Spawning of the Male Salmon Parr (*Salmo salar* Linn. juv.)

WE have watched the spawning behaviour of salmon in six successive seasons, using for the purpose an observation tank built by the Dee Fishery Board and the University of Liverpool on a tributary of the Welsh Dee; and we have long been convinced, on circumstantial evidence, that sexually mature male parr make a significant contribution to the fertilization of the eggs. It is, however, difficult in normal circumstances to get direct evidence, for the female only sheds her ova when accompanied by an adult male, and the cloud of sperm released simultaneously by him masks any contribution made by the parr.

This year (1951) we have observed apparently normal courting by adult males which had previously been sterilized by ligation of the sperm ducts. The orgasm also appeared normal in every way except that no sperm issued from the adult male, and it was therefore possible to observe whether any sexually mature parr which were present played their part. When the adult female deposited her eggs, no sperm was released from the adult male, but a cloud of sperm arose from the bottom of the bed where the sexually mature male salmon parr were lying; in one instance sperm was seen to issue from a salmon parr. Since it has already been shown[1] that sperm from stripped parr is as effective in the artificial fertilization of salmon eggs as is the sperm stripped from adults, it is no longer open to doubt that male parr, in natural conditions, may be responsible for the fertilization of a considerable fraction of the total of eggs which develop.

We have not yet observed normal courting by castrated adults, and attempts to induce it in castrates by injections of testosterone propionate have had only little success.

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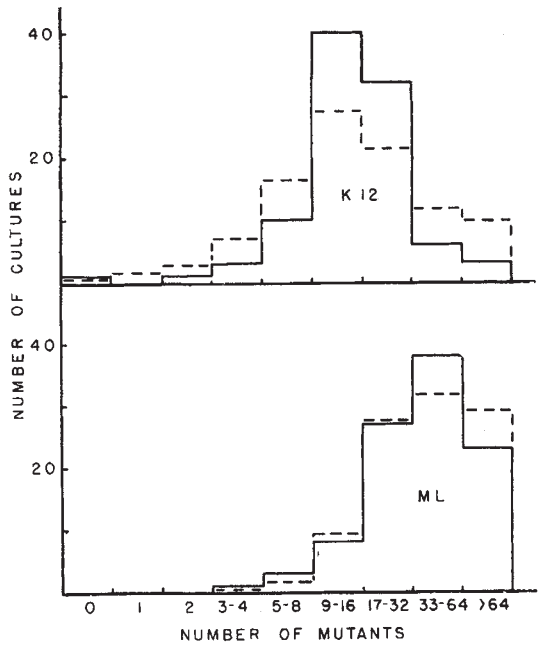
[1] Jones, J. W., and King, G. M., *Salmon and Trout Mag.*, No. 128 (January 1950).

Distribution of Numbers of Mutant Bacteria in Replicate Cultures

SINCE the publication by Lea and Coulson[1] of the expected distributions of numbers of bacterial mutants in replicate cultures, there has been only one limited attempt to compare this theory with experimental observations. Armitage[2] has shown that Lea and Coulson's unmodified theory will not allow a description of the pattern of mutants in *Escherichia coli* resistant[3,4] to phage *T*1. In the literature a number of other examples are available for comparison. Experiments on mutation to resistance to ultra-violet radiation[5] and to histidine independence[6] in *E. coli* can be shown to give distributions of mutants which, like those to phage resistance, do not agree with the distributions predicted by Lea and Coulson. On the other hand, mutations to penicillin resistance in *Staphylococcus aureus*[7], to tryptophane independence in *Eberthella typhosa*[8], and to streptomycin resistance in *E. coli*[9], give distributions fitting the theoretical one.

While working in the Service de Physiologie microbienne of the Pasteur Institute in Paris, I had occasion



to study the distributions of numbers of lactose-utilizing (*lac*$^+$) mutants among replicate cultures of two lactose non-utilizing (*lac*$^-$) strains of *E. coli* by methods described elsewhere[10]. The distributions are shown in the accompanying diagram. The dotted lines are the theoretical distributions calculated according to Lea and Coulson on the basis of an average number of mutations determined from the median of the observed distribution. The *ML* strain, at a level of $3{\cdot}2\ (\sigma = 0{\cdot}6) \times 10^7$ bacteria per culture, gave a good fit ($P = 0{\cdot}2$). Similarly, good fits were obtained at levels of $1{\cdot}3 \times 10^7$ ($P = 0{\cdot}3$) and $0{\cdot}9 \times 10^7$ ($P = 0{\cdot}3$) bacteria. On the contrary, the *K*12 strain, at a level of $3{\cdot}9\ (\sigma = 0{\cdot}4) \times 10^7$ bacteria, gave a poor fit ($P < 0{\cdot}01$).

The failure of the *K*12 strain to give mutants in the pattern predicted by Lea and Coulson demonstrates that the assumptions underlying their theoretical distribution are not met in this case. Their fundamental assumption is that the chance of mutation per bacterium per time unit is constant, so that the mutations are distributed randomly in space (within subclones of the culture) and in time (during the different growth phases of the culture). The first part of this assumption can be tested directly by experiment. It has been shown that the number of papillæ forming on surface colonies of *lac*$^-$ bacteria, grown on agar containing lactose and another carbon source, is a reflexion of the number of mutations to the *lac*$^+$ condition[10]. If the mutations are random, whether true back-mutations or suppressor mutations[11], their distribution should follow the Poisson formulation. The accompanying table shows that they do.

The only unique experiment ($P = 0{\cdot}01$) involved colonies of strain *K*12 which had unusually irregular surfaces, which may have resulted in too few colonies being counted as having no papillæ and too many as possessing one or more.

The failure of the *K*12 mutants to follow the Lea and Coulson distribution is not likely, then, to be due to spatial non-randomness. Temporal non-randomness would exist if the rate of mutation were