

# GNF-2

Catalog No: tcsc0012



## Available Sizes

**Size:** 5mg

**Size:** 10mg

**Size:** 50mg



## Specifications

**CAS No:**

778270-11-4

**Formula:**

$C_{18}H_{13}F_3N_4O_2$

**Pathway:**

Protein Tyrosine Kinase/RTK

**Target:**

Bcr-Abl

**Purity / Grade:**

>98%

**Solubility:**

DMSO :  $\geq 45$  mg/mL (120.22 mM)

**Observed Molecular Weight:**

374.32

## Product Description

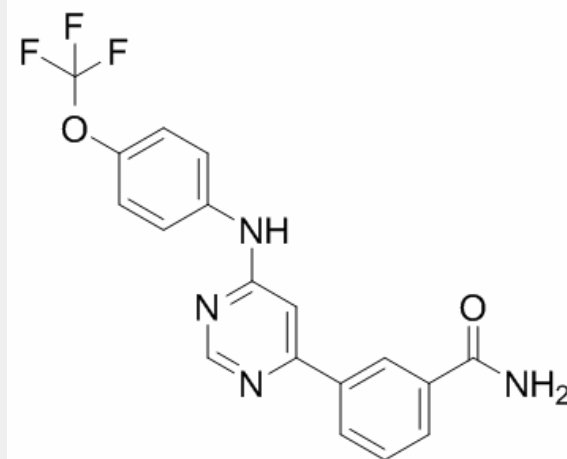
GNF-2 is a highly selective non-ATP competitive inhibitor of oncogenic Bcr-Abl activity (IC<sub>50</sub> = 0.14  $\mu$ M).

IC<sub>50</sub> value: 0.14  $\mu$ M [1]

Target: Bcr-Abl

in vitro: Ba/F3 cells harboring native or T315I mutated Bcr-Abl constructs were treated with GNF-2 and AKIs. We monitored the effect of GNF-2 with AKIs on the proliferation and clonogenicity of the different Ba/F3 cells. In addition, we monitored the auto-phosphorylation activity of Bcr-Abl and JAK2 in cells treated with GNF-2 and AKIs [2]. GNF-2 increased the effects of AKIs on unmutated BCR/ABL. Interestingly, the combination of Dasatinib and GNF-2 overcame resistance of BCR/ABL-T315I in all models used in a synergistic manner [3]. GNF-2 dose-dependently inhibited the proliferation of osteoclast precursors through the suppression of the M-CSFR c-Fms. In addition, GNF-2 accelerated osteoclast apoptosis by inducing caspase-3 and Bim expression. Furthermore, GNF-2 interfered with actin cytoskeletal organization and subsequently blocked the bone-resorbing activity of mature osteoclasts [4].

in vivo: Combining PDMP and GNF-2 eliminated transplanted-CML-T315I-mutants in vivo and dose dependently sensitized primary cells from CML T315I patients to GNF-2-induced proliferation inhibition and apoptosis[5].



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