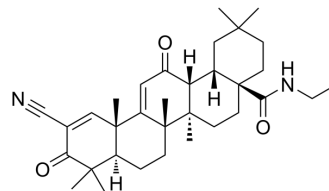


CDDO-EA

Cat. No.:	HY-12213		
CAS No.:	932730-51-3		
Molecular Formula:	C ₃₃ H ₄₆ N ₂ O ₃		
Molecular Weight:	518.73		
Target:	Keap1-Nrf2		
Pathway:	NF-κB		
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 : 16.67 mg/mL (32.14 mM; ultrasonic and warming and heat to 80°C)

Concentration	Solvent	Mass	Preparing Stock Solutions		
			1 mg	5 mg	10 mg
1 mM			1.9278 mL	9.6389 mL	19.2779 mL
5 mM			0.3856 mL	1.9278 mL	3.8556 mL
10 mM			0.1928 mL	0.9639 mL	1.9278 mL

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

In Vivo

- Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
Solubility: ≥ 1.67 mg/mL (3.22 mM); Clear solution
- Add each solvent one by one: 10% DMSO >> 90% corn oil
Solubility: ≥ 1.67 mg/mL (3.22 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CDDO-EA is an NF-E2 related factor 2/antioxidant response element (Nrf2/ARE) activator.

IC₅₀ & Target

Nrf2/ARE^[1]

In Vitro

CDDO-EA potently activates Nrf2/ARE in a cell culture model of ALS and in the G93A SOD1 mouse model of ALS^[1]. CDDO-EA is a potent inducer of apoptosis in A549 lung cancer cells, as shown both by PARP cleavage and Annexin staining. CDDO-EA is more potent than CDDO itself as inducers of heme oxygenase-1 (HO-1). In RAW264.7 macrophage-like cells, CDDO-EA is 7-fold more potent than CDDO as suppressors of the ability of IFN-γ to induce iNOS^[2].

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

In Vivo	<p>The survival analysis shows that G93A mice treated with CDDO-EA, compared to G93A littermate controls, lives significantly longer. CDDO-EA treatment increases the life-span by 20.6 days from 124.05±3.7 days to 144.72±8.1 days (16.6%) (p<0.001). In CDDO-EA-treated G93A mice, the age of death is 141.4±5.2 days and the duration from the age of onset to the age of death is 57.6±7.6 days, which means that the age of death from onset is prolonged by 17.5 days (43%)^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
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PROTOCOL

Cell Assay ^[1]	<p>Wild-type and Nrf2^{-/-} mouse embryonic fibroblasts are pre-treated with CDDO-EA or CDDO-TFEA at various concentrations (1, 10 and 100 nM in DMSO) for 18 hours and incubated with 2',7'-Dichlorodihydrofluorecein diacetate (H2DCFDA) for 30 min. Cells are challenged with 250 µM tBHP for 15-30 min and the mean fluorescence intensity for ~10,000 cells is analyzed by FACS flow cytometry using a 480-nm excitation wavelength and a 525-nm emission wavelength^[1].</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>
Animal Administration ^[1]	<p>Mice^[1]</p> <p>G93A SOD1 transgenic familial ALS mice (high copy number) B6SJL background strain (G93A SOD1, B6SJL-TgGur1) are used. G93A transgenic mice are assigned randomly to the control (vehicle, mouse chaw only) and to mouse chaw containing either CDDO-EA or CDDO-TFEA (400 mg/kg of food, n=30 in both groups). This dose corresponds to about 80 mg/kg body weight/day, assuming each mouse consumes 5 grams of food per day. We found mice can tolerate this dose. Treatments started at two different time regimens: 1) "Early" at 30 days of age, about two months prior to symptom onset; 2) "At Onset" from the onset of the phenotype (80-90 days of age). A diet consisting of either 400 mg of CDDO-TFEA per kg of food or 400 mg of CDDO-EA per kg of food, and a control lab diet, are prepared by Purina.</p> <p>MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

CUSTOMER VALIDATION

- CNS Neurosci Ther. 2021 Jan;27(1):82-91.
- J Cell Mol Med. 2019 Sep;23(9):6034-6047.

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REFERENCES

[1]. Neymotin A, et al. Neuroprotective effect of Nrf2/ARE activators, CDDO ethylamide and CDDO trifluoroethylamide, in a mouse model of amyotrophic lateral sclerosis. Free Radic Biol Med. 2011 Jul 1;51(1):88-96.

[2]. Liby K, et al. The synthetic triterpenoids CDDO-methyl ester and CDDO-ethyl amide prevent lung cancer induced by vinyl carbamate in A/J mice. Cancer Res. 2007 Mar 15;67(6):2414-9.

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

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