(-)-DHMEQ

Cat. No.: HY-14645

CAS No.: 287194-40-5

Molecular Formula: $C_{13}H_{11}NO_5$

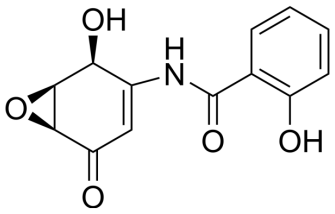
Molecular Weight: 261.23

Target: NF-κB

Pathway: NF-κB

Storage: -20°C, stored under nitrogen

* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (191.40 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.8280 mL	19.1402 mL	38.2804 mL
	5 mM	0.7656 mL	3.8280 mL	7.6561 mL
	10 mM	0.3828 mL	1.9140 mL	3.8280 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: 5 mg/mL (19.14 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

(-)-DHMEQ (Dehydroxymethylepoxyquinomicin) is a potent, selective and irreversible NF-κB inhibitor that covalently binds to a cysteine residue. (-)-DHMEQ inhibits nuclear translocation of NF-κB and shows anti-inflammatory and anticancer activity[1][2][3].

IC_{50} & Target	RelA	RelB

In Vitro

(-)-DHMEQ (Dehydroxymethylepoxyquinomicin; 2-10 μg/mL; 12-48 hours) treatment significantly reduces the viability of all cell lines in a dose- and time-dependent manner, whereas the effect is not significant in a control cell line K562 without constitutive NF-κB activity[2].

?(-)-DHMEQ (10 μg/mL; 0-48 hours; TL-Om1, MT-1 and K562 cells) treatment significantly increases the Annexin V-positive cells in MT-1 and TL-Om1 cell lines[2].

?(-)-DHMEQ (10 μg/mL; 4-16 hours; MT-1 cells) treatment down-regulates Bcl-xL, Bcl-2, c-myc, cyclin D1, Rb, and p53, and up-regulates proapoptotic genes such as caspase-3, -8, and-9[2].

?(-)-DHMEQ treatment increases cells in G0 /G1 phase in a time-dependent manner, demonstrating antiproliferative effects

of (-)-DHMEQ[2].
?(-)-DHMEQ binds to p65, cRel, RelB, and p50, but not to p52 at specific cysteine residues. (-)-DHMEQ inhibits not only DNA-binding of RelB, but also its interaction to importin. (-)-DHMEQ also induces instability of RelB[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[2]

Cell Line:	TL-Om1, MT-1, KK-1, ST-1 and K562 cells
Concentration:	2 μg/mL, 5 μg/mL, 10 μg/mL
Incubation Time:	12 hours, 24 hours, 48 hours
Result:	Significantly reduced the viability of all cell lines in a dose- and time-dependent manner.

Apoptosis Analysis[2]

Cell Line:	TL-Om1, MT-1 and K562 cells
Concentration:	10 μg/mL
Incubation Time:	0 hours, 24 hours, 48 hours
Result:	Annexin V-positive cells were significantly increased after 24 to 48 hours.

Western Blot Analysis[2]

Cell Line:	MT-1 cells
Concentration:	10 μg/mL
Incubation Time:	4 hours, 8 hours, 16 hours
Result:	Annexin V-positive cells were significantly increased after 24 to 48 hours.

In Vivo

(-)-DHMEQ (4 mg/kg or 12 mg/kg; intraperitoneal injection; on day 0 and 3 times a week; for one month; SCID mice) treatment shows a significant increase in the survival rate in mice[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male C.B17-scid/scid (5 weeks old) mice injected with MT-2 cells[2]
Dosage:	4 mg/kg or 12 mg/kg
Administration:	Intraperitoneal injection; on day 0 and 3 times a week; for one month
Result:	Showed a significant increase in the survival rate in mice.

CUSTOMER VALIDATION

- Biosens Bioelectron. 2020 Dec 30;176:112942.
- Theranostics. 2023 Apr 23;13(8):2588-2604.
- Theranostics. 2019 Jan 1;9(2):436-448.
- Redox Biol. 2021 Sep 30;47:102157.
- Br J Cancer. 2021 Sep 25.

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REFERENCES

[1]. Quach HT, et al. Eudesmane-Type Sesquiterpene Lactones Inhibit Nuclear Translocation of the Nuclear Factor κB Subunit RelB in Response to a Lymphotoxin β Stimulation. Biol Pharm Bull. 2017;40(10):1669-1677.

[2]. Yinzhi Lin, et al. Inhibition of Late and Early Phases of Cancer Metastasis by the NF-κB Inhibitor DHMEQ Derived from Microbial Bioactive Metabolite Epoxyquinomicin: A Review.

[3]. Mariko Watanabe, et al. Dual targeting of transformed and untransformed HTLV-1-infected T cells by DHMEQ, a potent and selective inhibitor of NF-kappaB, as a strategy for chemoprevention and therapy of adult T-cell leukemia. Blood. 2005 Oct 1;106(7):2462-71.

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