

## Product Datasheet

# Buparlisib

Catalog No. **BP-02464** · CAS **944396-07-0** · Form **free base** · Physical state **solid** · Color **White to off-white** · Version **1.0** · Revision **2026-09-10**

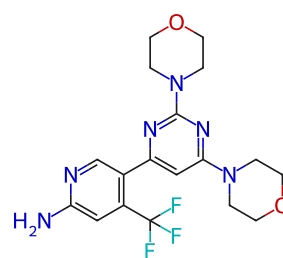
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|                                  |                                |
|----------------------------------|--------------------------------|
| Target<br><b>PI3K; Apoptosis</b> | Research area<br><b>Cancer</b> |
|----------------------------------|--------------------------------|

Molecular formula **C<sub>18</sub>H<sub>21</sub>F<sub>3</sub>N<sub>6</sub>O<sub>2</sub>**

Molecular weight **410.3935**

### STRUCTURE



## DESCRIPTION

Buparlisib (BKM120; NVP-BKM120) is a pan-class I PI3K inhibitor, with blood-brain barrier permeability. Buparlisib has IC50s of 52, 166, 116 and 262 nM for p110α, p110β, p110δ and p110γ, respectively .

## BIOLOGICAL ACTIVITY

### In Vitro

Buparlisib (NVP-BKM120) exhibits activity of 50–300 nM against class I PI3K (including the most common p110α mutant). In addition, NVP-BKM120 shows lower potency against class III and IV PI3K, with biochemical activities of 2, 5, >5 and >25 μM observed, respectively, for the inhibition of VPS34, mTOR, DNAPK and PI4K[1].

Buparlisib (≥10 μM, 24 h) induces apoptosis in multiple myeloma (MM) cells in a dose- and time-dependent manner[1].

Buparlisib (10 μM, 24 h) causes dose-dependent growth inhibition in all tested MM cell lines, with IC50 values between 1 and 10 μM for ARP-1, ARK, and MM.1R, while the IC50 value for MM.1S was <1 μM and for U266 it was between 10 and 100 μM[2].

### In Vivo

In A2780 xenograft tumors, oral dosing of Buparlisib (NVP-BKM120) at 3, 10, 30, 60, and 100 mg/kg results in a dose dependent modulation of pAKTSer473. Partial inhibition of pAKTSer473 is observed at 3 and 10 mg/kg, and near complete inhibition is observed at doses of 30, 60, or 100 mg/kg, respectively. Inhibition of pAKT (normalized to total AKT) tracked well with both plasma and tumor drug exposure[1].

Mice receiving Buparlisib (NVP-BKM120) (5 μM per kg per day for 15 days) treatment has significantly smaller tumor burdens as compare with control mice, which are measured as tumor volume (P<0.05) and level of circulating human kappa chain. In addition, NVP-BKM120 treatment significantly prolongs the survival of tumor-bearing mice

( $P < 0.05$ )[2].

Buparlisib has an excellent brain penetration that is unaffected by efflux transporters at the blood-brain barrier[5].

## SOLUBILITY

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Soluble in DMSO/H<sub>2</sub>O

## REFERENCES

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- [1].Burger MT, et al. Identification of NVP-BKM120 as a Potent, Selective, Orally Bioavailable Class I PI3 Kinase Inhibitor for Treating Cancer. *ACS Med Chem Lett.* 2011 Aug 26;2(10):774-9.
- [2].Zheng Y, et al. Novel phosphatidylinositol 3-kinase inhibitor NVP-BKM120 induces apoptosis in myeloma cells and shows synergistic anti-myeloma activity. *J Mol Med (Berl).* 2012 Jun;90(6):695-706.
- [3]. Ni J, et al. Combination inhibition of PI3K and mTORC1 yields durable remissions in mice bearing orthotopic patient-derived xenografts of HER2-positive breast cancer brain metastases. *Nat Med.* 2016 Jul;22(7):723-6.
- [4].Liu H, et al. Identifying and Targeting Sporadic Oncogenic Genetic Aberrations in Mouse Models of Triple Negative Breast Cancer. *Cancer Discov.* 2018 Mar;8(3):354-369.
- [5]. de Gooijer MC, et al. Buparlisib is a brain penetrable pan-PI3K inhibitor. *Sci Rep.* 2018 Jul 17;8(1):10784.

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