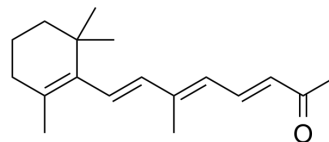


## β-Apo-13-carotenone

Cat. No.:	HY-101953
CAS No.:	17974-57-1
Molecular Formula:	C <sub>18</sub> H <sub>26</sub> O
Molecular Weight:	258.4
Target:	RAR/RXR
Pathway:	Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor
Storage:	-80°C, protect from light



### SOLVENT & SOLUBILITY

In Vitro	DMSO : 110 mg/mL (425.70 mM; Need ultrasonic)				
		Solvent Concentration	Mass		
	Preparing Stock Solutions		1 mg	5 mg	10 mg
		1 mM	3.8700 mL	19.3498 mL	38.6997 mL
		5 mM	0.7740 mL	3.8700 mL	7.7399 mL
	10 mM	0.3870 mL	1.9350 mL	3.8700 mL	
Please refer to the solubility information to select the appropriate solvent.					
In Vivo	1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: 2.75 mg/mL (10.64 mM); Suspended solution; Need ultrasonic				
	2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: ≥ 2.5 mg/mL (9.67 mM); Clear solution				
	3. Add each solvent one by one: 10% DMSO >> 90% corn oil Solubility: ≥ 2.5 mg/mL (9.67 mM); Clear solution				

### BIOLOGICAL ACTIVITY

Description	β-Apo-13-carotenone (D'Orenone) is a naturally occurring β-apocarotenoid functioned as an antagonist of RXRα.
In Vitro	β-apo-13-Carotenone is identified as enzymatic cleavage products of β-carotene in homogenates of intestinal mucosa of rat. β-apo-13-carotenone is found to antagonize the activation of RXRα by 9-cis-retinoic acid and is effective at concentrations as low as 1nM. Molecular modeling studies reveal that β-apo-13-carotenone makes molecular interactions like an antagonist of RXRα <sup>[1]</sup> . β-apo-13-carotenone competes for 9cRA binding to RXRα with an affinity (7–8 nM) identical to 9cRA itself. β-apo-13-carotenone antagonizes 9cRA activation of full-length hRXRα with a similar efficiency as the known antagonist UVI3003. β-apo-13-carotenone induces formation of the RXRα transcriptionally silent tetramer but does not inhibit coactivator recruitment to the isolated LBD <sup>[2]</sup> . The uptake and/or metabolism of β-apo-13-carotenone does not allow for accumulation

of these  $\beta$ -carotene metabolites in cells. 3T3-L1 adipocyte marker gene expression is induced by  $\beta$ -apo-carotenoid treatment<sup>[3]</sup>.

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

## PROTOCOL

### Kinase Assay <sup>[1]</sup>

Cos-1 cells are transfected in serum-free medium with three plasmids mixed in the following amounts per well, 0.05  $\mu$ g of pRL-TK, 2  $\mu$ g of pRXRE-luciferase, 2.5  $\mu$ g of pSG5-RXR $\alpha$  in triplicates. Following transfection, the plates are incubated at 37°C in 5% CO<sub>2</sub> for 4 h. The medium is then changed to complete DMEM. Charcoal stripped FBS has been treated with activated carbon to adsorb lipophilic compounds including retinoids. Twenty hours after transfection, cells are treated with test compounds ( $\beta$ -Apo-13-carotenone) that are dissolved in ethanol or 0.1% ethanol alone for an additional 24 h. Cells are washed once with PBS and lysed by incubation with 500  $\mu$ L passive lysis buffer for 15 min at room temperature. A 20- $\mu$ L aliquot of cell lysate is then assayed for luciferase activities using a kit<sup>[1]</sup>.

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

## REFERENCES

- [1]. Eroglu A, et al. The eccentric cleavage product of  $\beta$ -carotene,  $\beta$ -apo-13-carotenone, functions as an antagonist of RXR $\alpha$ . Arch Biochem Biophys. 2010 Dec 1;504(1):11-6.
- [2]. Sun J, et al.  $\beta$ -Apo-13-carotenone regulates retinoid X receptor transcriptional activity through tetramerization of the receptor. J Biol Chem. 2014 Nov 28;289(48):33118-24.
- [3]. Wang CX, et al. Actions of  $\beta$ -apo-carotenoids in differentiating cells: differential effects in P19 cells and 3T3-L1 adipocytes. Arch Biochem Biophys. 2015 Apr 15;572:2-10.

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

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