**Product Name**: BP-1-102  
**Catalog Number**: T3708  
**CAS Number**: 1334493-07-0  
**Molecular Formula**: C29H27F5N2O6S  
**Molecular Weight**: 626.15

**Description**: BP-1-102 is an orally active, effective and specific STAT3 inhibitor. BP-1-102 binds Stat3 (Kd: 504 nM), then blocks Stat3-phosphotyrosine (pTyr) peptide interactions and Stat3 activation (4-6.8 μM), and selectively inhibits migration, survival, growth, and invasion of Stat3-dependent tumor cells. The BP-1-102-mediated inhibition of aberrantly active Stat3 in tumor cells suppresses the expression of c-Myc, Bcl-xL, Cyclin D1, Survivin, and VEGF.

**Storage**: 2 years -80°C in solvent; 3 years -20°C powder;

<table>
<thead>
<tr>
<th>Solubility</th>
<th>DMSO</th>
<th>93 mg/mL (148.5 mM)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Ethanol</td>
<td>&lt;1 mg/mL</td>
</tr>
<tr>
<td></td>
<td>Water</td>
<td>&lt;1 mg/mL</td>
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</table>

( < 1 mg/ml refers to the product slightly soluble or insoluble )

| Receptor (IC50) | STAT3      | 504nM(Kd) |

**In vitro Activity**
BP-1-102 binds Stat3 with an affinity (KD) of 504 nM, blocks Stat3-phospho-tyrosine (pTyr) peptide interactions and Stat3 activation at 4-6.8 μM, and selectively inhibits growth, survival, migration, and invasion of Stat3-dependent tumor cells. BP-1-102-mediated inhibition of aberrantly active Stat3 in tumor cells suppresses the expression of c-Myc, Cyclin D1, Bcl-xL, Survivin, VEGF, and Krüppel-like factor 8, which is identified as a Stat3 target gene that promotes Stat3-mediated breast tumor cell migration and invasion. Treatment of breast cancer cells with BP-1-102 further blocks Stat3–NF-κB cross-talk, the release of granulocyte colony-stimulating factor, soluble intercellular adhesion molecule 1, macrophage migration-inhibitory factor/glycosylation-inhibiting factor, interleukin 1 receptor antagonist, and serine protease inhibitor protein 1, and the phosphorylation of focal adhesion kinase and paxillin, while enhancing E-cadherin expression. BP-1-102 inhibits Stat3 DNA-binding activity in vitro, with an IC50 value of 6.8±0.8 μM and preferentially inhibits Stat3-Stat3, over Stat1-Stat3, Stat1-Stat1, or Stat5-Stat5 DNA-binding activity. BP-1-102 has little or no effect on phospho-Shc, Src, Jak-1/2, Erk1/2, or Akt levels[1].

**In vivo Activity**
Intravenous or oral gavage delivery of BP-1-102 furnishes micromolar or microgram levels in tumor tissues and inhibits growth of human breast and lung tumor xenografts and modulates Stat3 activity, Stat3 target genes, and soluble factors in vivo. BP-1-102 selectively suppresses growth, survival, malignant transformation, migration, and invasion of malignant cells harboring constitutively active stat3. BP-1-102 is detectable at micromolar concentrations in plasma and in micrograms in tumor tissues[1].

**Cell Assay**
Proliferating cells in 6- or 96-well plates are treated once with 0–30 μM BP-1-102 for 24 h or with 10 μM BP-1-102 for up to 96 h. Viable cells are counted by trypan blue exclusion/phase-contrast microscopy or assessed by a CyQUANT Cell Proliferation Kit. (Only for Reference)

Cell line: Cultured MDA-MB-231, DU145, Panc-1, and NIH 3T3/v-Src cells harboring aberrantly active Stat3 and NIH 3T3, NIH 3T3/v-Ras, mouse thymus stromal epithelial cells, TE-71, Stat3-null mouse embryonic fibroblasts (Stat3−/− MEFs), cisplatin-sensitive ovarian cancer cells, A2780S cells

**Animal Experiment**
Animal Model: Athymic nude mice

**Reference**

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