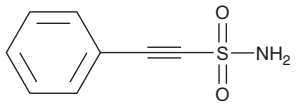
PRODUCT INFORMATION

Pifithrin-μ

Item No. 10748

CAS Registry No.:	64984-31-2
Formal Name:	2-phenyl-ethynesulfonamide
Synonyms:	PFT-μ, 2-Phenylethynesulfonamide
MF:	$C_8H_7NO_2S$
FW:	181.2
Purity:	≥98%
UV/Vis.:	λ_{max}: 247 nm 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

Pifithrin-μ (PFT-μ) is supplied as a crystalline solid. A stock solution may be made by dissolving the PFT-μ in the solvent of choice. PFT-μ is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of PFT-μ in these solvents is approximately 14 mg/ml.

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

Description

In addition to its transactivational functions, p53 mediates apoptosis by binding with the anti-apoptotic proteins Bcl-xL and Bcl-2 at the mitochondrial surface.[1,2] PFT-μ is an inhibitor of p53-mediated apoptosis, preventing p53 binding to Bcl-xL and Bcl-2 at the mitochondria without affecting p53 transactivational activities.[1] *In vitro*, PFT-μ binds both p53 (K_d = 0.82 mM) and Bcl-xL (K_d = 0.80 mM).[3] PFT-μ reduces p53-mediated apoptosis induced by γ-radiation in mouse thymocytes *in vitro* and protects mice from doses of radiation that cause lethal hematopoietic syndrome. At 25 μM, PFT-μ reduces apoptosis triggered by nutlin-3, which inhibits MDM2/p53 binding and potentiates p53-mediated growth arrest and apoptosis.[2] PFT-μ also interacts selectively with heat shock protein 70 (Hsp70), leading to disruption of the association between Hsp70 and many of its co-chaperones and substrate proteins.[4]

References

1. Strom, E., Sathe, S., Komarov, P.G., *et al*. Small-molecule inhibitor of p53 binding to mitochondria protects mice from gamma radiation. *Nat. Chem. Biol.* **2(9)**, 474-479 (2006).
2. Vaseva, A.V., Marchenko, N.D., and Moll, U.M. The transcription-independent mitochondrial p53 program is a major contributor to nutlin-induced apoptosis in tumor cells. *Cell Cycle* **8(11)**, 1711-1719 (2009).
3. Hagn, F., Klein, C., Demmer, O., *et al*. BclxL changes conformation upon binding to wild-type but not mutant p53 DNA binding domain. *J. Biol. Chem.* **285(5)**, 3439-3450 (2010).
4. Leu, J.I., Pimkina, J., Frank, A., *et al*. A small molecule inhibitor of inducible heat shock protein 70. Mol. *Cell* **36(1)**, 15-27 (2009).

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