:	C ₁₄ H ₂₂ O		
Molecular Weight:	206.32		
Target:	Endogenou	s Metabol	lite; Apoptosis; DNA/RNA Synthesis
Pathway:	Metabolic E	nzyme/Pi	rotease; Apoptosis; Cell Cycle/DNA Damage
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 : 100 mg/mL (484.68 mM; Need ultrasonic)					
		Mass Solvent Concentration	1 mg	5 mg	10 mg	
	Preparing Stock Solutions	1 mM	4.8468 mL	24.2342 mL	48.4684 mL	
		5 mM	0.9694 mL	4.8468 mL	9.6937 mL	
	10 mM 0.4847 mL	2.4234 mL	4.8468 mL			
	Please refer to the sol	ubility information to select the app	propriate solvent.			
In Vivo	1. Add each solvent o Solubility: ≥ 2.5 m	one by one: 10% DMSO >> 40% PEC g/mL (12.12 mM); Clear solution	G300 >> 5% Tween-8	0 >> 45% saline		
	2. Add each solvent o Solubility: ≥ 2.5 mg	one by one: 10% DMSO >> 90% (20 g/mL (12.12 mM); Clear solution	% SBE-β-CD in saline)			
	3. Add each solvent o Solubility: ≥ 2.5 m	one by one: 10% DMSO >> 90% cor g/mL (12.12 mM); Clear solution	n oil			

BIOLOGICAL ACTIVITY

Description

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4-tert-Octylphenol, a endocrine-disrupting chemical, is an estrogenic agent. 4-tert-Octylphenol is also a biodegradation product of non-ionic surfactants alkylphenol polyethoxylates. 4-tert-Octylphenol induces apoptosis in neuronal progenitor cells in offspring mouse brain. 4-tert-Octylphenol reduces bromodeoxyuridine (BrdU), mitotic marker Ki67, and phosphohistone H3 (p-Histone-H3), resulting in a reduction of neuronal progenitor proliferation. 4-tert-Octylphenol disrupts brain development and behavior in mice, which is promising for reserch of immune response, neuro-related diseases and ethology^{[1][2][3][4]}.

OH



In Vitro	 4-tert-Octylphenol (0.01 and 1 μM, 24 h) may inhibit proliferation and promote apoptosis of neuronal progenitor cells during the early stage of brain development^[1]. 4-tert-Octylphenol (10 μM, 6 h) down-regulates the expression of IL-12p35, IFN-γ2 and CXCb2 in LPS-stimulated monocytes/macrophages and decreases the levels of nitric oxide (NO) released from LPS-stimulated cells^[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only. Immunofluorescence^[1] 			
	Cell Line:	Primary cortical neurons		
	Concentration:	0.01 and 1 μM		
	Incubation Time:	24 h		
	Result:	The numbers of primary and secondary dendrites in the 4-tert-Octylphenol-treated groups were markedly higher than those in the vehicle group in neuron cells.		
	Immunofluorescence ^[3]			
	Cell Line:	LPS-stimulated monocytes/macrophages		
	Concentration:	0.1, 1, 10 μΜ		
	Incubation Time:	6 h		
	Result:	Down-regulated the expression of IL-12p35, IFN-γ2 and CXCb2 in LPS-stimulated monocytes/macrophages and decreased the levels of NO released from LPS-stimulated cells.		
	Cell Proliferation Assay ^[1]			
	Cell Line:	Primary cortical neuronal cells		
	Concentration:	0.01 and 1 μM		
	Incubation Time:	12 h		
	Result:	The percentage of BrdU ⁺ cells in the OP 1 μ M group was significantly lower than that in the OP 0.01 μ M group in primary cortical neuronal cells.		
In Vivo	4-tert-Octylphenol (10, 50 r dysfunction and impairs so 4-tert-Octylphenol (10, 50 r gyrus (DG) neural progenite 4-tert-Octylphenol (2.5 μg/ those in control infected ar MCE has not independently	ng/kg, s.c., a single dose) promotes cell death in offspring mouse brain, induces cognitive ociability and decreases social novelty preference in offspring mice ^[1] . ng/kg, s.c., a single dose for 24 h) promotes cell cycle exit and inhibits cell cycle reentry of dentate ors in embryonic and adult neurogenesis ^[1] . kg, feed, daily for 14 days) lowers numbers of peritoneal leukocytes/phagocytes compared to nimals in peritoneal and head kidney leukocytes of fish ^[3] . y confirmed the accuracy of these methods. They are for reference only.		
	Animal Model:	Pathogen-free C57BL/6J male and female mice (8 weeks old, 25-30 g) $^{[1]}$		
	Dosage:	10, 50 mg/kg		
	Administration:	s.c., a single dose		
	Result:	Elevated apoptosis in offspring mouse brain and impaired cognitive functioning in areas		

IC₅₀ & Target

Human Endogenous Metabolite

	such as spatial and nonspatial learning and memory.
Animal Model:	Sexually immature, young (9–12 months) common carp ^[3]
Dosage:	2.5 μg/kg food
Administration:	feed, daily for 14 days
Result:	Lowed numbers of peritoneal leukocytes/phagocytes in peritoneal and head kidney leukocytes of fish.

REFERENCES

[1]. Olaniyan LWB, et al. Environmental Water Pollution, Endocrine Interference and Ecotoxicity of 4-tert-Octylphenol: A Review[J]. Rev Environ Contam Toxicol. 2020;248:81-109.

[2]. Maciuszek M, et al. 17α-ethinylestradiol and 4-tert-octylphenol concurrently disrupt the immune response of common carp[J]. Fish Shellfish Immunol. 2020 Dec;107(Pt A):238-250.

[3]. Lee J, Zee S, et al. Effects of crosstalk between steroid hormones mediated thyroid hormone in zebrafish exposed to 4-tert-octylphenol: Estrogenic and anti-androgenic effects[J]. Ecotoxicol Environ Saf. 2024 Jun 1;277:116348.

[4]. Dinh Nam Tran, et al. 4-tert-Octylphenol Exposure Disrupts Brain Development and Subsequent Motor, Cognition, Social, and Behavioral Functions. Oxidative Medicine and Cellular Longevity, 2020.

Caution: Product has not been fully validated for medical applications. For research use only.