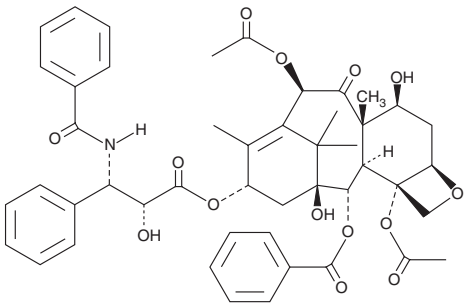
PRODUCT INFORMATION

Paclitaxel

Item No. 10461

CAS Registry No.: 33069-62-4
Formal Name: βS-(benzoylamino)-αR-hydroxy-benzenepropanoic acid, (2aR,4S,4aS,6R,9S,11S,12S,12aR,12bS)-6,12b-*bis*(acetyloxy)-12-(benzoyloxy)-2a,3,4,4a,5,6,9,10,11,12,12a,12b-dodecahydro-4,11-dihydroxy-4a,8,13,13-tetramethyl-5-oxo-7,11-methano-1H-cyclodeca[3,4]benz[1,2-b]oxet-9-yl ester
Synonym: NSC 125973
MF: $C_{47}H_{51}NO_{14}$
FW: 853.9
Purity: ≥98%
UV/Vis.: λ_{max}: 228 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

Paclitaxel is supplied as a crystalline solid. A stock solution may be made by dissolving the paclitaxel in the solvent of choice. Paclitaxel is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF), which should be purged with an inert gas. The solubility of paclitaxel in these solvents is approximately 1.5 mg/ml in ethanol and approximately 5 mg/ml in DMSO and DMF.

Paclitaxel is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, paclitaxel should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Paclitaxel has a solubility of approximately 0.1 mg/ml in a 1:10 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Paclitaxel, a potent disruptor of microtubules derived from the bark of the Pacific yew tree, is widely used as a chemotherapeutic compound. Tested against a panel of cervical (HeLa), lung (A549), breast (MCF-7), colon (HT-29), ovarian (OVG-1), and pancreatic (PC-Sh) carcinomas, paclitaxel demonstrates IC_{50} values ranging from 2.5-7.5 nM.[1] Paclitaxel disrupts multipolar spindle formation, inducing cell cycle arrest in various human cell cancer lines (IC_{50}s = 6.7-18.5 nM) at both prophase and G_1.[2] It initiates apoptosis of cancer cells through multiple mechanisms involving p53-dependent and -independent pathways, Bcl-2 family members, cyclin-dependent kinases, and c-Jun N-terminal kinases/stress-activated protein kinases.[3]

References

1. Liebmann, J.E., Cook, J.A., Lipschultz, C., *et al. Br. J. Cancer* **68**, 1104-1109 (1993).
2. Woods, C.M., Zhu, J., McQueney, P.A., *et al. Mol. Med.* **1(5)**, 506-526 (1995).
3. Wang, T., Wang, H.-S., and Soong, Y.-K. *Cancer* **88**, 2619-2628 (2000).

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