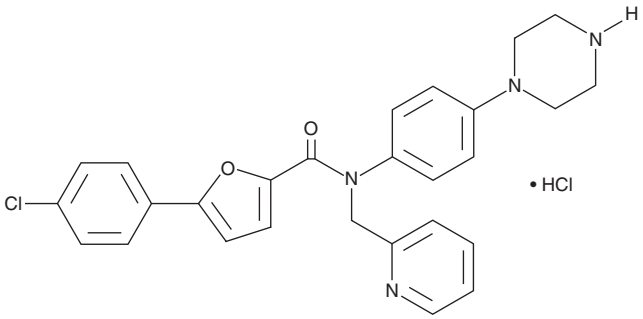
PRODUCT INFORMATION

MK2 Inhibitor IV

Item No. 14399

CAS Registry No.:	1314118-94-9
Formal Name:	5-(4-chlorophenyl)-N-[4-(1-piperazinyl) phenyl]-N-(2-pyridinylmethyl)-2-furancarboxamide, monohydrochloride
Synonym:	MK 25
MF:	$C_{27}H_{25}ClN_4O_2$ • HCl
FW:	509.4
Purity:	≥95%
UV/Vis.:	λ_{max}: 256, 312 nm
Supplied as:	A crystalline solid
Storage:	-20°C
Stability:	≥2 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

MK2 inhibitor IV is supplied as a crystalline solid. A stock solution may be made by dissolving the MK2 inhibitor IV in the solvent of choice, which should be purged with an inert gas. MK2 inhibitor IV is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide (DMF). The solubility of MK2 inhibitor IV in ethanol is approximately 1 mg/ml and approximately 30 mg/ml in DMSO and DMF.

MK2 inhibitor IV is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, MK2 inhibitor IV should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. MK2 inhibitor IV 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

Activation of the p38/mitogen-activated protein kinase-activated protein kinase 2 (MAPKAPK2 or MK2) pathway has been implicated in promoting pro-inflammatory cytokine production and related inflammatory diseases. MK2 inhibitor IV is a highly selective, non-ATP competitive MK2 inhibitor with an IC_{50} value of 0.11 µM.[1] MK2 inhibitor IV has been shown to inhibit pro-inflammatory cytokine secretion from the human THP-1 acute monocytic leukemia cell line, causing inhibition of LPS-stimulated TNF-α (IC_{50} = 4.4 µM) and IL-6 (IC_{50} = 5.2 µM) secretion.[1] It can also inhibit IL-1β-stimulated matrixmetalloprotease 13 secretion from the SW1353 chondrosarcoma cell line (IC_{50} = 5.7 µM) and human primary chondrocyte cultures (IC_{50} = 2.2 µM).[1]

Reference

1. Huang, X., Shipps, G.W., Jr., Cheng, C.C., *et al.* Discovery and hit-to-lead optimization of non-ATP competitive MK2 (MAPKAPK2) inhibitors. *ACS Med. Chem. Lett.* **2(8)**, 632-637 (2011).

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