

Antibiotic bacitracin induces hydrolytic degradation of nucleic acids.

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Background: Bacitracin is a polypeptide antibiotic active against Gram-positive bacterial strains. Its mechanism of action postulates disturbing the cell wall synthesis by inhibiting dephosphorylation of the lipid carrier. We have discovered that bacitracin induces degradation of nucleic acids, being particularly active against RNA.

Methods: In the examination of the nucleolytic activity of bacitracin several model RNA and DNA oligomers were used. The oligomers were labeled at their 5' ends with (32)P radioisotope and following treatment with bacitracin the cleavage sites and efficiency were determined.

Results and conclusions: Bacitracin induces degradation of RNA at guanosine residues, preferentially in single-stranded RNA regions. Bacitracin is also able to degrade DNA to some extent but comparable effects to those observed with RNA require its 10-fold higher concentration. The sites of degradation in DNA are very infrequent and preferentially occur near cytidine residues. Free radicals are not involved in the reaction, and which probably proceeds via a hydrolytic mechanism. The phosphate groups at the cleavage sites are present at the 3' ends of RNA products and at the 5' ends of DNA fragments. Importantly, the presence of EDTA does not influence RNA degradation but completely inhibits the degradation of DNA. For DNA degradation divalent metal ions like Mg(2+), Mn(2+) or Zn(2+) are absolutely necessary.

General significance: The ability of bacitracin to degrade nucleic acids via a hydrolytic mechanism was a surprising observation, and it is of interest whether these properties can contribute to its mechanisms of action during antibiotic treatment.

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