
Product Data Sheet

Product Name: MYCMI-6
Cat. No.: GC60259

Chemical Properties

Cas. No. 681282-09-7

SMILES NC1=NC(N)=CC=C1/N=N/C2=CC3=NC4=CC=C(OCC)C=C4C(N)=C3C=C2

Formula C20H19N7O M.Wt 373.41

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

MYCMI-6 (NSC354961) is a potent and selective endogenous MYC:MAX protein interactions inhibitor. MYCMI-6 blocks MYC-driven transcription and binds selectively to the MYC bHLHZip domain with a K_d of 1.6 μM. MYCMI-6 inhibits tumor cell growth in a MYC-dependent manner (IC₅₀<0.5 μM). MYCMI-6 is not cytotoxic to normal human cells. MYCMI-6 induces apoptosis[1].

MYCMI-6 (NSC354961) (6.25 μM; 48 hours) selectively suppresses MYC-driven tumor cell growth with high efficacy[1]. MYCMI-6 significantly inhibits growth of Burkitt's lymphoma cells (Mutu, Daudi and ST486) - another classical example of a MYC-driven tumor, having translocations of MYC to one of the immunoglobulin loci - in a dose-dependent manner with an average GI₅₀ of 0.5 μM. Treatment of MCF7 cells with the MYCMI-6 for 24 hours significantly decreased MYC:MAX isPLA signals to 7%. Titration showed an IC₅₀ for inhibition of MYC:MAX of less than 1.5 μM for MYCMI-6 by isPLA. MYCMI-6 inhibits the MYC:MAX heterodimer formation with an IC₅₀ of 3.8 μM. MYCMI-6 efficiently inhibits anchorage-independent growth of MYCN-amplified neuroblastoma cells with GI₅₀ values of <0.4 μM[1]. Cell Viability Assay[1] Cell Line: MYCN-amplified neuroblastoma cells (IMR-32, Kelly and SK-N-DZ), MYCN-non-amplified neuroblastoma cells (SK-N-F1, SK-N-AS

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

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and SK-N-RA)

MYCMI-6 (20 mg/kg; i.p.; daily for 1-2 weeks) induces massive apoptosis and reduces tumor cell proliferation, tumor microvasculature density and MYC:MAX interaction in a MYC-dependent xenograft tumor model[1]. Animal Model: 6-8 weeks old athymic nude mice (bearing MYCN-amplified SK-N-DZ neuroblastoma cells)[1]

[1]. Castell A, et al. A selective high affinity MYC-binding compound inhibits MYC:MAX interaction and MYC-dependent tumor cell proliferation. Sci Rep. 2018 Jul 3;8(1):10064.

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