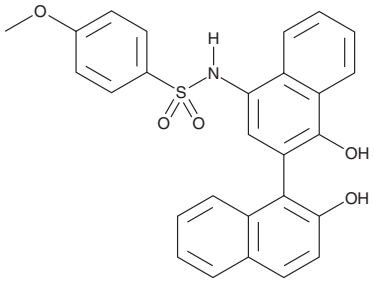
PRODUCT INFORMATION

C188-9

Item No. 30928

CAS Registry No.:	432001-19-9
Formal Name:	N-(1′,2-dihydroxy[1,2′-binaphthalen]-4′-yl)-4-methoxy-benzenesulfonamide
MF:	$C_{27}H_{21}NO_5S$
FW:	471.5
Purity:	≥98%
UV/Vis.:	λ_{max}: 229 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

C188-9 is supplied as a crystalline solid. A stock solution may be made by dissolving the C188-9 in the solvent of choice, which should be purged with an inert gas. C188-9 is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of C188-9 in these solvents is approximately 10 mg/ml.

C188-9 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, C188-9 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. C188-9 has a solubility of approximately 0.16 mg/ml in a 1:5 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

C188-9 is a STAT3 inhibitor.[1,2] It binds to the phosphotyrosyl peptide binding site in the STAT3 Src homology 2 (SH2) domain (K_i = 136 nM) and inhibits G-CSF-induced activation of STAT3 in patient-derived acute myeloid leukemia (AML) cells (IC_{50}s = 8-18 µM). C188-9 induces apoptosis in patient-derived AML cells (EC_{50}s = 6-50 µM) and reduces viability of HepG2, Huh7, and PLC/PRF/5 hepatoma cells (IC_{50}s = 10.19, 11.27, and 11.83 µM, respectively).[2,3] *In vivo*, C188-9 (100 mg/kg) reduces hepatic *Pten* deletion-induced hepatic macro- and microsteatosis, which reduces the development of hepatocellular carcinomas in mice. C188-9 (12.5 mg/kg) increases muscle fiber size in a murine Lewis lung carcinoma model of cancer cachexia.[1]

References

1. Silva, K.A.S., Dong, J., Dong, Y., *et al*. Inhibition of Stat3 activation suppresses caspase-3 and the ubiquitin-proteasome system, leading to preservation of muscle mass in cancer cachexia. *J. Biol. Chem.* **290(17)**, 11177-11187 (2015).
2. Redell, M.S., Ruiz, M.J., Alonzo, T.A., *et al*. Stat3 signaling in acute myeloid leukemia: Ligand-dependent and -independent activation and induction of apoptosis by a novel small-molecule Stat3 inhibitor. *Blood* **117(21)**, 5701-5709 (2011).
3. Jung, K.H., Yoo, W., Stevenson, H.L., *et al*. Multifunctional effects of a small-molecule STAT3 inhibitor on NASH and hepatocellular carcinoma in mice. *Clin. Cancer Res.* **23(18)**, 5537-5546 (2017).

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