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**Product Data Sheet**

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Product Name: MI-nc (hydrochloride)

Cat. No.: GC12173

**Chemical Properties**

Cas. No. 1934302-23-4

Chemical Name 6-ethyl-4-[4-(1,3,4-thiadiazol-2-yl)-1-piperaziny]-thieno[2,3-d]pyrimidine, dihydrochloride

SMILES CCC(S1)=CC(C1=NC=N2)=C2N3CCN(C4=NN=CS4)CC3.Cl.ClFormula  $C_{14}H_{16}N_6S_2 \cdot 2HCl$ 

M.Wt 405.4

Solubility  $\leq 0.1$ mg/ml in ethanol; 1.3mg/ml in DMSO; 0.5mg/ml in dimethyl formamideStorage Store at  $-20^{\circ}C$ General tips For obtaining a higher solubility , please warm the tube at  $37^{\circ}C$  and shake it in the ultrasonic bath for a while. Stock solution can be stored below  $-20^{\circ}C$  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**

MI-nc is a weak inhibitor of the menin-MLL fusion protein interaction with an  $IC_{50}$  of 193  $\mu M$ . It can be used as a negative control compound for tests involving MI-2, which showed more binding potency with menin, blocked the menin-MLL interaction with an  $IC_{50}$  of 0.45  $\mu M$ , and induced apoptosis in cells expressing MLL fusion proteins [1].

Menin, a product of the multiple endocrine neoplasia gene, is an essential co-factor of oncogenic MLL fusion proteins. MLL interacts with menin in a bivalent mode involving 2 N-terminal fragments of MLL, and the menin-MLL interaction is critical for development of acute leukemia. Inhibition of the menin interaction with MLL fusion proteins is a very promising strategy to reverse their oncogenic activity [2].

## 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.

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

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[2]. Yokoyama A, Somerville T C P, Smith K S, et al. The menin tumor suppressor protein is an essential oncogenic cofactor for MLL-associated leukemogenesis[J]. Cell, 2005, 123(2): 207-218.

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