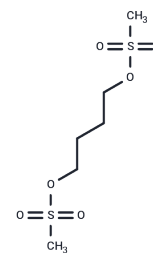


Busulfan

Chemical Properties

CAS No. :	55-98-1
Formula:	C ₆ H ₁₄ O ₆ S ₂
Molecular Weight:	246.30
Storage:	Store under nitrogen Powder: -20°C for 3 years In solvent: -80°C for 1 year <small>Actual storage temperature shall be subject to the COA.</small>



Biological Description

Description	Busulfan is an alkylating antineoplastic agent derived from dimethane sulfonate, with cytotoxic and immunosuppressive properties. It primarily acts by forming carbonium ions in vivo, which induce cross-linking between DNA strands or between DNA and proteins, leading to DNA damage, inhibition of DNA replication, and suppression of RNA transcription. In addition, Busulfan can inhibit thioredoxin reductase and induce apoptosis. It is commonly used as a myeloablative agent in preconditioning regimens for bone marrow transplantation and can also be used to establish anemia models.
Targets(IC50)	Apoptosis,DNA Alkylation,DNA Alkylator/Crosslinker
In vitro	Mice transplanted with busulfan exhibit slow and incomplete lymphoid engraftment. Mice treated with busulfan demonstrate a significant increase in apoptosis and a reduction in testicular weight. In NOD/SCID mice, busulfan treatment combined with irradiation achieves a detection sensitivity similar to that of limiting dilution assays. A dose-dependent lymphoid tissue reconstitution is provided in mice with 20 mg/kg to 100 mg/kg of busulfan. At 40 mg/kg, busulfan induces the maximum number of apoptotic cells while minimizing the number of necrotic cells.
In vivo	Busulfan, an alkylating agent that induces DNA damage through cross-linking DNA with DNA and proteins, triggers senescence in normal human diploid WI38 fibroblast cells via a cascade mediated by extracellular signal-regulated kinase (Erk) and p38 mitogen-activated protein kinase (p38 MAPK), independent of the p53-DNA damage pathway. Busulfan causes a transient reduction in glutathione levels, followed by a sustained increase in ROS production. The hypophosphorylation of Rb induced by Busulfan inhibits the expression of PCNA in testicular cells, preventing apoptosis of spermatogonial stem cells. Moreover, while Busulfan reduces the frequency of cobblestone area-forming cells, it does not significantly increase apoptosis in hematopoietic stem cells (HSC)-like cells and progenitor cells with similar phenotypes. Busulfan suppresses the hematopoietic function of HSC-like cells and progenitor cells through an apoptosis-dependent mechanism. Additionally, Busulfan-induced senescence in bone marrow hematopoietic cells correlates with a time-dependent increase in the expression of p16Ink4a and p19Arf.

Solubility Information

A DRUG SCREENING EXPERT

Solubility	Ethanol: < 1 mg/mL (insoluble or slightly soluble), DMSO: 50.00 mg/mL (203.00 mM),Sonication is recommended. (< 1 mg/ml refers to the product slightly soluble or insoluble)
In vivo Formulation	10% DMSO+40% PEG300+5% Tween-80+45% Saline: 3.00 mg/mL (12.18 mM) <i>Please add the solvents sequentially, clarifying the solution as much as possible before adding the next one. Dissolve by heating and/or sonication if necessary. Working solution is recommended to be prepared and used immediately. The formulation provided above is for reference purposes only. In vivo formulations may vary and should be modified based on specific experimental conditions.</i>

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	4.0601 mL	20.3004 mL	40.6009 mL
5 mM	0.812 mL	4.0601 mL	8.1202 mL
10 mM	0.406 mL	2.030 mL	4.0601 mL
50 mM	0.0812 mL	0.406 mL	0.812 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

- Meng A, et al. Cancer Res, 2003, 63(17), 5414-5419.
Probin V, et al. Free Radic Biol Med, 2007, 42(12), 1858-1865.
Choi YJ, et al. FEBS Lett, 2004, 575(1-3), 41-51.
Robert-Richard E, et al. Haematologica, 2006, 91(10), 1384.
Yeager AM, et al. Blood, 1991, 78(12), 3312-3316.

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