# Product Data Sheet

Product Name: MI-2 (hydrochloride)
Cat. No.: GC10388

## Chemical Properties

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|---|---|---|---|
| Cas. No. | | | |
| Chemical Name | 4-(4-(5,5-dimethyl-4,5-dihydrothiazol-2-yl)piperazin-1-yl)-6-propylthieno[2,3-d]pyrimidine, dihydrochloride | | |
| SMILES | CCCC(S1)=CC(C1=NC=N2)=C2N3CCN(C4=NCC(C)(C)S4)CC3.Cl.Cl | | |
| Formula | $C_{18}H_{25}N_5S_2$ • 2HCl | M.Wt | 448.5 |
| Solubility | ≤30mg/ml in ethanol;10mg/ml in DMSO;20mg/ml in dimethyl formamide | Storage | Store at -20°C |
| General tips | For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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 | MI-2 (hydrochloride) | | |

## Background

MI-2 (Menin-MLL Inhibitor) is a potent menin-MLL interaction inhibitor with an IC50 value of 446 ± 28 nM.

Menin is an oncogenic cofactor in leukemic transformations which could bind to the N-terminal fragment of MLL existed in all MLL fusion proteins. Menin is a highly specific and direct binding partner of MLL and MLL fusion proteins which is essential for regulation of their target genes. Disruption of the menin-MLL protein interaction abrogates oncogenic properties of MLL fusion proteins and blocks the development of acute leukemia [2].

In vitro: In HEK293 cells, MI-2 accessed the protein target menin-MLL and effectively inhibited the menin-MLL-AF9 interaction. MI-2 effectively blocked cell proliferation, and induced cell apoptosis in human MLL leukemia cell lines harboring different MLL translocations MLL-AF9 and MLL-ENL, with the GI50 value of about 5 μM for MI-2. MI-2


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showed little effect on the cell growth of E2A-HLF transduced BMC with the GI50 of > 50 μM. MI-2 specifically reduced the immortalization potential of cells transformed with MLL fusion oncoproteins by downregulating the expression of target genes required for MLL fusion protein oncogenic activity [1].

In vivo: After 7 days treatment with MI-2, MLL-AF9 transformed BMC showed great morphology changes and the expression of CD11b was greatly increased [1].

References:
[1]. Grembecka J, He S, Shi A, et al. Menin-MLL inhibitors reverse oncogenic activity of MLL fusion proteins in leukemia[J]. Nature chemical biology, 2012, 8(3): 277-284.
[2]. Borkin D, He S, Miao H, et al. Pharmacologic inhibition of the Menin-MLL interaction blocks progression of MLL leukemia in vivo[J]. Cancer Cell, 2015, 27(4): 589-602.