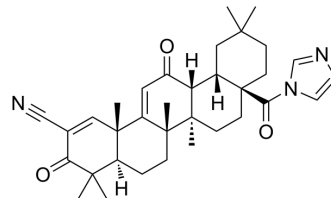


## CDDO-Im

<b>Cat. No.:</b>	HY-15725												
<b>CAS No.:</b>	443104-02-7												
<b>Molecular Formula:</b>	C <sub>34</sub> H <sub>43</sub> N <sub>3</sub> O <sub>3</sub>												
<b>Molecular Weight:</b>	541.72												
<b>Target:</b>	Keap1-Nrf2; PPAR; Ferroptosis												
<b>Pathway:</b>	NF-κB; Cell Cycle/DNA Damage; Metabolic Enzyme/Protease; Vitamin D Related/Nuclear Receptor; Apoptosis												
<b>Storage:</b>	<table border="0"> <tr> <td>Powder</td> <td>-20°C</td> <td>3 years</td> </tr> <tr> <td></td> <td>4°C</td> <td>2 years</td> </tr> <tr> <td>In solvent</td> <td>-80°C</td> <td>2 years</td> </tr> <tr> <td></td> <td>-20°C</td> <td>1 year</td> </tr> </table>	Powder	-20°C	3 years		4°C	2 years	In solvent	-80°C	2 years		-20°C	1 year
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 Concentration	Mass		
		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

- 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
- 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
- Add each solvent one by one: 10% DMSO >> 90% corn oil  
Solubility: ≥ 2.5 mg/mL (4.61 mM); Clear solution
- 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
- 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<sub>i</sub>s of 232 and 344 nM for PPARα and PPARγ<sup>[1][2]</sup>.

IC <sub>50</sub> & Target	PPAR $\alpha$ 232 nM (Ki)	PPAR $\gamma$ 344 nM (Ki)	Nrf2
<b>In Vitro</b>	<p>CDDO-Im is highly active in suppressing cellular proliferation of human leukemia and breast cancer cell lines (IC<sub>50</sub> 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<sup>[1]</sup>. 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<sup>[2]</sup>. 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<sup>[3]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>		
<b>In Vivo</b>	<p>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 <math>\mu</math>g) of CDDO-Im almost completely blocks the ability of IFN-<math>\gamma</math> to induce iNOS, and treatment with as little as 1 nmol of CDDO-Im (0.54 <math>\mu</math>g) is partially effective<sup>[1]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>		

## PROTOCOL

<b>Cell Assay</b> <sup>[3]</sup>	<p>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<sup>[3]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
<b>Animal Administration</b> <sup>[1]</sup>	<p>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-<math>\gamma</math>. CDDO and CDDO-Im are injected i.p. 30 min after IFN-<math>\gamma</math>. Macrophages are harvested 10 h later, cultured for 12 h, and then assayed for expression of iNOS protein and production of nitric oxide (NO)<sup>[1]</sup>.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

## 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.
- Exp Neurol. 2024 May 30:114822.

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## REFERENCES

- [1]. Place AE, et al. The novel synthetic triterpenoid, CDDO-imidazolidine, 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-imidazolidine, are potent inducers of heme oxygenase-1 and Nrf2/ARE signaling. Cancer Res. 2005 Jun

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

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