

Product Data Sheet

Product Name: CXJ2080

Cat. No.: GC80884

Chemical Properties

Cas. No.

Formula $C_{51}H_{68}ClN_{11}O_6S_2$

M.Wt 1030.74

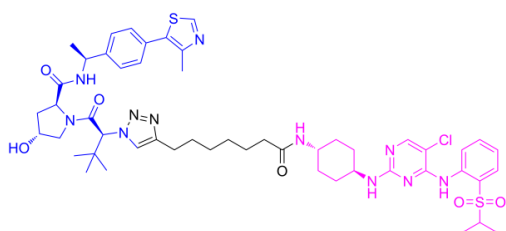
Solubility

Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37 °C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution : ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Background

CXJ2080 is a selective PROTAC-based CDK7 degrader with a DC_{50} of 0.88 nM. CXJ2080 recruits VHL E3 ligase to induce ubiquitin-proteasome-dependent CDK7 degradation, disrupts the CDK7-cyclin H-MAT1 complex, suppresses CDK7-dependent phosphorylation of RNA polymerase II CTD Ser5, CDK1 Thr161, and CDK2 Thr160. CXJ2080 activates the p53 - p21 axis, suppresses MYC - driven signaling, induces leukemia cell cycle arrest, apoptosis, and differentiation, reduces CD117 expression, spares platelets and normal PBMCs, maintains sustained CDK7 degradation post-washout. CXJ2080 can be used for the research of acute leukemia ^[1]. (Pink: CDK7 ligand; Blue: VHL ligand; Black: linker).

Caution: Product has not been fully validated for medical applications. For research use only.

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In Vivo, CXJ2080 (20-50 mg/kg; i.v.; every other day; 2 weeks) administered at 50 mg/kg every other day via intravenous injection achieves potent tumor growth inhibition with selective CDK7 degradation in RS4;11 xenograft tumors, while sparing PBMCs^[1]. CXJ2080 (20-50 mg/kg; i.v.; every other day; 3 weeks) administered at 50 mg/kg every other day via intravenous injection achieves potent tumor growth inhibition with selective CDK7 degradation in MV4-11 xenograft tumors, while sparing PBMCs and maintaining normal platelet parameters^[1]. CXJ2080 (20 mg/kg; i.v.; every other day; 21 days) administered at 20 mg/kg every other day via intravenous injection significantly improves survival in mice with disseminated Molm13 acute myeloid leukemia^[1].

In Vitro, CXJ2080 (0.5-200 nM; 6 h) potently degrades CDK7 in MV4-11 acute myeloid leukemia cells with a DC₅₀ of 0.88 nM and >98% maximum degradation efficiency^[1]. CXJ2080 (72 h) potently inhibits the proliferation of RS4;11 acute lymphoblastic leukemia cells (IC₅₀ = 17.29 nM) and MV4-11 acute myeloid leukemia cells (IC₅₀ = 4.31 nM)^[1]. CXJ2080 (72 h) inhibits the proliferation of primary acute myeloid leukemia patient-derived cell samples^[1]. CXJ2080 (100 nM; 4 h pretreatment) induces sustained CDK7 degradation in MV4-11 acute myeloid leukemia cells, with suppression maintained for 48 h post-washout^[1]. CXJ2080 (5-100 nM; 8 h) induces dose-dependent CDK7 degradation, p53 accumulation, Myc downregulation, and a biphasic p21 protein response in MV4-11 acute myeloid leukemia cells^[1]. CXJ2080 (5-100 nM; 12 h) induces dose-dependent cell cycle arrest in MV4-11 acute myeloid leukemia cells, with increased SubG1 phase accumulation at higher concentrations^[1]. CXJ2080 (5-100 nM; 48 h) induces dose-dependent apoptosis in MV4-11 acute myeloid leukemia cells^[1]. CXJ2080 (5-100 nM; 24 h) significantly reduces CD117 (c-KIT) stemness marker expression in MV4-11 acute myeloid leukemia cells^[1].

References:

[1]. Tu Y, et al. Discovery of a selective CDK7 PROTAC against acute leukemia with low platelet toxicity. *Leukemia*. Published online March 25, 2026.

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