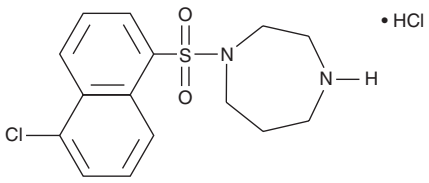
PRODUCT INFORMATION

ML-9

Item No. 10010236

CAS Registry No.: 105637-50-1
Formal Name: 1-[(5-chloro-1-naphthalenyl)sulfonyl] hexahydro-1H-1,4-diazepine, monohydrochloride
MF: $C_{15}H_{17}ClN_2O_2S \bullet HCl$
FW: 361.3
Purity: ≥98%
UV/Vis.: λ_{max}: 229, 300 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-9 is supplied as a crystalline solid. A stock solution may be made by dissolving the ML-9 in the solvent of choice, which should be purged with an inert gas. ML-9 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of ML-9 in ethanol is approximately 0.5 mg/ml and approximately 30 mg/ml in DMSO and DMF.

ML-9 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, ML-9 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. ML-9 has a solubility of approximately 0.5 mg/ml in a 1:1 solution of DMSO:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

ML-9 was originally identified as a selective Ca^{2+}-calmodulin-dependent myosin light chain kinase inhibitor. Concentrations from 10-100 µM are effective at inhibting vascular smooth muscle tension and reducing intracellular Ca^{2+} concentrations.[1] ML-9 also inhibits PKB/Akt activity with an IC_{50} range of 10-50 µM in rat primary adipocytes. This results in a specific inhibition of insulin-stimulated glucose transport and GLUT4/IGF II receptor translocation to the plasma membrane yet does not interfere with the antilipolytic effect of insulin.[2] Additionally, ML-9 inhibits other serine/threonine kinases including PKA (IC_{50} = ~20 µM), p90 S6 (IC_{50} = ~50 µM), and MAP kinase (IC_{50} = ~35 µM).[2]

References

1. Ito, S., Kume, H., Honjo, H., *et al.* ML-9, a myosin light chain kinase inhibitor, reduces intracellular Ca^{2+} concentration in guinea pig trachealis. *Eur. J. Pharmacol.* **486**, 325-333 (2004).
2. Smith, U., Carvalho, E., Mosialou, E., *et al.* PBK inhibition prevents the stimulatory effect of insulin on glucose transport and protein translocation but not the antilipolytic effect in rat adipocytes. *Biochem. Biophys. Res. Commun.* **268**, 315-320 (2000).

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