# LSD1-IN-25

**Cat. No.:** HY-149968

**CAS No.:** 2911585-60-7

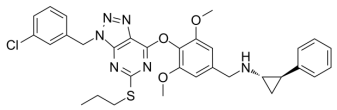
**Molecular Formula:** $C_{32}H_{33}ClN_6O_3S$

**Molecular Weight:** 617.16

**Target:** Histone Demethylase; Apoptosis

**Pathway:** Epigenetics; Apoptosis

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



## BIOLOGICAL ACTIVITY

**Description**

LSD1-IN-25 (Compound 9j) is a potent, selective and orally active $LSD_1$ inhibitor with an $IC_{50}$ of 46 nM ($K_i$ = 30.3 nM). LSD1-IN-25 induces cancer cell apoptosis[1].

| $IC_{50}$ & Target | | |
|---|---|---|
| | LSD1<br>46 nM ($IC_{50}$) | LSD1<br>30.3 nM (Ki) |

**In Vitro**

LSD1-IN-25 (Compound 9j; 0-20 μM; 72 h) inhibits solid tumor cell proliferation[1].

LSD1-IN-25 (1-4 μM; 24 h) induces the elevation of cellular H3K4me2 and inhibits the EMT process of H1650 cells[1].

LSD1-IN-25 (1-4 μM; 24 h) induces apoptosis and S arrest in H1650 cells[1].

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

Cell Proliferation Assay[1]

| | |
|---|---|
| Cell Line: | MGC-803, MGC-803$^{LSD1-KO}$, SGC-7901, GES-1, MCF-7, H1650, A549, H460, PC-3 and EC-109 |
| Concentration: | 0-20 μM |
| Incubation Time: | 72 h |
| Result: | Antiproliferative activity of LSD1-IN-25 (Compound 9j) on solid tumor cell lines[1] |

| Cell line | Origin | $IC_{50}$ (μM) |
|---|---|---|
| MGC-803 | Gastric cancer | 5.1 ± 0.7 |
| MGC-803$^{LSD1-KO}$ | Gastric cancer | >20 |
| SGC-7901 | Gastric cancer | 9.5 ± 2.1 |
| GES-1 | Normal gastric epithelial cell | >20 |

| | | |
|---|---|---|
| MCF-7 | Breast cancer | 13.4 ± 2.4 |
| H1650 | Lung cancer | 4.3 ± 0.9 |
| A549 | Lung cancer | 7.9 ± 1.5 |
| H460 | Lung cancer | 13.6 ± 4.2 |
| PC-3 | Prostate cancer | 7.5 ± 1.1 |
| EC-109 | Esophageal cancer | 15.2 ± 2.9 |

Western Blot Analysis[1]

| | |
|---|---|
| Cell Line: | H1650 cell |
| Concentration: | 1, 2 and 4 μM |
| Incubation Time: | 24 h |
| Result: | Induced the elevation of cellular H3K4me2. Elevated the expression of the epithelial marker E-cadherin, decreased the expression of the mesenchymal markers such as N-cadherin, slug and vimentin. |

Apoptosis Analysis[1]

| | |
|---|---|
| Cell Line: | H1650 cell |
| Concentration: | 1, 2 and 4 μM |
| Incubation Time: | 24 h |
| Result: | Induced an evident increase in cell apoptosis, with the percentage of apoptotic cells of 43.9% (1 μM), 44.5% (2 μM) and 45.7% (4 μM), respectively, in comparison with 12.7% of the control. |

Cell Cycle Analysis[1]

| | |
|---|---|
| Cell Line: | H1650 cell |
| Concentration: | 1, 2 and 4 μM |
| Incubation Time: | 24 h |
| Result: | The percentage of cells in S phase were 29.97% 33.32%, 39.81%, 43.26% at concentrations of 0, 1 μM, 2 μM, 4 μM, respectively. |

**In Vivo**

LSD1-IN-25 (Compound 9j; 10 and 20 mg/kg; oral; once daily for 21 days) shows antitumor activity in a mouse xenograft model of H1650 cells[1].

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

| | |
|---|---|
| Animal Model: | Nude mice, xenograft model of H1650 cells[1] |
| Dosage: | 10 mg/kg and 20 mg/kg |
| Administration: | Oral, once daily for 21 days |
| Result: | Presented a remarkable reduction of average tumor weight by 41.5% and 64.0% at dosages of 10 and 20 mg/kg, respectively. Evidently prolonged the mice's survival. |

## REFERENCES

[1]. Li Z, et al. Design, synthesis and in vitro/in vivo anticancer activity of tranylcypromine-based triazolopyrimidine analogs as novel LSD1 inhibitors. Eur J Med Chem. 2023 May 5;253:115321.



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