BACTERIOLOGY

Chairman: GORDON MALLETT, Eli Lilly and Company

RICHARD D. SMITH, Miles Ames Research Laboratory, Elkhart, was elected chairman for 1963

The inhibition of an RNA Bacteriophage by Streptomycin, using Host Bacteria Resistant to the Antibiotic.[1] THOMAS D. BROCK, Indiana University.—In most studies on the effect of antibiotics on virus growth, the host cells used have been sensitive to the inhibitory agent, so that a specific antiviral effect was not achieved. Growth of an RNA phage (MS2) of *Escherichia coli* is inhibited by 250 ug./ml. of streptomycin, even when the host cells used are resistant to the antibiotic. Streptomycin has no effect on free virus particles, or on adsorption of virus to cells. If antibiotic is added at the time of infection, no virus growth occurs, and there is a slow loss of infected centers. If antibiotic is not added until 5 minutes after infection (latent period of virus is about 15 minutes), it does not inhibit virus growth. Since streptomycin probably does not get into the interior of resistant bacteria, it is proposed that after adsorption of virus, there is a transient period during which free viral RNA is present at the periphery of the cell, where it can combine with streptomycin. If viral RNA has already passed into the cell before the antibiotic is added, it is now protected, and replication may proceed normally. Thus streptomycin may be a useful agent in analyzing the penetration of viral RNA into the cell. A variety of DNA phages of *E. coli* (Including a singlestranded DNA phage) are not affected by streptomycin, although a DNA phage of *Streptococcus faecium* is inhibited. In addition to its fundamental interest, this work raises the hope that specific antiviral agents for other viruses might be found.

The Concentrative Uptake of α-Methyl Glucoside by *Escherichia coli* D. P. KESSLER and H. V. RICKENBERG, Indiana University.—The concentrative uptake of α-MG by Escherichia coli strain ML was studied. The uptake system was stereospecific and of the carbohydrates tested only glucose inhibited α-MG uptake competitvely. The affinity of the uptake system was considerably higher for glucose than for α-MG suggesting that this particular transport system is in fact responsible for glucose transport under physiological conditions. Concentrative uptake of α-MG required energy. α-MG was not utilized by this strain of *E. coli* and the intracellular glycoside appeared to be in equilibrium with the α-MG of the medium. Unlabeled α-MG or glucose displaced the intracellular labeled α-MG.

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