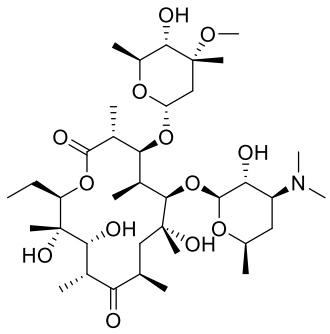
Product data sheet

MedKoo Cat#: 540123 Name: Erythromycin CAS#: 114-07-8 Chemical Formula: $C_{37}H_{67}NO_{13}$ Exact Mass: 733.4612 Molecular Weight: 733.93	
Product supplied as:	Powder
Purity (by HPLC):	≥ 98%
Shipping conditions	Ambient temperature
Storage conditions:	Powder: -20°C 3 years; 4°C 2 years.
	In solvent: -80°C 3 months; -20°C 2 weeks.

1. Product description:
Erythromycin is an inhibitor of protein translation and mammalian mRNA splicing. It inhibits growth of gram negative and gram positiove bacteria.

2. CoA, QC data, SDS, and handling instruction
SDS and handling instruction, CoA with copies of QC data (NMR, HPLC and MS analytical spectra) can be downloaded from the product web page under "QC And Documents" section. Note: copies of analytical spectra may not be available if the product is being supplied by MedKoo partners. Whether the product was made by MedKoo or provided by its partners, the quality is 100% guaranteed.

3. Solubility data

Solvent	Max Conc. mg/mL	Max Conc. mM
DMSO	87.33	118.99
DMF	15.0	20.44
Ethanol	88.5	120.58
Ethanol:PBS (pH 7.2) (1:1)	0.5	0.68
Water	2.0	2.73

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	1.36 mL	6.81 mL	13.63 mL
5 mM	0.27 mL	1.36 mL	2.73 mL
10 mM	0.14 mL	0.68 mL	1.36 mL
50 mM	0.03 mL	0.14 mL	0.27 mL

5. Molarity Calculator, Reconstitution Calculator, Dilution Calculator
Please refer the product web page under section of "Calculator"

6. Recommended literature which reported protocols for in vitro and in vivo study
In vitro study

1. Liu J, Zhong X, He Z, Zhang J, Bai J, Liu G, Liang Y, Ya L, Qin X. Erythromycin Suppresses the Cigarette Smoke Extract-Exposed Dendritic Cell-Mediated Polarization of CD4+ T Cells into Th17 Cells. J Immunol Res. 2020 Jan 21;2020:1387952. doi: 10.1155/2020/1387952. PMID: 32411785; PMCID: PMC7201779.
2. Zhang H, Qiu SL, Tang QY, Zhou X, Zhang JQ, He ZY, Bai J, Li MH, Deng JM, Liang Y, Zhong XN. Erythromycin suppresses neutrophil extracellular traps in smoking-related chronic pulmonary inflammation. Cell Death Dis. 2019 Sep 12;10(9):678. doi: 10.1038/s41419-019-1909-2. PMID: 31515489; PMCID: PMC6742640.

In vivo study

1. Tamura H, Maekawa T, Domon H, Hiyoshi T, Hirayama S, Isono T, Sasagawa K, Yonezawa D, Takahashi N, Oda M, Maeda T, Tabeta K, Terao Y. Effects of Erythromycin on Osteoclasts and Bone Resorption via DEL-1 Induction in Mice. Antibiotics (Basel). 2021 Mar 17;10(3):312. doi: 10.3390/antibiotics10030312. PMID: 33803007; PMCID: PMC8002756.

2. Maekawa T, Tamura H, Domon H, Hiyoshi T, Isono T, Yonezawa D, Hayashi N, Takahashi N, Tabeta K, Maeda T, Oda M, Ziogas A, Alexaki VI, Chavakis T, Terao Y, Hajishengallis G. Erythromycin inhibits neutrophilic inflammation and mucosal disease by upregulating DEL-1. JCI Insight. 2020 Aug 6;5(15):e136706. doi: 10.1172/jci.insight.136706. PMID: 32603314; PMCID: PMC7455085.

7. Bioactivity

Biological target:

Erythromycin acts by binding to bacterial 50S ribosomal subunits and inhibits RNA-dependent protein synthesis by blockage of transpeptidation and/or translocation reactions, without affecting synthesis of nucleic acid.

In vitro activity

The Th17 cells in the CSE-exposed DC/MLR group increased significantly compared with those in the control DC/MLR group ($P < 0.05$). Moreover, Th17 cells in the CD40-blocked CSE-exposed DC/MLR group and EM-treated CSE-exposed DC/MLR group were reduced compared with those in the CSE-exposed DC/MLR group ($P < 0.05$). Thus, these findings suggested that EM suppressed the CSE-exposed DC-mediated polarization of $CD4^+$ T cells into Th17 cells and that this effect may be mediated through inhibition of the CD40/CD40L pathway.

Reference: J Immunol Res. 2020; 2020: 1387952. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7201779/

In vivo activity

Additionally, the ERM (erythromycin) treatment significantly reduced bone loss compared to the PC and JSM treatment groups (Figure 1b,c). Compared to the EtOH group, ERM, JSM, and PC treatment significantly decreased the gene expression of *Nfatc1* and *RANK*, which are osteoclast differentiation-related factors in the gingiva (Figure 2a,b). Furthermore, ERM and JSM treatment significantly downregulated the expression of bone resorption activity-related factors (*Acp5*, *Ctsk*) (Figure 2c,d). Interestingly, ERM treatment significantly upregulated *Del1* expression, compared to the EtOH group (Figure 2e).

Reference: Antibiotics (Basel). 2021 Mar; 10(3): 312. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8002756/