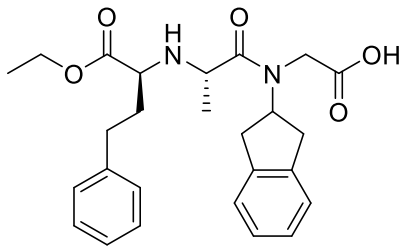
Product data sheet

MedKoo Cat#: 561619 Name: Delapril CAS#: 83435-66-9 Chemical Formula: $C_{26}H_{32}N_2O_5$ Exact Mass: 452.2311 Molecular Weight: 452.55	
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:
Delapril is an angiotensin-converting enzyme (ACE) inhibitor that blocks the conversion of angiotensin I to angiotensin II.

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
H2O	0.005	0.01

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	2.21 mL	11.05 mL	22.10 mL
5 mM	0.44 mL	2.21 mL	4.42 mL
10 mM	0.22 mL	1.10 mL	2.21 mL
50 mM	0.04 mL	0.22 mL	0.44 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. Zalvidea S, André L, Loyer X, Cassan C, Sainte-Marie Y, Thireau J, Sjaastad I, Heymes C, Pasquié JL, Cazorla O, Aimond F, Richard S. ACE inhibition prevents diastolic Ca2+ overload and loss of myofilament Ca2+ sensitivity after myocardial infarction. Curr Mol Med. 2012 Feb;12(2):206-17. doi: 10.2174/156652412798889045. PMID: 22280358; PMCID: PMC3472404.
2. Ichihara A, Hayashi M, Hirota N, Okada H, Koura Y, Tada Y, Kaneshiro Y, Tsuganezawa H, Saruta T. Angiotensin II type 2 receptor inhibits prorenin processing in juxtaglomerular cells. Hypertens Res. 2003 Nov;26(11):915-21. doi: 10.1291/hypres.26.915. PMID: 14714584.

In vivo study

1. Zalvidea S, André L, Loyer X, Cassan C, Sainte-Marie Y, Thireau J, Sjaastad I, Heymes C, Pasquié JL, Cazorla O, Aimond F, Richard S. ACE inhibition prevents diastolic Ca2+ overload and loss of myofilament Ca2+ sensitivity after myocardial infarction. Curr Mol Med. 2012 Feb;12(2):206-17. doi: 10.2174/156652412798889045. PMID: 22280358; PMCID: PMC3472404.
2. Nakaya H, Sasamura H, Hayashi M, Saruta T. Temporary treatment of prepubescent rats with angiotensin inhibitors suppresses the development of hypertensive nephrosclerosis. J Am Soc Nephrol. 2001 Apr;12(4):659-666. doi: 10.1681/ASN.V124659. PMID: 11274226.

7. Bioactivity
Biological target:
Delapril is an ACE inhibitor indicated in the treatment of essential hypertension.

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In vitro activity

The effect of delapril on cardiomyocytes isolated from the non-infarcted area comprised of viable cells subjected to cardiac remodeling following the MI (myocardial infarction) was investigated. In unloaded intact single cardiomyocytes from MI mice (vs. Sham), sarcomere length (SL) shortening, reflecting cell contraction during field stimulation, was decreased (Fig. 2A,B). Contraction and relaxation velocities were also reduced (Fig. 2C). Delapril treatment prevented all of these changes. Diastolic Ca2+ using the ratiometric indicator indo-1 was further investigated. Diastolic Ca2+ was higher in MI mice than in Sham mice, an increase that was completely prevented by delapril (Fig. 3C).Delapril treatment thus, had a notable effect on cell relaxation, Ca2+-transient decay and diastolic Ca2+. Delapril treatment had a powerful preventive effect on changes in both maximal active tension and myofilament Ca2+ sensitivity (pCa50: 5.74±0.02, n=22; P<0.05 for MI-D vs. MI). Delapril treatment had no effect on the reduction of SERCA2a abundance but remarkably, prevented the increase of PLB protein and phosphorylation levels. In conclusion, early therapy with delapril after MI preserved the normal contraction/relaxation cycle of surviving cardiomyocytes.

Reference: Curr Mol Med. 2012 Feb; 12(2): 206–217. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3472404/

In vivo activity

The effects of the ACE-I delapril on cardiomyocytes in a mouse model of heart failure (HF) after MI (myocardial infarction) were investigated. Mice were randomly assigned to three groups: Sham, MI, and MI-D (6 weeks of treatment with a non-hypotensive dose of delapril started 24h after MI). Delapril maintained cardiomyocyte contraction and relaxation, prevented diastolic Ca2+ overload and retained the normal Ca2+ sensitivity of contractile proteins. Delapril maintained SERCA2a activity through normalization of P-PLB/PLB (for both Ser16-PLB and Thr17-PLB) and PLB/SERCA2a ratios in cardiomyocytes, favoring normal reuptake of Ca2+ in the sarcoplasmic reticulum. In addition, delapril prevented defective cTnI function by normalizing the expression of PKC, enhanced in MI mice. In conclusion, early therapy with delapril after MI preserved the normal contraction/relaxation cycle of surviving cardiomyocytes with multiple direct effects on key intracellular mechanisms contributing to preserve cardiac function.

Reference: Curr Mol Med. 2012 Feb; 12(2): 206–217. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3472404/