# c-Met-IN-22

**Cat. No.:** HY-157387

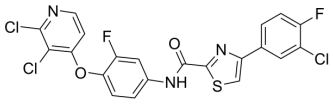
**Molecular Formula:** $C_{21}H_{10}Cl_3F_2N_3O_2S$

**Molecular Weight:** 512.74

**Target:** c-Met/HGFR; Apoptosis

**Pathway:** Protein Tyrosine Kinase/RTK; Apoptosis

**Storage:** Please store the product under the recommended conditions in the Certificate of Analysis.



## BIOLOGICAL ACTIVITY

**Description**

c-Met-IN-22 (compound 51am) is an orally active inhibitor against c-Met with an $IC_{50}$ value of 2.54 nM. c-Met-IN-22 has antiproliferative and antitumor activities. c-Met-IN-22 induces cell apoptosis[1].

**$IC_{50}$ & Target**

| | | | |
|---|---|---|---|
| c-Met$^{wild\ type}$<br>2.54 nM ($IC_{50}$) | c-Met$^{H1094R}$<br>93.6 nM ($IC_{50}$) | c-Met$^{D1228H}$<br>29.4 nM ($IC_{50}$) | c-Met$^{Y1230H}$<br>45.8 nM ($IC_{50}$) |
| c-Met$^{Y1235D}$<br>54.2 nM ($IC_{50}$) | c-Met$^{M1250T}$<br>26.5 nM ($IC_{50}$) | c-Met$^{kit}$<br>4.94 nM ($IC_{50}$) | c-Met$^{Ron}$<br>3.83 nM ($IC_{50}$) |
| c-Met$^{PDGFR\alpha}$<br>425 nM ($IC_{50}$) | c-Met$^{PDGFR\beta}$<br>513 nM ($IC_{50}$) | c-Met$^{VEGFR-2}$<br>527 nM ($IC_{50}$) | c-Met$^{Fit-3}$<br>6.12 nM ($IC_{50}$) |
| c-Met$^{Fit-4}$<br>276 nM ($IC_{50}$) | | | |

**In Vitro**

c-Met-IN-22 shows antiproliferative activity against MKN-45, A-549, HT-29, MDA-MB-231, HUVEC and FHC with $IC_{50}$s of 0.092, 0.83, 0.68, 3.94,2.54 and 8.63 μM, respectively[1].

c-Met-IN-22 shows anti-drug resistence against tH1094R, D1228H, Y1230H, Y1235D, and M1250T, with $IC_{50}$ values of 93.6, 29.4, 45.8, 54.2 and 26.5 nM,respectively[1].

c-Met-IN-22 (0, 2.5, 5.0, and 10.0 μM, 24 h) inhibits c-Met phosphorylation in MKN-45 in a dose-dependent manner[1] .

c-Met-IN-22 (0.4, 0.8, and 1.2 μM, 24 h) induces cell cycle arrest at G2 phase and apotosis of MNK-45 in a dose-dependent manner[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Apoptosis Analysis[1]

| | |
|---|---|
| Cell Line: | MNK- 45 cells |
| Concentration: | 0.4, 0.8, 1.2 μM |
| Incubation Time: | 24 h |
| Result: | Induced cell apoptosis in a dose-dependent manner. |

Cell Cycle Analysis[1]

| | |
|---|---|
| Cell Line: | MNK- 45 cells |
| Concentration: | 0.4, 0.8, 1.2 μM |
| Incubation Time: | 24 h |
| Result: | Induced cell cycle arrest at G2 phase in a dose-dependent manner. |

Western Blot Analysis[1]

| | |
|---|---|
| Cell Line: | MNK- 45 cells |
| Concentration: | 0, 2.5, 5, 10 μM |
| Incubation Time: | 24 h |
| Result: | Inhibited c-Met phosphorylation in a dose-dependent manner. |

**In Vivo**

c-Met-IN-22 (10mg/kg;p.o.;single dose) exhibits good oral bioavailability (F=69%) in BALB/c mice, with elimination half-time of 5.6h and clearance of 0.87 L/h•kg[1].
c-Met-IN-22 (1.5mg/kg;i.v.) exhibits an elimination half-time of 3.2h[1].

Pharmacokinetic Analysis of c-Met-IN-22 in BALB/c mice[1]

| Route | Dose (mg/kg) | $AUC_{0\to\infty}$ (μg·h/mL) | $T_{1/2}$ (h) | $T_{max}$ (h) | $C_{max}$ (ng/mL) | Cl (L/h·kg) | F (%) |
|---|---|---|---|---|---|---|---|
| i.v. | 1.5mg/kg | 2.5 | 3.2 | / | 552 | 0.6 | / |
| p.o. | 10mg/kg | 11.5 | 5.6 | 4.1 | 1756 | / | 69 |

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

| | |
|---|---|
| Animal Model: | Pharmacokinetic Analysis in BALB/c mice |
| Dosage: | p.o.10 mg/kg; i.v. 1.5 mg/kg |
| Administration: | Intravenous (i.v.) injection, Oral administration (p.o.) |
| Result: | Characterised good maximum concentration, plasma exposure and elimination half-time in pharmacokinetics. |

## REFERENCES

[1]. Xiang Nan,et al. Design, synthesis, and biological evaluation of thiazole/thiadiazole carboxamide scaffold-based derivatives as potential c-Met kinase inhibitors for cancer treatment. J Enzyme Inhib Med Chem. 2023 Dec;38



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