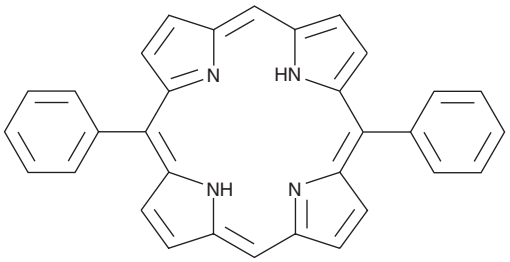
PRODUCT INFORMATION

5,15-DPP

Item No. 16090

CAS Registry No.:	22112-89-6
Formal Name:	5,15-diphenyl-21H,23H-porphine
Synonyms:	5,15-Diphenylporphyrin, STAT3 Inhibitor VIII
MF:	$C_{32}H_{22}N_4$
FW:	462.6
Purity:	≥95%
UV/Vis.:	λ_{max}: 402 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

5,15-DPP is supplied as a crystalline solid. A stock solution may be made by dissolving the 5,15-DPP in the solvent of choice. 5,15-DPP is soluble in dimethyl formamide at a concentration of approximately 2 mg/ml.

5,15-DPP is sparingly soluble in aqueous solutions. To enhance aqueous solubility, dilute the organic solvent solution into aqueous buffers or isotonic saline. If performing biological experiments, ensure 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.

Description

Signal transducer and activator of transcription 3 (STAT3) is a cytokine-inducible transcription factor with roles in inflammation and cancer.[1,2] 5,15-DPP is a cell-permeable porphyrin derivative that selectively binds STAT3 (K_d = 880 nM).[3] This inhibits STAT3 dimerization *via* Src homology 2 (Sh2) domains (IC_{50} = 280 nM), preventing nuclear translocation and DNA binding.[3] 5,15-DPP poorly inhibits STAT1 (IC_{50} = 10 µM) and does not affect Grb2.[3] It reduces IL-6-dependent STAT3 activation and consequent c-myc expression in MDA-MB-468 cells and blocks TRAIL-induced migration and invasion in A549 cells.3,4 Furthermore, inhibition of JAK/STAT signaling in satellite cells *via* 5,15-DPP and the JAK2 inhibitor AG-490 (Item No. 10010311) has been used to stimulate muscle regeneration in a model of aging skeletal muscle deterioration.[5]

References

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2. Yu, H. and Jove, R. The stats of cancer - new molecular targets come of age. *Nat. Rev. Cancer* **4**, 97-105 (2004).
3. Uehara, Y., Mochizuki, M., Matsuno, K., *et al.* Novel high-throughput screening system for identifying STAT3-SH2 antagonists. *Biochem. Biophys. Res. Commun.* **380(3)**, 627-631 (2009).
4. Azijli, K., Yuvaraj, S., Peppelenbosch, M.P., *et al.* Kinome profiling of non-canonical TRAIL signaling reveals RIP1-Src-STAT3-dependent invasion in resistant non-small cell lung cancer cells. *J. Cell Sci.* **125(Pt 19)**, 4651-4661 (2012).
5. Price, F.D., von Maltzahn, J., Bentzinger, C.F., *et al.* Inhibition of JAK-STAT signaling stimulates adult satellite cell function. *Nat. Med.* **20(10)**, 1174-1181 (2014).

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