with *Aerobacter aerogenes*[1], *Escherichia coli* and *Pseudomonas fluorescens* have shown that starvation in aerated saline buffer at 37° results in a considerable loss of protein during a period when viability of the populations remains at 95–100 per cent. Since analyses of ultracentrifugal fractions separated from *A. aerogenes* after various periods of starvation showed decreases in the protein content of the insoluble, ribosomal and soluble fractions[3], the loss apparently represents a depletion of various cell proteins. It is interesting, therefore, that induced β-galactosidase in *Escherichia coli* was found to be relatively stable while other proteins were being rapidly degraded during turnover[4]. The relative stability of induced β-galactosidase in a wild-type strain of *E. coli* during starvation has been investigated and this communication records the relevant data.

Escherichia coli (M.R.E. 162: from human faeces) was grown at 37° for 16 h in shaken flasks (2 l.) containing a defined ammonium-salts medium[1] (100 ml.) with lactose (1 per cent w/v) as the sole source of carbon. Stationary phase organisms were separated from the culture and washed twice with saline phosphate buffer (0·055 M NaCl and KH_2PO_4 + K_2HPO_4 (0·01 M PO_4), *p*H 6·5) by centrifugation. The β-galactosidase concentration in these bacteria was about 90 units whereas bacteria grown in a medium with mannitol as the sole source of carbon contained less than 0·2 units of enzyme. The bacteria were re-suspended (1·5–2·5 mg dry weight/ml.) in saline buffer and the suspension was aerated with washed air in a Dreschel bottle at 37°; effluent air escaped through a water reflux condenser fitted with an air filter. At intervals samples were removed for determinations of total protein by a Biuret method[5] (colour obeyed Beer's law in the range used; coefficient of variation, 1·1 per cent for fifteen replicates), β-galactosidase with *O*-nitrophenyl-galactoside[6] (*O*-nitrophenol colour obeyed Beer's law; coefficient of variation, 2·6 per cent for twenty-two replicates), viability by viable plate count (coefficient of variation, 4 per cent for twelve replicates with mean count of 835 colonies) and viability by slide culture[7] (coefficient of variation, 1 per cent for twelve replicates with mean of 95·7 per cent). The enriched agar medium for viable counts and slide culture was as described by Postgate, Crumpton and Hunter[7] except that glucose (0·5 per cent w/v) replaced glycerol.

The results (Table 1) show that during 30 h at 37°, when loss of viability was 5 per cent, losses of total protein and β-galactosidase activity were 21 and 29 per cent of the initial concentrations, respectively; the total protein-loss accounted for 56 per cent of the dry-weight loss and one of the products was ammonia. When starvation was continued for 48 h, further small losses of total protein and galactosidase activity occurred accompanied by a further small loss of viability. At each time-interval, β-galactosidase was determined in the whole suspension and in the bacteria separated by centrifugation, and re-suspended in saline buffer; under the latter conditions results were slightly lower in some cases due, perhaps, to incomplete recovery of bacteria or failure to re-suspend to exactly the same volume. When bacteria were starved in saline buffer containing thiomethylgalactoside[8] (TMG; 1 mM), β-galactosidase activity remained at the initial level for at least 24 h, while loss of protein was similar to that in the absence of inducer.

Thus, while under some conditions, β-galactosidase and indeed other induced enzymes in non-growing bacteria may be relatively stable (see ref. 4), the present results indicate that in the absence of nitrogen and carbon sources at growth temperature, induced β-galactosidase activity in *E. coli* is lost at a higher rate than the average rate of degradation of all cellular protein. Viability, according to two methods of assay, was maintained at a level that excludes bacterial lysis as the cause of losses of protein or β-galactosidase activity[4]. Regarding the method used for protein determination, parallel assays of *A. aerogenes* after various periods of starvation, by the present method, and by a copper-iodometric α-amino nitrogen method (after hydrolysis of the bacteria with 6 N hydrochloric acid for 20 h at 105°), gave similar results for protein losses. The stabilizing effect of TMG on β-galactosidase in starved *E. coli* may be due to the formation of a stable inducer-enzyme complex or to resynthesis of enzyme stimulated by the inducer.

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VIROLOGY

A Cytopathogenic Virus from the Brain of Fowl

A virus has been isolated from the brains of chickens which have died or been killed while suffering from a disease indistinguishable by clinical signs or histopathological lesions from infectious avian encephalomyelitis ('epidemic tremor').

The virus has multiplied in monkey kidney cell culture and has been repeatedly passaged in that system, in which it produces a cytopathic effect similar to that produced by an enterovirus. The agent appears to be completely resistant to the effect of ether. An antiserum produced against the Van Roekel strain of infectious avian encephalomyelitis virus has been shown to have a neutralizing effect against this virus in monkey kidney cell culture.

Further investigations of the virus are in progress, the results of which will be published in the near future.

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GENETICS

Evolution of Over-dominance during the Initial Spread of New Alleles

Bodmer and Parsons[1–3] have discussed a model for the initial increase in the frequency of newly arising, favourable mutations in a large undivided population and under different mating systems. They argued that over-dominance (higher fitness of heterozygotes over the homozygotes) has evolved in outbreeding species during this process as follows: (1) a new allele, say A_1, at locus A_1–A_2 occurs principally in the heterozygotes when it is initially at a low frequency in the population; (2) selection favouring heterozygotes allows a rapid rate of increase in frequency of A_1; (3) hence, the modifiers that primarily enhance the fitness of A_1A_2, particularly when linked, would themselves increase rapidly in frequency and accumulate to form the linked gene complexes; (4) such gene combinations would exhibit so-called segmental (or associative[4]) over-dominance maintaining a balanced polymorphism.

Thus, Bodmer and Parsons[5] concluded that "polymorphic over-dominant complexes have almost certainly evolved by the accumulation of linked interacting modifiers". Their theoretical analysis involved approximate