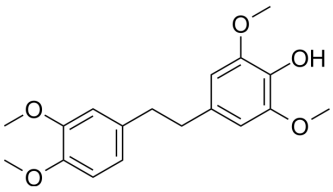
Chrysotoxine

Cat. No.:	HY-123298
CAS No.:	156951-82-5
Molecular Formula:	$C_{18}H_{22}O_5$
Molecular Weight:	318.36
Target:	Src; Akt; Apoptosis
Pathway:	Protein Tyrosine Kinase/RTK; PI3K/Akt/mTOR; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Chrysotoxine is a dual inhibitor of Src/Akt. Chrysotoxine suppresses cancer stem cells (CSCs) phenotypes by down-regulating Src/Akt signaling. Chrysotoxine reduces cell viability and increases apoptosis level in H460 and H23 cells instead of non-tumor cell lines. Chrysotoxine shows rapid excretion and low bioavailability in rats. Chrysotoxine is used in cancer research[1][2].

In Vitro

Chrysotoxine (50 nM, 24 h) reduces cell viability and increases apoptosis level in H460 and H23 cells[1].
Chrysotoxine (5-20 nM, 72 h) suppresses the CSC populations in H460 and H23 cells[1].
Chrysotoxine (0-20 nM, overnight) decreases the stemness of H460 and H23 cells by suppressing the Src-Akt activating mechanism[1].

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

Western Blot Analysis[1]

Cell Line:	460, H23 cells
Concentration:	0-20 μM
Incubation Time:	Overnight
Result:	Significant decreased the p-Src and p-Akt expression in a dose-dependent manner instead of Src and Akt. Significantly reduced the down-stream stem cell transcription factor Sox2 as the decline of p-Src in H460 and H23 cells.

Cell Viability Assay[1]

Cell Line:	460, H23 cells
Concentration:	0, 1, 5, 10, 20 and 50 μM
Incubation Time:	24 h
Result:	Significantly reduced cell viability and increased H460 and H23 cells cell apoptosis at 50 μM with IC_{50}s of 127.34 and 145.47 μM, respectively. Showed no cytotoxic effect on non-tumor cell lines at all tested concentrations.

Cell Differentiation Assay[1]

Cell Line:	460, H23 cells
Concentration:	5-20 μM
Incubation Time:	72 h
Result:	Decreased approximately 30, 60, 90 and 95% of the H460 CSC spheroid size at day 7 with treated 1, 5,10 and 20 μM Chrysotoxine, respectively. Decreased approximately 40, 60, 80 and 92% of the H23 CSC spheroid size at day 7 with treated 1, 5,10 and 20 μM Chrysotoxine, respectively.

In Vivo

Chrysotoxine (25 mg/kg for i.v; 100 mg/kg for p.o;once) rapidly excreted and has low bioavailability in Sprague-Dawley rats model[2]. Pharmacokinetic parameters of Chrysotoxine in Sprague-Dawley rats[2]

Parameters	Intravenous	Oral
AUC_{0-t} (μg h/L)	1257.6 ± 570.7	172.8 ± 118.9
$AUC_{0-\infty}$ (μg h/L)	1270.1 ± 560.6	202.5 ± 123.8
MRT_{0-t} (μg h/L)	0.467 ± 0.056	1.2 ± 0.46
$MRT_{0-\infty}$ (μg h/L)	0.59 ± 0.21	2.4 ± 1.8
$t_{1/2Z}$ (h)	1.4 ± 0.76	1.7 ± 1.1
T_{max} (h)	/	0.098 ± 0.040
CL_Z /F (L/h/kg)	22.9 ± 11.2	668.7 ± 396.9
V_Z /F (L/kg)	55.3 ± 54.1	1443.2 ± 943.0
C_{max} (μg/L)	4961.2 ± 3254.8	408.8 ± 160.5
F (%)	/	3.4 ± 2.4

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REFERENCES

[1]. Bhummaphan N, et al. Cancer Stem Cell-Suppressing Activity of Chrysotoxine, a Bibenzyl from Dendrobium pulchellum. J Pharmacol Exp Ther. 2018 Feb;364(2):332-346.

[2]. Fan J, et al. Determination of chrysotoxine in rat plasma by liquid chromatography-tandem mass spectrometry and its application to a rat pharmacokinetic study. J Chromatogr B Analyt Technol Biomed Life Sci. 2014 Sep 15;967:57-62.

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

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