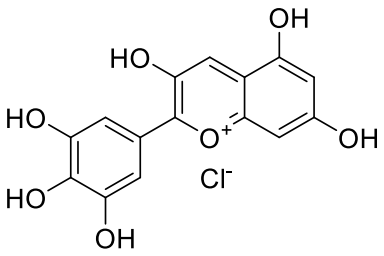
Product data sheet

MedKoo Cat#: 563782 Name: Delphinidin Chloride CAS#: 528-53-0 (Cl) Chemical Formula: $C_{15}H_{11}ClO_7$ Exact Mass: 338.0193 Molecular Weight: 338.7	
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:
Delphinidin Chloride is an inhibitor of BDNF-induced migration and invasion in SKOV3 ovarian cancer cells.

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	46.0	135.81

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.95 mL	14.76 mL	29.52 mL
5 mM	0.59 mL	2.95 mL	5.90 mL
10 mM	0.30 mL	1.48 mL	2.95 mL
50 mM	0.06 mL	0.30 mL	0.59 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

1. Chen Y, Ge Z, Huang S, Zhou L, Zhai C, Chen Y, Hu Q, Cao W, Weng Y, Li Y. Delphinidin attenuates pathological cardiac hypertrophy via the AMPK/NOX/MAPK signaling pathway. Aging (Albany NY). 2020 Mar 25;12(6):5362-5383. doi: 10.18632/aging.102956. Epub 2020 Mar 25. PMID: 32209725; PMCID: PMC7138591.
2. Kang SH, Bak DH, Chung BY, Bai HW, Kang BS. Delphinidin enhances radio-therapeutic effects via autophagy induction and JNK/MAPK pathway activation in non-small cell lung cancer. Korean J Physiol Pharmacol. 2020 Sep 1;24(5):413-422. doi: 10.4196/kjpp.2020.24.5.413. PMID: 32830148; PMCID: PMC7445475.

In vivo study

1. Chen Y, Ge Z, Huang S, Zhou L, Zhai C, Chen Y, Hu Q, Cao W, Weng Y, Li Y. Delphinidin attenuates pathological cardiac hypertrophy via the AMPK/NOX/MAPK signaling pathway. Aging (Albany NY). 2020 Mar 25;12(6):5362-5383. doi: 10.18632/aging.102956. Epub 2020 Mar 25. PMID: 32209725; PMCID: PMC7138591.
2. Han B, Peng X, Cheng D, Zhu Y, Du J, Li J, Yu X. Delphinidin suppresses breast carcinogenesis through the HOTAIR/microRNA-34a axis. Cancer Sci. 2019 Oct;110(10):3089-3097. doi: 10.1111/cas.14133. Epub 2019 Sep 16. PMID: 31325197; PMCID: PMC6778627.

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7. Bioactivity

Biological target:

Delphinidin chloride, an anthocyanidin, shows endothelium-dependent vasorelaxation as well as modulates JAK/STAT3 and MAPKinase signaling to induce apoptosis in HCT116 cells.

In vitro activity

Neonatal rat cardiomyocytes (NRCMs) were treated with angiotensin II (Ang II) and delphinidin in vitro. The cardiac hypertrophy-related increase in cell size after Ang II stimulation was blocked by delphinidin (50 μM) (Figure 4B). Furthermore, delphinidin (50 μM) significantly prevented Ang II-induced increases in Anp, Bnp and β-MHC mRNA expression levels (Figure 4C). In addition, DHE and DCF-DA fluorescence images showed that the marked increase in ROS production in cardiomyocytes in response to Ang II stimulation was attenuated by delphinidin (50 μM) (Figure 4D and 4E). Consistent with these findings, delphinidin (50 μM) reduced the increased myocardial NOX activity after Ang II stimulation (Figure 4F). Delphinidin significantly blocked the increase in cell proliferation induced by Ang II, judging by the results of cell counting and CCK-8 assays (Supplementary Figure 2A, 2B). Moreover, the results of scratch wound and Transwell migration assays suggested that delphinidin administration abrogated the increased cell migration induced by Ang II stimulation (Supplementary Figure 2C, 2D). Furthermore, the administration of delphinidin dramatically suppressed Ang II-induced increases in the mRNA levels of the fibrotic markers collagen I, collagen III, and CTGF (Supplementary Figure 2E). In summary, all the above results showed that delphinidin protected against pathological cell growth, oxidative stress and activation in cardiomyocytes and cardiac fibroblasts induced by Ang II in vitro.

Reference: Aging (Albany NY). 2020 Mar 31; 12(6): 5362–5383. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7138591/

In vivo activity

To determine the effect of delphinidin on cardiac hypertrophy in vivo, wild-type (WT) mice underwent TAC or sham operation; after 3 days, the mice were assigned to receive delphinidin or DMSO for 8 weeks. Delphinidin at the highest dosage used (15 mg/kg/day) significantly reversed TAC-induced cardiac hypertrophy, which manifested as decreased heart weight/body weight (HW/BW) and heart weight/tibia length (HW/TL) ratios (Figure 2A). In addition, the decreased left ventricular end-systolic dimension (LVESd) and left ventricular end-diastolic dimension (LVEDd) and increased left ventricular ejection fraction (LVEF) and left ventricular shortening rate (LVFS) compared with those of the sham surgery group further confirmed the effect of delphinidin at the high dosage on cardiac function (Figure 2B). Moreover, marked myocyte hypertrophy was observed at 8 weeks after surgery. Consistently, TAC increased myocardial mRNA expression of hypertrophic markers atrial natriuretic factor (Anp), brain natriuretic peptide (Bnp), and β-myosin heavy chain (β-MHC) in WT mice, but these changes were ameliorated by delphinidin treatment at the high dosage, but not by delphinidin at the low dosage (Figure 2F). These results suggested that treatment with delphinidin at the high dosage could inhibit pathological hypertrophy, oxidative stress, and cardiac dysfunction caused by pressure overload.

Reference: Aging (Albany NY). 2020 Mar 31; 12(6): 5362–5383. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7138591/