Correlation Between Degradation of Bacteriophage T2 Deoxyribonucleic Acid and the Resistance of *Escherichia coli* to Infection

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The ability of certain strains of *Escherichia coli* to degrade T2 deoxyribonucleic acid to acid-soluble fragments is correlated with their high capacity to survive T2 infection.

While studying the mechanism of the abortive infection of *Escherichia coli* strain W by bacteriophage T2, it was observed that about 30 to 50% of the infecting viral deoxyribonucleic acid (DNA) was rapidly converted to acid-soluble fragments and that 50 to 60% of the interactions between virus and bacteria failed to result in death of the bacteria (4). A relationship between the degradation of the viral genome and the capacity to survive infection was suggested. The availability of strains derived from *E. coli* W which did not restrict viral multiplication, or which did so without acid solubilization (3), allowed us to study this relationship.

The properties of *E. coli* strain W (Waksman strain, ATCC 9637) and the derived strain WS have been described (2-4). Strain W adsorbs T2 but fails to produce progeny, whereas strain WS undergoes an apparently normal infection. Strains WS(ω)0, WS(ω)1C, and WS(ω)2B, which are lysogenic strains derived from strain WS, undergo abortive infection with T2 (3). Strains WS(ω)1C and WS(ω)2B, but not WS(ω)0, were found to degrade infecting T2 DNA. This report describes an experiment in which the number of cells surviving infection as a function of T2 multiplicity was determined for these strains of bacteria.

Standard phage techniques were used to measure T2 concentration, viable bacteria, and unadsorbed phage (1). Duplicate dilutions were performed in all cases. T2 was assayed on tryptone plates with *E. coli* B as the indicator strain (4). Bacteria were grown in a minimal medium and colony counts were made on nutrient-agar plates. Anti-T2 serum was present when bacteria surviving infection were measured. Unadsorbed T2 was determined by treating a sample of the infected culture with chloroform prior to the assay for virus. The multiplicity of infection was

calculated from the number of phage added to the culture and the number of phage remaining unadsorbed to the cells at 10 min, the time when

FIG. 1. Killing curves of derivatives of Strain W. Theoretical curve described by e^{-x} . Symbols: ○, data obtained with strain WS(ω)0; ●, data obtained with strain WS; △, data obtained with strain WS(ω)2B; ▲, data obtained with strain WS(ω)1C. Duplicate measurements are shown for strains WS(ω)1C and WS(ω)2B.

surviving bacteria were determined. At this time, less than 5% of the virus was unadsorbed. The number of bacteria retaining the capacity

to produce colonies as a function of viral multiplicity is shown in Fig. 1. The curve describing the killing of strains WS and WS(ω)0 are close approximations to the theoretical curve predicted by the Poisson distribution (1). With these strains, every interaction between T2 and the bacterial cell results in killing. Strains WS(ω)1C and WS(ω)2B gave killing curves like strain W, in which the slope of the line was approximately half that predicted. This result is interpreted to mean that 50% of the T2-cellular interactions failed to kill the bacterial cells. Since strains WS(ω)1C and WS(ω)2B, but not strain WS(ω)0, degrade T2 DNA, the correlation between acid solubilization and reduced killing is maintained and the hypothesis is supported that the ability of the cells to survive infection is contingent on their capacity to destroy the viral DNA.

Strain WS(ω)0 permits viral synthesis to continue longer than the other lysogens (3). The possibility should be considered that inefficient killing of strains W, WS(ω)1C, and WS(ω)2B results from reduced production of a viral protein

necessary for the loss in viability. At higher multiplicities, a dosage effect might compensate for the limited viral syntheses. This explanation appears unlikely since production of the viral enzyme deoxycytidylate hydroxymethylase in strain W did not show a dependency on the multiplicity of infection (H. Smith, Ph.D. Thesis, Univ. of Pennsylvania, 1967).

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