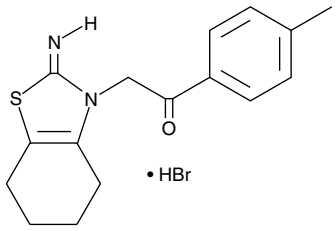
Product Information

Pifithrin-α

Item No. 13326

CAS Registry No.: 63208-82-2
Formal Name: 1-(4-methylphenyl)-2-(4,5,6,7-tetrahydro-2-imino-3(2H)-benzothiazolyl)-ethanone, monohydrobromide
Synonym: PFT-α
MF: $C_{16}H_{18}N_2OS \bullet HBr$
FW: 367.3
Purity: ≥95%
Stability: ≥2 years at -20°C
Supplied as: A crystalline solid
UV/Vis.: λ_{max}: 259 nm



Laboratory Procedures

For long term storage, we suggest that pifithrin-α be stored as supplied at -20°C. It should be stable for at least two years.

Pifithrin-α is supplied as a crystalline solid. A stock solution may be made by dissolving the pifithrin-α in an organic solvent purged with an inert gas. Pifithrin-α is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of pifithrin-α in these solvents is approximately 1 mg/ml.

Pifithrin-α is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, pifithrin-α should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. Pifithrin-α has a solubility of approximately 0.1 mg/ml in a 1:10 solution of DMSO:PBS (pH 7.2) using this method.

If aqueous stock solutions are required for biological experiments, they can best be prepared by diluting the organic solvent into aqueous buffers or isotonic saline. Ensure that the residual amount of organic solvent is insignificant, since organic solvents may have physiological effects at low concentrations. We do not recommend storing the aqueous solution for more than one day.

Pifithrin-α is an inactivator of p53 that blocks p53-dependent transcriptional activation and apoptosis.[1] It prevents p53-mediated apoptosis induced by cytotoxic compounds in C8 cells at 10 µM [1] and in human umbilical vein endothelial cells at 30 µM.[2] Pifithrin-α can also protect cells from DNA damage-induced apoptosis by a p53-independent mechanism that might involve cyclin D1.[3]

References

1. Komarov, P.G., Komarova, E.A., Kondratov, R.V., *et al.* A chemical inhibitor of p53 that protects mice from the side effects of cancer therapy. *Science* **285**, 1733-1737 (1999).
2. Lorenzo, E., Ruiz-Ruiz, C., Quesada, A.J., *et al.* Doxorubicin induces apoptosis and CD95 gene expressionin human primary endothelial cells through a p53-dependent mechanism. *J. Biol. Chem.* **277(17)**, 10883-10892 (2002).
3. Sohn, D., Graupner, V., Neise, D., *et al.* Pifithrin-α protects against DNA damage-inducedapoptosis downstream of mitochondria independent of p53. *Cell Death and Differentiation* **16**, 869-878 (2009).

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