The cold variant appears to be heat labile and its growth at 40° C can be used as a marker of differentiation. A marked reduction in mortality of infected mice was observed. The cold variant appears to infect mice without overt pathological response, while stimulating good antibody responses. Thus a significant degree of attenuation has been achieved.

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H. F. MAASSAB

Department of Epidemiology,
School of Public Health,
University of Michigan,
Ann Arbor, Michigan.

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Do Certain Colicines and Phages share Common Receptors?

THE receptors of bacterial cells for colicines have not so far been studied in great detail, but there is some evidence[1,2] for the assumption that the role of these receptors is to fix colicines specifically on to the bacterial surface and to enable them to have inhibitive effects like phage receptors[3]. Although it is generally accepted, this hypothesis has never been proved[4]. The main support for it came from Fredericq's later experiments, which showed a cross-resistance between certain colicines and phages. Consequently they are both supposed to be adsorbed on to common receptors. Thus colicine *K* should share a common receptor with the phage *T*6 (ref. 5), colicine *M* with the phages *T*1 and *T*5 (ref. 6), etc.

All the receptors of *T*-phages are localized in the bacterial cell wall. Attempts have been made to define them in terms of the biochemical composition of the *Escherichia coli B* cell wall: the *T*6 receptor is situated in the protein part of its lipoproteid layer[7], while the *T*5 receptor seems to be a lipopolysaccharide-lipoprotein complex[8]. The *T*1 receptor has not yet been defined chemically.

From experiments with stable L-forms of *Proteus mirabilis* it is known that these cells which are devoid of their cell walls have lost their phage receptors and therefore are absolutely resistant to the action of phages[9].

Recently a stable L-form of *E. coli B* has been isolated[10]. Preliminary experiments showed that this stable L-form, too, is resistant to the phages of the *T*-series. Thus it became possible to compare the action of both colicine *K* and phage *T*6 on normal rods and on the cells of the L-form, that is, on cells with and without cell walls.

Plates of 1·5 per cent meat-peptone agar containing 1·5 per cent horse blood serum, 1·5 per cent or 3 per cent sucrose and 0·1 per cent magnesium sulphate ($MgSO_4.7H_2O$) were used in our tests. On to these, 6–8 days old broth culture of the stable L-form of *E. coli B* was densely seeded for every experiment and (*a*) broth suspension of the phage *T*6 (10^6, 10^8, 10^{10} particles/ml.), (*b*) broth solution of the colicine *K* (10^{10}, 10^{11} lethal units/ml.), and (*c*) broth (control) were dropped on to the dry surface. (For colicine *K* we used a filtrate of a broth culture of the strain *E. coli K* 235; the participation of the other colicine produced by this strain[11] was excluded by cultivating the producer in meat-peptone broth under heavy aeration.) The experiment was repeated with various modifications.

In all cases the results were the same: the stable L-form of *E. coli B* is completely resistant towards the phage *T*6 as well as towards all other phages of the *T*-series; however, at the same time, it remains fully sensitive towards colicine *K*. Together with its cell wall the L-form has evidently lost its receptors for *T*-phages, but not its colicine *K* sensibility, which means that the action of the colicine does not depend on previous adsorption on to a specific receptor in the cell wall.

Unfortunately, it is hardly possible to perform a similar experiment with the colicine *M* and phages *T*1 and *T*5; it would be necessary to have a stable L-form of *E. coli B* resistant to colicine *V*, from which the colicine *M* cannot be separated. But the colicine *M* does not attack normal cells of *E. coli B*, which are specifically resistant to colicine *V*, in spite of the fact that the *B* strain is the classical indicator for *T*-phages. In this case, therefore, it is not possible to suppose a common receptor.

We conclude that the analogous sensibility of some strains towards certain colicines and phages cannot be simply explained by the assumption of common receptors. Reeves[12] has also stated that this assumption is open to serious criticism. The cross-resistance has never been found in 100 per cent of the strains covered.

The observation that specific cell wall receptors are not needed for colicine action at all—at least with certain colicines—will be thoroughly discussed elsewhere.

These experiments were performed at the Institute of Microbiology and Experimental Therapy, Jena.

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J. ŠMARDA

Department of Biology, Medical Faculty,
J. E. Purkyně University,
Brno, Czechoslovakia.

EDITH SCHUHMANN

German Academy of Sciences,
Institute of Microbiology and
Experimental Therapy,
Jena.

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Bacteriocinogeny in Strains of *Providence* and *Proteus morganii*

BACTERIOCINS[1] are a distinctive class of antibiotics produced by bacteria. Bacteriocins are proteinaceous and their action is limited to strains of the same or closely related species as the producer organisms.

Strains of a number of different families produce bacteriocins (bacteriocinogenic), but until the recent discovery of bacteriocin production in strains of *Proteus hauseri*[2] the *Proteus–Providence* group were regarded as unique among the Enterobacteriaceae in being non-bacteriocinogenic[1]. This communication describes the presence of these antibiotics in strains of *Providence* and *P. morganii*. The organisms used comprised the seventeen *P. morganii*, twenty-two *P. rettgeri*, twenty-four *Providence*, twenty-eight *P. hauseri* and other strains of Enterobacteriaceae previously used[3,4]. A further seventy-