## Product Datasheet

# LSD1-IN-24

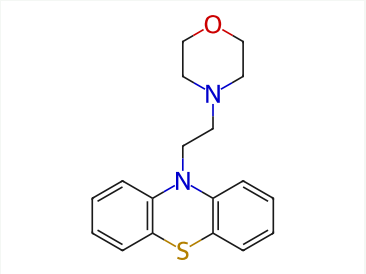
Catalog No. **BP-02423** · CAS **4734-59-2** · Form **free base** · Physical state **solid** · Color **Pale purple to purple** · Version **1.0** · Revision **2026-09-11**

**FOR RESEARCH USE ONLY. NOT FOR HUMAN OR VETERINARY USE.**

| Target | Research area |
|---|---|
| **Histone Demethylase; TLK; Apoptosis; PD-1/PD-L1** | **Cancer** |

| | |
|---|---|
| Molecular formula | $C_{18}H_{20}N_2OS$ |
| Molecular weight | **312.4290** |

**STRUCTURE**




## DESCRIPTION

LSD1/TLK1-IN-1 is an orally active LSD1, TLK1, TLK2, TTK inhibitor with an LSD1 IC50 of 0.247 μM. LSD1/TLK1-IN-1 suppresses phosphorylation of Nek1 at T141 and Rad9 at S328, abrogates the TLK1>Nek1>ATR>Chk1 axis, protects H3K4me1/2 from demethylation, and does not affect LSD2, MAO-A, or MAO-B. LSD1/TLK1-IN-1 induces apoptosis, bypasses cell-cycle arrest, suppresses tumor growth, downregulates PD-L1 expression, enhances T-cell killing response, inhibits gastric cancer cell proliferation. LSD1/TLK1-IN-1 can be used for the research of prostate cancer and gastric cancer .

## BIOLOGICAL ACTIVITY

In Vitro
LSD1/TLK1-IN-1 (compound J3-54) (20 μM; range; 30 minutes) potently inhibits recombinant human TLK1B kinase activity in vitro, with a Km of 31.31 μM in competitive ATP-binding assays.

LSD1/TLK1-IN-1 (6-20 μM; 72 h) induces weak dose-dependent proliferation inhibition in multiple prostate cancer cell lines, causes dose-dependent viability loss in LNCaP and TRAMP-C2 cells over 72 hours, and has no effect on normal RWPE1 prostate cells.

LSD1/TLK1-IN-1 (2-3 weeks) alone reduces colony formation in LNCaP, VCaP, and TRAMP-C2 androgen-sensitive prostate cancer cells, and causes a 4- to 5-fold greater suppression when combined with bicalutamide.

LSD1/TLK1-IN-1 (5-10 μM; 12-24 h) alone causes a modest S-phase reduction in LNCaP cells, and when combined with Bicalutamide (HY-14249), induces apoptosis and bypasses G1 cell cycle arrest in LNCaP, VCaP, and TRAMP-C2 cells, while suppressing TLK1B-mediated DNA damage response signaling.

LSD1/TLK1-IN-1 (compound 3S) potently inhibits recombinant LSD1 with an IC50 of 0.247 μM.

LSD1/TLK1-IN-1 (10 μM) shows high selectivity for LSD1, with less than 10% inhibitory activity against LSD2, MAO-A, and MAO-B at 10 μM.

LSD1/TLK1-IN-1 (0.5 μM; 6 h) directly binds to cellular LSD1 in BGC-823 gastric cancer cells when treated at 0.5 μM for 6 h, as demonstrated by enhanced LSD1 thermal stability in CETSA.

LSD1/TLK1-IN-1 (5-20 μM) dose-dependently suppresses PD-L1 expression via reducing PD-L1 mRNA transcription in an LSD1-dependent manner in BGC-823 and MFC gastric cancer cells when treated at 5, 10, 20 μM, with no effect on LSD1 KO cells.

LSD1/TLK1-IN-1 (5-20 μM) dose-dependently enhances T-cell killing response against BGC-823 gastric cancer cells via an LSD1- and PD-L1-dependent manner when treated at 5, 10, 20 μM, increasing IFNγ and TNFα secretion and reducing PD-1 binding.

LSD1/TLK1-IN-1 does not alter the proliferation of BGC-823 or MFC gastric cancer cells.
In Vivo
LSD1/TLK1-IN-1 (compound J3-54) (5 mg/kg; i.p.; biweekly) administered intraperitoneally at 5 mg/kg biweekly significantly suppresses LNCaP xenograft tumor growth and promotes apoptosis by inhibiting the TLK1B > pNek1 DNA damage response pathway, with enhanced efficacy when combined with bicalutamide.

LSD1/TLK1-IN-1 (compound 3S) (10-50 mg/kg; p.o.; daily; 14 days) dose-dependently inhibits gastric cancer tumor growth in immunocompetent mice, enhances intratumoral T-cell infiltration and activity, and reduces intratumoral PD-L1 expression without significant systemic toxicity.

## SOLUBILITY

Soluble in DMSO : 100 mg/mL

## REFERENCES

[1].Singh V, et al. Generation of Phenothiazine with Potent Anti-TLK1 Activity for Prostate Cancer Therapy. iScience. 2020;23(9):101474. Published 2020 Aug 20.

[2].Dai XJ, et al. Phenothiazine-Based LSD1 Inhibitor Promotes T-Cell Killing Response of Gastric Cancer Cells. J Med Chem. 2023;66(6):3896-3916.