

Product Datasheet

CDDO-EA

Catalog No. **BP-02107** · CAS **932730-51-3** · Form **free base** · Physical state **other** · Color **white to off-white** · Version **1.0** · Revision **2026-09-14**

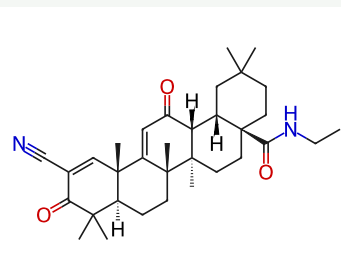
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Target Keap1-Nrf2	Research area Cancer
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Molecular formula **C₃₃H₄₆N₂O₃**

Molecular weight **518.7299**

STRUCTURE



DESCRIPTION

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

BIOLOGICAL ACTIVITY

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.

In Vivo

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%).

SOLUBILITY

Soluble in DMSO

In vitro DMSO : 16.67 mg/mL

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.

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Lot CoA and SDS are separate documents.