

Now that it is possible, as the writer has shown, to detect telomeric fusion of chromosomes the way is open for the experimental study of the mechanisms and biological role of telomeric interaction between chromosomes in health and disease.

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INDEPENDENT INTEGRATION OF GENES CONTROLLING THE INVASIVE PROPERTIES AND STREPTOMYCIN RESISTANCE OF ENTEROPATHOGENIC STRAIN *Escherichia coli* 0124

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Crossing *Escherichia coli* K12 Hfr AB313 with an enteropathogenic strain of *E. coli* of the serological group 0124 yielded recombinants which had lost their invasiveness. The loss of invasiveness of these recombinants was not due to the acquisition of genes controlling resistance to streptomycin.

KEY WORDS: enteropathogenic strains of *E. coli*; invasiveness; streptomycin resistance; transmission of genes.

One of the factors in the pathogenicity of shigellas and certain serological groups of enteropathogenic escherichias is invasiveness. Investigations [1-3] have shown that replacement of the streptomycin region of invasive strains of shigellas and escherichias by the corresponding streptomycin-resistant region of noninvasive escherichias in conjugation experiments leads as a rule to loss of invasiveness. This suggested the existence of a special gene (or genes) controlling this property. However, the difficulty in the interpretation of these findings is that incorporation of the streptomycin-resistant region can itself cause loss of virulence because of disturbance of synthetic processes.

In this investigation an attempt was made to discover whether the loss of invasiveness is connected with the acquisition of genes of the streptomycin resistant region or of genes outside it.

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TABLE 1. Genetic Groups of Recombinants of *E. coli* 0124

№	Genotype	Number of strains
1	nad ⁺	127
2	nad ⁺ mot ⁺	2
3	nad ⁺ dul ⁺	38
4	nad ⁺ str ^r	17
5	nad ⁺ dul ⁺ mot ⁺	7
6	nad ⁺ dul ⁺ str ^r	5
7	nad ⁺ mot ⁺ dul ⁺ str ^r	1
Total . . .		197

TABLE 2. Virulence of Recombinants Allowing for Inheritance of Individual Markers

Marker	Result of eye test	% of strains losing invasiveness
nad	37/113	33
mot	0/6	0
dul	4/20	20
str ^r	3/19	16
pil	0/7	0

Legend. Numerator, number of strains losing invasiveness; denominator, number of strains studied.

EXPERIMENTAL METHOD

The standard streptomycin-resistant strain *E. coli* K12 Hfr AB313 was used and was conjugated with a freshly isolated streptomycin-sensitive strain of *E. coli* of serological group 0124:K72(B17). Strain AB313 is well known in the literature and the transmission of its chromosomes starts with the gene controlling mannitol utilization. Strain 0124 has not been mapped. Recombinants were selected for their ability to synthesize nicotinic acid (nad), and the unselective markers chosen were those controlling motility (mot), fimbria synthesis (pil), ability to ferment the carbohydrates rhamnose and dulcitol (rha, dul), and invasiveness, defined as the ability of the strain to induce keratoconjunctivitis in guinea pigs [4].

EXPERIMENTAL RESULTS

Depending on the inheritance of the various markers, all the recombinants obtained could be subdivided into seven genetic groups (Table 1).

Most of the recombinants (127) inherited none of the unselective markers whereas the rest inherited them in various combinations.

To conclude whether a given marker acquired during conjugation influenced the loss of invasiveness, the virulence of the recombinants was studied allowing for inheritance of individual markers (Table 2).

It will be clear from Table 2 that 33% of the recombinants lost their invasiveness without inheriting any of the unselective markers studied. It would be natural to expect that if a given marker influenced the loss of invasiveness, this percentage should have increased. In fact, however, it was lower in every case.

Analysis of the recombinants thus showed that the genes controlling dulcitol utilization, the synthesis of flagella and fimbriae, and also resistance to streptomycin were inherited independently of the genes controlling invasive properties.

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