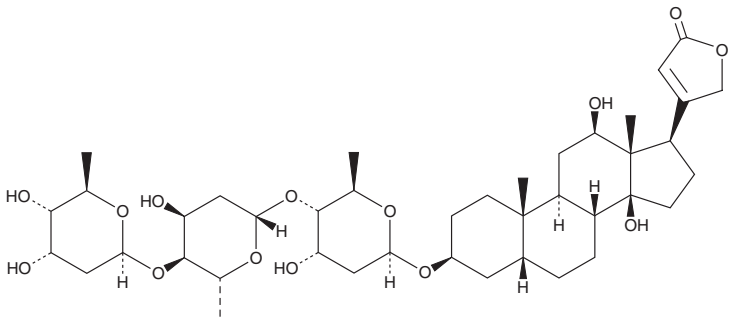
PRODUCT INFORMATION

Digoxin

Item No. 22266

CAS Registry No.:	20830-75-5
Formal Name:	(3β,5β,12β)-3-[(O-2,6-dideoxy-β-D-ribo-hexopyranosyl-(1→4)-O-2,6-dideoxy-β-D-ribo-hexopyranosyl-(1→4)-2,6-dideoxy-β-D-ribo-hexopyranosyl)oxy]-12,14-dihydroxy-card-20(22)-enolide
Synonym:	NSC 95100
MF:	$C_{41}H_{64}O_{14}$
FW:	780.9
Purity:	≥98%
UV/Vis.:	λ_{max}: 218 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

Digoxin is supplied as a crystalline solid. A stock solution may be made by dissolving the digoxin in the solvent of choice, which should be purged with an inert gas. Digoxin is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of digoxin in these solvents is approximately 30 mg/ml.

Description

Digoxin is a cardiac glycoside and metabolite of digitoxin that binds to and inhibits the Na^+/K^+-ATPase in cardiac tissues in an ATP- and Mg^{2+}-dependent manner.[1] This inhibition results in loss of the transmembrane Na^+ gradient, which decreases activity of the Na^+/Ca^{2+} exchanger, increasing intracellular Ca^{2+} levels, inotropy, and cardiac force.[2] It increases activity of mitochondrial ATPase and actomyosin ATPase in rat hearts, which is directly correlated with increased myofibrillar contractile strength.[3] *In vivo*, digoxin also decreases right atrial pressure and increases cardiac output in a canine model of congestive heart failure produced by pulmonary artery constriction.[4] Formulations containing digoxin have been used to treat atrial fibrillation.[5]

References

1. Matsui, H. and Schwartz, A. Mechanism of cardiac glycoside inhibition of the (Na^+-K^+)-dependent ATPase from cardiac tissue. *Biochim Biophys. Acta.* **151(3)**, 655-663 (1968).
2. Neves, C.H., Tibana, R.A., Prestes, J., *et al.* Digoxin induces cardiac hypertrophy without negative effects on cardiac function and physical performance in trained normotensive rats. *Int. J. Sports Med.* **38(4)**, 263-269 (2017).
3. Hamrick, M.E. and Fritz, P.J. Enzymatic adaptation: Molecular basis for cardiac glycoside action? 1. Increase in rat heart actomyosin and mitochondrial ATPase specific activities following digoxin injection. *Biochem. Biophys. Res. Commun.* **22(5)**, 540-546 (1966).
4. Davis, J.O., Howell, D.S., and Hyatt, R.E. Effects of acute and chronic digoxin administration in dogs with right-sided congestive heart failure produced by pulmonary artery constriction. *Circ. Res.* **3(3)**, 259-263 (1955).
5. Kotecha, D., Calvert, M., Deeks, J.J., *et al.* A review of rate control in atrial fibrillation, and the rationale and protocol for the RATE-AF trial. *BMJ Open* **7(7)**, e015099 (2017).

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