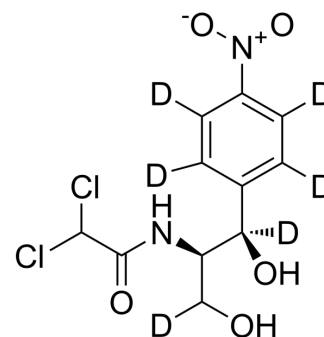


## Threo-Chloramphenicol-d<sub>6</sub>

<b>Cat. No.:</b>	HY-B0239S2
<b>Molecular Formula:</b>	C <sub>11</sub> H <sub>6</sub> D <sub>6</sub> Cl <sub>2</sub> N <sub>2</sub> O <sub>5</sub>
<b>Molecular Weight:</b>	329.17
<b>Target:</b>	JNK; Apoptosis; VEGFR; MMP; Bacterial; Akt; Autophagy; HIF/HIF Prolyl-Hydroxylase; Antibiotic; Beclin1
<b>Pathway:</b>	MAPK/ERK Pathway; Apoptosis; Protein Tyrosine Kinase/RTK; Metabolic Enzyme/Protease; Anti-infection; PI3K/Akt/mTOR; Autophagy
<b>Storage:</b>	Please store the product under the recommended conditions in the Certificate of Analysis.



### BIOLOGICAL ACTIVITY

<b>Description</b>	Threo-Chloramphenicol-d <sub>6</sub> is the deuterium labeled Chloramphenicol[1]. Chloramphenicol is an orally active, potent and broad-spectrum antibiotic. Chloramphenicol shows antibacterial activity. Chloramphenicol represses the oxygen-labile transcription factor and hypoxia inducible factor-1 alpha (HIF-1 $\alpha$ ) in hypoxic A549 and H1299 cells. Chloramphenicol suppresses the mRNA levels of vascular endothelial growth factor (VEGF) and glucose transporter 1, eventually decreasing VEGF release. Chloramphenicol can be used for anaerobic infections and lung cancer research[2][3][4].
<b>In Vitro</b>	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 <sup>[1]</sup> . MCE has not independently confirmed the accuracy of these methods. They are for reference only.

### REFERENCES

- [1]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. *Ann Pharmacother.* 2019 Feb;53(2):211-216.
- [2]. Hsu HL, et al. Chloramphenicol Induces Autophagy and Inhibits the Hypoxia Inducible Factor-1 Alpha Pathway in Non-Small Cell Lung Cancer Cells. *Int J Mol Sci.* 2019 Jan 3;20(1):157.
- [3]. Yuan ZR, et al. Chloramphenicol induces abnormal differentiation and inhibits apoptosis in activated T cells. *Cancer Res.* 2008 Jun 15;68(12):4875-81.
- [4]. Li CH, et al. Chloramphenicol causes mitochondrial stress, decreases ATP biosynthesis, induces matrix metalloproteinase-13 expression, and solid-tumor cell invasion. *Toxicol Sci.* 2010 Jul;116(1):140-50.
- [5]. Turton JA, et al. Characterization of the myelotoxicity of chloramphenicol succinate in the B6C3F1 mouse. *Int J Exp Pathol.* 2006 Apr;87(2):101-12.
- [6]. Bartlett JG. Chloramphenicol. *Med Clin North Am.* 1982;66(1):91-102.

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

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