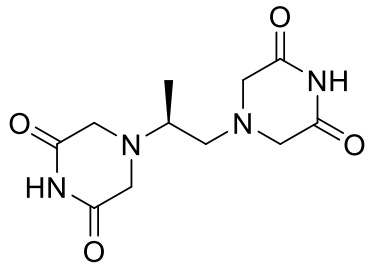
Product data sheet

MedKoo Cat#: 100260 Name: Dexrazoxane CAS#: 24584-09-6 Chemical Formula: $C_{11}H_{16}N_4O_4$ Exact Mass: 268.11716 Molecular Weight: 268.27	
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:
Dexrazoxane is a bisdioxopiperazine with iron-chelating, chemoprotective, cardioprotective, and antineoplastic activities. After hydrolysis to an active form that is similar to ethylenediaminetetraacetic acid (EDTA), dexrazoxane chelates iron, limiting the formation of free radical-generating anthracycline-iron complexes, which may minimize anthracycline-iron complex-mediated oxidative damage to cardiac and soft tissues. This agent also inhibits the catalytic activity of topoisomerase II, which may result in tumor cell growth inhibition.

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	54	201.29

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	3.73 mL	18.64 mL	37.28 mL
5 mM	0.75 mL	3.73 mL	7.46 mL
10 mM	0.37 mL	1.86 mL	3.73 mL
50 mM	0.07 mL	0.37 mL	0.75 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. Yu X, Ruan Y, Shen T, Qiu Q, Yan M, Sun S, Dou L, Huang X, Wang Q, Zhang X, Man Y, Tang W, Jin Z, Li J. Dexrazoxane Protects Cardiomyocyte from Doxorubicin-Induced Apoptosis by Modulating miR-17-5p. Biomed Res Int. 2020 Mar 1;2020:5107193. doi: 10.1155/2020/5107193. PMID: 32190669; PMCID: PMC7071803.

In vivo study

1. Polanski AK, Ebner A, Ebner B, Hofmann A, Steinbronn N, Brandt A, Forkmann M, Tausche AK, Morawietz H, Strasser RH, Wunderlich C. Dexrazoxane prevents the development of the impaired cardiac phenotype in caveolin-1-disrupted mice. J Cardiovasc Pharmacol. 2013 Jun;61(6):545-52. doi: 10.1097/FJC.0b013e31828de47c. PMID: 23474841.

2. Yu X, Ruan Y, Shen T, Qiu Q, Yan M, Sun S, Dou L, Huang X, Wang Q, Zhang X, Man Y, Tang W, Jin Z, Li J. Dexrazoxane Protects Cardiomyocyte from Doxorubicin-Induced Apoptosis by Modulating miR-17-5p. Biomed Res Int. 2020 Mar 1;2020:5107193. doi: 10.1155/2020/5107193. PMID: 32190669; PMCID: PMC7071803.

7. Bioactivity

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Biological target:

Dexrazoxane (ICRF-187) is a cardioprotective agent.

In vitro activity

To determine whether dexrazoxane exerts cardioprotective effect through its direct action on the cardiomyocytes, dexrazoxane (200 μM) was applied to neonatal mouse ventricular myocytes before doxorubicin treatment. Then cell viability was examined with MTT assay. The outcome illustrated that treatment with dexrazoxane prior to doxorubicin exposure substantially increased the cell viability (Fig. 3G). In addition, LDH release was detected, another index of cellular damage. Doxorubicin induced a remarkable increase in LDH leakage, which was blocked by dexrazoxane treatment (Fig. 3H). DNA electrophoresis assay also proved that dexrazoxane could attenuate doxorubicin-induced cell death (Fig. 3I). Our study infers that dexrazoxane could increase cardiomyocyte viability and mitigate cardiotoxicity after doxorubicin treatment.

Reference: Biochem Biophys Res Commun. 2020 Feb 26;523(1):140-146. https://linkinghub.elsevier.com/retrieve/pii/S0006-291X(19)32347-2

In vivo activity

Dexrazoxane treatment was evaluated for 6 weeks in cav1 mice and wild-type controls. This study provides the first evidence for a reduced reactive oxygen species formation in the vessels of dexrazoxane-treated cav1 mice. This reduced oxidative stress resulted in a markedly reduced rate of apoptosis, which finally was translated into a significantly improved heart function in dexrazoxane-treated cav1 mice. These hemodynamic improvements were accompanied by significantly lowered proatrial natriuretic peptide levels. Notably, these protective properties of dexrazoxane were not evident in wild-type animals. Taken together, these novel findings indicate that dexrazoxane significantly reduces vascular reactive oxygen species formation cav1. Because this is paralleled by an improved cardiac performance in cav1 mice, these data suggest dexrazoxane as a novel therapeutic strategy in this specific cardiomyopathy.

Reference: J Cardiovasc Pharmacol. 2013 Jun;61(6):545-52. https://doi.org/10.1097/FJC.0b013e31828de47c