Product Data Sheet

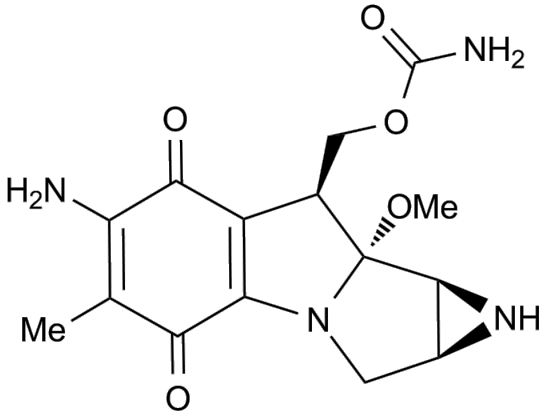
Product Name: Mitomycin C

Cat. No.: GC12353

Chemical Properties

Cas No.	50-07-7		
化学名	((1aS,8S,8aR,8bS)-6-amino-8a-methoxy-5-methyl-4,7-dioxo-1,1a,2,4,7,8,8a,8b-octahydroazirino[2',3':3,4]pyrrolo[1,2-a]indol-8-yl)methyl carbamate		
Canonical SMILES	NC(C1=O)=C(C)C(C2=C1[C@@H](COC(N)=O)[C@]3(OC)N2C[C@H]4[C@@H]3N4)=O		
分子式	$C_{15}H_{18}N_4O_5$	分子量	334.33
溶解度	≥ 16.7mg/mL in DMSO	储存条件	4°C, protect from light
General tips	For obtaining a higher solubility , please warm the tube at 37 ℃ and shake it in the ultrasonic bath for a while.Stock solution can be stored below -20℃ 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, 2]:

Cell lines	HCT116, HT-29
Preparation Method	Ten millimolar Mitomycin C is prepared in 100% dimethyl sulfoxide, stored as small aliquots at -80°C and then diluted as needed in cell culture medium.
Reaction Conditions	5 μM,12 or 24h
Applications	Mitomycin C is a mitomycin that is used as a chemotherapeutic agent by virtue of its antitumour activity. Mitomycin C not only potentiates TRAIL-induced apoptosis in HCT116 (p53−/−) colon cancer cells but also sensitizes TRAIL- resistant colon cancer cells HT-29 to the cytokine. Mitomycin C inhibits HT-29 with IC_{50} of 40 nM.

Animal experiment [1]:

Animal models	Nude mice (6 weeks) injected subcutaneously with 1 × 106 HCT116 (p53−/−) or 2 × 10^6 HT-29 cells mixed with Matrigel
Preparation Method	Ten millimolar Mitomycin C is prepared in 100% dimethyl sulfoxide, stored as small aliquots at -80°C and then diluted as needed in cell culture medium.

Caution: Producthasnot been fully validated for medical applications. For research use only.

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Dosage form	1 mg/kg, Intraperitoneal injection
Applications	Mitomycin C suppresses tumor growth significantly and does not impact the weight of the mice with TRAIL, indicating that the therapeutic combination of Mitomycin C and TRAIL is well-tolerated and has anti-tumor activity in vivo.

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]. Hodgkinson TJ, et al. Chemical synthesis and cytotoxicity of some azinomycin analogues devoid of the 1-azabicyclo[3.1.0]hexane subunit. Bioorg Med Chem Lett. 2000 Feb 7;10(3):239-41.

Background

Mitomycin C, a kind of antibiotic isolated from Streptomyces caespitosus or Streptomyces lavendulae, inhibits DNA synthesis through covalent mitomycin C-DNA adduct with EC50 values of 0.14μM in PC3 cells.

Mitomycin C is an antibiotic that has demonstrated antitumor activity in preclinical and clinical studies and is widely used to treat various cancers. Mitomycin C is known to act synergistically with capecitabine and irinotecan. Some studies suggested that the combination of 5-FU plus Mitomycin C is more active in vitro than mono-therapy in colorectal cancer. The efficacy of the combination of Mitomycin C with other cytotoxic agents such as capecitabine and raltiterxed for colorectal cancer has been reported.[1]

Mitomycin C not only potentiates TRAIL-induced apoptosis in HCT116 (p53−/−) colon cancer cells but also sensitizes TRAIL-resistant colon cancer cells HT-29 to the cytokine. At a mechanistic level, Mitomycin C downregulates cell survival proteins, including Bcl2, Mcl-1 and Bcl-XL, and upregulates pro-apoptotic proteins including Bax, Bim and the cell surface expression of TRAIL death receptors DR4 and DR5. Besides, the result of cell experiment indicates that Mitomycin C inhibits HT-29 with IC_{50} of 40 nM. [1,2]

Mitomycin C also plays an effective role in antitumor in vivo. For in vivo experiment, Mitomycin C suppressed tumor growth significantly and did not impact the weight of the mice with TRAIL, indicating that the therapeutic combination of Mitomycin C and TRAIL is well-tolerated and has anti-tumor activity in vivo. [1]

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]. Hodgkinson TJ, et al. Chemical synthesis and cytotoxicity of some azinomycin analogues devoid of the 1-azabicyclo[3.1.0]hexane subunit. Bioorg Med Chem Lett. 2000 Feb 7;10(3):239-41.

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