

GANT 58

Catalog No: tcsc0507



Available Sizes

Size: 5mg

Size: 10mg

Size: 50mg

Size: 100mg



Specifications

CAS No:

64048-12-0

Formula:

$C_{24}H_{16}N_4S$

Pathway:

Stem Cell/Wnt

Target:

Gli

Purity / Grade:

>98%

Solubility:

DMSO : 9.09 mg/mL (23.16 mM; Need ultrasonic)

Alternative Names:

NSC 75503

Observed Molecular Weight:

392.48

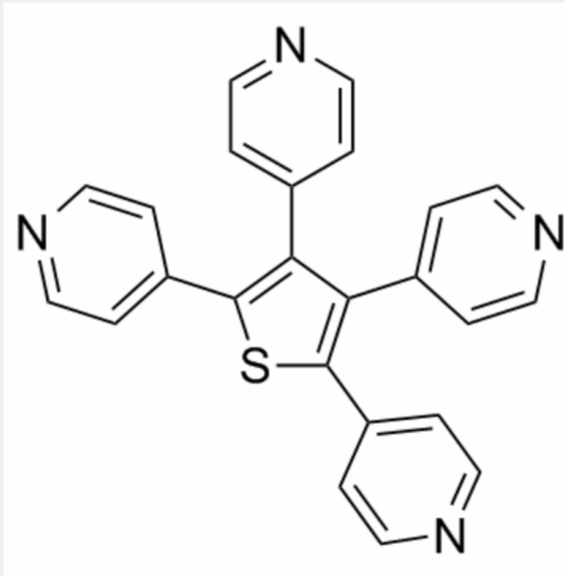
Product Description

GANT 58 is a potent **Gli** antagonist that inhibits GLI1-induced transcription with **IC₅₀** of 5 μ M.

IC50 & Target: IC50: 5 μ M (Gli)^[1]

In Vitro: GANT58 is a downstream inhibitor of Hh signaling. GANT58 is an indeed inhibitor of Hh signaling downstream of Smo and Sufu. GANT58 mainly acts at the nuclear level because transcription induced by GLI1 with a mutated nuclear export signal is still blocked. GANT58 efficiently inhibits in vitro tumor cell proliferation in a GLI-dependent manner and successfully blocks cell growth using human prostate cancer cells harboring downstream activation of the Hh pathway^[1]. GANT58 (NSC75503) has been shown to inhibit transcriptional activation by GLI1 (as well as by the other GLI species). GANT58 has been shown to inhibit GLI transactivation [2].

In Vivo: Nude mice are injected s.c. with GLI1-positive 22Rv1 prostate cancer cells, and tumors are established (median size \approx 250 mm³). Nude mice are treated with daily s.c. injections at a concentration of 50 mg/kg of cyclopamine, GANT61, GANT58, or solvent only (n=4-5 for each group). However, after 3 days, cyclopamine-treated animals presented with severe ulcerations at the injection sites. Therefore, changing the treatment regimen to injections only every second day. To be able to compare all compounds, this protocol is also introduced for the GANTs, although mice treated with these compounds showed no such signs of toxicity. All injections are done 2-3 cm away from the tumors. During an 18-day treatment period, suppression of tumor cell growth is observed for all compounds. Treatment with cyclopamine or GANT58 results in the inhibition of additional xenograft growth and limited the increase in tumor size^[1].



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