when multiplicity and degree of irradiation were varied.

Later, Dulbecco[2] subjected the theory to a stricter test in the range where prolonged irradiation of the particles makes subsequent multiplicity reactivation a very rare event. According to the proposed theory, as the time of irradiation is increased the slope of the curve of survival of multicomplexes against time of irradiation should approach the slope of the curve of survival of single particles. In fact, the decline in survival of multicomplexes was shown to occur much less rapidly. Since no simple process of unit substitution could be more efficient than the proposed one, the recombination theory of multiplicity reactivation was therefore regarded as disproved.

Variation in bacterial size would produce exactly this effect. In an extreme case, the decline in survival of multicomplexes with increasing irradiation would merely represent decline in the number of cells large enough to have collected enough particles to ensure the presence of one complete set of undamaged units. We have therefore recalculated expectations for Dulbecco's experiments, incorporating the frequency distribution of cell-sizes given by Dulbecco in an appendix to the earlier paper. We have assumed that there is a Poisson distribution of bacteriophage particles over the bacterial surfaces, that the bacterial lengths given by Dulbecco can be equated with surface areas, and that there is no loss through lysis-from-without. We have also had to assume that a frequency distribution of cell-sizes, based upon measurement of 764 cells, is adequate for predicting events which have a frequency of 10^{-4} to 10^{-7}.

As shown in Fig. 1, the agreement between observation and recalculated expectation is surprisingly good, considering the uncertainties of the calculation. The observed survival of multicomplexes is now seen to be close to, or rather lower than, expected. Therefore, it seems no longer necessary to seek an alternative to the recombination theory of multiplicity reactivation.

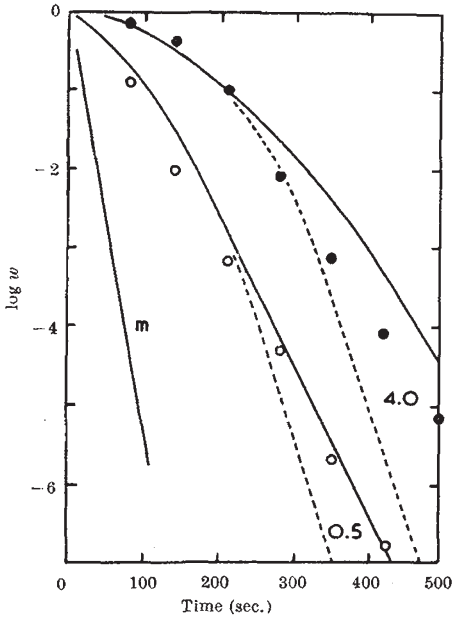

Fig. 1. Effect of increasing irradiation on the survival of multicomplexes. The proportion of surviving multicomplexes (w) is plotted against the time of irradiation for the two multiplicities, 0·5 and 4·0 bacteriophage particles per bacterium. Broken lines represent the expected survival as calculated by Dulbecco, assuming a Poisson distribution of particles among bacteria; solid lines are for the recalculated distribution, assuming a Poisson distribution of particles over the bacterial surfaces. Each particle was taken to contain 25 units. The points and the curve for survival of single particles (m) are from Dulbecco's paper

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Composition of a Hypertensin Peptide

The isolation of a homogeneous pressor peptide (hypertensin or angiotonin) made by the action of rabbit renin on ox serum was previously reported by me[1]. Further quantitative study on the composition of this pressor peptide has shown the empirical structure to be :

One residue : leucine, phenyl alanine, tyrosine, proline, aspartic acid and arginine.

Two residues : valine and histidine.

This gives a minimum molecular weight of 1,445. The distinction between aspartic acid and asparagine has not yet been made.

Only one N-terminal amino-acid, aspartic, was found, using the dinitrofluorobenzene technique[2], and this was supporting evidence of homogeneity.

The hypertensin peptide recently described by Skeggs, Marsh, Kahn and Shumway[3] differed only by the presence of *iso*leucine in place of one of the two valine residues. This would presumably represent a species difference, since these workers used pig renin and horse serum substrate. The N-terminal amino-acid was not stated, and no further comparison is possible.

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Effect of Antibody on the Respiratory Rate of *Trypanosoma vivax*

The West African N'Dama breed of cattle shows a remarkable tolerance to trypanosomiasis, as was demonstrated by Chandler[1]. In order to investigate the nature of this tolerance, it was necessary to develop a technique which would give a quantitative estimation of antibody titre. Methods employed in the study of bacteriological immunity are not generally applicable to the study of the immune response as it occurs in African trypanosomiases. Tests such as the mouse protection test employed by Fiennes[2] in studying *T. congolense* infections do not readily give a quantitative estimate of antibody titre and are operative only when the trypanosomes survive in the host as a patent or latent infection.

It was noted that the oxygen consumption of two strains of *T. vivax*, one cyclically transmitted and maintained in sheep and the other adapted to rats and passaged by syringe, showed a marked decrease