

Product Data Sheet

Product Name: Methylnitronitrosoguanidine(wetted with ca. 50% Water)

Cat. No.: GC36598

Chemical Properties

Cas. No. 70-25-7

SMILES N=C(N[N+])([O-])=O)N(C)N=O

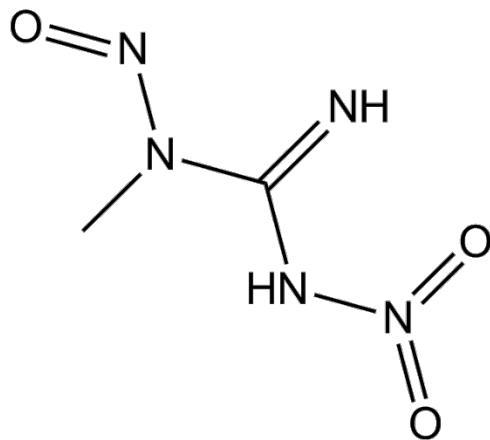
Formula C2H5N5O3 M.Wt 147.09

Solubility DMSO : 125 mg/mL (849.82 mM) Storage Store at -20°C,unstable in solution, ready to use.

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

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

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OC3 cells at passage 24 (OC3-P24) were treated with the carcinogen MethylNitronitrosoguanidine (MNNG, final concentration 3 µg/mL in medium) and protected from light. After 24 h, the media containing MNNG was removed and cells were rinsed 3 times with phosphate buffered saline (PBS). The process was repeated 15 times and cells were then cultured in regular medium and passaged Preparation Method when necessary. The induced cell line was designated as OC3W3-15, and it corresponds to regular cultured passage 38 of OC3 cells (OC3-P38) which served as control. Cell growth and morphology of both cell lines were regularly monitored with an inverted microscope. The ultrastructural changes during transformation were observed under the H-600 transmission electronic microscope (TEM).

Reaction Conditions 3 µg/mL, 24 hours for 15 times

Applications After treatment with the carcinogen MethylNitronitrosoguanidine 15 times, the OC3W3-15 took on a long- or short-fusiform shape in a multilayer, overlapping and in a confused arrangement. The cells showed karyokinesis or even abnormal karyokinesis.

Animal experiment [2]:

Animal models Male Wistar albino rats

Control rats were assisted for group I for 20 weeks. Group II was stimulated oral gave administration of MNNG (200 mg/kg bw) at 0 and 14th days. 1 ml of NaCl for each rats was given trice a day for 6 weeks for saturation and kept till the Preparation Method experimental period. Group III were stimulated with MNNG + Corilagin (30 mg/kg bw) and treated for 20 weeks with effective dosage. Corilagin (30 mg/kg bw) alone was taken as group IV. The experiment was carried out for 20 weeks and all the animals were sacrificed by cardiac puncture.

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Dosage form Oral, 200 mg/kg, 2 weeks.

Applications In group II, the rat body weights were significantly decreased compared with normal group whereas, tumor weight was increased and also 100% gastric cancer incidence rats were also recorded. The rat body weight was increased and tumor weights significantly decreased in corilagin administered group compared to group II.

References:

- [1]: Ding L, Ding Y N, Lin Y, et al. Proteins related to early changes in carcinogenesis of hepatic oval cells after treatment with methylnitronitrosoguanidine[J]. Experimental and Toxicologic Pathology, 2014, 66(2-3): 139-146.
- [2]: Zhang L, Jia B, Velu P, et al. Corilagin induces apoptosis and inhibits HMBG1/PI3K/AKT signaling pathways in a rat model of gastric carcinogenesis induced by methylnitronitrosoguanidine[J]. Environmental Toxicology, 2022, 37(5): 1222-1230.

Background

Methylnitronitrosoguanidine (MNNG) is an orally active alkylating agent that shows toxic and mutagenic effects. Methylnitronitrosoguanidine is often used as a carcinogen and mutagen in vivo and in vitro [1,2]. The Methylnitronitrosoguanidine (MNNG) mechanism of action is owing to its electrophilic compound degradations, where ammonium ion is the mutagen and leads to the

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electrophilic impact on DNA base pairs, specifically on its nucleophilic sites [3].

Following treatment with Methylnitronitrosoguanidine (3 µg/mL), the ultrastructure of the OC3W3-15 cells possessed larger nuclei with enlarged and prominent nucleoli, usually more than 2 in number. The amount of heterochromatin decreased while euchromatin increased. The cytoplasm was filled with abundant rough endoplasmic reticulum, free ribosomes, Golgi apparatus, and well-developed mitochondria, yet the nuclear-to-cytoplasmic ratio remained high [2]. Methylnitronitrosoguanidine (3 µg/mL) treatment increased the proliferative rate of hepatic oval cells, and more cells were in prophase for rapid proliferation than in the control group [2]. 2×10^{-4} mol/L

Methylnitronitrosoguanidine could increase the proliferation, promote invasion and migration, and suppress the apoptosis of GES-1 cells. The expression levels of NF-κB p65 mRNA and protein were upregulated in GES-1 cells after treatment with MNNG [4].

Methylnitronitrosoguanidine (200 mg/kg, 2 weeks) treatment significantly decreased the Male Wistar albino rat's body weights, increased tumor weight, and gastric cancer was induced in 100% of the rats [1]. Methylnitronitrosoguanidine (200 mg/kg, 2 weeks) reduced protein levels of Bax, caspase-3, and -9 [1].

References:

- [1]. Zhang L, Jia B, Velu P, et al. Corilagin induces apoptosis and inhibits HMBG1/PI3K/AKT signaling pathways in a rat model of gastric carcinogenesis induced by methylnitronitrosoguanidine[J]. Environmental Toxicology, 2022, 37(5): 1222-1230.
- [2]. Ding L, Ding Y N, Lin Y, et al. Proteins related to early changes in carcinogenesis of hepatic oval cells after treatment with methylnitronitrosoguanidine[J]. Experimental and Toxicologic Pathology, 2014, 66(2-3): 139-146.
- [3]. Zhu, F., Xu, Y., Pan, J., Li, M., Chen, F., & Xie, G. (2021). Epigallocatechin gallate protects against MNNG-induced precancerous lesions of gastric carcinoma in rats via the PI3K/Akt/mTOR pathway. Evidence-Based Complementary and Alternative Medicine, 2021.
- [4]. XU J, ZHANG X, SUN D, et al. Effect of methylnitronitrosoguanidine (MNNG) on the malignant transformation of human gastric mucosa GES-1 cells and its mechanism[J]. Medical Journal of Chinese People's Liberation Army, 2016, 41(11): 887-891.

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