Dimethylcurcumin

Cat. No.: HY-15194

CAS No.: 52328-98-0

Molecular Formula: $C_{23}H_{24}O_6$

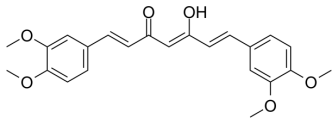
Molecular Weight: 396.43

Target: Androgen Receptor

Pathway: Vitamin D Related/Nuclear Receptor

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (126.13 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.5225 mL	12.6126 mL	25.2251 mL
5 mM	0.5045 mL	2.5225 mL	5.0450 mL
10 mM	0.2523 mL	1.2613 mL	2.5225 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.17 mg/mL (5.47 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Dimethylcurcumin (ASC-J9) is an androgen receptor degradation enhancer that effectively suppresses castration resistant prostate cancer cell proliferation and invasion.

In Vitro

Dimethylcurcumin (ASC-J9) is able to degrade fAR and AR3 in a dose-dependent manner in various human PCa cells. Dimethylcurcumin (ASC-J9) can also effectively suppress AR-targeted genes in CWR22Rv1-fARKD cells. Dimethylcurcumin (ASC-J9) (5 or 10 μM) significantly suppresses the DHT-induced cell growth in all three PCa cell lines. Dimethylcurcumin (ASC-J9) suppresses AR-targeted genes and cell growth by degradation of fAR and ectopic AR3 in C81 and C4-2 cells[1]. Dimethylcurcumin (ASC-J9) selectively promotes AR degradation by disrupting the interaction between AR and AR coregulators. ASC-J9 reduces the AR aggregated AR-112Q in cells. Dimethylcurcumin (ASC-J9) suppresses the aggregation of AR-112Q in SBMA PC12/AR-112Q cells[2].

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

In Vivo	Dimethylcurcumin (ASC-J9) (75 mg/kg, i.p.) degrades both fAR and AR3 in the xenografted tumors in vivo, and SC-J9-treated tumors has significantly decreased Ki67-positive cells[1]. Dimethylcurcumin (ASC-J9) (50 mg/kg every 48 h, i.p.) substantially ameliorates the SBMA symptoms in AR-97Q mice, and ameliorates neuromuscular pathological findings. The Dimethylcurcumin (ASC-J9)-treated SBMA mice have relatively normal serum testosterone concentrations[2]. ASC-J9-treated mice show significantly smaller prostate tumor sizes when compared with those receiving classic ADT/castration with little serum androgen[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	For the cell survival assay, the PC12/AR-112Q and PC12/AR-10Q cells are cultured as described previously and incubated cells in the presence of 10 μg/mL doxycycline for 24 h. Then the cells are treated with vehicle, 5 μM Dimethylcurcumin (ASC-J9) or 10 μM Dimethylcurcumin (ASC-J9), along with 1 nM DHT, and determined cell viability using Trypan blue staining at specific time intervals. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	CWR22Rv1 cells (1×10^6 cells per site) are injected into both anterior prostates of castrated nude mouse after 2 weeks of implantation. The mice are randomLy divided into two groups (four mice/eight tumors each group) and either receives 75 mg/kg Dimethylcurcumin (ASC-J9) intraperitoneal injection or vehicle control every other day. After 4 weeks of treatment, all mice are killed to examine the tumor growth. Body weights and mice activity are measured weekly. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Clin Transl Med. 2022 Apr;12(4):e797.
- Biol Sex Differ. 2020 Mar 29;11(1):12.
- Biochem Pharmacol. 2017 Sep 15;140:73-88.
- Mol Cancer Ther. 2016 Jul;15(7):1702-12.
- Lab Invest. 2023 Apr 12;100148.

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REFERENCES

[1]. Yamashita S, et al. ASC-J9 suppresses castration-resistant prostate cancer growth through degradation of full-length and splice variant androgen receptors. Neoplasia. 2012 Jan;14(1):74-83.

[2]. Yang Z, et al. ASC-J9 ameliorates spinal and bulbar muscular atrophy phenotype via degradation of androgen receptor. Nat Med. 2007 Mar;13(3):348-53.

[3]. Lee SO, et al. New therapy targeting differential androgen receptor signaling in prostate cancer stem/progenitor vs non-stem/progenitor cells. J Mol Cell Biol. 2012 Jul 24.

[4]. Ma W, et al. Targeting androgen receptor with ASC-J9 attenuates cardiac injury and dysfunction in experimental autoimmune myocarditis by reducing M1-like macrophage. Biochem Biophys Res Commun. 2017 Apr 15;485(4):746-752. doi: 10.1016/j.bbrc.2017.02.123.

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

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