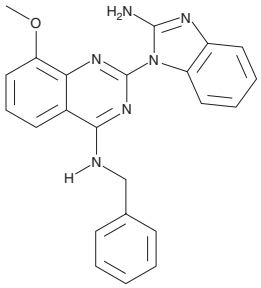
PRODUCT INFORMATION

ML-240

Item No. 17373



CAS Registry No.: 1346527-98-7
Formal Name: 2-(2-amino-1H-benzimidazol-1-yl)-8-methoxy-N-(phenylmethyl)-4-quinazolinamine
MF: $C_{23}H_{20}N_6O$
FW: 396.4
Purity: ≥95%
UV/Vis.: λ_{max}: 254, 304 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-240 is supplied as a crystalline solid. A stock solution may be made by dissolving the ML-240 in the solvent of choice, which should be purged with an inert gas. ML-240 is soluble in organic solvents such as DMSO and dimethyl formamide. The solubility of ML-240 in these solvents is approximately 1 mg/ml.

ML-240 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, ML-240 should first be dissolved in DMSO and then diluted with the aqueous buffer of choice. ML-240 has a solubility of approximately 0.25 mg/ml in a 1:3 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-240 is an ATP-competitive inhibitor of the D2 domain of the ATPase p97 (IC_{50} = 0.11 µM; K_i = 0.22 µM).[1,2] It disrupts the endoplasmic reticulum-associated degradation (ERAD) and autophagy pathways, preventing the degradation of p97-dependent proteasome substrates (IC_{50} = 0.9 µM) and causing accumulation of ubiquitin conjugates in nuclear membrane and cytosolic compartments at 5-10 µM.[1] ML-240 has also been shown to block proliferation of HCT15 and SW403 colon cancer cell lines (GI_{50}s = 0.76 and 0.5 µM, respectively) and to rapidly mobilize caspase-3 and -7, inducing apoptosis.[1]

References

1. Chou, T.-F., Li, K., Frankowski, K.J., *et al.* Structure-activity relationship study reveals ML240 and ML241 as potent and selective inhibitors of p97 ATPase. *ChemMedChem.* **8(2)**, 297-312 (2013).
2. Chou, T.-F., Bulfer, S.L., Weihl, C.C., *et al.* Specific inhibition of p97/VCP ATPase and kinetic analysis demonstrate interaction between D1 and D2 ATPase domains. *J. Mol. Biol.* **426(15)**, 2886-2899 (2014).

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