

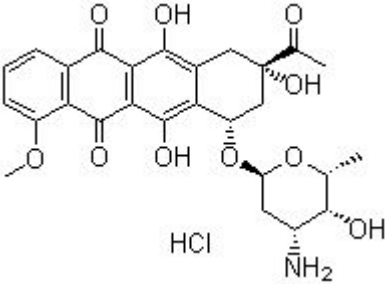


## Product Introduction

### Daunorubicin HCl

Daunorubicin HCl inhibits both DNA and RNA synthesis and inhibits DNA synthesis with  $K_i$  of 0.02  $\mu\text{M}$ .

#### Technical Data:

<b>Molecular Weight (MW):</b>	563.98	
<b>Formula:</b>	$\text{C}_{27}\text{H}_{29}\text{NO}_{10} \cdot \text{HCl}$	
<b>Solubility (25°C)</b>	DMSO 113 mg/mL	
<b>* &lt;1 mg/ml means slightly soluble or insoluble:</b>	Water 113 mg/mL	
	Ethanol 13 mg/mL	
<b>Purity:</b>	>98%	
<b>Storage:</b>	3 years -20°C Powder 6 months-80°C in DMSO	
<b>CAS No.:</b>	23541-50-6	

#### Biological Activity

At drug concentrations that reflect the peak plasma concentration after Daunorubicin administration, the primary mechanism is likely to be through interaction with topoisomerase II, which may be a primary triggering event for growth arrest and/or cell killing through a signalling pathway leading to apoptosis, at least in leukemic cells and thymocytes. The quinone structure permits daunorubicin to act as electron acceptors in reactions mediated by oxoreductive enzymes including cytochrome P450 reductase, NADH dehydrogenase, and xanthine oxidase. At Daunorubicin concentrations exceeding approximately 2–4  $\mu\text{M}$ , free radical mediated toxicity and DNA cross-linking may become evident. Daunorubicin inhibits both DNA and RNA syntheses in HeLa cells over a concentration range of 0.2 through 2  $\mu\text{M}$ . Daunorubicin inhibits

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both DNA syntheses in Ehrlich ascites tumor cells over a concentration range of 4  $\mu$ M. Daunorubic triggers apoptosis at concentrations of 0.5 and 1  $\mu$ M in either HL-60 or U-937 human leukemic cells. [1] Daunorubicin stimulates ceramide elevation and apoptosis in P388 and U937 cells through de novo synthesis via activation of the enzyme ceramide synthase. [2] Daunorubicin dose-dependently increases the phosphatidylserine exposure and consequent procoagulant activity of human umbilical vein endothelial cells. Daunorubicin (0.2 mM) significantly enhances the release of endothelial microparticles which are highly procoagulant in human umbilical vein endothelial cells. [3]

## References

- [1] Gewirtz DA, et al. *Biochem Pharmacol*, 1999, 57(7), 727-741.
- [2] Bose R, et al. *Cell*, 1995, 82(3), 405-414.
- [3] Fu Y, et al. *Thromb Haemost*, 2010, 104(6), 1235-1241.
- [4] Tardi P, et al. *Leuk Res*, 2009, 33(1), 129-139.



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