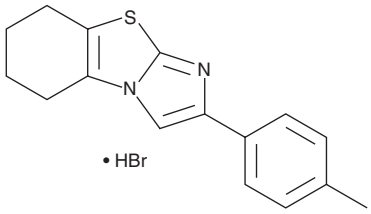
PRODUCT INFORMATION

Cyclic Pifithrin-α (hydrobromide)

Item No. 14748

CAS Registry No.: 511296-88-1
Formal Name: 5,6,7,8-tetrahydro-2-(4-methylphenyl)-imidazo[2,1-b] benzothiazole, monohydrobromide
Synonyms: Cyclic PFT-α, PFT-β, Pifithrin-β
MF: $C_{16}H_{16}N_2S \bullet HBr$
FW: 349.3
Purity: ≥95%
UV/Vis.: λ_{max}: 260 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

Cyclic Pifithrin-α (PFT-α) (hydrobromide) is supplied as a crystalline solid. A stock solution may be made by dissolving the cyclic PFT-α (hydrobromide) in the solvent of choice, which should be purged with an inert gas. Cyclic PFT-α (hydrobromide) is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of cyclic PFT-α (hydrobromide) in ethanol and DMSO is approximately 0.5 mg/ml and approximately 10 mg/ml in DMF.

Cyclic PFT-α (hydrobromide) is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, cyclic PFT-α (hydrobromide) should first be dissolved in DMF and then diluted with the aqueous buffer of choice. Cyclic PFT-α (hydrobromide) has a solubility of approximately 0.14 mg/ml in a 1:6 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Pifithrin-α (PFT-α; Item No. 13326) is a reversible inhibitor of p53-dependent transcription and apoptosis.[1] Cyclic PFT-α, also known as PFT-β, is a stable analog of PFT-α, formed by the condensation of pifithrin-α in solution.[2] It inhibits the growth of the cancer cell lines IGROV-1, A2780, and HCT116 (IC_{50} = 23, 77, and 103 µM, respectively).[2,3] At doses lower than those that inhibit growth, cyclic PFT-α induces autophagy in HCT116 cells and sensitizes IGROV-1 and H460 cells to anti-microtubule agents.[4,5] In addition, 10 µM cyclic PFT-α blocks p53-dependent protection from DNA damage, induced by hydrogen peroxide or ultraviolet radiation, in melanocytes.[6]

References

1. Komarov, P.G., Komarova, E.A., Kondratov, R.V., *et al. Science* **285(5434)**, 1733-1737 (1999).
2. Walton, M.I., Wilson, S.C., Hardcastle, I.R., *et al. Mol. Cancer Ther.* **4(9)**, 1369-1377 (2005).
3. Christodoulou, M.S., Colombo, F., Passarella, D., *et al. Bioorg. Med. Chem.* **19(5)**, 1649-1657 (2011).
4. Tasdemir, E., Maiuri, M.C., Orhon, I., *et al. Cell Cycle* **7(19)**, 3006-3011 (2008).
5. Zuco, V. and Zunino, F. *Neoplasia* **10(6)**, 587-596 (2008).
6. Kadekaro, A.L., Chen, J., Yang, J., *et al. Mol. Cancer Res.* **10(6)**, 778-786 (2012).

CAYMAN CHEMICAL
1180 EAST ELLSWORTH RD
ANN ARBOR, MI 48108 · USA
PHONE: [800] 364-9897
[734] 971-3335
FAX: [734] 971-3640
CUSTSERV@CAYMANCHEM.COM
WWW.CAYMANCHEM.COM