# Busulfan

**Cat. No.:** HY-B0245

**CAS No.:** 55-98-1

**Molecular Formula:** $C_6H_{14}O_6S_2$

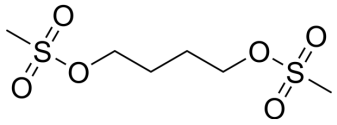
**Molecular Weight:** 246.3

**Target:** DNA Alkylator/Crosslinker; Apoptosis

**Pathway:** Cell Cycle/DNA Damage; Apoptosis

**Storage:**

| | | |
|---|---|---|
| Powder | -20°C | 3 years |
| | 4°C | 2 years |
| In solvent | -80°C | 6 months |
| | -20°C | 1 month |



## SOLVENT & SOLUBILITY

**In Vitro**

DMSO : 50 mg/mL (203.00 mM; Need ultrasonic)

Methanol : 1 mg/mL (4.06 mM; ultrasonic and warming and heat to 60°C)

$H_2O$ : < 0.1 mg/mL (insoluble)

| Preparing Stock Solutions | Mass / Solvent / Concentration | 1 mg | 5 mg | 10 mg |
|---|---|---|---|---|
| | 1 mM | 4.0601 mL | 20.3004 mL | 40.6009 mL |
| | 5 mM | 0.8120 mL | 4.0601 mL | 8.1202 mL |
| | 10 mM | 0.4060 mL | 2.0300 mL | 4.0601 mL |

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

**In Vivo**

1. Add each solvent one by one: 15% Cremophor EL >> 85% Saline
   Solubility: 6.25 mg/mL (25.38 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: corn oil
   Solubility: 3.12 mg/mL (12.67 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (8.44 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (8.44 mM); Clear solution
5. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (8.44 mM); Clear solution

## BIOLOGICAL ACTIVITY

**Description**

Busulfan is a potent alkylating antineoplastic agent. Busulfan causes DNA damage by cross-linking DNAs and DNA and

proteins. Busulfan inhibits thioredoxin reductase. Busulfan induces apoptosis. Busulfan is an immunosuppressive and myeloablative chemotherapeutic agent[1][2][3].

**In Vitro**

Busulfan (120 μM; 24 h) incited a moderate p53 activation, but strong Erk, p38, and JNK phosphorylation, in a time-dependent manner[1].

Busulfan (120 μM; 24 h) results in premature senescence in WI38 cells via the Erk and p38 MAPK pathway, reduces GSH and increases ROS production, but the production can be suppressed by NADPH oxidase[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

| | |
|---|---|
| Cell Line: | WI38 cells |
| Concentration: | 120 μM |
| Incubation Time: | 24 hours |
| Result: | Incited a moderate p53 activation, but strong Erk, p38, and JNK phosphorylation, in a time-dependent manner.<br>Elicited an immediate up-regulation of p21 expression, which subsided by day 11. |

**In Vivo**

Busulfan can be used in animal modeling to establish models of acute aplastic anemia. Busulfan (40 mg/kg; i.p; single dose) increases apoptosis and decreases the testis weight in mice[4].

Busulfan (2.5, 5.0 mg/kg, i.p.) causes earlier occurrence of persistent esturs in a dose dependent manner in rats.

Busulfan (5.0 mg/kg) also increases the incidence of uterine adenocarcinomas and multiplicity of uterine neoplastic lesions[5].

AUC is 220 ± 34 h·nmol·mL$^{-1}$ after 16.5 mg/kg and 604 ± 87 h·nmol·mL$^{-1}$ after 33 mg/kg at the first injection in mice[7].

Induction of aplastic anemia model[6]

- Background

  Busulfan causes DNA damage by cross-linking DNA as well as DNA and proteins.

- Specific Mmodeling Methods

  ICR mice : male • 18-22 g, 6-8 weeks

  Administration: 20 mg/kg busulfan+40 mg/kg cyclophosphamide• i.p. • one time per day for 12 days.

  Note

  (1) Twenty-four hours after the last intraperitoneal injection, tail vein blood was collected from eight mice randomly selected from each group for blood test.

  (2) the mice are sacrificed by cervical dislocation and one femur was surgically dissected. After removing epiphysis from the femur, bone marrow cells are washed off using 1 ml PBS to prepare bone marrow cell suspension.

- Modeling Indicators

  The peripheral blood cells, hemoglobin, and bone marrow nucleated cells decreased significantly.
  Histopathological: the proliferation of bone marrow hematopoietic tissues and cells was inhibited, and non-hematopoietic cells (fat cells) were significantly increased.

- Correlated Product(s): Cyclophosphamide (HY-17420)
- Opposite Product(s):

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

| | |
|---|---|
| Animal Model: | ICR male mice ranging in age from 8 to 12 weeks (30-40 g) [4] |
| Dosage: | 40 mg/kg (in sesame oil) |
| Administration: | IP; single dose |
| Result: | Increased apoptosis and decreased the testis weight in mice.<br>Exhibited higher level of pRB expression, inhibited Rb phosphorylation and PCNA expression compared to the control. |

## CUSTOMER VALIDATION

- Biomark Res. 2023 Jan 24;11(1):8.
- J Neuroinflammation. 2023 Nov 17;20(1):270.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.
- Stem Cells Dev. 2020 Apr 15;29(8):475-487.
- Stem Cells Dev. 2019 Oct 1;28(19):1322-1333.

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## REFERENCES


[1]. Mattan Levi, et al. Treosulfan induces distinctive gonadal toxicity compared with busulfan. Oncotarget. 2018 Apr 10;9(27):19317-19327.

[2]. Janka Reimer, et al. Antineoplastic agent busulfan regulates a network of genes related to coagulation and fibrinolysis. Eur J Clin Pharmacol. 2012 Jun;68(6):923-35.

[3]. Chen YF, et al. The role of RIP1 and RIP3 in the development of aplastic anemia induced by cyclophosphamide and busulphan in mice. Int J Clin Exp Pathol. 2014 Dec 1;7(12):8411-20.

[4]. Bouligand J, et al. Induction of glutathione synthesis explains pharmacodynamics of high-dose busulfan in mice and highlights putative mechanisms of drug interaction. Drug Metab Dispos. 2007 Feb;35(2):306-14.

[5]. Probin V, et al. Busulfan-induced senescence is dependent on ROS production upstream of the MAPK pathway. Free Radic Biol Med. 2007 Jun 15;42(12):1858-65. Epub 2007 Mar 31.

[6]. Choi YJ, et al. Murine male germ cell apoptosis induced by busulfan treatment correlates with loss of c-kit-expression in a Fas/FasL- and p53-independent manner. FEBS Lett. 2004 Sep 24;575(1-3):41-51.

[7]. Yoshida M, et al. Reduction of primordial follicles caused by maternal treatment with busulfan promotes endometrial adenocarcinoma development in donryu rats. J Reprod Dev. 2005 Dec;51(6):707-14. Epub 2005 Sep 22.



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