The Specificity of Lamboid Phage Late Gene Induction
(Lamboid Phage Late Gene Specificity)

The purpose of the work reported here was to determine the specificity of late gene induction of phages λ and φ80 by various temperate coliphages. As there is a large and variously related family of coliphages (1, 2), it is of interest to determine whether any of them are able to induce the late genes of their relatives.

These measurements were made possible by the properties of phage φ80dada and λφ80dada. In both of these phages, genes of the arabinose operon of Escherichia coli have replaced some phage late genes and are now controlled as phage late genes (3). On φ80dada they are under control of the φ80 late gene promoter, and on λφ80dada they are under control of the λ late gene promoter (3). Heteroimmune infection of φ80dada or λφ80dada lysogens was used to test for late gene induction by the other phages. If the infecting phage can grow on the lysogen and induce the late genes of the prophage, arabinose isomerase will be synthesized, resulting from transcription of the arabinose operon by an RNA polymerase which initiated at the phage late gene promoter under control of the phage Q protein (3). The Q induced synthesis of arabinose isomerase from λφ80dada has been shown to be independent of Nα (product of the λ X gene), an inducer of λ early protein, once Qα is present (3). Figure 1 shows data leading to a similar conclusion, here λimm2α infected lysogens of λφ80dada and φ80dada. The λimm2α was able to grow on both lysogens, but Qα was able to induce only the late genes of λ. As λimm2α cannot complement a λN−, i.e., λimm2α does not possess an Nα (4, 5), Nα is not directly required for inducing λ late genes.

Table 1 shows the ability of various lamboid phages to induce arabinose isomerase from λφ80dada and φ80dada. The first entries, λ, λimm4α, and λvir, are as expected; those phage able to grow in a λ lysogen but possessing Qα induce isomerase from the phage with arabinose genes under control of a λ late gene promoter, Myφ80dada. The Qα does not induce the arabinose operon of φ80dada, a result in agreement with the results of Spirier and Brachet, who showed by other means that the Qα does not induce the late genes of φ80 (6). Phages 381, 424, and 434vir do not induce λ or φ80 late genes. The same is seen for λimm44α qinA3 (7) and λimm44αvir, two phages apparently having replaced their Q gene with a Q-like gene and promoter from another prophage (8).

The inability of 434 to induce the late genes of φ80 implies that it does not produce a Q product identical to the lambdoid product. This allows a refinement in the location of the Q gene on the λ chromosome. Doeffer, Egan, and Black have determined that the Q gene is close to the λ 434vir from φ80. In fact, DNA heteroduplexes between λimm44α and φ80 show homology from 87.5 to 94.7% (9), results showing Qα ≈ QGQα imply that part of the lambda Q gene lies to the left of λ 434vir. This conclusion is subject to the reservation that a difference in biological activity could be generated by base sequence apparently homologous in electron electron observation of DNA heteroduplexes.

Interestingly, phages φ81 and φ82 induce the late genes of φ80. Phage φ81 was found in the same isolate of E. coli, and therefore both phages could belong to the same system for inducing late gene expression. However, the induction of φ80 late genes is less expected, since φ82 shows no significant homology to λ and may not be more closely related to λ than φ80.

It is possible that the induction...
**TABLE 1**

The heterimmune induction and plating efficiencies of lambdoid phage on *A80dara* and *A80b1ara* lysogens.

<table>
<thead>
<tr>
<th>Phage</th>
<th>Induction of <em>A80dara</em> lysogen</th>
<th>Plating efficiency on <em>A80dara</em> lysogen relative to nonlysogen</th>
<th>Induction of <em>A80b1ara</em> lysogen</th>
<th>Plating efficiency on <em>A80b1ara</em> lysogen relative to nonlysogen</th>
</tr>
</thead>
<tbody>
<tr>
<td>λ</td>
<td>&lt;.1</td>
<td>.0</td>
<td>&lt;.1</td>
<td>1.1</td>
</tr>
<tr>
<td>λ<em>le</em></td>
<td>1</td>
<td>1.2</td>
<td>&lt;.1</td>
<td>1.0</td>
</tr>
<tr>
<td>λ<em>le</em>μ</td>
<td>1</td>
<td>&lt;.1</td>
<td>&lt;.1</td>
<td>.7</td>
</tr>
<tr>
<td>λμ<em>le</em></td>
<td>1</td>
<td>.9</td>
<td>&lt;.1</td>
<td>.9</td>
</tr>
<tr>
<td>434<em>le</em></td>
<td>&lt;.1</td>
<td>1.0</td>
<td>&lt;.1</td>
<td>1.8</td>
</tr>
<tr>
<td>421</td>
<td>&lt;.1</td>
<td>1.0</td>
<td>&lt;.1</td>
<td>1.0</td>
</tr>
<tr>
<td>381</td>
<td>&lt;.1</td>
<td>.6</td>
<td>&lt;.1</td>
<td>.2</td>
</tr>
<tr>
<td>λμ<em>le</em>μ<em>ginB3</em></td>
<td>&lt;.1</td>
<td>.8</td>
<td>&lt;.1</td>
<td>.7</td>
</tr>
<tr>
<td>λμ<em>le</em>μ<em>psl</em></td>
<td>&lt;.1</td>
<td>.9</td>
<td>&lt;.1</td>
<td>1.0</td>
</tr>
<tr>
<td>80C<em>mut</em></td>
<td>&lt;.1</td>
<td>.5</td>
<td>&lt;.1</td>
<td>.0</td>
</tr>
<tr>
<td>80<em>le</em>μ</td>
<td>&lt;.1</td>
<td>&lt;.001*</td>
<td>1</td>
<td>1.0</td>
</tr>
<tr>
<td>81</td>
<td>&lt;.1</td>
<td>.06*</td>
<td>1</td>
<td>.9</td>
</tr>
<tr>
<td>82</td>
<td>&lt;.1</td>
<td>.7</td>
<td>.4 ± .2</td>
<td>.4</td>
</tr>
</tbody>
</table>

* The procedure described in Fig. 1 was followed. Phage φ82 was used within 2 hr of making a plate stock as it was highly unstable.

* The λ and φ80 were from Mark Ptashne; the rest were from Ethan Signer via Ira Herskowitz.

* Induction of a lysogen is calculated from the slope of the isomerase accumulation following infection relative to the slope following infection of the *A80dara* lysogen by λ*le*.

* In cross-streaking a lysogen of *A80dara* against phage stocks at 1-5 × 10⁶/ml, φ80*le*μ does nothing, whereas φ81 weakly kills the cells.

The genes of λ imply that it does not possess a Q product identical to the lambda Q product. This allows a refinement in the possible location of the Q gene on the λ map. Hogness, Doolittle, Egan, and Black (9) determined that the Q gene is close to 90.0. However, DNA heteroduplexes between λ and φ80 show homology from 87.3 to 90.7 and homology from 90.7 to 94.7 (10). My results showing Qφ = Qφ80 imply that at least part of the lambda Q gene lies to the right of 90.7. This conclusion is subject to the reservation that a difference in biological activity could be generated by base sequences apparently homologous in electron microscopic observation of DNA heteroduplexes.

Interestingly, phages φ81 and φ82 both induce the late genes of φ80. Phage φ81 was found in the same isolate of *E. coli* as φ80 and therefore both phages could have similar systems for inducing late genes (11); however, the induction of φ80 late genes by φ82 is less expected, since φ82 shows considerable homology to λ and may therefore be more closely related to λ than φ80 (10).

It is possible that the induction by φ81 and φ82 of the late genes of φ80 is a result of Qφ or Qφ80 initiating an RNA polymerase at the late gene promoter, but simply from recombination between φ81 or φ82 and φ80. Such recombination would then place the arabinose genes on φ80 under direct control of φ81 or φ82. Without Q gene mutants in phage φ81 or φ82, this possibility cannot be excluded; it seems unlikely, though, since 444 did not detectably induce λ late genes, and yet λ and 444 possess extensive homology through most of their late gene regions (10).

These results show that phages φ81 and φ82 are able to induce the late genes of φ80; whereas the other temperate coliphages tested are not able to turn on late genes of λ or φ80.

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A Comparison of Certain Properties of a Turnip Mosaic Virus with Those of a Turnip Yellow Mottle Virus

Walters and Scott (5) isolated a turnip yellow mosaic virus (TYMV) from turnip, and a turnip mosaic virus (TVM) from turnip yellow mosaic virus (TYMV) and Turnip Yellow Mottle Virus (TYMV), both of which are transmitted by the Cornell potato aphid, Macrosiphum euphorbiae. The virus used in this study was isolated by transitory action of the aphid on turnip yellow mosaic virus (TYMV) and Turnip Yellow Mottle Virus (TYMV), both of which are transmitted by the Cornell potato aphid, Macrosiphum euphorbiae. The virus used in this study was isolated by transitory action of the aphid on turnip yellow mosaic virus (TYMV) and Turnip Yellow Mottle Virus (TYMV), both of which are transmitted by the Cornell potato aphid, Macrosiphum euphorbiae. The virus used in this study was isolated by transitory action of the aphid on turnip yellow mosaic virus (TYMV) and Turnip Yellow Mottle Virus (TYMV), both of which are transmitted by the Cornell potato aphid, Macrosiphum euphorbiae. 

The TYMV isolate was passed three local lesion transfers on a "Desmodium" sp. (1). Virus was purified from infected bean (Phaseolus vulgaris L., "Northern") harvested and frozen after inoculation. Infected tissue was ground in 0.2 M NaH2PO4 (1 g) and the homogenate squeezed through cheesecloth. The extract was adjusted to 5.0 with 0.1 N HCl, stirred for 1 hour, and subjected to alternate low speed (8000 g for 10 minutes) and high speed (100,000 g for 90 minutes) centrifugations. Virus pellets were resuspended in 0.01 M phosphate buffer, pH 7.4, and purified from Chinese cabbage (Brassica chinensis L.) by the same procedure.

The schlieren pattern obtained from TYMV preparations in the Model E analytical ultracentrifuge was typical of members of the TMV species. Two components with sedimentation coefficients of about 54 S and 114 S, respectively, were observed. Markham's graphical method (3) was used to determine the components.

Antisera were developed in rabbits by repeated intramuscular injections of TYMV and TYMV. The virus was mixed with Freund's incomplete adjuvant. Comparisons of the two viruses were made using homologous and heterologous antisera in gel diffusion tests in 1% agarose gels in water with 0.02% sodium azide in reciprocal spur formation. Figure 1 shows the reactions of TYMV antisera with TYMV and DYMV antigens.