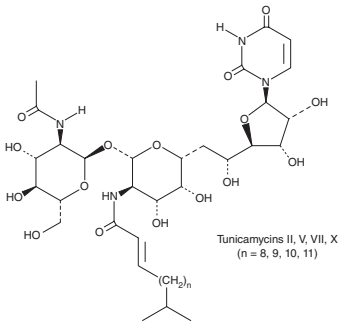
PRODUCT INFORMATION

Tunicamycin Mixture

Item No. 11445



CAS Registry No.: 11089-65-9
MF: $C_{39}H_{64}N_4O_{16}$ (for Tunicamycin VII)
FW: 845.0
Purity: ≥95% (mixture of congeners)
UV/Vis.: λ_{max}: 208, 260 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

Tunicamycin is supplied as a crystalline solid. A stock solution may be made by dissolving the tunicamycin in the solvent of choice which should be purged with an inert gas. Tunicamycin is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of tunicamycin in these solvents is approximately 20 mg/ml.

Tunicamycin is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, tunicamycin should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Tunicamycin has a solubility of approximately 0.25 mg/ml in a 1:3 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Tunicamycin mixture is a mixture of tunicamycins with variable *trans*-2,3-unsaturated branched chain fatty acid (BFCA) chain lengths. Tunicamycins are anti-microbial agents that are active against Gram-positive bacteria, fungi, and viruses.[1] They inhibit the N-acetylhexosamine (HexNAc) phosphotransferase family of enzymes in bacteria and prevent peptidoglycan biosynthesis.[2] In eukaryotes, they inhibit N-acetylglucosamine (GlcNAc) phosphotransferase (GPT), preventing the first step in N-linked glycosylation and inducing the unfolded protein response and cell death.[2-4] The cellular toxicity of tunicamycins is linked to the *trans*-2,3-unsaturated BCFA, and saturated BCFA-containing tunicamycin derivatives, such as TunR1 (Item No. 31537) and TunR2 (Item No. 31538), have reduced toxicity.[2,5] Tunicamycins impair glycosylation of the receptor tyrosine kinases EGFR, HER2, HER3, and IGF-1R, which prevents their translocation out of the endoplasmic reticulum and Golgi apparatus and reduces their protein levels and activity.[6] Tunicamycin sensitizes EGFR inhibitor-resistant U251 glioma and Bx/PC-3 pancreatic adenocarcinoma cells to irradiation.[6]

References

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6. Contessa, J.N., Bhojani, M.S., Freeze, H.H., *et al.* Inhibition of N-linked glycosylation disrupts receptor tyrosine kinase signaling in tumor cells. Cancer Res. 68(10), 3803-3809 (2008).

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