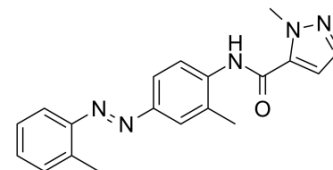


CH-223191

Cat. No.:	HY-12684
CAS No.:	301326-22-7
Molecular Formula:	C ₁₉ H ₁₉ N ₅ O
Molecular Weight:	333.39
Target:	Aryl Hydrocarbon Receptor
Pathway:	Immunology/Inflammation
Storage:	Powder -20°C 3 years 4°C 2 years



* The compound is unstable in solutions, freshly prepared is recommended.

SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 35 mg/mL (104.98 mM)
* "≥" means soluble, but saturation unknown.

Concentration	Solvent	Mass		
		1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM	2.9995 mL	14.9974 mL	29.9949 mL
	5 mM	0.5999 mL	2.9995 mL	5.9990 mL
	10 mM	0.2999 mL	1.4997 mL	2.9995 mL

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
Solubility: ≥ 2.5 mg/mL (7.50 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 2.5 mg/mL (7.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CH-223191 is a potent and specific antagonist of aryl hydrocarbon receptor (AhR).CH-223191 inhibits TCDD-mediated nuclear translocation and DNA binding of AhR, and inhibits TCDD-induced luciferase activity with an IC₅₀ of 0.03 μM^[1].

In Vitro

CH-223191 (0.1-10 μM; pre-treated 1 hour) inhibits TCDD-caused cytochrome P450 1A1 mRNA expression in a in dose-dependent manner^[1].
CH-223191 (0.1-10 μM; pre-treated 1 hour) causes a concentration-dependent inhibition of TCDD-induced cytochrome P450 enzyme activity^[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.
RT-PCR^[1]

	<table border="1"> <tr> <td>Cell Line:</td> <td>HepG2 cells</td> </tr> <tr> <td>Concentration:</td> <td>0.1-10 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>1 hour</td> </tr> <tr> <td>Result:</td> <td>Caused inhibition of TCDD-induced cytochrome P450 mRNA expression.</td> </tr> </table> <p>Western Blot Analysis^[1]</p> <table border="1"> <tr> <td>Cell Line:</td> <td>HepG2 cells</td> </tr> <tr> <td>Concentration:</td> <td>0.1-10 μM</td> </tr> <tr> <td>Incubation Time:</td> <td>1 hour</td> </tr> <tr> <td>Result:</td> <td>Decreased TCDD-caused cytochrome P450 1A1 protein Treatment.</td> </tr> </table>	Cell Line:	HepG2 cells	Concentration:	0.1-10 μ M	Incubation Time:	1 hour	Result:	Caused inhibition of TCDD-induced cytochrome P450 mRNA expression.	Cell Line:	HepG2 cells	Concentration:	0.1-10 μ M	Incubation Time:	1 hour	Result:	Decreased TCDD-caused cytochrome P450 1A1 protein Treatment.
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In Vivo	<p>CH-223191 (10 mg/kg; once a day; 25 days) suppresses cytochrome P450 1A1 expression and the intrahepatocyte fat content in liver, reduces activity of AST and ALT in TCDD-treated mice^[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p> <table border="1"> <tr> <td>Animal Model:</td> <td>Male ICR mice (6 weeks old)^[1]</td> </tr> <tr> <td>Dosage:</td> <td>10 mg/kg</td> </tr> <tr> <td>Administration:</td> <td>10 mg/kg; once a day; 25 days</td> </tr> <tr> <td>Result:</td> <td>Prevented TCDD-elicited cytochrome P450 induction, liver toxicity, and wasting syndrome in mice.</td> </tr> </table>	Animal Model:	Male ICR mice (6 weeks old) ^[1]	Dosage:	10 mg/kg	Administration:	10 mg/kg; once a day; 25 days	Result:	Prevented TCDD-elicited cytochrome P450 induction, liver toxicity, and wasting syndrome in mice.								
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CUSTOMER VALIDATION

- J Hazard Mater. 2020 Mar 5;385:121521.
- Brain Behav Immun. 2020 Aug;88:471-481.
- Sci Total Environ. 2020 Jun 1;719:135097.
- Cell Death Dis. 2019 Jan 15;10(2):38.
- Cancers (Basel). 2020 Oct 10;12(10):2915.

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REFERENCES

- [1]. Zhao B, et al. CH223191 is a ligand-selective antagonist of the Ah (Dioxin) receptor. Toxicol Sci. 2010 Oct;117(2):393-403.
- [2]. Kim SH, et al. Novel compound 2-methyl-2H-pyrazole-3-carboxylic acid (2-methyl-4-o-tolylazo-phenyl)-amide (CH-223191) prevents 2,3,7,8-TCDD-induced toxicity by antagonizing the aryl hydrocarbon receptor. Mol Pharmacol. 2006 Jun;69(6):1871-8.

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

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