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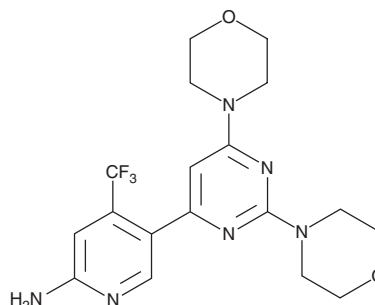
PRODUCT INFORMATION



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: C₁₈H₂₁F₃N₆O₂
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 (IC₅₀ = 52 nM) and p110β (IC₅₀ = 166 nM).¹ It is selective for these isoforms over the class III PI3K Vps34 (IC₅₀ = 2,410 nM), the mammalian target of rapamycin (mTOR; IC₅₀ = 2,866 nM), PI4Kβ (IC₅₀ = >25,000 nM), and a variety of kinases (IC₅₀s = >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 (IC₅₀s = 1.28 and 2.08 μM, respectively).^{2,3} It halts the cell cycle in the G₂/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).

WARNING

THIS PRODUCT IS FOR RESEARCH ONLY - NOT FOR HUMAN OR VETERINARY DIAGNOSTIC OR THERAPEUTIC USE.

SAFETY DATA

This material should be considered hazardous until further information becomes available. Do not ingest, inhale, get in eyes, on skin, or on clothing. Wash thoroughly after handling. Before use, the user must review the [complete](#) Safety Data Sheet, which has been sent via email to your institution.

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