Biochemists interested in the mode of action of antibiotics will welcome this second edition appearing 9 years after the first. The work has been extensively rewritten, and provides an excellent and up to date survey of the subject. It is a pity that the first chapter containing information on arsenicals and sulphonamide antibacterials has not been revised. It contains no references more recent than 1968 and thus misses later work which has thrown new light on the actions of these compounds. The greater part of the book consists of five substantial chapters on: inhibitors of cell wall synthesis; compounds affecting the cytoplasmic membrane; inhibitors of nucleic acid synthesis; inhibitors of ribosome function; and bacterial resistance. The book concludes with a balanced and well-written chapter entitled 'Perspectives'.

The authors do not claim comprehensive coverage of antibiotics, but most of those of any consequence are included. One surprise is the lack of information on antibiotics affecting the respiratory pathway. There is no mention of antimycin or piericidin and only a brief paragraph on oligomycin. Another less important omission is of antibiotics, such as borrelidin and indolmycin, which inhibit aminoacyl tRNA formation. On the other hand, plant alkaloids and substances such as diphtheria toxin, ricin and emetine, not normally regarded as antibiotics, are mentioned. Some synthetic antibacterials are included but there is no systematic treatment of such compounds.

Among individual groups of antibiotics, the β-lactams occupy a long section of 58 pages. This is entirely appropriate to their outstanding importance, and to the complexity of the problems concerning their exact mode of action. Even more space is devoted to compounds intercalating with DNA, a coverage out of proportion to the importance of the antibacterials concerned. By contrast, the tetracyclines are dealt with in 5 pages. In other respects the coverage of different topics is adequate and well balanced. Multiple authorship has allowed each author to concentrate authoritatively on his specialist chapter.

However, it has led to variations in style and some overlapping of material. Chemical structures reappear in different chapters, often in different guises which may create difficulties for the student. The book is well set out with clear print and excellent diagrams. A few errors were noticed in structural formulae.

The book is thoroughly recommended to advanced students and research workers. Not many will wish to afford its high price, but they should see that their libraries buy it.

G. A. Snow

**Gene Amplification and Analysis, Volume 1**

Edited by J. G. Chirikjian
Elsevier Biomedical; Amsterdam, New York, 1981
xii + 246 pages. $46.25

There are few scientific advances which are rapidly taken up by the public at large — transistors, lasers, micro-chips, the double helix, the pill, heart transplants and test tube babies are perhaps the more obvious since the war. In the last decade 'genetic engineering' must have a good claim to evoke the

In or about 1943: Discovery of an enzyme in bacteria that could break off the molecular core of penicillin, the beta-lactam ring. XIX. In 1944: Several researchers (Howard W. Florey, Ernst H. Chain, H. W. Florey) decried the misuse of antibiotics. 

Antibiotics can be distinguished on the basis of their mode(s) of action that is on their ability to interfere with the metabolic machinery of microbes. The microbe needs this machinery to thrive, hold itself together, or make duplicate versions of it. Antibiotics are supposed to thwart bacterial not human cells through one or more of the following modes of action. However, the precise mechanism of action and the molecular basis for membrane selectivity are still a matter of debate. 

We have designed a new peptide antibiotic (NK-2) with enhanced antimicrobial activity based on an effector protein of mammalian immune cells (NK-lysin). Antibiotics with enhanced antimicrobial activity have emerged as compounds with potentially significant therapeutic applications against human pathogens. Antimicrobial peptides are typically small (15 to 50 amino acid residues) but have a diverse fold, such as α-helices, β-sheets and cyclic structures. 

Compared with the antibiotic group, maternal synbiotic supplementation decreased the plasma levels of glucose, gastrin, and Ala, as well as abundances of Pasteurella and RFN20 and propionate level in the colonic content. Expression of genes coding for E-cadherin, Occludin, ZO-1, ZO-2, IL-10, and interferon-α were down-regulated in the colonic mucosa. The acyl-enzyme complex is found lying in a deep thermodynamic sink, and deacylation is indeed the severely rate-limiting step, leading to suicide inhibition of the peptidoglycan cross-linking. 

The usage of QM-cluster models is a promising technique to understand, improve, and design antibiotics to disrupt function of the Streptomyces R61 DD-peptidase. Mechanisms of Antibiotic Action. Download PDF Copy. By Jack Davis, B.Sc. Reviewed by Dr. Surat P, Ph.D. Antibiotics are used in medicine and agriculture against bacterial infections and bacterial growth in food. These antibiotics are characterized by a beta-lactam ring in the molecule’s center, and function by interfering with the synthesis of the bacterial cell wall. 

β-lactams stop peptide chains from cross-linking during the formation of a new peptidoglycan chain which is a major component of the bacterial cell wall. Jack is a freelance scientific writer with research experience in molecular biology, genetics, human anatomy and physiology, and advanced analytical chemistry. He is also highly knowledgeable about DNA technology, drug analysis, human disease, and biotechnology.