

Product Data Sheet

Product Name: CWI1-2
Cat. No.: GC68916

Chemical Properties

Cas. No. 2408590-36-1

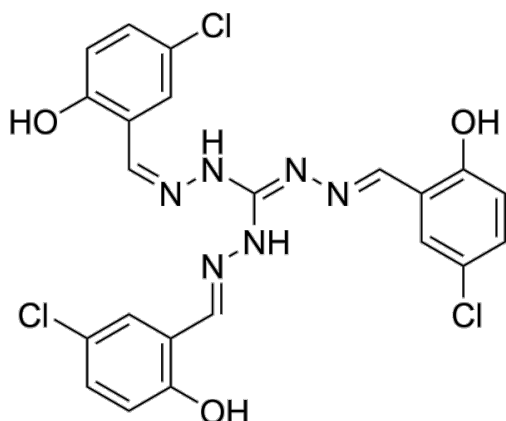
Formula $C_{22}H_{17}Cl_3N_6O_3$ M.Wt 519.77

Solubility DMSO : 100 mg/mL (192.39 mM; Need ultrasonic) 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



Protocol

Cell experiment [1]:

Cell lines THP1

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

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Preparation Method Cells were treated with CWI1-2 (0.1-1 μ M) for 48 h, cell viability was then evaluated by MTT assay. The medium was then changed with MTT solution (0.5mg/ml) in DMEM or RPMI without phenol red and FBS; cells were incubated at 37°C for 30 min and violet precipitate was dissolved with isopropanol. Absorbance at 550nm was measured through an EnSpire Multimode Plate reader.

Reaction Conditions 0.1 to 1 μ M, 48 hours

Applications Treatment of THP1 cells with CWI1-2 induced dramatic cell differentiation and apoptosis in a concentration-dependent manner

Animal experiment [2]:

Animal models B6.SJL (CD45.1) mice

Preparation Method B6.SJL (CD45.1) mice were performed bone marrow transplants (BMTs) using blast cells from MAP-induced leukemia mice or C1498 murine AML cells. Following the transplantation, CWI1-2 is administered at a dosage of 5 mg/kg daily via intravenous injection for a period of 7 to 10 days. Peripheral blood (PB) samples are then collected, and flow cytometric analysis is conducted to assess leukemia engraftment.

Dosage form 5 mg/kg/d, 7-10 days, i.v.

Applications CWI1-2 treatment significantly delayed leukemia onset and prolonged the survival of BMT recipient mice

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References:

[1]. Weng H, Huang F, Yu Z, et al. The m⁶A reader IGF2BP2 regulates glutamine metabolism and represents a therapeutic target in acute myeloid leukemia. *Cancer Cell*. 2022 Dec 12;40(12):1566-1582.e10. doi: 10.1016/j.ccell.2022.10.004.

Background

CWI1-2 is a selective inhibitor of the m⁶A reader protein IGF2BP2, disrupting the interaction between IGF2BP2 and m⁶A-modified target transcripts [1] [2]. The IC₅₀ at the cellular level ranges from 203.1 to 781.6 nM [1]. This inhibitor is primarily used for research in acute myelocytic leukemia (AML) due to its promising anti-leukemic properties [1].

CWI1-2 exerts anti-leukemic effects by targeting IGF2BP2, inducing apoptosis and differentiation in leukemia cells. The IC₅₀ at the cellular level ranges from 203.1 to 781.6 nM [1]. In AML cell lines, CWI1-2 at concentrations of 0.1 to 1 μM over 48h inhibits cell growth and induces apoptosis [3].

CWI1-2 (5 mg/kg, intravenous injection, daily for 7-10 days) significantly reduced leukemia progression and prolonged survival in BMT recipient mice. Treatment results in a marked reduction in leukemia cell burden without significant changes in body weight [3].

References:

[1]. Zhu TY, Hong LL, Ling ZQ. Oncofetal protein IGF2BPs in human cancer: functions, mechanisms and therapeutic potential. *Biomark Res*. 2023 Jun 6;11(1):62. doi: 10.1186/s40364-023-00499-0.

[2]. Wang M, Chao M, Han H, et al. Hinokiflavone resists HFD-induced obesity by promoting apoptosis in an IGF2BP2-mediated Bim m⁶A modification dependent manner. *J Biol Chem*. 2024 Sep;300(9):107721. doi: 10.1016/j.jbc.2024.107721. Epub 2024 Aug 29. PMID: 39214307; PMCID: PMC11465056.

[3]. Weng H, Huang F, Yu Z, et al. The m⁶A reader IGF2BP2 regulates glutamine metabolism and represents a therapeutic target in acute myeloid leukemia. *Cancer Cell*. 2022 Dec 12;40(12):1566-1582.e10. doi: 10.1016/j.ccell.2022.10.004.

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