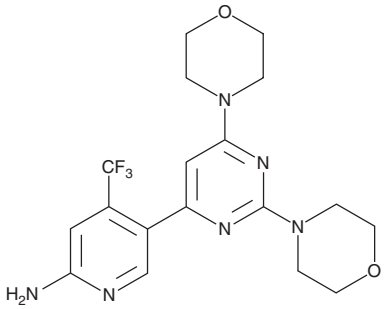
PRODUCT INFORMATION

NVP-BKM120

Item No. 11587

CAS Registry No.: 944396-07-0
Formal Name: 5-(2,6-di-4-morpholinyl-4-pyrimidinyl)-4-(trifluoromethyl)-2-pyridinamine
Synonym: Buparlisib
MF: $C_{18}H_{21}F_3N_6O_2$
FW: 410.4
Purity: ≥95%
UV/Vis.: 232, 254, 318 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

NVP-BKM120 is supplied as a crystalline solid. A stock solution may be made by dissolving the NVP-BKM120 in the solvent of choice. NVP-BKM120 is soluble in organic solvents such as ethanol, DMSO, and dimethyl formamide, which should be purged with an inert gas. The solubility of NVP-BKM120 in these solvents is approximately 17, 14, and 12 mg/ml, respectively.

NVP-BKM120 is sparingly soluble in aqueous buffers. For maximum solubility in aqueous buffers, NVP-BKM120 should first be dissolved in ethanol and then diluted with the aqueous buffer of choice. NVP-BKM120 has a solubility of approximately 0.5 mg/ml in a 1:1 solution of ethanol:PBS (pH 7.2) using this method. We do not recommend storing the aqueous solution for more than one day.

Description

NVP-BKM120 is an orally bioavailable inhibitor of the class I PI3K isoforms p110α (IC_{50} = 52 nM) and p110β (IC_{50} = 166 nM).[1] It is selective for these isoforms over the class III PI3K Vps34 (IC_{50} = 2,410 nM), the mammalian target of rapamycin (mTOR; IC_{50} = 2,866 nM), PI4Kβ (IC_{50} = >25,000 nM), and a variety of kinases (IC_{50}s = >10,000 nM). NVP-BKM120 inhibits proliferation of human tumor and glioma cell lines, with p53 wild-type glioma cells being more sensitive than p53 mutant/deleted glioma cells (IC_{50}s = 1.28 and 2.08 µM, respectively).[2,3] It halts the cell cycle in the G_2/M phase in both p53 wild-type and p53 mutant/deleted glioma cells, but p53 mutant/deleted cells reenter the cell cycle, progress into mitosis, and die *via* mitotic catastrophic cell death. NVP-BKM120 (1-5 mg/kg) crosses the blood brain barrier and selectively decreases phosphorylation of the PI3K target protein Akt.[4] It increases survival in a U87 glioma mouse xenograft intracranial tumor model when administered orally at doses of 20 and 40 mg/kg once per week.[2]

References

1. Maira, S.-M., Pecchi, S., Huang, A., *et al. Mol. Cancer Ther.* **11(2)**, 317-328 (2012).
2. Koul, D., Fu, J., Shen, R., *et al. Clin. Cancer Res.* **18(1)**, 184-195 (2012).
3. Park, E., Park, J., Han, S.-W., *et al. Int. J. Oncol.* **40(4)**, 1259-1266 (2012).
4. de Gooijer, M.C., Zhang, P., Buil, L.C.M., *et al. Sci. Rep*. **8(1)**, 10784 (2018).

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