Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

NVP-BGT226

Catalog No: tcsc0583

Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg

Specifications

CAS No:
1245537-68-1

Formula:
$C_{32}H_{29}F_3N_6O_6$

Pathway:
PI3K/Akt/mTOR;Autophagy

Target:
PI3K;Autophagy

Purity / Grade:
>98%

Solubility:
DMSO : 103.3 mg/mL (158.78 mM; Need ultrasonic)

Observed Molecular Weight:
650.6

Product Description

NVP-BGT226 is a potent pan-class I **PI3K** and mTOR catalytic inhibitor with **IC_{50}**s of 4 nM, 63 nM and 38 nM for PI3Kα, PI3Kβ and

Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

PI3Kγ.

IC50 & Target: IC50: 4 nM (PI3Kα), 63 nM (PI3Kβ), 38 nM (PI3Kγ)[1]

In Vitro: NVP-BGT226 (BGT226) specifically inhibits p110α, p110β, p110δ, and p110γ, with a preference for the α-isoform (wild type and mutated), and mTOR, with no significant inhibitory activity against other tested kinases. The IC_{50} of NVP-BGT226 for the individual isoforms of the class I PI3Ks is (±standard deviation) PI3Kα 4 nM (±1), PI3Kβ 63 nM (±10), and PI3Kγ 38 nM (±23) as determined by filter-binding assay. In preclinical studies, NVP-BGT226 inhibits cancer cell proliferation with IC_{50} values in the low nanomolar range and inhibits solid tumor growth in various mouse xenograft models, including glioblastoma multiforme, breast, and prostate cancer[1]. To evaluate the antiproliferative activity of NVP-BGT226 (BGT226) against head and neck cancer cells, 9 HNSCC cell lines from different sites of the oral cavity and 1 NPC cell line with its cisplatin-resistant variant are used. NVP-BGT226 displays potent growth-inhibitory activity against all tested cell lines, with the IC_{50} ranging from 7.4 to 30.1 nM. Notably, both Detroit 562 and HONE-1 cells, which express *PIK3CA* mutation H1047R, are still sensitive to the growth-inhibitory effect of NVP-BGT226 treatment. In addition, the sensitivity to NVP-BGT226 between HONE-1 cells and its cisplatin-resistant variant is almost identical[2].

In Vivo: NVP-BGT226 (BGT226) is rapidly absorbed after single-dose oral administration, with median peak plasma concentrations (C_{max}) observed between 1.0 and 4.6 h post dose (T_{max}). Interpatient variability in C_{max} and area under the plasma concentration time curve (AUC_{0-48}) is relatively high and ranged (CV%) from 50% to 100% and from 30% to 200%, respectively, for most cohorts. Systemic exposure to BGT226 increased with increasing dose. It is, however, generally lower than expected based on preclinical data, and this finding is also supported by lower than expected C_{max} values, particularly at the 100 mg/day dose. The median terminal elimination half-life ranges from 6 to 9 h after the first dose[1]. Whether oral administration of NVP-BGT226 (BGT226) is effective is determined in vivo. NVP-BGT226 inhibits tumor growth in a dose-dependent manner in a FaDu cell xenografted mouse model. Oral administration of NVP-BGT226 at 2.5 and 5 mg/kg for 3 weeks caused 34.7% and 76.1% reduction of the tumor growth on day 21, respectively (compared with control). NVP-BGT226 causes a diminishment of the cytoplasmic immunoreactivity of p-p70S6K in a dose-dependent manner. Furthermore, results of the electronic microscopic analysis shows the formation of autophagosome as indicated by the formation of double membrane vacuoles and internal debris in mice treated with NVP-BGT226[2].