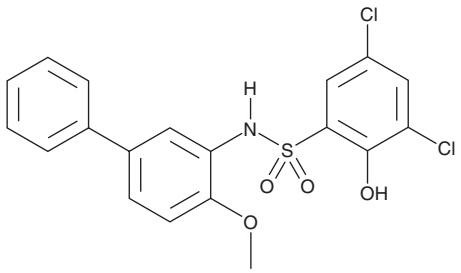
PRODUCT INFORMATION

BMS 303141

Item No. 16239

CAS Registry No.: 943962-47-8
Formal Name: 3,5-dichloro-2-hydroxy-N-(4-methoxy[1,1'-biphenyl]-3-yl)-benzenesulfonamide
MF: $C_{19}H_{15}Cl_2NO_4S$
FW: 424.3
Purity: ≥98%
UV/Vis.: λ_{max}: 208, 261 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

BMS 303141 is supplied as a crystalline solid. A stock solution may be made by dissolving the BMS 303141 in the solvent of choice, which should be purged with an inert gas. BMS 303141 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of BMS 303141 in these solvents is approximately 15, 25, and 30 mg/ml, respectively.

BMS 303141 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, BMS 303141 should first be dissolved in DMF and then diluted with the aqueous buffer of choice. BMS 303141 has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMF:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

ATP citrate lyase (ACL) catalyzes the synthesis of acetyl-CoA and oxaloacetate using citrate, CoA, and ATP as substrates and Mg^{2+} as a cofactor.[1] The ACL-dependent synthesis of acetyl-CoA is important for the *de novo* synthesis of fatty acids and cholesterol.[2] Furthermore, as a key enzyme for linking glucose and lipid metabolism, ACL is thought to contribute to the Warburg effect in cancer cells.[3] BMS 303141 is a cell-permeable, 2-hydroxy-N-arylbenzenesulfonamide that inhibits ACL with an IC_{50} value of 0.13 µM.[4] At an oral dose of 100 mg/kg/day, BMS 303141 has been reported to reduce weight gain and lower plasma cholesterol, triglycerides, and glucose in a mouse model of hyperlipidemia.[4]

References

1. Ma, Z., Chu, C.-H., and Cheng, D. A novel direct homogeneous assay for ATP citrate lyase. *J. Lipid Res.* **50(10)**, 2131-2135 (2009).
2. Dufort, F.J., Gumina, M.R., Ta, N.L., *et al.* Glucose-dependent de novo lipogenesis in B lymphocytes: A requirement for ATP-citrate lyase in lipopolysaccharide-induced differentiation. *J. Biol. Chem.* **289(10)**, 7011-7024 (2014).
3. Zu, X.-Y., Zhang, Q.-H., Liu, J.-H., *et al.* ATP citrate lyase inhibitors as novel cancer therapeutic agents. *Recent Pat. Anticancer Drug Discov.* **7(2)**, 154-167 (2012).
4. Li, J.J., Wang, H., Tino, J.A., *et al.* 2-hydroxy-N-arylbenzenesulfonamides as ATP-citrate lyase inhibitors. *Bioorg. Med. Chem. Lett.* **17(11)**, 3208-3211 (2007).

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