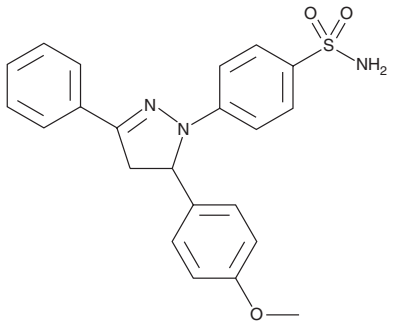
PRODUCT INFORMATION

ML-141

Item No. 18496

CAS Registry No.:	71203-35-5
Formal Name:	4-[4,5-dihydro-5-(4-methoxyphenyl)-3-phenyl-1H-pyrazol-1-yl]-benzenesulfonamide
Synonym:	CID-2950007
MF:	$C_{22}H_{21}N_3O_3S$
FW:	407.5
Purity:	≥98%
UV/Vis.:	λ_{max}: 229, 274, 357 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

ML-141 is supplied as a crystalline solid. A stock solution may be made by dissolving the ML-141 in the solvent of choice, which should be purged with an inert gas. ML-141 is soluble in organic solvents such as DMSO and dimethyl formamide (DMF). The solubility of ML-141 in these solvents is approximately 20 and 30 mg/ml, respectively.

ML-141 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, ML-141 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. ML-141 has a solubility of approximately 0.2 mg/ml in a 1:4 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

Cdc42 is a member of the Rho GTPase subfamily that has roles in cytoskeleton organization, cell cycle progression, signal transduction, and vesicle trafficking. It cycles between a GTP-bound active state and a GDP-bound inactive state. ML-141 is a noncompetitive, allosteric inhibitor of Cdc42 (EC_{50} = 2.1 µM) with no inhibitory action against Rho, Ras, Rab, or Rac.[1,2] It also inhibits the constitutively active Cdc42 Q61L mutant (EC_{50} = 2.6 µM).[1] ML-141 blocks Cdc42-dependent filopodia formation in 3T3 cells and migration of ovarian cancer cells, prevents Cdc42-regulated virus infection, and inhibits the signaling pathway of VLA-4 integrin.[1] It is a useful tool to interrogate the role of Cdc42 intracellular signaling and cell activity.[3,4]

References

1. Hong, L., Kenney, S.R., Phillips, G.K., *et al.* Characterization of a Cdc42 protein inhibitor and its use as a molecular probe. *J. Biol. Chem.* **288(12)**, 8531-8543 (2013).
2. Surviladze, Z., Waller, A., Strouse, J.J., *et al.* A potent and selective inhibitor of Cdc42 GTPase, National Center for Biotechnolog Information, in Probe Reports from the NIH Molecular Libraries Program, (2010).
3. Kalwat, M.A., Yoder, S.M., Wang, Z., *et al.* A p21-activated kinase (PAK1) signaling cascade coordinately regulates F-actin remodeling and insulin granule exocytosis in pancreatic β cells. *Biochem. Pharmacol.* **85(6)**, 808-816 (2013).
4. Pinzaglia, M., Montaldo, C., Polinari, D., *et al.* EIF6 over-expression increases the motility and invasiveness of cancer cells by modulating the expression of a critical subset of membrane-bound proteins. *BMC Cancer* **15**, 131-146 (2015).

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