

described by Garvie, Gregory and Mabbitt¹¹. Since a variety of strains and possibly more than one species were being examined, the growth of each strain was observed after 24- and 48-hr. incubation at 30° C. in the medium with and without folic acid. Two of the strains, *N.C.D.O.* 1250 and 1247, grew equally well in all tubes and had no requirement for folic acid. The other strains, *N.C.D.O.* 1248 and *N.C.D.O.* 1249, gave excellent growth in the presence of folic acid at 24 hr., but none in its absence. However, at 48 hr. in the absence of folic acid, growth sometimes occurred. Thus, with these strains, folic acid has a marked stimulatory effect on growth, although under conditions at present undefined, they grew in its absence, but only after a prolonged lag phase. The control, ATCC 8081, showed no growth in the absence of folic acid after 48 hr. incubation.

These results show that a requirement for folic acid is not restricted to typical strains of *Pedococcus cerevisiae*. Until more strains have been examined, it is not possible to decide if the growth requirements which have been discussed are useful in classification, or if they demonstrate merely variations within the species *Pedococcus cerevisiae*.

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A Bacteriophage for Motile Bacteria

In the first stage of phage infection, the phage particle is adsorbed to a specific receptor which is usually on the bacterial body¹. However, an exceptional phage has been reported² the host-range of which appeared to be determined by flagella, and these are structurally distinct from the bacterial surface proper³.

Four conditions have now been found to determine bacterial sensitivity to this *Salmonella* phage, named χ .

(1) The bacteria must be flagellated. Non-flagellated strains are invariably resistant, and resistant non-flagellated variants are selected from sensitive strains by the phage.

(2) The flagella must carry certain antigens. Sensitivity was shown to depend on the flagellar (*H*) antigen, and not on the somatic (*O*) antigen, by tests on (a) 524 naturally occurring motile *Salmonella* strains belonging to 27 different *O* groups and carrying most of the known *H* antigens; and (b) sets of artificial derivatives of single strains, differing in their *H* antigens, which had been prepared by phage-mediated transduction⁴. Strains with most *H* antigens could be sensitive, but all 53 naturally occurring strains with antigens of the *g* complex (*g,m, g,p, g,s,t, etc.*) were

resistant: transduction of antigen *g* . . . to sensitive strains made them resistant and resistant strains naturally carrying *g* . . . were often sensitive when another antigen was substituted. The wild-type phage did not attack strains with *H* antigen *l* . . . (*l,z₁₃, l,v, or l,w*), but could mutate to a form able to do so. Host-range mutants were also isolated for bacteria with *H* antigens *e,h* and Arizona 13. Diphasic strains carrying *g* . . . , *l* . . . or *e,h* in one phase could be sensitive to wild-type phage in the alternate phase.

(3) The flagella must be active: 10/10 naturally occurring paralysed (that is, flagellated but non-motile) strains were resistant while their motile derivatives were sensitive. Also, 8/80 resistant variants selected by the χ phage from sensitive strains turned out to be paralysed. Reversion to motility was accompanied by return of sensitivity.

(4) The bacterial strain itself must be suitable, for not all motile bacteria with appropriate *H* antigens were sensitive. The importance of the bacterial strain was clearly seen with transduction of an antigen, for the strain from which the antigen had been transduced often reacted differently from the recipient.

Resistance due to absent or inactive flagella, or to flagella of inappropriate antigenic type, could be shown to be associated with impaired adsorption of the phage, by using sets of variants of suitable strains. Adsorption was prevented by detaching the flagella from sensitive bacteria, and returned progressively to normal as the flagella were regenerated. Adsorption was also impaired when sensitive bacteria were artificially paralysed by treatments such as thorough washing, or exposure to 2,4-dinitrophenol.

No adsorption to detached flagella was detected, probably because they were inactive.

Electron micrographs, kindly prepared by Mrs. H. Ozeki and by Dr. E. H. Mercer, showed phage particles attached by the tips of their tails all along the length of the flagella; none was seen on flagella carrying antigens associated with resistance and few or none were found on paralysed bacteria. Other experiments showed that infection could follow adsorption to distal as well as proximal parts of a flagellum.

If, as it appears, isolated⁵ and paralysed⁶ flagella are structurally similar to active flagella, then the latter do not seem to possess any unique structure that might be the phage receptor. The necessity for flagellar movement to promote phage infection may thus mean that the activity is necessary in itself and not merely a concomitant of a new flagellar structure. The means by which the phage genome passes from its primary site of adsorption on a flagellum to the bacterial body is not clear, but the flagella do not seem to act merely by 'activating' the phage so that it can attach itself to the bacterial body afterwards. These points and the detailed results are discussed elsewhere.

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