Mitomycin C

Cat. No.: HY-13316

CAS No.: 50-07-7

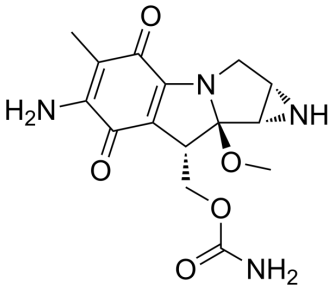
Molecular Formula: $C_{15}H_{18}N_4O_5$

Molecular Weight: 334.33

Target: DNA Alkylator/Crosslinker; DNA/RNA Synthesis; ADC Cytotoxin; Apoptosis; Bacterial; Antibiotic

Pathway: Cell Cycle/DNA Damage; Antibody-drug Conjugate/ADC Related; Apoptosis; Anti-infection

Storage: Please store the product under the recommended conditions in the Certificate of Analysis.



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (149.55 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.9911 mL	14.9553 mL	29.9106 mL
5 mM	0.5982 mL	2.9911 mL	5.9821 mL
10 mM	0.2991 mL	1.4955 mL	2.9911 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (6.22 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (6.22 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Mitomycin C (Ametycine) is a DNA cross-linking agent and induces DNA damaging. Mitomycin C is an antitumor agent and antibiotic that shows extraordinary ability to inhibit DNA synthesis. Mitomycin C is an ADC Cytotoxin and induces apoptosis [1][2][3].

IC_{50} & Target

Traditional Cytotoxic Agents

In Vitro

The HCT116 ($p53^{-/-}$) cells are minimally sensitive to either Mitomycin C (Ametycine) or TRAIL alone. However, surprisingly, combination treatment with MMC and TRAIL decreases cell viability significantly. Although Mitomycin C and TRAIL alone are moderately effective, Mitomycin C substantially enhances the effect of TRAIL on suppression of the cell proliferation.

	Mitomycin C and TRAIL treatment alone induces 9.5% and 35.0% apoptosis, respectively. However, combination treatment with Mitomycin C and TRAIL enhances apoptosis to 66.6%[1]. ?Mitomycin C is a cytotoxic chemotherapeutic agent that causes DNA damage in the form of DNA cross-links as well as a variety of DNA monoadducts and is known to induce p53[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Mice bearing xenografted HCT116 ($p53^{-/-}$) colon tumors and HT-29 colon tumors are treated with Mitomycin C (Ametycine; i.p., 1 mg/kg) and TRAIL (i.v., 100 μg) every other day. Animals are treated with 10 consecutive cycles of the combination therapy regimen. The combination therapy suppresses tumor growth significantly and does not impact the weight of the mice, indicating that the therapeutic combination of Mitomycin C and TRAIL is well-tolerated and has anti-tumor activity in vivo[1]. ?Intravesical Mitomycin C instillations has an effect on body weight. After 3 consecutive weekly instillations of 1 mg/mL Mitomycin C there is almost no weight gain, whereas rats in the other 3 groups has a statistically significant weight gain compared with MMC treated rats[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Colon adenocarcinoma HCT116 and HT-29 human colon cancer cells are used. The CellTiter-Glo Luminescent Cell Viability Assay is used to measure cell viability, which use a unique, stable form of luciferase to measure ATP as an indicator of viable cells, and the luminescent signal produced is proportional to the number of viable cells present in culture. Cells are pretreated with Mitomycin C (5 μM) for 12 h or 24 h, and then exposed to different concentrations of TRAIL for 12 h. An equal volume (100 μL) of CellTiter-Glo™ reagent is added and the solution is mixed gently for 2 min on an orbital shaker. Mixture is incubated at room temperature for 10 min to allow luminescent signal to stabilize, and then imaging is performed using the Xenogen IVIS system to quantify the cell viability[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1][3]	Mice[1] Four- to 6-wk-old NCr nude mice are treated with Mitomycin C (1 mg/kg) by intraperitoneal injection for 24 h, followed by one intravenous dose of purified rhTRAIL (100 μg). As a negative control, a subset of the mice are injected (i.p. and i.v.) with saline (vehicle) at the same frequency of treatment. Animals are treated for 3 consecutive weeks. The tumor size is monitored every week using caliper measurements of the tumor volume. Rats[3] Young adult female Wistar rats at age 13 weeks with a median weight of 217 g (range 187 to 255) are randomized into 4 groups of 10 each, namely a normal group with no instillations, an NaCl 0.9% or placebo group that received instillations with the solvent of the chemotherapeutic agent, Mitomycin C (1 mg/mL) group. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Discov. 2019 May 28;5:29.
- J Hematol Oncol. 2018 Aug 13;11(1):102.
- J Hematol Oncol. 2018 Mar 20;11(1):44.
- Nat Microbiol. 2021 May;6(5):682-696.
- Nat Microbiol. 2018 Nov;3(11):1266-1273.

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REFERENCES

[1]. Cheng H, et al. Mitomycin C potentiates TRAIL-induced apoptosis through p53-independent upregulation of death receptors: Evidence for the role of c-Jun N-terminal kinase activation. Cell Cycle. 2012 Sep 1;11(17): 3312-23.

[2]. Abbas T, et al. Differential activation of p53 by the various adducts of mitomycin C. J Biol Chem. 2002 Oct 25;277(43):40513-9.

[3]. Michielsen D, et al. Mitomycin C: functional bladder damage in rats after repeat intravesical instillations. J Urol. 2005 Jun;173(6):2166-70.

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