Chlorambucil

Cat. No.: HY-13593

CAS No.: 305-03-3

Molecular Formula: $C_{14}H_{19}Cl_2NO_2$

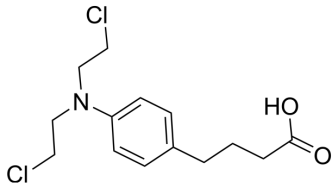
Molecular Weight: 304.21

Target: DNA Alkylator/Crosslinker

Pathway: Cell Cycle/DNA Damage

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 : ≥ 24 mg/mL (78.89 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.2872 mL	16.4360 mL	32.8720 mL
5 mM	0.6574 mL	3.2872 mL	6.5744 mL
10 mM	0.3287 mL	1.6436 mL	3.2872 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: ≥ 2.5 mg/mL (8.22 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.22 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.22 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Chlorambucil (CB-1348), an orally active antineoplastic agent, is a bifunctional alkylating agent belonging to the nitrogen mustard group. Chlorambucil can be used for the research of lymphocytic leukemia, ovarian and breast carcinomas, and Hodgkin's disease[1][2][3][4].

IC_{50} & Target

DNA Alkylator[1]

In Vitro	Chlorambucil can deprive the function of complementary strands of DNA molecules via alkalization-induced cross interaction, and then inhibits tumor cell proliferation. Chlorambucil (0, 2.5, 5, 10 μM) exhibits slight inhibitory effect on Raji cell apoptosis, but potently increases DR4 and DR5 mRNA expression in Raji cells. Chlorambucil (10 μM) in combination with Tumor necrosis factor (TNF) related apoptosis inducing ligand (TRAIL, 80 ng/ml) has synergistic effect on Raji cell apoptosis and inhibition on proliferation[1]. ?Chlorambucil is a DNA alkylator at high doses and, at lower doses, acts as an inhibitor of synthesis of nuclear proteins, particularly histones. Increasing doses are associated with a higher frequency of apoptosis, whereas long-term maintenance therapy has been associated with mutations of the p53 gene, leading to secondary malignancies[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Chlorambucil (0.2 mg/kg, p.o.) in combination with levamisole (5 mg/kg) has enhanced anti-cancer effect on Ehrlich ascites carcinoma which elevates apoptosis of Ehrlich ascites carcinoma and the survival rate of the mice. However, Chlorambucil exhibits adverse effects on the liver and kidneys of mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	Cultured cells at log-growth phase are digested by trypsin into single cell suspension and are seeded into 96-well plate at 1000 per well density. The plate is placed in a 37°C chamber with 5% CO_2. After attached growth for 24 h, cells are treated with TRAIL at 0, 20, 40 and 80 ng/mL or Chlorambucil at 0, 2.5, 5 and 10 μM for 48 h. 10 μL CCK-8 reagent is added to each well, followed by incubation at 37°C for 4 h. Absorbance values at 450 nm are then measured by a micro-plate reader. Six parallel samples are performed in each treatment group. Cell proliferation rate (%) = mean value of experimental group/mean value of control group × 100%[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Female Swiss mice are divided randomly into five group (20 mice per group). Group 1 is kept as the control group, Group 2 receives intraperitoneal injection of by 2.5×10^6 Ehrlich ascites carcinoma cell, Gropu 3 is treated orally with Chlorambucil 0.2 mg/kg body weight, Group 4 is treated orally with levamisole (5 mg/kg body weight) and Group 5 is treated orally with a combination of Chlorambucil and levamisole each day, using a bent stainless steel stomach tube[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Autoimmun. 2020 Aug;112:102465.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.
- Mol Biol Cell. 2023 Mar 29;mbcE22110518.

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REFERENCES

[1]. Guo JX, et al. Synergistic effects of chlorambucil and TRAIL on apoptosis and proliferation of Raji cells. Eur Rev Med Pharmacol Sci. 2017 Oct;21(20):4703-4710.

[2]. Salem FS, et al. Biochemical and pathological studies on the effects of levamisole and chlorambucil on Ehrlich ascites carcinoma-bearing mice. Vet Ital. 2011 Jan-Mar;47(1):89-95.

[3]. Mohamed D, et al. Chlorambucil-adducts in DNA analyzed at the oligonucleotide level using HPLC-ESI MS. Chem Res Toxicol. 2009;22(8):1435-1446.

[4]. Birnbaum AD, et al. Chlorambucil and malignancy. Ophthalmology. 2010;117(7):1466-1466.e1.

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