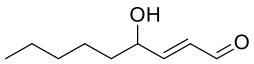
Product data sheet

MedKoo Cat#: 556019 Name: 4-hydroxynonenal CAS#: 75899-68-2 Chemical Formula: $C_9H_{16}O_2$ Exact Mass: 156.115 Molecular Weight: 156.23	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:

4-hydroxynonenal is a lipid peroxidation product derived from oxidized ω-6 polyunsaturated fatty acids. 4-hydroxy Nonenal is widely used as a marker of lipid peroxidation. 4-Hydroxynonenal Contributes to Fibroblast Senescence in Skin Photoaging Evoked by UV-A Radiation. 4-Hydroxynonenal induces Cx46 hemichannel inhibition through its carbonylation.

2. CoA, QC data, SDS, and handling instruction

SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	>50	320.04
DMF	>50	320.04
Ethanol	>50	320.04
PBS pH 7.2	>1	6.4

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	6.40 mL	32.01 mL	64.01 mL
5 mM	1.28 mL	6.40 mL	12.80 mL
10 mM	0.64 mL	3.20 mL	6.40 mL
50 mM	0.13 mL	0.64 mL	1.28 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator

Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study

In vitro study

Hu Z, Zhang H, Yi B, Yang S, Liu J, Hu J, Wang J, Cao K, Zhang W. VDR activation attenuate cisplatin induced AKI by inhibiting ferroptosis. Cell Death Dis. 2020 Jan 29;11(1):73. doi: 10.1038/s41419-020-2256-z. PMID: 31996668; PMCID: PMC6989512.

In vivo study

Lou B, Boger M, Bennewitz K, Sticht C, Kopf S, Morgenstern J, Fleming T, Hell R, Yuan Z, Nawroth PP, Kroll J. Elevated 4-hydroxynonenal induces hyperglycaemia via Aldh3a1 loss in zebrafish and associates with diabetes progression in humans. Redox Biol. 2020 Oct;37:101723. doi: 10.1016/j.redox.2020.101723. Epub 2020 Sep 16. PMID: 32980661; PMCID: PMC7519378.

7. Bioactivity

Biological target:

Na+, K+-ATPase activity,
Target IC50: 120 μM against Na+, K+-ATPase activity

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In vitro activity

By using ferroptosis inhibitor ferrostatin-1 and measurement of ferroptotic cell death phenotype in both in vivo and in vitro cisplatin induced AKI model, the decreased blood urea nitrogen, creatinine, and tissue injury by ferrostatin-1 were observed, hence validated the essential involvement of ferroptosis in cisplatin induced AKI. VDR agonist paricalcitol could both functionally and histologically attenuate cisplatin induced AKI by decreasing lipid peroxidation (featured phenotype of ferroptosis), biomarker 4-hydroxynonenal (4HNE), and malondialdehyde (MDA), while reversing glutathione peroxidase 4 (GPX4, key regulator of ferroptosis) downregulation. VDR knockout mouse exhibited much more ferroptotic cell death and worsen kidney injury than wild type mice. And VDR deficiency remarkably decreased the expression of GPX4 under cisplatin stress in both in vivo and in vitro, further luciferase reporter gene assay showed that GPX4 were target gene of transcription factor VDR. In addition, in vitro study showed that GPX4 inhibition by siRNA largely abolished the protective effect of paricalcitol against cisplatin induced tubular cell injury. Besides, pretreatment of paricalcitol could also alleviated Erastin (an inducer of ferroptosis) induced cell death in HK-2 cell. These data suggested that ferroptosis plays an important role in cisplatin induced AKI. VDR activation can protect against cisplatin induced renal injury by inhibiting ferroptosis partly via trans-regulation of GPX4.

Reference: Hu Z, Zhang H, Yi B, Yang S, Liu J, Hu J, Wang J, Cao K, Zhang W. VDR activation attenuate cisplatin induced AKI by inhibiting ferroptosis. Cell Death Dis. 2020 Jan 29;11(1):73. doi: 10.1038/s41419-020-2256-z. PMID: 31996668; PMCID: PMC6989512.

In vivo activity

Aldh3a1-/- zebrafish larvae displayed retinal vasodilatory alterations, impaired glucose homeostasis, which can be aggravated via pdx1 silencing induced hyperglycaemia. Unexpectedly, MG was not altered, but 4-hydroxynonenal (4-HNE), another prominent lipid peroxidation RCS exhibited high affinity with Aldh3a1, was increased in aldh3a1 mutants. 4-HNE was responsible for the retinal phenotype via pancreas disruption induced hyperglycaemia and can be rescued via l-Carnosine treatment. Furthermore, in type 2 diabetic patients, serum 4-HNE was increased and correlated with disease progression. Thus, our data suggest impaired 4-HNE detoxification and elevated 4-HNE concentration as biomarkers but also the possible inducers for diabetes, from genetic susceptibility to the pathological progression.

Reference: Lou B, Boger M, Bennewitz K, Sticht C, Kopf S, Morgenstern J, Fleming T, Hell R, Yuan Z, Nawroth PP, Kroll J. Elevated 4-hydroxynonenal induces hyperglycaemia via Aldh3a1 loss in zebrafish and associates with diabetes progression in humans. Redox Biol. 2020 Oct;37:101723. doi: 10.1016/j.redox.2020.101723. Epub 2020 Sep 16. PMID: 32980661; PMCID: PMC7519378.

Note: The information listed here was extracted from literature. MedKoo has not independently retested and confirmed the accuracy of these methods. Customer should use it just for a reference only.