CDDO-Im

Cat. No.: HY-15725

CAS No.: 443104-02-7

Molecular Formula: $C_{34}H_{43}N_3O_3$

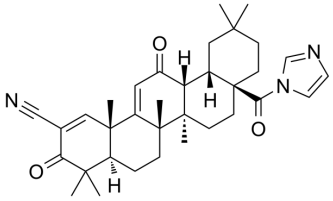
Molecular Weight: 541.72

Target: Keap1-Nrf2; PPAR; Ferroptosis

Pathway: NF-κB; Cell Cycle/DNA Damage; Vitamin D Related/Nuclear Receptor; Apoptosis

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 : 25 mg/mL (46.15 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.8460 mL	9.2299 mL	18.4597 mL
	5 mM	0.3692 mL	1.8460 mL	3.6919 mL
	10 mM	0.1846 mL	0.9230 mL	1.8460 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: 2.5 mg/mL (4.61 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (4.61 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.61 mM); Clear solution
4. Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
   Solubility: 2.5 mg/mL (4.61 mM); Suspended solution; Need ultrasonic
5. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (4.61 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description CDDO-Im (RTA-403) is an activator of Nrf2 and PPAR, with K_is of 232 and 344 nM for PPARα and PPARγ[1][2].

IC_{50} & Target	PPARα	PPARγ	Nrf2

	232 nM (Ki)	344 nM (Ki)	

In Vitro

CDDO-Im is highly active in suppressing cellular proliferation of human leukemia and breast cancer cell lines (IC_{50} approximately 10-30 nM). In U937 leukemia cells, CDDO-Im also induces monocytic differentiation as measured by increased cell surface expression of CD11b and CD36[1]. Treatment with CDDO-Im elevates protein levels of Nrf2, a transcription factor previously shown to bind ARE sequences, and increases expression of a number of antioxidant and detoxification genes regulated by Nrf2[2]. CDDO-Im is one of the most potent synthetic triterpenoids shown to induce growth inhibition and apoptosis in various human cancer cells, including multiple myeloma, lung, pancreas and breast cancer. CDDO-Im treatment markedly induces cell cycle arrest at G2/M-phase and apoptosis in the triple-negative breast cancer cell lines, SUM159 and MDA-MB-231. The CD24−/EpCAM+ cells in SUM159 tumorspheres are significantly inhibited by CDDO-Im treatment. CDDO-Im also significantly decreases sphere forming efficiency and tumorsphere size in both primary and secondary sphere cultures[3].

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

In Vivo

CDDO-Im is a potent inhibitor of de novo inducible nitric oxide synthase expression in primary mouse macrophages. Moreover, CDDO-Im inhibits growth of B16 murine melanoma and L1210 murine leukemia cells in vivo. Injection of 10 nM (5.4 μg) of CDDO-Im almost completely blocks the ability of IFN-γ to induce iNOS, and treatment with as little as 1 nmol of CDDO-Im (0.54 μg) is partially effective[1].

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

PROTOCOL

Cell Assay [3]

CDDO-Im is dissolved in DMSO. SUM159 and MDA-MB-231 cells are seeded into each well of 96-well plates (1,000 cell/well) and treated the next day with vehicle control or CDDO-Im (1, 10, 50, 100 and 200 nM) for given incubation time. The absorbance is measured with a spectrophotometer to determine cell proliferation rate[3].

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Animal Administration [1]

Mice: Mice are injected i.p. with thioglycollate, and the resulting resident peritoneal macrophages are activated 3 days later with an i.p. injection of IFN-γ. CDDO and CDDO-Im are injected i.p. 30 min after IFN-γ. Macrophages are harvested 10 h later, cultured for 12 h, and then assayed for expression of iNOS protein and production of nitric oxide (NO)[1].

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

CUSTOMER VALIDATION

- Cell Death Differ. 2022 Nov 3.
- Environ Pollut. 2019 Jan 3;247:293-301.
- Free Radic Biol Med. 2022.
- Eur J Med Chem. 2021 Feb 15;212:113030.
- Biogerontology. 2021;67(1):91-100.

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REFERENCES

[1]. Place AE, et al. The novel synthetic triterpenoid, CDDO-imidazolide, inhibits inflammatory response and tumor growth in vivo. Clin Cancer Res. 2003 Jul;9(7):2798-806.

[2]. Liby K, et al. The synthetic triterpenoids, CDDO and CDDO-imidazolide, are potent inducers of heme oxygenase-1 and Nrf2/ARE signaling. Cancer Res. 2005 Jun 1;65(11):4789-98.

[3]. So JY, et al. A synthetic triterpenoid CDDO-Im inhibits tumorsphere formation by regulating stem cell signaling pathways in triple-negative breast cancer. PLoS One. 2014 Sep 17;9(9):e107616.

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