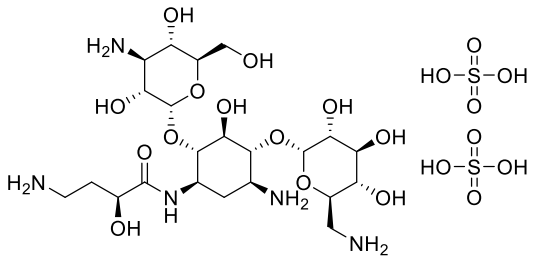
Product data sheet

MedKoo Cat#: 100955 Name: Amikacin sulfate CAS#: 39831-55-5 (sulfate) Chemical Formula: $C_{22}H_{47}N_5O_{21}S_2$ Molecular Weight: 781.752	
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:

Amikacin is an antibiotic used for a number of bacterial infections. This includes joint infections, intraabdominal infections, meningitis, pneumonia, sepsis, and urinary tract infections. It is also used for the treatment of multidrug-resistant tuberculosis. It is used either by injection into a vein or muscle. Amikacin is in the aminoglycoside family of medications. It works by blocking the function of the bacteria's 30S ribosomal subunit, making it unable to make protein.

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	55.0	70.35

4. Stock solution preparation table:

Concentration / Solvent Volume / Mass	1 mg	5 mg	10 mg
1 mM	1.28 mL	6.40 mL	12.79 mL
5 mM	0.26 mL	1.28 mL	2.56 mL
10 mM	0.13 mL	0.64 mL	1.28 mL
50 mM	0.03 mL	0.13 mL	0.26 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. Kuti JL, Wang Q, Chen H, Li H, Wang H, Nicolau DP. Defining the potency of amikacin against Escherichia coli, Klebsiella pneumoniae, Pseudomonas aeruginosa, and Acinetobacter baumannii derived from Chinese hospitals using CLSI and inhalation-based breakpoints. Infect Drug Resist. 2018 May 25;11:783-790. doi: 10.2147/IDR.S161636. PMID: 29872328; PMCID: PMC5975598.
2. Sutherland CA, Verastegui JE, Nicolau DP. In vitro potency of amikacin and comparators against E. coli, K. pneumoniae and P. aeruginosa respiratory and blood isolates. Ann Clin Microbiol Antimicrob. 2016 Jun 17;15(1):39. doi: 10.1186/s12941-016-0155-z. PMID: 27316973; PMCID: PMC4912699.

In vivo study

1. Pezzanite L, Chow L, Hendrickson D, Gustafson DL, Russell Moore A, Stoneback J, Griffenhagen GM, Piquini G, Phillips J, Lunghofer P, Dow S, Goodrich LR. Evaluation of Intra-Articular Amikacin Administration in an Equine Non-inflammatory Joint Model to Identify Effective Bactericidal Concentrations While Minimizing Cytotoxicity. Front Vet Sci. 2021 May 21;8:676774. doi: 10.3389/fvets.2021.676774. PMID: 34095281; PMCID: PMC8175670.
2. Bugnon D, Potel G, Xiong YQ, Caillon J, Kergueris MF, Le Conte P, Baron D, Drugeon H. In vivo antibacterial effects of simulated human serum profiles of once-daily versus thrice-daily dosing of amikacin in a Serratia marcescens endocarditis experimental model. Antimicrob Agents Chemother. 1996 May;40(5):1164-9. doi: 10.1128/AAC.40.5.1164. PMID: 8723459; PMCID: PMC163284.

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7. Bioactivity

Biological target:

Amikacin disulfate is bactericidal, acting directly on the 30S and 50S bacerial ribosomal subunits to inhibit protein synthesis.

In vitro activity

For Escherichia coli, including extended-spectrum beta-lactamase (ESBL)-producing isolates (45.7% of population), amikacin demonstrated excellent activity (93.0%–94.7% susceptible) similar to tigecycline, piperacillin/tazobactam, and the carbapenems. Against Klebsiella pneumoniae, only tigecycline retained susceptibility >90%; amikacin inhibited 83.7% and 71.1% of the total and ESBL-producing (24.2%) populations at its breakpoint, respectively.

Reference: Infect Drug Resist. 2018; 11: 783–790. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5975598/

In vivo activity

Macrophages engulfing amikacin-killed cells demonstrated a reduced release of pro-inflammatory cytokines (IL-1β, TNF-α, and IL-6) and a concomitant increased production of anti-inflammatory cytokines (TGF-β) (Figure 6). Similarly, amikacin-killed chondrocytes also induced more TGF-β than did heat-killed chondrocytes ($p = 0.03$). In contrast, less IL-1β was produced by macrophages incubated with amikacin-killed cells than by macrophages incubated with heat-killed cells ($p = 0.006$). Similar responses were noted for TNF-α ($p < 0.0001$) and IL-6 ($p < 0.0001$) production between amikacin- vs. heat-killed cells. These findings indicate that cell death induced by amikacin is inherently anti-inflammatory compared to the pathways associated with necrosis. These findings in vitro help explain why SF from amikacin-treated horses manifested a relatively benign response to ongoing cellular death and tissue injury locally within the joint.

Reference: Front Vet Sci. 2021; 8: 676774. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8175670/