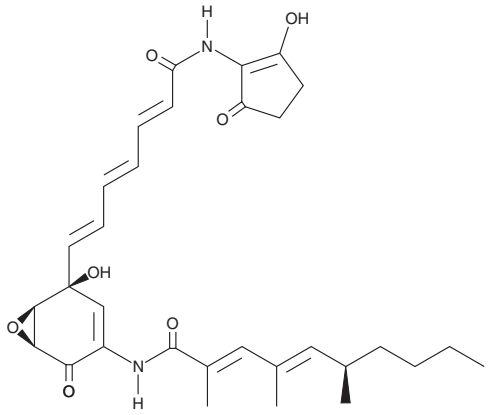
PRODUCT INFORMATION

Manumycin A

Item No. 10010497

CAS Registry No.:	52665-74-4
Formal Name:	N-[(1S,5S,6R)-5-hydroxy-5-[(1E,3E,5E)-7-[(2-hydroxy-5-oxo-1-cyclopenten-1-yl)amino]-7-oxo-1,3,5-heptatrien-1-yl]-2-oxo-7-oxabicyclo[4.1.0]hept-3-en-3-yl]-2E,4E,6R-trimethyl,2,4-decadienamide
Synonyms:	NSC 622141, UCF 1C
MF:	$C_{31}H_{38}N_2O_7$
FW:	550.7
Purity:	≥98%
UV/Vis.:	λ_{max}: 279, 318 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥2 years



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

Laboratory Procedures

Manumycin A is supplied as a crystalline solid. A stock solution may be made by dissolving the manumycin A in an organic solvent purged with an inert gas. Manumycin A is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of manumycin A in these solvents is approximately 5, 10, and 20 mg/ml, respectively.

Manumycin A is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, manumycin A should first be dissolved in DMF and then diluted with the aqueous buffer of choice. Manumycin A has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Manumycin A is an antibiotic that acts as a potent and selective farnesyltransferase (FTase) inhibitor with anti-tumor activity.[1,2] It inhibits rat brain FTase with a K_i value of 1.2 µM, thereby preventing Ras activation which requires farnesylation at the C-terminus for membrane attachment.[1] It exhibits significant antitumor activity against K_i-ras-activated solid tumors in mice at a dose of 6.3 mg/kg.[1] Manymycin A inhibits IκB kinase (IKK), independent of FTase inhibition, in an number of cells types with effective concentrations of 2-10 µM.[3] In ApoE-deficient mice, Manumycin A treatment for 22 weeks at 5 mg/kg reduced aortic fatty streak lesion size to 43% of vehicle-treated animals, indicating FTase inhibition as a potential target for prevention or treatment of atherosclerosis.[4]

References

1. Hara, M., Akasaka, K., Akinaga, S., *et al. Proc. Natl. Acad. Sci. USA* **90**, 2281-2285 (1993).
2. Hara, M. and Han, M. *Proc. Natl. Acad. Sci. USA* **92**, 3333-3337 (1995).
3. Bernier, M., Kwon, Y.-K., Pandey, S.K., *et al. J. Biol. Chem.* **281(5)**, 2551-2561 (2006).
4. Sugita, M., Sugita, H., and Kaneki, M. *Arterioscler. Thromb. Vasc. Biol.* **27**, 1390-1395 (2007).

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