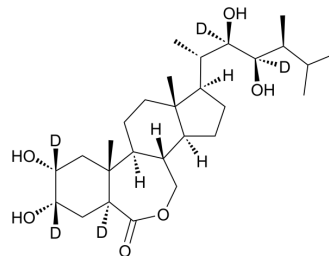


Brassinolide-d₅

Cat. No.:	HY-N0273S
Molecular Formula:	C ₂₈ H ₄₃ D ₅ O ₆
Molecular Weight:	485.71
Target:	Isotope-Labeled Compounds; Apoptosis
Pathway:	Others; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Brassinolide-d ₅ (Brassin lactone-d ₅) is the deuterated labeled Brassinolide (HY-N0273). Citric acid is a natural preservative and food tartness enhancer. Citric acid induces apoptosis and cell cycle arrest at G2/M phase and S phase in HaCaT cells. Citric acid cause oxidative damage of the liver by means of the decrease of antioxidative enzyme activities. Citric acid causes renal toxicity in mice ^{[1][2][3]} .
IC₅₀ & Target	plant growth modulator ^[1]
In Vitro	<p>Brassinolide is a plant sterol first isolated from pollen of rape (<i>Brassica napus</i> L.). Brassinolide can induce a time and concentration-dependent cytotoxicity in PC-3 cells. The mode of cell death appears to be predominately apoptosis, as shown by flow-cytometric analysis, fluorescence and transmission electron microscopes. Caspase-3 activity is obviously increased after Brassinolide treatment. Western blot studies indicate that treatment with Brassinolide triggered a time-dependent decrease in the expression of anti-apoptotic protein Bcl-2, which suggests that Brassinolide can induce cytotoxicity in PC-3 cells by triggering apoptosis. Brassinolide might therefore be a promising candidate for the treatment of prostate cancer^[1]. Brassinolide is a plant growth modulator, on multidrug resistance (MDR) of human T lymphoblastoid cell line CCRF-VCR 1000 which is obtained by progressively addition of vincristine (VCR) to sensitive CCRF-CEM cells, and to explore preliminarily the mechanism of reversing action. After treatment of Brassinolide under the concentration of 0.001-10 µg/mL, the resistance of CCRF-VCR is reversed partly with the reversing folds respectively as 4.4-11.6. The intracellular accumulation of rhodamine 123 is significantly reduced in the resistant cells. After treatment of Brassinolide, the accumulation increased, the level of fluorescent dye is situated between resistant cells and sensitive cells. No alteration of the catalytic activity of topoisomerase II is found among three groups. The level of protein expression of p53 in resistant cells is higher than that of sensitive cells. After Brassinolide treatment, the expression of p53 in CCRF-VCR cells restored to the level of sensitive cells. Brassinolide can effectively reverse the resistance of CCRF-VCR cells by inhibiting the effusion of drug transported by P-glycoprotein. To down regulate the abnormal expression of p53 maybe one of the mechanisms of reversing MDR for Brassinolide^[2].</p> <p>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^[3].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

REFERENCES

[1]. Xian LJ, et al. Reversing effect of brassinolide on multidrug resistance of CCRF-VCR1000 cells and a preliminary investigation on its mechanisms. Yao Xue Xue Bao. 2005

Feb;40(2):117-21.

[2]. Wu YD, et al. Brassinolide, a plant sterol from pollen of Brassica napus L., induces apoptosis in human prostate cancer PC-3 cells. Pharmazie. 2007 May;62(5):392-5.

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