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Drugs

ANTIBIOTICS AND PROTEIN SYNTHESIS Most antibiotics, with the exception of those that act on cell wall assembly, exert their bacteriostatic and bacteriocidal effects by virtue of their inhibitory effects on protein synthesis. Protein synthesis represents the end-result of three processes: 1) deoxyribonucleic acid (DNA) synthesis, or replication; 2) DNA-dependent ribonucleic acid (RNA) synthesis, or transcription; 3) RNA-dependent protein synthesis or translation. Antibiotics which inhibit any of these processes will inhibit protein synthesis, but those that inhibit primarily translation are the most effective clinically. It has been assumed that agents in this category inhibit bacterial protein synthesis without producing similar effects on host protein. However, in the past ten years it has become increasingly apparent that several antibiotics may inhibit mammalian systems and therefore threaten the host as well as the invading bacteria. Mammalian cells that are replicating rapidly or undergoing new protein synthesis are particularly susceptible to inhibition. Chloramphenicol, tetracyclines, the aminoglycoside family (*e.g.*, streptomycin, oleandomycin), and other generic antibiotic types have been implicated in this regard. (*Beard, N. S., Jr., Armentrout, S. A., and Weisberger, A. S.: Inhibition of Mammalian Protein Synthesis by Antibiotics, Pharmacol. Rev. 21: 213 (Sept.) 1969.*) ABSTRACTER'S COMMENT: Of special interest to anesthesiologists are the observations that antibiotic-induced inhibition of protein synthesis has been shown to prolong sleep time from barbiturates; to delay recovery from conduction blockade produced by local anesthetics, and perhaps to shorten induction time and prolong recovery time following exposure to halothane.