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SZABO-SCANDIC HandelsgmbH

Quellenstraße 110, A-1100 Wien

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

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

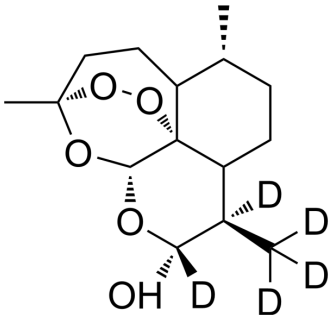
mail@szabo-scandic.com

www.szabo-scandic.com

linkedin.com/company/szaboscandic

Dihydroartemisinin-d_5

Cat. No.:	HY-N0176S3
Molecular Formula:	$C_{15}H_{19}D_5O_5$
Molecular Weight:	289.38
Target:	Apoptosis; Parasite; Autophagy; NF-κB; Isotope-Labeled Compounds
Pathway:	Apoptosis; Anti-infection; Autophagy; NF-κB; Others
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Dihydroartemisinin-d_5 is deuterated labeled Dihydroartemisinin (HY-N0176). Dihydroartemisinin is a potent anti-malaria agent.
In Vitro	Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs[1]. ,Dihydroartemisinin (DHA) is an antimalarial agent. Dihydroartemisinin treatment effectively up-regulates the cytosolic RelA/p65 protein level and down-regulates the nuclear RelA/p65 protein level. Dihydroartemisinin blocks the nuclear translocation of RelA/p65 from the cytosol rather than suppressing RelA/p65 protein synthesis. Dihydroartemisinin induces autophagy in RPMI 8226 cells. Dihydroartemisinin suppresses NF-κB activation in RPMI 8226 cells. The NF-κB Dihydroartemisinin -binding activity is examined by EMSA assay. RPMI 8226 cells are exposed to various concentrations of Dihydroartemisinin (10, 20 and 40 μM) for 12 h, and TNF-α is introduced as a positive control for NF-κB activation. Dihydroartemisinin suppresses NF-κB activation in a dose-dependent manner in contrast with TNF-α[2]. Dihydroartemisinin (DHA) can enhance the anti-tumor effect of photodynamic therapy (PDT) on esophageal cancer cells, and cell viability is investigated using the MTT assay. Eca109 and Ec9706 cells are treated with Dihydroartemisinin (80 μM), PDT (25 and 20 J/cm ^{2}, respectively), or their combination. A single treatment with Dihydroartemisinin or PDT causes a 37±5% or 34±6% reduction in viability in Eca109 cells and a 33±7% or 34±6% reduction in Ec9706 cells, respectively. However, when PDT is combined with Dihydroartemisinin, the cell viability is reduced by 59±6% or 61±7% in the cell lines, respectively[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	,Single oral doses of Dihydroartemisinin (at 200, 300, 400 or 600 mg/kg), given once on each of day 6-8 post-infection, reduce total-worm burdens by 69.2%-90.6% and female-worm burdens by 62.2%-92.2%, depending on dosage in the first experiment. Similar treatments given on day 34-36 post-infection reduce total-worm burdens by 73.9%-85.5% and female-worm burdens by 83.8%-95.3%[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Hu W, et al. Dihydroartemisinin induces autophagy by suppressing NF-κB activation. Cancer Lett. 2014 Feb 28;343(2):239-48.

[2]. Li HJ, et al. Dihydroartemisinin-praziquantel combinations and multiple doses of dihydroartemisinin in the treatment of Schistosoma japonicum in experimentally infected mice. Ann Trop Med Parasitol. 2011 Jun;105(4):329-33.

[3]. Li YJ, et al. Dihydroartemisinin accentuates the anti-tumor effects of photodynamic therapy via inactivation of NF-κB in Eca109 and Ec9706 esophageal cancer cells. Cell Physiol Biochem. 2014;33(5):1527-36.

[4]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019 Feb;53(2):211-216.

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